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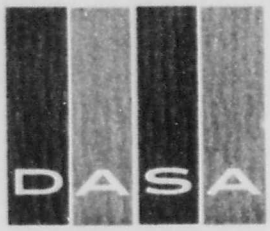

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PROCEEDINGS OF THE FIRST INTERDISCIPLINARY CONFERENCE ON SELECTED EFFECTS OF A GENERAL WAR

FEBRUARY 1968

DASIAC SPECIAL REPORT 67

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Proceedings:
OF THE FIRST INTERDISCIPLINARY CONFERENCE ON SELECTED EFFECTS OF A GENERAL WAR

VOLUME I

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SUMMARY

The conference convened under the cochairmanship of Dr. Austin M. Brues and Dr. Arthur C. Upton for discussion of somatic, genetic, and psychosocial effects of exposure to nuclear explosions and the subsequent radiation hazards. At the outset, Dr. Stafford L. Warren gave a graphic description, with motion pictures of several nuclear detonations, of the nuclear explosion phenomena.

Dr. Clayton S. White, drawing on both the experience in Hiroshima and Nagasaki and laboratory experiments, discussed blast effects, both direct and indirect. He cautioned against attempting to view the several effects as discrete phenomena, pointing out the inter-relationships between direct and indirect blast damage, between blast and thermal injury, and between blast or thermal injury and subsequent somatic damage from exposure to radiation. With this caution well in mind, however, he went on to summarize the results of a considerable number of analyses and experiments in which attempts were made to isolate the damaging blast forces and to relate their impact to the condition and situation of the target. He concluded with the observation that while steady and solid progress has been made in understanding blast effects, so much still remains to be learned that the prospect is at once discouraging and exciting.

Dr. Warren discussed thermal effects, both primary effects from infrared radiation and secondary effects from overturned stoves, short-circuits, etc. At Hiroshima and Nagasaki the former inflicted extensive burn damage on the exposed skin of unshielded human targets, but appeared to have limited damaging effect on structures because of the follow-up blast effect. The latter, however, was responsible for extensive fire damage in both cities. The group also considered the results of analyses of more localized burn damage, as to the eyes, hair, etc., and of possible later effects.

For the discussion of acute radiation effects, the group was given a background summary by Dr. John Auxier. He summarized the

findings and uncertainties of the several studies of the Hiroshima and Nagasaki experience, and recounted attempts made in laboratory and Nevada Test Site experiments to narrow the range of uncertainties. In the discussion that followed, it was emphasized that lack of knowledge of the degree to which individuals in Hiroshima and Nagasaki were shielded at the time of exposure makes impossible a precise kind of conclusion with respect to that experience. Knowledge of such phenomena as shielding and scattering is, however, being refined by later tests and experiments.

Dr. Upton initiated the discussion of chronic radiation effects, pointing particularly to the relation between leukemia incidence and radiation dose as one of the most significant findings of studies of the Japanese A-bomb survivors. He added that the relation correlates nicely with the observations of Drs. Court Brown and Doll on patients given large radiation exposures.

As the group turned its attention to genetic effects, Dr. William J. Schull took over as discussion initiator. He cautioned at the outset against treating somatic effects as distinctly different from genetic ones, pointing out that somatic response to irradiation is under a fair measure of genetic control.

Dr. Robert W. Miller turned from somatic to genetic effects by discussing findings of damage to children, chiefly in Hiroshima and Nagasaki, who were exposed in utero to substantial levels of radiation. One sharp finding of the studies of the Atomic Bomb Casualty Commission was that all eight Hiroshima women who were between the fifth and thirteenth week of pregnancy, and survived less than 1,200 meters from the point of detonation, had children who were retarded in intelligence and whose head circumferences were much smaller than normal. The experience of others in the same pregnancy time frame, but at increasing distances from the hypocenter, bears out the dose-response relationship as distance from the hypocenter increases. Dr. Miller also discussed findings of studies of growth rate and mortality of survivors under 20 years of age when exposed to the bomb detonation.

Dr. William Court Brown followed with an interesting discussion of ionizing radiation and chromosomal changes. He reported on the behavior of stable and unstable aberrations after radiotherapy, drawing particularly on the experience in Great Britain with studies of individuals so treated for ankylosing spondylitis. He also described the status of efforts to develop automated techniques which could

represent an important breakthrough in reducing the sheer drudgery of counting and analyzing chromosomes in a cell and increasing the productiveness of individual technicians by perhaps a factor of ten.

Dr. Schull and Dr. Seymour Abrahamson summarized for the group the present status of radiation genetics as it applies to man and experimental organisms—particularly Drosophila and the mouse. Laboratory experiments conducted in the two decades prior to the atomic bombings of Hiroshima and Nagasaki had led to certain hypotheses about radiation-induced mutations which served as the background for the formulations of genetic studies in these cities—the largest yet on man. The findings from Hiroshima and Nagasaki, though inconclusive, permit the setting of certain confidence limits. Moreover, they provide the most meaningful insight into the "doubling dose" appropriate to man. The uncertainties surrounding these estimates are such, however, that Dr. Schull concluded that though the increased mutational burden of the exposed populations of these two cities will ultimately manifest itself in some form, the rate at which this manifestation will appear is not clear.

Dr. Robert Lifton led the discussion of psychosocial effects of the bombings at Hiroshima and Nagasaki. He described his six-month study, consisting of interviews in depth of a sample of hibakusha (explosion-affected people) in Hiroshima, of the psychological response to that shattering experience. Among the important effects he found was a find of psychic numbing as a result of confrontation with so much death, widespread fear and uncertainty with respect to invisible contamination, post-crisis convergence behavior such as is commonly found in disaster situations, and a continuing tendency to see the root causes of subsequent misfortunes in the bomb experience.

Dr. Scott Matsumoto then reviewed briefly the major events which have occurred each year since 1945 in Hiroshima and Nagasaki and the influence which mass media coverage of these events has had in shaping the social impact on the two cities. He went on to present statistics on health, life span, marriage, divorce, migration, and other social factors. An interesting point examined was the different responses of the two cities, Hiroshima seeking to perpetuate its experience and identification and Nagasaki tending almost to submerge the experience into its past.

The group concluded its three-day discussion by raising questions and developing tentative and speculative hypotheses concerning the attitudinal implications of the experience for other populations.

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SESSION I

INTRODUCTORY SESSION

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OPENING REMARKS

FREMONT-SMITH: I am delighted to welcome you to the first of what we hope will be five conferences on this topic. This conference is under the auspices of the New York Academy of Sciences and run in the style of the Macy Conferences. I am going to call on Colonel Hemler to tell us how this all happened, how the Defense Atomic Support Agency (DASA) happened to turn us loose on this exciting topic.

We have been, for many years, and even more so today, inundated with masses of new information pouring in from all the outposts of information, of research around the whole periphery of knowledge. All this information has been developed by people on peninsulas protruding from the great area of common knowledge into the sea of ignorance. Each of these peninsulas is occupied by a group of subspecialists of subspecialists who have developed a special language, concepts, and acronyms—adequate for communicating with the peninsulas of advanced knowledge but very inadequate for communication with anyone who is on another peninsula or who is in the central body of knowledge.

The other difficulty is that people in these peninsulas are so used to talking to each other in their special language that they forget that they are using a special language; they are using the only natural language for them to talk in. They are not aware of the fact that they are not understood very well and often not at all by people in the other disciplines.

Back in 1936, when I joined the Macy Foundation, it was evident then, and is even more so today, that much more time and energy would need to be spent in the future in reintegrating the fragments of information which are pouring in upon us.

Another problem is to educate young people in science. The medical students in particular, but students in general, are overwhelmed by the mass of information which has poured in. With each

new discovery a professor will say, "But the students have got to get that and they've got to get this and they've got to get that." As a result, the real meaning of "educo" has been lost and it is now being translated as, "I feed in as rapidly as possible." This is one of the things to which many students object, because this is not the way in which their interest is stimulated.

A good many years ago, it came to us that we needed to teach by conceptual frames of reference which could act as skeletons upon which students could hang new information as it came along. It wasn't necessary to illustrate a conceptual frame of reference by all the families of facts that would fit into it but by a few examples. We made one other basic assumption: that nature is all of one piece, that all aspects of nature are related to all other aspects, and that no one aspect will ever be entirely understood until its relationships to the many other aspects are also involved and understood. Therefore, we have plenty of time ahead of us in which we can afford to be relatively humble as to what we know.

A dean of a medical school is supposed to have said recently to his students on opening day, "There is an enormous mass of facts which we're going to present to you in the next four years and unfortunately about half these facts are going to turn out not to be true. We have to say with great regret that we don't know which half is going to turn out not to be true!"

I like to talk a good deal about the evanescence of facts. I think facts are all being changed, and I would go so far as to challenge anybody to provide me with any fact which is not based on at least three unproved assumptions if one goes back a little way into its history.

Another aspect of the problem is illustrated very nicely by a report of the White House Conference on Education that appeared in the Saturday Review. This Conference took place a little over a year ago, with John Gardner as its chairman. According to the report, in opening the Conference he urged the participants to take big bites, barracuda bites, of the problems of education, because the problems of education were really in a bad state and needed this kind of vigorous attack.

The report went on to say that unfortunately the educational community didn't seem to be organized so that it could attack the problem

of education in the way Dr. Gardner had suggested; that perhaps some vigorous interchange may have taken place in the corridors, but that the Conference itself seemed much more like a game of golf in which each man continued to hit his own ball, rather than a game of tennis in which the ball was tossed back and forth across the net.

We decided long ago that communication takes place not by speaking at but by conversing with, if one really wants to understand this tossing of the ball back and forth across the net. As a crucial element in our conferences, we have moved from the standard procedure of having a number of speeches with a little conversation or discussion between them, to the point where we have, with the exception perhaps of my speech, no speeches at all. The plan is to bring into the conference room that which normally takes place in the corridors and in the bar. Our general attitude is that if anybody wants to make a speech, with the exception of me, we send him to the bar! [Laughter] This we find doesn't work too badly.

When somebody makes a speech or gives a lecture, if he interests his audience at all, he is bound to arouse ideas, doubts, challenges, questions. It is not polite to interrupt a lecture; thus people spend a great deal of time and energy in repressing every idea that comes to mind. If they don't do that, they doodle and think about other things. But the speech itself is pretty frustrating and fatiguing; that's why people get so exhausted listening to a speech. A conversation, on the other hand, is a mutually corrective feedback system in which those involved keep each other on the same wavelength or know very promptly if they are not on the same wavelength.

M. L. Abercrombie-Johnson of London pointed out in her little volume, Anatomy of Judgment, that an old idea that most of us were brought up on—that we first observe phenomena and then interpret them—is really false; that actually there is a built-in interpretation in the initial observation of anything; that the only way anybody can accommodate any new information is in the light of his past; and that since each person has a different past, each person observes the new phenomena slightly differently and sometimes very differently. Thus, one can almost be sure that if somebody just gives a lecture, with the exception of those very rare people who give magnificent lectures and capture the group completely, he will be misunderstood, ill-understood, or wrongfully understood by a large portion of the audience, with their different points of view. There will never be a chance to correct such misunderstandings unless interruptions are welcomed.

The longer a misunderstanding resides in the brain, the more difficult it is to get it corrected. Therefore, one of the points we make in our conferences is that it is important to interrupt at the moment when you feel the need of interruption. Our motto is, "don't speak when I am interrupting," and we hope the group will carry this mood throughout the meeting.

We have found that the attitude of people coming to a conference makes a lot of difference in the degree of their understanding one another and that the greater the degree of anxiety or hostility in the group, the greater the difficulty in communication. Everyone comes to a meeting with his own distorting lenses, with his blind spots, with his special, prejudiced points of view; and the more hostility or anxiety there is in the group, the more distorting the lenses are, the more tightly they are fixed to the eyes, and the larger the blind spots become. I am happy to say that we have developed a special magic aerosol, very scientific, which we put into the atmosphere and which we beg of you to inhale deeply. If you do, you will find that your distorting lenses become quite loosened and you can even see other people put their lenses right on the table! The blind spots begin to shrink and you actually see Arthur [Upton] as he really is—he looks quite nice! [Laughter] This aerosol is called free-floating security. It is my own invention, as the White Knight said, and it is, of course, the antithesis of the psychiatrist's free-floating anxiety. So if you will partake of our free-floating security, we believe that communication will flow.

Now Colonel John Hemler will tell us a little about how it happened that we are here.

HEMLER: I was commenting to Dr. Fremont-Smith and to Dr. de Boer earlier, that we seem to have come quite a distance from where we were 14 months ago when the germ of this idea was first born. At that time Dr. de Boer had enticed me to visit his radiobiology facility in Albuquerque, and as I found out later, for very ulterior motives. As we were walking down a dusty road, he explained the idea of the Interdisciplinary Communications Program and described what Dr. Fremont-Smith had been doing.

Some 2,000 miles east, Dr. Taylor, who was at that time with us in the Defense Atomic Support Agency (DASA), had been thinking along these same lines and was working in some of the particular areas with which we were concerned. So I might say I acted as a

catalyst or a carrier and brought the idea of the New York Academy of Sciences Communications Program together with Dr. Taylor's idea of getting some of the various peoples in the several involved disciplines together on long-range nuclear effects. When Dr. Taylor departed for Austria this past September, he more or less said to me, "Get going!" Of course, he didn't know at that time that we were planning to put him on the invitation list to bring him right back.

At DASA our mission within the Department of Defense is to do test and research work on the immediate effects of nuclear weapons. However, we recognize, and Dr. Taylor in particular is the one who has been strong on this, the need to look at some of the longer range or long-term effects of nuclear war or nuclear explosions. This is really the basic reason why we have all gathered together for this three-day conference and for the next several conferences, we certainly hope.

Shortly after I talked with Dr. de Boer in November of last year, we were able to get Dr. Fremont-Smith to come to Washington. After about two hours of his spellbinding on his communications program, we were pretty well sold on the idea, although it did take approximately another year to germinate it completely and to bring it to fruition.

We are very happy to contribute toward bringing all of you together and are very hopeful about the benefits of the information that each of you has to contribute to the Conference.

FREMONT-SMITH: I thank you very much indeed. I wonder whether Dr. Taylor would like to say something on this.

TAYLOR: Just one comment. I think there is a strong connection between the ideas that were occurring to Colonel Hemler and to Dr. de Boer in Albuquerque and to me in Washington. It was a case of spontaneous generation of a set of ideas, that not enough had been done to try and separate what is known to be untrue, or what is not known at all about the effects of a nuclear war. This has run the gamut from saying that all life on earth in all forms would be destroyed to the thought that it would be something not terribly different from World War II. Those of us that thought this was an important subject for clarification have been quite sure that the answer is somewhere in between. This is a trivial statement, I think. Some other people don't think it is trivial, and that is why we thought this was an important conference to have—because some people do believe that one extreme or the other is correct.

I think that this one observation has really been the basis for the idea that a group of people who have been concerned about some of these questions should get together and try to shed some light on the subject. Then, having done this, to try to pick out those parts of the subject that have been clarified and at least display them to other people, and what is not clear, perhaps even to display that more definitely.

MC CULLOCH: What are the ground rules? We are, by and large, interested in the biological effects, the long-term biological effects of nuclear warfare. Is that right?

FREMONT-SMITH: Biomedical and psychosocial.

MC CULLOCH: Yes, but does this include our looking at, say, an area where we have exploded bombs to see what has happened to the level of ground water?

FREMONT-SMITH: I don't think anything is excluded.

MC CULLOCH: Let me ask about the effects on rocks and the subsequent weathering, which is certainly going to be a consequence of it and which would certainly in a large measure upset the ecological conditions of all forms of life in the area.

FREMONT-SMITH: Which will be part of the biomedical and psychosocial impact.

MC CULLOCH: Yes, but only indirectly.

FREMONT-SMITH: Only indirectly, but I think that we are free to talk about those aspects of the problem that we wish to, and the very fact that we are in an interrupting mood means that any of us can really set his own rules by interrupting and speaking up.

MC CULLOCH: I think it would be quite within the province of this organization to ask the U.S. Geological Survey or some such group of trained scientists and some good crystallographers to look at the area of Alamogordo and find out what has happened to the rocks, to the water tables, etc. I would like to put this before you as a suggestion as to the kind of thing that we should come up with by way of questions to put to other scientists who will not be represented here.

FREMONT-SMITH: A very good idea. There may be similar ones.

BRUES: The unanswered questions will be written into the record, I believe.

FREMONT-SMITH: Yes, indeed. Not only this, but we can pull them together and even decide on which group they should be referred to. I think it is an excellent idea.

SELF-INTRODUCTION

BRUES: I think the next order of business is self-introductions. These can be either very long or very short and they should be only moderately short.

I will introduce myself first. I took my original training in the classics and then in medicine at Harvard. I did a little radiology and a little neuropsychiatry. It was in the latter that I first met Dr. Fremont-Smith. I was in cancer research and clinical hematology for awhile in Boston and then I was brought into the atomic energy business during the war, went to Chicago, eventually ending up as Director of Biology and Medicine at the Argonne Laboratory. I managed to work my way out of that job to be a simple scientist about three years ago.

UPTON: I, too, work in one of the national atomic energy laboratories, at Oak Ridge, in the Biology Division. I, too, was trained in medicine, at Michigan, where I stayed on after graduation, and studied pathology. I went to Oak Ridge in 1951 to work with Jacob Furth on radiation injury. Since that time, I have stayed on there, very much absorbed in the study of radiation injury, particularly in the late somatic effects of radiation. I have tended to concentrate on radiation carcinogenesis in my work, especially on the induction of leukemia.

BRILL: I was trained in medicine originally at the University of Utah, took a Ph. D. in biophysics at the University of California after having spent two years in Japan working at the Atomic Bomb Casualty Commission. During that time I had the unique opportunity to join the group of scientists who have collected and analyzed the leukemia data, and, in addition, to focus attention on the in utero exposed children, who, in 1957-59, were entering puberty.

When I came back to the States, I stayed with the Public Health Service for five more years to assist with the ABCC operation from

Stateside, and attempted to identify and study other radiobiologically significant population groups. Two years ago I returned to academic medicine at Vanderbilt University in diagnostic nuclear medicine, working on the beneficial applications rather than the deleterious effects of low-level radiations.

LIFTON: I mostly do psychiatric research at Yale now. I am concerned with ...

FREMONT-SMITH: I thought you just came back from England.

LIFTON: Well, I am sort of a peripatetic psychiatric researcher, concerned with psychology and history. That cosmic area includes everything. The first study I did was one of Chinese "thought reform," or so-called "brainwashing," which I conducted in Hong Kong. There seem to be some echoes, if not of my study, at least of the process, going on these days. Later on, I returned to the Far East and did a study of Japanese youth, again from the standpoint of individual psychology and historical change. While there, in 1962, completing that study, I became interested in Hiroshima and in the psychological effects of the atomic bomb mostly in Hiroshima itself, in that investigation. It became a very absorbing matter, and I have spent most of my time since then thinking about it, and then going on to related subjects, perhaps even more difficult, of involving psychology aspects of death symbolism and of the impact of nuclear weapons.

COURT BROWN: I am from Edinburgh, Scotland. I was graduated in medicine in Scotland, and for the last 15 years I have worked for the British Medical Research Council; for the last 10 years I have directed one of their research units, the Clinical Effects of Radiation Research Unit.

I have done a certain amount of work on the induction of leukemia in irradiated human populations with a colleague, Richard Doll, and for the last eight or nine years I have been primarily interested in human cytogenetics. Within human cytogenetics, my main interests are in human population cytogenetics and in radiation cytogenetics.

BUSTAD: I took some of my work at Washington State University in animal nutrition and veterinary medicine. I then joined Hanford Laboratories as a sheep herder, the first and probably the last sheep herder General Electric ever hired. I stayed with sheep until I recognized the value of swine. We developed a strain of miniature swine that became more famous than I by a country mile.

I took some time out to become a mouse house manager at the University of Washington School of Medicine, where I took a Ph. D. in physiology and biophysics. I returned to General Electric's Hanford Laboratory, which was recently taken over by Battelle Northwest. Although both of these are fine organizations, I followed Randy Brill's example. I, too, decided to return to academic life, so I went to the University of California at Davis. Davis is one of the nine campuses of the University of California. It is the northern and sane campus of the University. There is another sane campus, and Dr. Stafford Warren will mention that when his turn comes. I have really gone to the dogs, for at the University of California, I am chief kennel boy for over 1,000 beagles. My title is Professor of Radiation Biology and Director of the Radiobiology Laboratory.

I have been interested in the late effects of irradiation and I have spent a good deal of my time on radioiodine and the thyroid.

MILLER: I am a pediatrician by birth, and after finishing training in pediatrics I didn't feel quite finished. So I was a postdoctoral fellow in radiation effects for the Atomic Energy Commission and then tried to combine what I learned from that experience with pediatrics. I went to Japan, where I was responsible for the pediatric studies at the ABCC. That led me to epidemiology. ABCC is really an epidemiologic study. I trained in epidemiology somewhat formally and am now in charge of the branch concerned with that discipline at the National Cancer Institute. My interests are in leukemia, congenital malformations, and radiation effects.

ABRAHAMSON: I am now at the University of Wisconsin. I got my Ph. D. at Indiana University in Drosophila genetics, and since 1956 have been doing radiation genetic studies with the fruit fly. I am interested primarily in mutation rates and chromosome rearrangements induced by radiation.

JOHNSON: I am an internist. I received my training in internal medicine and cardiology at New Haven, where I was until 1964, at which time I left to join the Atomic Bomb Casualty Commission. I have been there since as Chief of Medicine. Concurrently, I am Director of the Division of Epidemiology at the Cornell University Medical School.

SCHULL: My interest is in human genetics. Perhaps one of my few claims to fame is that, save for a single word, I might have been one of J. H. Dofjansky's students many years ago at the time

when I was exploring the possibility of graduate training. I wrote to him trying to find out what was available at Columbia University at that time, and he wrote back a very cordial letter, saying that both he and L. C. Dunn were involved with graduate students. In those days, "involved" had a somewhat different connotation, and I wasn't sure whether that was where I wanted to get my training. [Laughter] Instead, I ended up with Larry Snyder at Ohio State University, and my choice of a professor also led to a choice of an organism other than Drosophila.

I found that I was interested in a beast that is less at home in the air, namely, man, and the next thing I got involved in was the geography of Japan. This has led to a long sequence of events of which an interest in radiation biology is only secondary to an interest in a country.

MC CULLOCH: I started life expecting to be a theologian. I went to college at Haverford. I ran into the most marvelous Quaker philosopher, Rufus Jones, who put me in a corner and asked me what I wanted to be. I told him I didn't know. He asked me what I wanted to do. I told him I didn't know. I just knew I wanted to know what a number was that man might know it and a man that he might know a number, and he said to me: "Thee'll be busy as long as thee lives, and happy!"

I am not a Quaker. I went to war, joined the Navy, and became a second-class seaman and was shipped to Yale as an instructor in Marlinespike Seamanship and Semaphore. That's topology and communication as far as I am concerned. I have been with them ever since.

I went through training in philosophy at Yale and training in psychology at Columbia. Then I studied neurophysiology, neuroanatomy, and so on. I am an M. D., an old Bellevue Hospital doctor. I went back and studied more mathematics and physics and then I went to Rockland State Hospital during the depression, where I came to know crazy people and to love them.

I went back to the laboratory at Yale, the Laboratory of Neurophysiology and from there I went to the University of Illinois as a professor of psychiatry and built a team of 50 people working on physics, chemistry, anatomy, and physiology. I did a lot of work in chemical warfare on the German war gases, etc. Then the U. S. Navy stole

from me one S. N. Stein, a neurosurgeon who had run my gang for me for nine of the eleven years I was with them, a marvelous fellow. Navy took him on at Bethesda to run neurophysiology for them; and I knew my neck was broken unless I got out in a hurry.

I went to MIT, to the Research Laboratory of Electronics, under one condition, that I never had to take an academic status of any kind. I have worked there happily from that time until the present moment. In the Research Laboratory of Electronics, I head a group in neurophysiology and my principal job there is no longer with the experimental detail—the boys are way ahead of me—but with an attempt to develop a decent circuit theory for the activity of the nervous system.

Things are going ahead beautifully. I have had a marvelous team of youngsters. I have got two years to go before they throw me out, after which time I am going to have even more fun.

Thanks to Frank Fremont-Smith, I was for 10 sessions in the chair of the early meetings of cybernetics. Those were about the most exciting meetings I ever attended, and they were very brutal. We had one rule that we always took two people from any one discipline so that every guy knew that there was somebody else in the audience who understood him when he spoke laboratory slang. Yet they were brutal. The first five were certainly not publishable. The last five became publishable, but only by dint of good editing of our remarks.

In the old days people left our meetings in tears and never came back. That goes for men and women. To talk across disciplines of just what you have to do is extremely difficult. You can count on making a fool of yourself, and I hope everybody here will feel free to do so. I know I have many times.

The art of communication in a group like this is an old story to Frank. We have learned to know it from him, and the main rule is that you are completely welcome to say the craziest and stupidest things you can imagine and count on somebody else to stop you. So I would say my main contribution in being here is not that I know anything about radiation biology. I got my hands burned in 1927 in Bellevue Hospital, but I don't know much about radiobiology except firsthand. I know nothing about the long-range effects on the biological system. I am here as an ignoramus and ready to make a fool of myself.

FREMONT-SMITH: We all hope to join you. [Laughter]

WHITE: I was christened Clayton Samuel White, and I grew up as Sam. This little fact has been useful to me because I am often asked who Sam White is, or do you know Clayton White, and vice versa. I listen very carefully and then I confess I am one or the other, whichever happens to be appropriate for my self-esteem. My wife calls me Clayton only when she is angry at me, but my English friends learned to call me Clayton when I was in school there. All my close friends in this country call me Sam.

I grew up in northern Colorado. I majored in psychology at the University of Colorado and minored in mathematics and physics. Through psychology, I became interested in psychiatry and was horrified to find out that a medical degree was required. Being young and silly, I said, "Well, I might as well get one." I started this exercise in Great Britain at Oxford, where I had the good fun of going to school for three years. I did a degree in physiology and also a bit of medicine at the same time. I finished my medicine at Colorado University.

By this time the war was on and I spent four or five years in the Navy during which I learned a new profession, Aviation Medicine. During that time I became acquainted with Dr. Randy Lovelace at some intraservice meetings and went to Albuquerque in 1947 to join the staff at the Lovelace Clinic. When the Lovelace Foundation was formed I slid over to look after the research effort of that organization.

The present form of the Lovelace Clinic and Foundation was organized in 1947 in somewhat—well, the legal work was similar—the same manner as the Mayo Clinic-Mayo Foundation. During the growth of the Lovelace Clinic and the Foundation, we have had the opportunity to put teams of people together to participate in some of the exciting things that nuclear energy made feasible. Some of these began with experiences in 1953 and continued in 1955 and 1957 at the Nevada Test Site doing research in the blast effects area. Subsequently, the Foundation has been able to continue work that was initiated in this area through the support of the Atomic Energy Commission along with the support of the Defense Atomic Support Agency beginning in 1959-60. At the same time, I and others on the staff have maintained some interest in selected aspects of weapons effects through the continued support of the Division of Biology and Medicine.

The Lovelace Foundation research programs are rather diverse and they touch about all of the disciplines that you find in an ordinary medical center or medical school. Most of the significant research programs touch three, four, five, or six disciplines, and many of you here understand the difficulties that develop when one tries to work and communicate between the disciplines.

However, as progress is made, the problems scientists have on their backs grow more complex, perhaps just because good research asks more questions than it answers. Also pertinent is that one of the sicknesses in science is a lack of flow of information from the lab to the people who can use it. But this should not, it seems to me, bother the man who really is interested in getting fundamental information. He has the good fun of doing this, and if he is really a talented individual, his motivation is the same as that of an artist. His media of expression are just different.

I am not a very wise fellow; I think you will find this out during this Conference. But I realize the problems that are set down on these pieces of paper as the topics of discussion here don't do anything but frighten the devil out of me. They are difficult, highly technical, and can hardly be handled adequately in the time alloted for the discussions here. If you will join me in this belief, fine. If you won't, I will try to see that you do! [Laughter]

FREMONT-SMITH: That is part of your wisdom.

WARREN: I am the oldest one here, I am sure, and I think I can claim longer contact with experimentation in radiation because I was a Hooper Fellow in 1916 at the University of California under Dr. Whipple. As a sophomore medical student, I worked on the effects of total body and partial body radiation, and studied actively (Reference 1) the damage of the crypt cells in the small intestine, the destruction of which by radiation was possibly the mechanism which caused the death of the dog by what was then called "proteose intoxication." This was a nice catch-all term, and probably the exact mechanism is nearly as unknown today. Anyway, it was a good start, and it is just about 40 years ago today.

After my medical degree, I took some more pathology and what amounted to about a four-year residency in medicine and then became persuaded to go to the new University of Rochester School of Medicine as a radiologist. There wasn't much time left for this training. So

I had three months' training in x-ray diagnosis and in therapy and then toured radiology clinics in Europe for three months on a Rockfeller Fellowship. With this background, I became an assistant professor of radiology and medicine in 1926 and had to build a department from scratch.

Dean George Whipple, when I arrived, gave me a piece of chalk, a 3-foot ruler, and said, "Go upstairs and mark the x-ray department on the floor and they'll built it!" [Laughter] And then I had to go out and buy the equipment, and so on. So I started the hard way. I admire the precision with which many of you gentlemen can speak of your own discipline because of the high quality of your training. I have had essentially no training in the basic sciences but I have had a lot of experience and I hope I have been a good observer.

I take full credit for the invention of biophysics and in the late 1930's I trained the first Ph. D. in biophysics in Rochester, New York. He is Professor William Bale. In the early days he built with baling wire and few dollars a mass spectograph which he then used for the measurement of Urey's deuterium to follow the development of hemoglobin in the anemic dogs that Whipple was working on. We built our own geiger counters and almost all of the equipment that was used at that time. We got some isotopes from Lee DuBridge's Cyclotron in Rochester's Physics Department which we helped to build and supplied a good deal of "junk" that went into the making of it. We got many isotopes from Robley Evans of MIT. Just before the war, we began to get interested in what might happen if there was contamination with radioactive materials, first in the laboratories and then buildings and crop areas. We learned that a 3-foot concrete wall was not a 100 percent shield against gamma rays, for there was a percentage that got through, and that scattering of intense beams was a very important factor to be guarded against. It was about this time that George Casarett got involved in the team.

In 1943, I was invited to become the medical chief of the Manhattan District or else I would be drafted! [Laughter]

There was a very interesting episode. General Groves and Colonel Marshall had me for lunch on the invitation of Dr. Gerald Chapman of Eastman Kodak at the local club. They tried to ply me with drink, which I fortunately refused. We went upstairs afterward. They went into a room and dismissed Dr. Chapman and shut the door and looked out the transom and out the window and under the bed. Then they said,

"We want you to work for us on a secret program. We can't tell you what it is but we have asked your President and he's agreed; we've asked the Dean and he's agreed." That was the way I was introduced into the medical section of the Manhattan Engineering District atomic bomb program.

Afterward, I had to go to Japan on special orders from the War Department to survey the effects of the atom bombs before we got troops in. We didn't expect to come back, but then we did very nicely.

Then there was Radiological Safety at Bikini and after that I was the civilian chief of the Division of Biology and Medicine in Washington until the civilian AEC was created and Shields Warren then took over as the chief of that division.

In 1947 I thought that, as the dean of a new medical school at the Los Angeles branch of the University, mentioned by Dr. Bustad earlier, I might be able to put research in its proper place, because at that time research was not recognized as an official obligation of medical schools; in fact it was almost a dirty word in the University budgeting process. Space costs money and research is not always returnable, at a dollar value anyway. It took 15 years of constant effort to achieve a balance of teaching, research, and service which I hope will stick.

At UCLA I was fortunate to be an AEC contractor to do some field work after the war for the Alamogordo follow-up on the very first bomb test, and, like Dr. White and others, until recently I have been engaged in fallout and other studies at Frenchman's Flat.

However, time moved on rapidly and in 1963 I had to retire. Fortunately for me and my successors, I was "drafted" to help President Kennedy on the mental retardation legislation and the politicking of getting that program along. So I left my successors a free hand by going to Washington for almost three years. Upon returning home, I decided to return to research. So I have now become an unregistered graduate student. [Laughter] This means that I am starting all over again. I have read furiously in radiation biology for a year and I think this communication problem is a tough one, because I was so brash as to ask Medlars for a few citations in a restricted area of the radiation biology literature since 1962, and I got 1,007 references, obviously too much to read profitably. I am exploring paleoradiation biology or the influence of the radiation background in the development

of the biological systems. I have found where a lot of gaps are and some very interesting places to work and I think I can begin to hold my own soon. At least I can understand what you experts are going to talk about in the next three days. Anyway, as an initiator, I am only supposed to ask you irritating questions and not to speak! [Laughter]

I have enjoyed looking forward to this meeting because there are a few old timers here and a lot of more recent friends too. Frank was at Bikini, and Austin was one of the Chicago reactor biology team during the war. He has been in and out of various ventures in Japan and other critical places. Sam has practically got himself blown up working with blast in Albuquerque and at Frenchman's Flat. Casarett was on the early Rochester team.

AYRES: The most relevant comment about my background is that I have never studied medicine of any kind. I studied physics at the University of Chicago, the University of Maryland, and the University of London—only theoretical physics. Also, I haven't been in a laboratory since my first year course. So my principal reason for being here is to make sure that this Conference is interdisciplinary by slightly diluting the concentration of medical doctors.

For four years, up until last summer, I was with Hudson Institute working on a project supported by the Office of Civil Defense. The purpose of this study is to look at the long-range ecological and environmental effects of possible nuclear war. I am no longer working full-time on this subject; I am now working on air pollution. But hopefully the results of this four-year study will eventually be published by the local press here, if the manuscript is ever completed.

WARREN: What is the Hudson Institute?

AYRES: It is a research organization founded by some refugees from RAND Corporation. The director is Herman Kahn, who was formerly at RAND. I think he spent a year here at Princeton in between the two. The Institute spent, I suppose, most of its effort in the first four years of its existence working in national security affairs, a good part of that on nuclear strategy and civil defense. It is still working on those things but at a lower level, and has gone into other activities. However, policy research still remains its primary concern.

AUXIER: I am from the Health Physics Division of the Oak Ridge National Laboratory. I seem to prove that physicists, like street cars, come in groups. I approach this field from the physical side at least. I also seem to have spent a good part of my life following, by 10 or 15 years, Dr. Stafford Warren and a few other people, in pursuing solutions to the problems associated with the bombings of Hiroshima and Nagasaki. This continued to Frenchman's and Yucca Flats. Back when Dr. Warren was taking his initial looks at Hiroshima and Nagasaki, I was still bending the throttles of the planes that were supposed to deliver bombs. But I found that working on the dosimetry and the radiation aspects of radiobiology and health physics under K. Z. Morgan is just as exciting as nuclear weaponry. I have been at the laboratory for about 13 years and find that it gets more exciting each year. Physical dosimetry and physical radiobiology are the things that interest me most.

FOUNTAIN: At present, I am assigned to DASA and am chief of the Medical Effects Branch. My training was at Cornell. I am a veterinarian, with graduate training in radiobiology at Reed College.

SPEAR: I will try to dispel the puzzlement that may have come over you when you found someone from an outfit called Public Administration Service represented. The fact that I am with Public Administration Service is probably irrelevant to our endeavor, but then most of my experience has been irrelevant to our endeavor, and I will pass over it briefly.

English major at Yale in 1932, which was a poor year for English majors! [Laughter] After a couple of years of sitting it out reading books, I took my M. A. in public administration at Syracuse; went from there to Chicago for nine years with the American Public Welfare Association; a short hitch in the Navy; came out to serve in a very improbable sounding bureau of our Federal Government—the Foreign Relations Service of the Veterans Administration.

If I can skip a little in the sequence here, about three years ago I retired from the Federal Government and went with the Public Administration Service, a nonprofit consulting firm which works only for government, and there the irrelevancy multiplied rapidly. I found myself concerned with the education of the deaf, with local building codes, with laws relating to our public lands, with data processing requirements of vocational schools, with traffic safety, and a number of other very interesting things.

To go back to the period I skipped, in 1951 I quite accidentally found myself in the Federal Civil Defense Administration, and for some 12 or 13 years I was involved in one way or another with nonmilitary defense. Part of my office was responsible for some of the civil defense effects tests in Nevada. I also was instrumental in setting up, under the National Research Council, a behavioral science advisory group that worked with us. I was also the representative of our agency, which changed its name a couple of times over the years, on the Planning Board of the National Security Council.

While there, I got caught up in an endeavor with Frank Fremont-Smith and Staff Warren in another group, of which this reminds me a good deal. It was an interdisciplinary look at the human effects of nuclear weapons development and it was probably from that experience that Frank thought of me as coeditor of this Conference Series.

CASARETT: After studies at the University of Toronto, in June, 1941, I came back to my hometown, Rochester, New York, with the intent of going either into graduate studies in medical sciences or to the Medical School. There I met Dr. Staff Warren, who infected me, as he did so many other young people, with his enthusiasm for research. He hired me in his Biophysics Division of the Radiology Department and I worked with a number of enthusiastic medical students who he had talked into dropping out for a year or two to do research on the problem of secondary shock. I would like to say now that he was a wonderful and inspiring man to work for.

When staff went into the Manhattan Project, I went into the Manhattan Project laboratory at Rochester, in the pathology and hematology divisions, and also continued studies which led to a Ph. D. in a combination of anatomy and pathology.

My interests have been in radiation pathology all of these years, with a special interest in long-term effects of radiation and their mechanisms.

When the Atomic Energy Commission was formed, its project at the University of Rochester became established as the Department of Radiation Biology of the Medical School and subsequently as the Department of Radiation Biology and Biophysics. I am a professor in that department, with a section of experimental radiation pathology. I have a secondary appointment in the Radiation Therapy Division of the Radiology Department, and I direct the radiobiological research in that division.

DE BOER: I am a Dutchman by birth and an American by choice. I am probably the junior in the field of radiation biology. I am a product of the Texas school system. I have one distinction that I am sure none of you have: I am a master cook and baker and have the papers to prove it in Holland. I came to this country at the age of 28 and was given the opportunity to go to school. I pursued this course for the next 10 years and finished with a Ph. D. from Texas A & M University. Two of the six years of graduate studies were spent at the Texas Medical Center and Baylor University in the physiology and biochemistry departments.

I function presently at the Air Force Weapons Laboratory as a project officer on a Nuclear Weapons Effects project. One of the reasons I have been interested in this Conference is because often I am asked for a so-called expert opinion, but since I am not an expert, I would like to learn more. Another reason why I am here is to learn firsthand about nuclear weapons effects, particularly since I have had no previous experience with nuclear weapons, per se. I have not been in Japan nor at the Nevada Test Site. During my stay at the Air Force Weapons Laboratory some testing has been done at the Nevada Test Site, but few civilians have been taken out there. I find myself often asked for facts and opinions about nuclear weapons effects on man, but at this time little is known and all has to be investigated through experimental animal research. I feel that the lack of communication between the people who provide the instruments of destruction and those who are supposed to tell them what the effects are on a population is very great, and both sides need to show much more understanding.

Just being frightened is not enough and it should not keep us from trying to get the best possible answers. This is another reason why I am here. I feel very strongly that this Conference can have beneficial effects, and I hope it will.

HEMLER: Dr. Stafford Warren, I hope the medical people will excuse my horrible pun, but when you were first getting started in 1916, I really wasn't even a gene in my father's eye. [Laughter]

I was graduated from the Military Academy at West Point in the early 1950's and after a fairly short tour in the Army as an artilleryman, the Army sent me to nuclear weapons school where I began to learn about nuclear weapons in 1957. Then, in the effort that the Army and the other services are now making to try to bridge the

information and the language gap between the military and the scientific communities, they sent me to Graduate School at the University of Arizona for a two-year education in nuclear physics and mathematics.

I finished that and was assigned as the physicist at the Air Defense School at Fort Bliss for a couple of years. Actually it was supposed to have been three years, but DASA decided to borrow me, as they often had others in the past, for a nuclear test series. The original borrowing period was supposed to have been 30 days. It started in April of 1962 and I returned home in late December of 1962 after spending a good summer at the Nevada Test Site with the Dominic Series. From there I was sent to Korea where I had occasion to work in the Army Headquarters on the employment techniques and the tactics for nuclear weapons.

After Korea, I was sent to one of the schools of learning in the Army at Fort Leavenworth and from there to DASA where I am now with the Deputy Director (Scientific). You might say that I am a generalist in the very purest sense of the word.

ANGEVINE: I think, Frank, it has been 17 years since I bared my soul to an audience like this, at the beginning of a Macy conference on connective tissue that continued for five years. If this proves to be as pleasant as that was, it probably will be worth the trip East.

I was graduated from McGill in medicine and took a good deal of my medical training at the University of Pennsylvania Hospital and then went to Cornell when they opened the Cornell Medical Center in 1932—which sort of dates me—and trained under Dr. Opie. Following 10 years in New York, I went to the Alfred I. du Pont Institute for Crippling Diseases in Children. This was a fine opportunity to study the diseases of connective tissue and I have been interested in rheumatic diseases since.

I then went overseas and was the pathologist, in the European Theater of Operation, for the First Medical General Laboratory. When I was in Paris, Dr. Middleton asked me if I would go to Wisconsin as professor of pathology. I wasn't sure I wanted to go west of the Appalachians, but after due deliberation I did go to Madison.

I have done some work on the long-term effects of radiation on purebred beagles. This was before the beagle became as popular as it is at present.

In 1962 I went to Hiroshima for one year with the Atomic Bomb Casualty Commission and had a very profitable and interesting year. In my spare time I edit the Archives of Pathology.

MATSUMOTO: I am with the Department of Medical Sociology, Atomic Bomb Casualty Commission in Hiroshima. I received my doctorate from American University in Washington, D. C., followed by a year of further graduate work at Harvard University.

During the war I worked with the Office of War Information (OWI) in the Pentagon, studying the morale factors of the Japanese military forces. With the termination of the war, I served as a civilian with the Army Air Force on the U. S. Strategic Bombing Survey, traveling throughout Japan interviewing respondents, and later helping with the analysis. Under the Allied Occupation, I returned again to Japan and worked with the Public Opinion and Sociological Research Unit of the Civil Information and Education Section in Tokyo. After two years, I decided to return to the States and complete my studies. While attending graduate school, I also worked part-time for Princeton University, receiving my pay checks for almost a two-year period, although I was never here in Princeton. At that time I worked for the Office of Population Research (of Princeton) with Dr. Irene Taeuber in Washington, D. C., on the population of Japan. After I received my degree, I went back to Hiroshima and Nagasaki where I have been for the past 10 years. Perhaps it indicates a long time, because many American scientists who come to visit ABCC always ask me why my English is so good! [Laughter]

TAYLOR: I did my undergraduate work at Cal Tech. Because of the severe dislocation of having been put through Cal Tech by the Navy through the V-12 Program, I proceeded to use the GI Bill of Rights to do my graduate work at Berkeley, which has always seemed to me sort of an injustice. I received my Ph. D. in physics at Cornell.

From 1949 to 1956 I mostly worked on the design of nuclear weapons, following which I spent six or seven years with the General Atomic Division of General Dynamics Corporation in San Diego. Most of the time there I spent promoting a pretty wild-sounding project for propelling a spaceship with a series of nuclear explosions. This project has been called by some people a disarmament project in disguise, because they thought that the only conceivable reason anybody would suggest putting a bunch of bombs on the structure and then

firing them through a hole in the center of it and setting them off beneath it was simply to get rid of the bombs in very large numbers! [Laughter]

In 1964 I went to DASA for two years, and since I left the Pentagon in September I have been in Vienna working as an independent consultant, which seems to me like someone who doesn't know quite what to do next. I have been working there on problems of international control of atomic energy in close proximity to, but not as part of, the International Atomic Energy Agency. I am here as an expert in essentially nothing at all, but very much concerned about the imprecision in what people have said about what would happen if we had a nuclear war.

FRANK: I am a psychiatrist at Johns Hopkins Medical School. I received my original training in psychology at Harvard and then couldn't get away from the place and took an M. D. also. I went to Johns Hopkins for my psychiatric training and then joined the Hopkins Unit in the South Pacific area; was in the Philippines at the time the atomic bomb was dropped in Hiroshima. It began to haunt me then but didn't interfere seriously with my career. I came back to Hopkins and did my job of taking care of the mentally ill until about 10 years ago, when it suddenly surfaced, and it occurred to me that one should take seriously what all the world's leaders have been saying about the need for eliminating wars, as the only hope for civilization. They haven't taken it seriously yet and it seems to me that they should. During the past 10 years much of my time has been devoted to the very difficult, perhaps hopeless, problem of how war can be eliminated. Conventional war would have to be included, because one can never be sure that a conventional war won't escalate into a nuclear one. I think the time will come when the Department of Defense will see its function as the prevention of war, and I hope that the deliberations of this Conference will help to move it in that direction.

KNORR: I was born and brought up in Germany, which I left in 1937 for reasons of political incompatibility. Since I had only a law degree, I decided to go back to graduate school. I went to the University of Chicago, where I received a Ph. D. in economics. I was an economist at Stanford and later on at Yale. I am now a professor of economics at Princeton and Director of the Center for International Studies, which is a multidisciplinary research organization concerned with studying various international problems.

Although I am an economist, I have not really worked in the field of economics for some time, 10 years or more. More recently, I have done research and writing in the field of military strategy, problems of arms control and disarmament. Recently, I published a book on the uses of military power in the nuclear age. I know absolutely nothing about the subject matter of the Conference. My interest in the subject matter is, well, it is unformed at the moment.

Referring to the chairman's guidelines, I think I bring no hostility to the Conference, and my free-floating anxiety is slightly positive but unstable! [Laughter]

JABLON: I am with the staff of the National Academy of Sciences. I started out by training in mathematics at Columbia and then moved first to mathematical statistics and finally into medical or biostatistics.

For the past 20 years we have been working, at the Academy, on a program of longitudinal medical studies of veterans, and it is something of a coincidence that the Academy also is responsible for the Atomic Bomb Casualty Commission in Hiroshima and Nagasaki, which is also a longitudinal medical study of humans. My associations with ABCC date back to 1955 and I spent three years there, from 1960 to 1963.

FREMONT-SMITH: Because Stafford Warren is going to show us a film, I will refrain from giving you that long and delightful story of my life, which I would so much like to do! [Laughter] But I will tell you a few small points.

I had training in medicine at Harvard Medical School, pathology and medicine and neurology, psychosomatic medicine, and some psychiatry. I insist upon telling my friends that I am really only a half-baked psychiatrist, and if they ask me why half-baked, I say, "Well, of course, you know, to be fully baked you have to lie quite flat on a couch during your analysis and I was in a semireclining position the whole time, so I'm only half-baked!" [Laughter]

Then in 1936 I was invited to join the Josiah Macy Jr. Foundation, but before they would accept me, the trustees had to look me over. One of them, a very delightful gentleman, sat me in a chair in his apartment, took a seat directly opposite me, and just looked at me for the longest time. Finally he said, "Well, Dr. Fremont-Smith,

do you believe in conferences?" Well, I hadn't even thought about conferences. In fact, I practically had never been to one and I didn't know what in the world to say, yes or no, yes or no; better to say yes. I said, "Yes, sir, I do," and then I thought this was quite inadequate and I would have to add something more. I couldn't think what in the world I should say. I said, "Yes, sir, I do, if they last long enough." This is why we have been having conferences that I hope last long enough.

Staff Warren told us about the very important occasion in which he didn't have a cocktail, but I also remember an important occasion in which he had two cocktails and undoubtedly if he hadn't had those two cocktails, we wouldn't be holding this Conference.

This was when he emerged from the Manhattan Project. He had absolutely disappeared—nobody had seen or hear of him for years and years and years. All of a sudden he appeared at the New York Academy of Medicine to give a lecture on the atomic bomb. Of course, I went up to the dinner preceding the lecture and there he was, surrounded by a group of people, drinking his third cocktail, I think it was. I approached him with my first one in my hand. I heard him saying to the group, "And I want to have some people come out to Bikini who will really be interested in the full significance of this experiment." Then he turned to me and said, "Frank, you are just the kind of man I want to have." So I said, "Staff, don't say that. Don't kid me. I couldn't stand it." He said, "I'm not kidding." I said, "All right, try to get rid of me!" He tried to, but he never has succeeded, and that is how I happened to be out at Bikini.

I want you to imagine the expression on the face of the skipper of the destroyer U. S. S. Ingraham, when a civilian employee of the Army climbed aboard, who was to be responsible for the movement and safety of the ship and crew—which was my position out there. However, I was careful not to rub this in very hard, because I didn't think it would have had any impact on him if I had. We did get rained upon and through another fluke we were able to bring back the first radioactive rainfall that had ever been collected.

I think you all know what a specialist is—a person who gets to know more and more about less and less until eventually he knows practically everything about almost nothing. I am just the opposite; I am the generalist, one who gets to know less and less about more and more until he knows practically nothing about almost everything! [Laughter] This is where I now stand.

To close our session tonight and to arrange the setting for our meeting tomorrow, Staff Warren has some films of nuclear explosions to show us. Would you like to tell us what to look for, Staff?

DESCRIPTION OF NUCLEAR DETONATIONS

WARREN: I ought to tell you about it first, because it goes so rapidly that, unless you are warned, you may not see the phenomena that are of interest. First, there is just a strip of two little drone planes and the mother plane and then of a drone plane landing on an airfield. This was an historic episode, because it was the first successful use of drone planes going into the radioactive cloud, hopefully to collect a sample of radioactive material on filters. This tells a physicist what the efficiency of the yield was and gives the radiological group some idea of the intencities too. This worked very well, although they did not risk sending the drones into the very highest concentration where the turbulence and perhaps the heat might have destroyed the planes.

There is a brief shot of the Bikini coral and beach for background color before showing the camera towers which are on the white, sandy shore. The towers were essentially three miles from the zero point of the detonations in the target center of ships of all kinds. You will recognize pictures taken by them because they are relatively close up, using telephoto lenses. These towers had lead shields in front to protect the film from the ionizing radiation. The shields parted after the initial prompt radiation was over to allow the lens to look at the scene as it took place.

MC CULLOCH: What kind of bomb?

WARREN: These are two nominal atomic bomb shots, the first one, the air drop, is called Able, and the second, underwater, is Baker. These tests were carried out at Bikini Atoll in 1946. It was a highly organized experiment to obtain every kind of information possible from the effects as a weapon and the basic physics. About 70,000 persons attended, including the scientists, military contingents and crews, and newspaper representatives. The large target fleet consisted of obsolete warships of all kinds and even the latest German and Japanese designs.

The first view is from nine miles away from the target fleet so that not much is seen, except the flash through the rigging of the Mt. McKinley and then the rising column of peach-colored hot gases which seem to come up rather slowly at this distance. These are actual elapsed times. The column eventually reaches over five miles into the sky.

In the telephoto tower views, you will see some of the furious action of the updraft as these incandescent and hot gases rise from above the lagoon and the nitrogen oxides form from the intense ionization of the air to give an intense red and brown color and later a peach color to the vapors.

There is one view at the base of the first shot which shows the fleet le ing over, and it is quite obvious that some of the vessels that were broadside to the blast front keel over almost enough to allow the gunwales to dip into the water. Then just as they come back to the upright position, the black soot knocked from the smoke stacks obscures the picture. Later, as the column rises in the air, the winds at the various levels through which it passes shift it, but not enough to change its general upward course.

The top of the Able column reaches a height which is determined by the reduction of the initial energy of the detonation as it rises through the layers of colder air, so that eventually it stops rising. In this first test of a nominal bomb, the top of the column is somewhere in the order of 60,000 to 65,000 feet, and, of course, even from nine miles it reaches an appreciable height and is visible as a very large object. I mention this, because in looking at such a distance at sea, the proportions are apt to be lost. This has been true also in the desert.

As this column goes through very cold areas, very fine ice crystals may form on the surface. It looks as if a veil is being pulled across the top (at 50,000 feet). This takes a tremendous amount of energy (heat) out of that top so that it slows down very appreciably at this point.

The passage of the positive part of the blast wave compresses the air and then is followed by the negative part of the wave which expands it vigorously. This produces in this humid air a Wilson cloud chamber effect which you can see to some extent in the Able air blast as a condensation or mist which travels outward very rapidly at about

the speed of sound. It was rapidly broken up and dispersed because there wasn't enough pressure or moisture in the air to keep it intact for more than perhaps a radius of five or six miles.

The rest of the film is of the Baker shot or underwater detonation. The underwater blast emerges from the surface like the head of an asparagus tip and then immediately becomes a column and then a weeping willow tree about five miles in diameter. The column rises rapidly like a projectile to about 12,000 feet high. There must be a great deal of solid water in it from above the bomb for it to go so high. Immediately, but clearly later, the blast reflected from the bottom of the lagoon takes the water that was below the bomb up through the center of the column reaching a height of about 15,000 feet. It spreads out into a huge weeping willow tree and begins to fall back to the lagoon.

The water falls down much like what you see in a big waterfall, either Niagara Falls or Yosemite Falls, in great big rockets of water that keep shooting outward and down.

The stem of this huge mass of water is about 2,000 feet across and presumably the stem is hollow because the gases are vented up through the stem. They caused turbulence and icing and the spread of the water and vapor at the top.

The ejection was so rapid that a blast wave was formed—a compression wave in the air. It is like the emergence of a bullet from a rifle barrel. In this case there was only a rumble because the frequency of this pulse is quite low. But the resulting Wilson cloud chamber effect is very clear here. In fact, it obscures your view almost immediately as it travels outward and you will see views from the air which show the top of this column, which is probably now mostly foam, causing a blast wave and a Wilson cloud chamber effect on top, and you will see this come off like a lid.

If you look at the lagoon surface from the air, you will see that the water is compressed on the surface by the bottom of the standing blast wave front as it travels outward. It causes the wave pattern to disappear making the surface look smooth in an expanding disk.

The radioactive materials are now mixed in all of this water and foam which rains down over a diameter of six miles. A mist or ghost is left behind which has a high content of radioactive materials.

It drifted out to sea and contaminated the ocean for quite a long distance. Wave action caused it to be diluted quickly and it was not measurable on the surface after three to four hours. It is my opinion that this underwater test produces the worst possible kind of problem because of the large amount of contaminated water and mist and the large size of the base surge. The Weapons Handbook (Reference 2) puts it at about 100 feet, somewhere between 50 and 100 feet of water and foam as this column of water descends and then surges upward and outward in the lagoon.

The size of this base surge as it migrated outward, particularly toward the shore, was limited by the depth of the lagoon which was about 250 feet. The lagoon remained about that depth for about one-third of the distance to the shore, that is, about one mile. Then the water shallowed and about half a mile off shore it became 15 feet deep. Thus the 50-foot high fluid water portion of the base surge, which essentially was a standing wave, had an additional 200 feet or more of foam on top of it. If the water had been deep enough, it might have come ashore full height and engulfed the island, but it stubbed its toe, you might say, on the bottom and decayed. Thus it came ashore only about 5 to 10 feet deep and did not destroy much of the shore installations or even bother the towers. But in places with very deep harbors, this standing wave might travel a long distance. One of the reasons for not doing Charley (the third one) first, which was supposed to be anchored in the deep water to the west of the atoll, was that the standing wave might have gone a long distance. You know that standing waves from earthquakes have traveled from Alaska to Chile or Hawaii.

The target fleet was restrained by anchors, to some extent; yet when the base surge engulfed them, they were tossed around with great abandon. This was enough to break loose the ladders from one deck to another, to cast loose coffee and other machines that were bolted down, and to upset furniture. Contaminated coral mud and seawater covered a great deal of the target fleet.

The amount of contamination on the fleet after this was over could be measured from airplanes passing 400 or 500 feet above the height of the mast. There were lethal amounts on the decks for many days and the clean-up presented insuperable problems to the extent that many of the target ships had to be abandoned.

SESSION II

SOMATIC EFFECTS

(Initiator: Stafford L. Warren)
(Chairman: Austin M. Brues)

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INTRODUCTION

BRUES: This is the first of a series of five interdisciplinary conferences on the selected effects of a general war. This series will take place over the next three years.

As to the first Conference, the organizing group decided to orient the meeting to the one instance where there is actually much definitive information, and this, of course, is the aftermath of the nuclear weapon drops in Japan. It can be hoped that the picture which will be drawn at this meeting on the immediate and later effects, in their many aspects, may supply a basis for future discussions of the possible nature of nuclear warfare of other types, which we can hope would not be encountered.

The first discussion will be initiated by Dr. Stafford Warren, who, I think, was the first person in this group to witness directly any of the consequences of the atomic bombings on location. He will start with a statement from which I hope we can, within the day, get into a substantial discussion of the late somatic effects on populations.

The material on genetic and related effects of nuclear weapons will be dealt with in depth tomorrow, although there is no reason why the word "genetics" should not be mentioned today. I am just indicating that there will be opportunity for a full discussion of that later on in the meeting.

Dr. Fremont-Smith, do you have any additional comments before we start?

FREMONT-SMITH: I might say that we had one conference on marine biology and one whole day was devoted to the giant ameba. This may seem unrelated, but not wholly unrelated to the topic of today, and by the end of the day it was quite clear that nobody understood what was going on inside the giant ameba or why—enormous amounts of plasma streaming and nobody had any idea as to the

origins of this motion. At the very end of the day somebody inadvertently mentioned the official name of the giant ameba, chaos chaos! [Laughter] So I hung on to that. I called it "chaos squared."

It may seem that the aim of our conferences is to have "chaos squared" and to some extent this is true. We would like to expose the degree of "chaos possible" and see where we can go from there.

Going back to Austin's [Brues] remarks, it is for this reason that, although our agenda shows some slight degree of organization, actually it is very tenuous and there is no reason why the genetic effects or the psychosocial effects should not come in today or the somatic effects come back into the meeting on Saturday morning.

LIFTON: Could I add just one thought before Dr. Warren begins?

FREMONT-SMITH: What! Interrupt him before he starts! [Laughter]

LIFTON: That's what I want to do. I was a bit troubled last night by what he said and what we didn't say about the purposes of the Conference. A few of us discussed the question later on. Some things, it seems, have to be discussed over beer anyhow. It was said yesterday that the idea of the Conference began with the purpose of trying to designate an in-between area, that is, between speculation that nuclear warfare would eliminate all of human life on the one hand, or is nothing but another version of conventional warfare on the other. This seemed to me an inadequate motivation for the Conference, nor do I think it was really the kind of motivation that I heard about in talks with Frank Fremont-Smith earlier which stimulated my own interest in the Conference.

What I would say very simply is that to have a conference on this subject is different. It is similar in some ways to having a conference on, say, connective tissue or group processes or cybernetics, because we are all scientists who have worked in a common area and we do want to exchange ideas across disciplinary barriers, and this is an exciting enterprise intellectually. The intellectual excitement is not to be minimized or tossed off lightly. But there is something more here. If we are scientists who work with problems of nuclear warfare, this is something different from, say, journalists dealing with nuclear warfare. We presumably bring a potential depth of understanding, a special grasp of things. And I think it is important

from the beginning to have at least a sense of our moral involvement as scientists, our sense of purpose in what we are doing in addition to our purely scientific function.

All of us have this sense in one way or another; each of us may express it or feel it differently. We may not share entirely what our visions are of what we are doing, and perhaps our various attitudes will come out more vividly during the course of the Conference. Nor am I proposing that we have a large discussion right now on the moral responsibilities of the scientist in regard to nuclear weapons, but I do think we should keep them in mind. I think these things are extremely relevant for this Conference. If they were not, I for one would not be here, and maybe others would not be here as well.

FREMONT-SMITH: Bob, I'm very glad you said this, and I most completely concur with you. I'm happy to say that DASA, in asking us to hold the Conference, said that it is up to us to make of it what we can and wish to, and we have been given no directives and no constraints. For my part, it is in exactly the frame of reference that you describe that I am interested in holding this Conference. I would hope that in some small way both the moral responsibilities of the scientists would come out, be evident from the discussions, and that we would make some contribution, small as it may be, toward the prevention of war, which is my primary concern in holding the meeting.

A second concern, which is primary in all our conferences, is cross-discipline communication, and we have the opportunity to do this around a topic of ghastly significance for the future of mankind.

LIFTON: Perhaps one question to keep in mind, and it is a difficult question to answer, is to what extent full accurate information about this subject, insofar as that is possible, adds to the general capacity for mastering these weapons; that is, avoiding their use, as Frank said. I wish it were a one-to-one relationship, that full knowledge then meant control and management of them. But I'm assuming that it at least contributes to it, even though the relationship is not simple.

FREMONT-SMITH: I think we would need to add full knowledge of nature to full knowledge of the weapons to make a one-to-one relationship and we haven't got full knowledge of human nature, I suspect.

WARREN: I might say that while I'm essentially a disordered, undisciplined person myself, I would like to approach things in a somewhat orderly fashion, because otherwise I get so involved in details that I never get the subject covered. In my country-boy fashion, I queried the administration of the Conference about an agenda, because in looking at the outline on somatic effects, one could spend a great deal of his lifetime in exploring different aspects of the problem that is created by the detonation of nuclear weapons. So I'm just going to make a few guidelines, I think, to start out with an I'll put one of them on the board:

Chronic somatic effects:

1. Blast effects
2. Thermal effects

We must, in effect, concentrate on chronic somatic effects, long-term ones on individuals and mass casualties with effects on large numbers of people. We should start out with blast and the thermal effects, these two being apparently of less long-term consequence (although not altogether) as will come out in our discussion. Hopefully, we may get these done by the coffee break so that we can then take a big breath and after the caffeine stimulation launch ourselves upon the more complex and more difficult effects of the penetrating radiation.

The nuclear detonation, in my view, differs from conventional detonations in essentially two or three things. One is that the magnitude can be adjusted to any size that you wish, for any purpose that you may have, whether it is to destroy a city or to create a harbor or build a canal, or just to find out about the intimate family life of the nucleus and what it might do under differential situations. I think that the physicists have had a heyday since the first detonation. In fact, they had a heyday all during the Manhattan District. They ran on excitement which made them work around the clock, because here was a new field for exploration entirely theoretical and mostly unknown.

As you saw from the movies, the detonation occurs with great speed but the actual detonation is a slower phenomenon than other methods of producing a high-speed expansion of hot gases. This is one of the interesting phenomena, that the pulse lasts long enough so that the kinetic energy of the wave front can be, say, translated from

the pane to the window frame and the frame can be torn out. This is not so true of dynamite or barytal, or other explosions which occur with such a short pulse that only the glass is fractured.

FREMONT-SMITH: You mean the glass can be torn out whole?

WARREN: The window frame and the glass can go almost simultaneously. There is an interval, but the interval, while very, very short, is sufficient to help translate the energy to the window frame or the door frame.

I had no evidence from inquiry among the Japanese, a month to six weeks afterward, of the movement of people as missiles, such as occurred in the Texas City fertilizer ship explosion, where in the middle of the wharf one man was lifted and transported completely over a warehouse, a distance of 150 feet, and his only injury, outside of the shock and his strange behavior, was a fracture of the ankles when he landed. Another man was stripped of his clothing. This is not an uncommon thing around explosions, to have clothing torn off without much other damage.

I saw areas where the buildings obviously had been plunged down by the front of the blast wave below the zero point and you could see some buildings, particularly in Nagasaki, that looked as if a big hammer had hit the corner of the building and had sent reverberations of the pulse through the building and had fractured the weak points of the structure.

The problem here is what happened to a large number of the individuals who were not otherwise subjected to lethal radiation exposure. In the open and close in there are essentially three bullets in the head, for you have not only the blast but intense heat and large amounts of several kinds of penetrating radiation.

BRUES: May I interrupt with a figure which illustrates one of your points, Staff?

WARREN: Good. Let's have it.

BRUES: This is a building in Nagasaki (Figure 1).

LIFTON: Roughly about how far from the hypocenter?

BRUES: It was less than 500 meters from the hypocenter.

WARREN: Yes, a little more than 1,000 feet. That terrain is very complex. There is a stone wall and at some places the front of the blast wave went down the canal wall and lifted one half of a bridge without...

SCHULL: Excuse me. Is that Nagasaki? I don't quite recognize it. This is in the direction of the prison; where are we to orient ourselves?

BRUES: I think we're looking across the valley from the area of the medical school.

WARREN: I think so. At right angles to the prison. The prison would be to the right. I don't know what's left there now because I haven't been back since.

BRUES: This picture was made a year and a half later. This point is shown even more clear in Figure 2, which shows a shrine that was destroyed by the blast. This was also within 500 meters of the hypocenter.

FRANK: There is a lot of vegetation.

WARREN: Yes. That has come back.

One half of the bridge was lifted up and turned over and cast into the stream bed. This damage (Figure 3) was done without harm to the standing part and you could not have done a better job with a crane. In another place that kind of terrace wall protected a building that was one-story high, a very expensive dwelling and not a window was gone and the tiles on the roof were not disturbed. Yet immediately behind it, buildings were all flattened.

FREMONT-SMITH: How do you explain this focus of pressure or force?

WARREN: These are reflections of the wave front, and at the end I will try to summarize this by showing you pictures of wave front reflections that will explain some of these things if Sam White doesn't steal my thunder first.

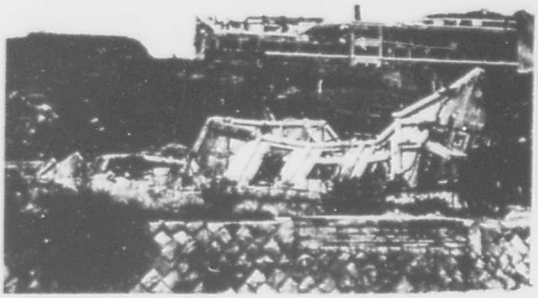


Figure 1. Building in Nagasaki damaged by blast. Photograph was taken in December, 1946.

Figure 2. Ruins of a shrine in Nagasaki near the hypocenter. Photograph was taken in December, 1946.

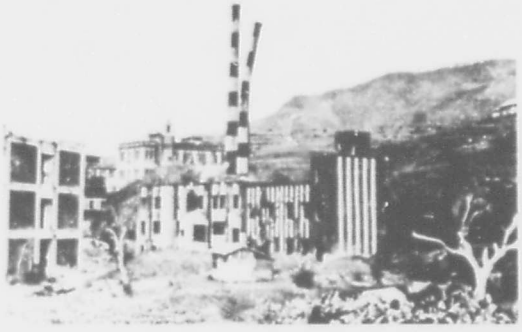


Figure 3. Ruins of Nagasaki Medical School. Photograph was taken in December, 1946.

One of the things that may not be mentioned here is that people have amnesia with any cataclysmic experience which may or may not be accompanied by trauma, actual injury. There was a lot of this. Many of the professors and the doctors in the Nagasaki medical school who survived because they were well inside the building found themselves out in the country 20 or 30 miles away in the late afternoon and could only say, "Well, at that time of day I should have been in such-and-such a place in the building seeing patients." Very often teachers and businessmen who were in the periphery of the actual damage area had the same experience.

I think we can avoid any discussion of the intense light except for the fact that the scotomas are apt to be very severe. Of course, the closer you are, the worse it might be, and it would last quite a while. The evidence on this is rather sketchy. There were burros at Alamogordo that were wandering around apparently unable to see because they had been facing the detonation or almost immediately faced it and apparently they had scotoma which lasted four or five hours.

FREMONT-SMITH: Is this an afterimage or a straight scotoma, straight blindness?

WARREN: A scotoma interferes with vision because of the intense afterimage resulting from the temporary bleaching of the visual purple of the retina.

UPTON: This is not a retinal burn?

WARREN: No, this is not a retinal burn.

So we are left with blast problems in the population that are existent today or have existed for some time, and the most obvious ones, from blast alone, are trauma. These could be of innumerable varieties, depending upon the approach that the blast front had to the individual and his immediate situation with relation to flying objects caused by the passage of the blast wave, and so on.

I would like to ask Sam if he would say something about the mechanisms of the blast as it affects the individual. Sam has, perhaps, the world's greatest experience with animals and equipment at the Nevada Test Site in shelter mockups.

BLAST EFFECTS

WHITE: I told you, Staff, yesterday that I was a little horrified, and not really from the "scare" point of view, but bothered by the breadth of this problem. You just asked me to talk for two or three days and I know that there are around 25 people here and there are something like 20 hours scheduled for the meeting. So by simple division, there are about 45 to 50 minutes available for each person to speak. I can't even get started on the problem in that time, but I'll try to say a few things to the point of your question.

First, so far as the classification of blast effects is concerned, the indirect and direct divisions seem sound to me (References 2 and 3). The direct ones are those due to variations in environmental pressure that emanate radially from an explosive source. The indirect ones are related with translational events involving (1) blast-energized debris that can be penetrating or not, as far as the body is concerned; and (2) the whole body displacement problem. I will talk about the latter later, for decelerative impact can be a very hazardous experience. Finally, there are certain miscellaneous indirect effects such as: (1) dust, radioactive or not, even inside underground structures, and this can occur if they are not buried deeply enough; (2) non-line-of-sight thermal phenomena such as burns from hot, dust-laden air being driven into open structures; and (3) blast-induced fires. One has to contemplate the factors that control the effects of something that is as involved as the above. Some of them are as follows.

As far as primary blast is concerned, the magnitude, rate, and character of the pressure rise and fall are all significant. There are other factors besides the magnitude, rate, and character of the rise and fall of pressure (Reference 4); the ambient pressure at which exposure occurs (Reference 5); the orientation and the geometric conditions of exposure (References 6 and 7); the species, the age, and the condition of the animal of interest (References 8 and 9); and

the postexposure events of importance, such as the activity or exercise immediately after the exposure, medical therapy or the lack of it, and complications such as infections, particularly in the chest and the abdomen (References 9–13).

For the indirect effects involving blast-produced missiles, one must consider the velocity, mass, density, and character of blast-energized debris; the area of the body involved and whether penetration occurs; the condition of the host; and again postexposure events. For indirect effects associated with whole body translation, the controlling factors are velocity of impact; the time and distance over which accelerative or decelerative events take place; the area of the body involved in impact; the condition of the subjects; and again postexposure events. I won't say anything in detail about the miscellaneous indirect effects now.

One cannot approach the problems, it seems to me, of chronic effects which you put on the board, Staff, without being fully aware that there are potentially very hazardous immediate effects, particularly from whole body impact and from high pressures, if the rate of rise of pressure is sufficient. For both cases the mortality-time curve rises very quickly under certain circumstances (Reference 4). The survivors of the immediate effects become those that are candidates for the intermediate and long-term effects, and it is impossible to understand either of the latter two without knowing what is occurring early, because the organism is not the same. The animal or man is not the same after it has had this experience and there are only a few studies of chronic and intermediate effects in primary blast (Reference 14).*

I think one of the difficulties in understanding secondary and tertiary effects has been the lack of delineation of the biophysical events that occur during such experiences as high-speed automobile accidents and other trauma including penetrating and nonpenetrating debris. For example, in indirect and direct blast effects, early significant events that take place pressurewise in the body occur in microseconds and milliseconds. Only recently has this been understood. Although some relevant data are of World War II vintage, many of the studies were not continued since then.

*From unpublished data of R. K. Jones, D. R. Richmond, and T. L. Chiffelle. DASA blast and shock biology and combined injury programs. Lovelace Foundation for Medical Education and Research. Albuquerque, New Mexico.

I can tell you now that one of the most interesting things that occurred in my experience in Nevada was to be able to carry out experiments ir houses and in underground shelters exposed to from about 2 to 90 psi at ranges near 10,500 to 1,050 feet from a 29-KT explosion, and take living creatures even from the most forward open structures (References 15 through 17). This was a very surprising thing to me. Also it was surprising to go back to the Nevada Test Site after a series, look out across the desert, and see that the greenest acres were the regions where the towers had been. I was naive enough to believe that every living thing would be destroyed and nothing would grow, but this was not the case. The green areas were tumbleweeds. They grew first on the desert. They shielded the aftercoming vegetation, and in two or three years, the places looked very much as they did before, except for some glazed areas if there happened to be sand around under the towers.

It became obvious to me that nuclear explosions were not really as destructive as I had imagined or had been led to think that they were. I recall reading, during the Nevada test days, some figures from Oughterson and Warren's text (Reference 18) concerning the 255,000 people said to be at risk in Hiroshima. Fatalities on the first day totaled around 45,000 and approximately 20,000 died later, for total fatalities of 65,000 in four months. That meant really that 255,000 minus 45,000 or around 210,000 people got out of the city the first day. They either walked out ahead of the fire storm or somebody helped them out. From the figures available, the surviving casualties numbered 72,000, and 119,000 people were uninjured (Reference 18).

There has to be a reason for the data I have just quoted. I want to talk a little bit about what some of those reasons are and it will help you understand why I think the problem of assessing the effects of nuclear explosions is very complex indeed.

Figure 4 deals with some of the physically related problem areas and Figure 5 with the more biologically oriented ones.

Starting with the energy source and the related uncertainties shown in Figure 4, one works out range-yield effects relationships. Circular slide rules (References 2 and 19) and the laws of physics and mathematics can be used to lay out as a function of range the major weapon effects parameters, assuming flat terrain and the absence of buildings. This is noted in Figure 4 as free-field scaling and provides an estimate of the biological variations of interest if an individual or an animal were to be exposed on flat terrain right out in the open.

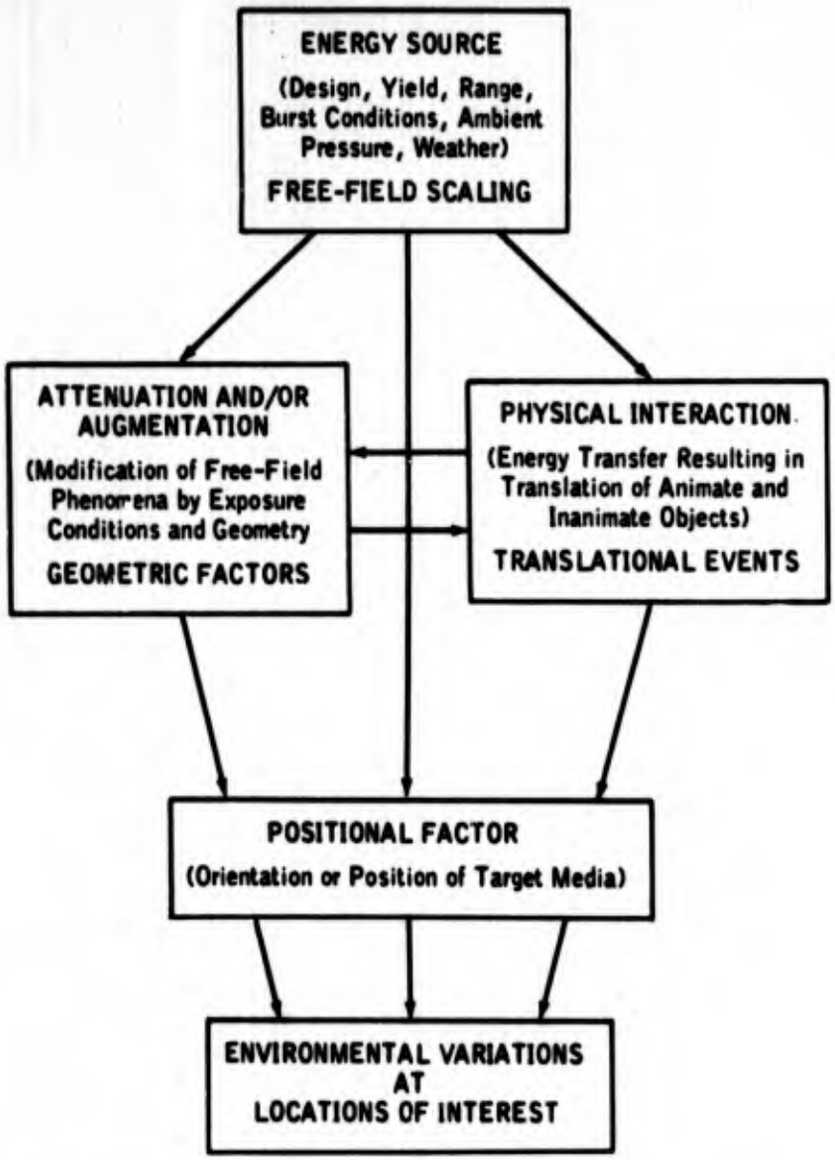


Figure 4. Physically oriented problem areas (Reference 3).

But even so, note in Figure 4 the central arrow pointing down. There are positional factors at play and the orientation or the position of the target, be it man or animal, changes the quantitative response (References 3 and 6). As depicted in the left-central part of the figure, conditions of exposure might involve buildings or structures. The free-field effects can either be attenuated or augmented or the same as those that occur outside the structure. They may be greater with regard to pressure, for example, because reflections in certain

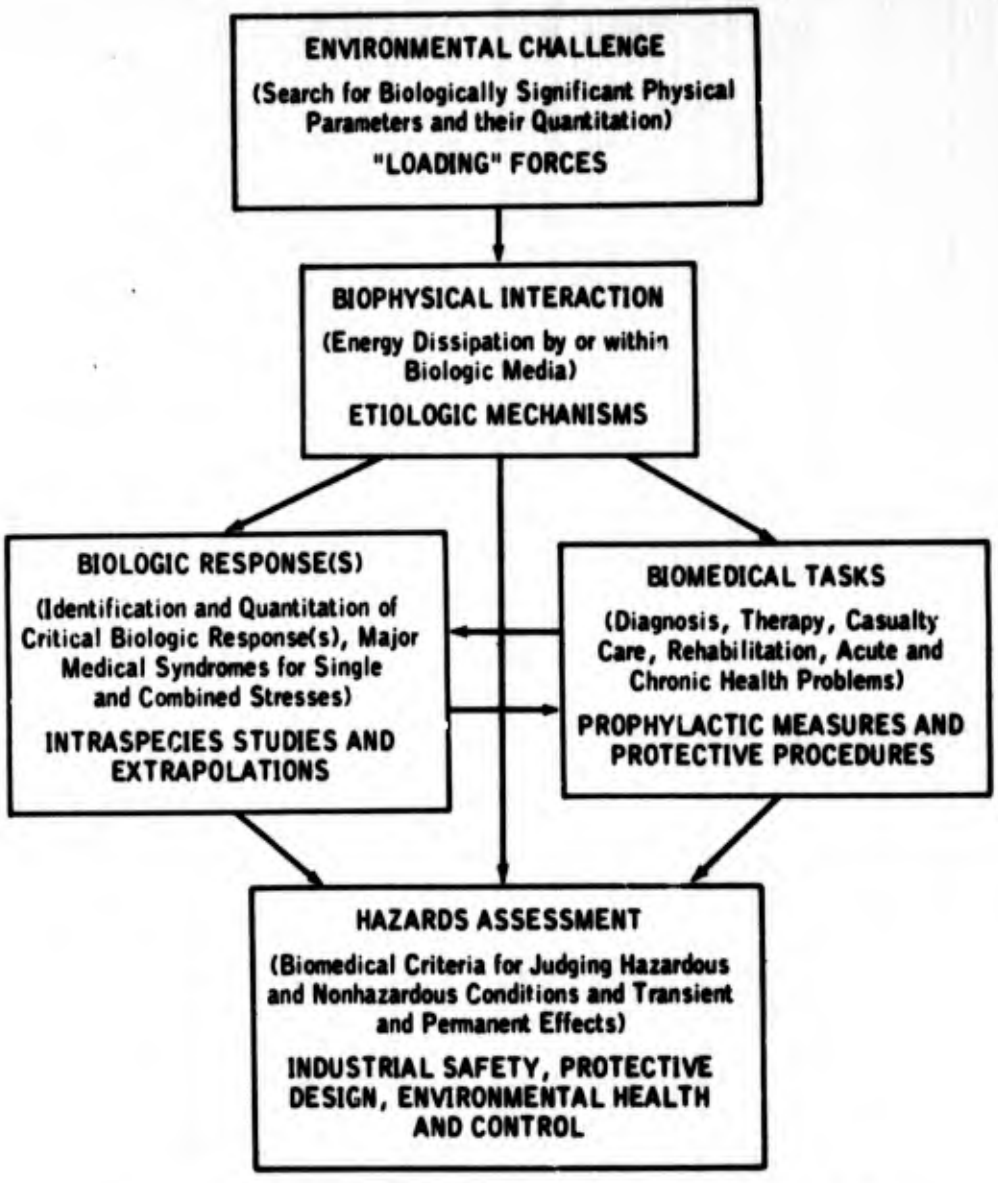


Figure 5. Biomedically oriented problem areas (Reference 3).

geometries can increase the maximum pressure severalfold even up to five or ten times at higher pressure. If the conditions of exposure involve a large enclosed volume with air feeding through a small opening, the pressures may be much less than outside. These geometric factors emerge as very major factors in determining the outcome of exposure.

For example, Oughterson and Warren (Reference 18) cited average survival data as a function of range for individuals in the open, for

individuals in wooden structures, and for individuals in seismic, heavy concrete buildings.

Now, these were not small differences. The 50 percent survival ranges were like 1.3 miles for people in the open in Hiroshima, like 0.8 mile for the average if all exposed were lumped together, around 0.45 mile for people in wooden buildings, and something around 0.2 to 0.3 mile for 85 percent immediate survival for people in concrete buildings (References 3 and 18).

What this means is that there can be differences by factors of, like 10, in the range of survival, depending upon where an individual happens to be at the time that hazardous explosive events occur. The significance of the geometric factors of exposure has really not been widely appreciated at all. Certainly they deserve a great deal more attention than they have gotten. Finally, even in a structure, the positional factors and the orientation factors are important. There is quite a difference whether one is in front of a window or beside it, as far as wounding by glass is concerned, as implied by the box labeled physical interaction in the right-hand side of Figure 4. Involved is energy transfer to debris or animate objects. For whole body displacement there can be hazardous accelerative or decelerative experiences with the factors controlling that I mentioned earlier. Also, for translational events, positional factors may be definitive.

By hindsight, looking back on the Nevada experience, this kind of thing is something that should have been appreciated very early, but wasn't. In 1953 animals that were exposed in long, tubular, underground structures 50 feet long and 7 feet in diameter with 15 psi maximum overpressure "going by" outside, and 20 to 25 psi maximum occurring inside, survived very well. However, they were restrained so that translational events did not complicate the experiment. Animals in the 1953 structures had no radiation problems (Reference 16). In 1953 the opportunity was taken to work in close where free-field outside pressures were 90 or 91 psi with maximum inside pressures in the forward open structure being around 60 to 65 psi. All large animals were recovered alive except for one fatality due to impact following translation. This animal was seriously burned by hot, dust-laden air driven into the shelter by the blast wave (Reference 15).

UPTON: When you speak of these pressures, are you referring to the peak of the positive overpressure or the difference between the positive and the negative?

WHITE: The peak of the positive overpressure, above the local ambient. However, the internal pressures measured in Nevada were monitored with wall pressure gauges. This unfortunately did not necessarily tell exactly what was happening where a given animal was located.

Consider Figure 5. If one really wants to develop a quantitative understanding of biological response when an environmental variation is imposed on an animal, one should try to measure the pressure either on the animal's surface or very close by to determine the environmental challenge or the loading forces (see top of Figure 5). If these are changes in pressure with time, the effects may have to be studied for a considerable time to find out what portions of the pulse are significant biologically. This has been done to some extent and explains why I said earlier that the rate, character, and magnitude of the pressure rise and fall were significant factors. At any rate, if one doesn't know what physical parameter is appropriate to correlate with a significant biological effect, it will be necessary to find out. Such exercises have been very frustrating experiences as far as the effects of variations in environmental pressure are concerned, as I will try to tell you later on.

But assuming that some of the things important to the animal are known and one learns how to reproduce a desired environmental variation repetitively and quantitatively, then one is in a position to search for the etiologically significant, biological effects. Often these are obscure at first.

The simplest thing to do is to look at lethality-dose or survival-dose relationships. One really needs to look quantitatively at what is going on in the animal, try to understand the energy transferred to the target, what the target "does" with the energy it receives, and what significance this has in relation to quantitative biological responses. Note the left side of Figure 5. One attempts to use all the wisdom learned from clinical medicine and from interspecies scaling studies. Because one really can't expose people to hazardous events and must depend, as Jelle de Boer said earlier, on animal studies, one uses everything he knows from the diagnosis and therapy to help assess casualties. If fortunate, one may eventually get enough information to establish tentative and relevant criteria and, as hinted in the bottom portion of Figure 5, may make significant contributions to industrial safety, protective design, and environmental health and control.

Crude tentative biological criteria are now available for primary, secondary, and tertiary blast effects (Reference 3). They are far from complete. They have a lot of uncertainties, but they are available and they are useful.

I have two boxes of slides to help summarize around 12 years of work in the blast field if anyone wants to talk in depth. However, right now, if I can have about five or ten more minutes, I'll mention some of the exciting and complicated things that one really needs to know to begin combating the schizophrenia that is involved in weapons effects today. It is not enough to know just about blast or thermal or ionizing radiation effects; one must know about them all, because the challenge may be one or the other alone or combinations of two or three. Assessing combinations of effects is going to be the exciting thing in the future. Whether one can gain a quantitative understanding depends much on the "state of the art" in individual effects, but it is becoming possible now to do more combined effects work. For example, one knows that a pressure produced by a given explosion decreases in magnitude but increases in duration as the pulse moves radially away from the source. Thus an understanding of tolerance as a function of range requires a knowledge of the effects of variation in both the magnitude and duration of the overpressure.

In a plot of p-max (psi maximum) against the duration of a classical pulse having no significant disturbance of the rise of the pressure, tolerance for 50 percent survival varies as shown in Figure 6. For a dog it takes very high pressures of a short duration to be lethal, and, within limits, the longer the duration, the less the pressure required. The periods of times involved are like 2 msec for 220 psi and 12 msec for 75 psi. For longer durations of 400 msec—certainly applicable to a long-duration nuclear wave—the pressure for 50 percent survival is around 50 psi.

The only variables here, for a standard target of the same age, are just the magnitude and duration of the pulse. Schardin noted this a number of years ago when he published a charge-distance diagram, shown in Figure 7. Note the relationships between the iso-momentum and iso-pressure lines and the characteristic lines of destruction for animate and inanimate objects. All targets are pressure sensitive for the long-duration pulses; they are impulse sensitive for the short-duration case (at the nearer ranges). For dogs, we don't know yet today whether tolerance at the near ranges really remains parallel with the iso-impulse lines. The data just aren't good enough to know.

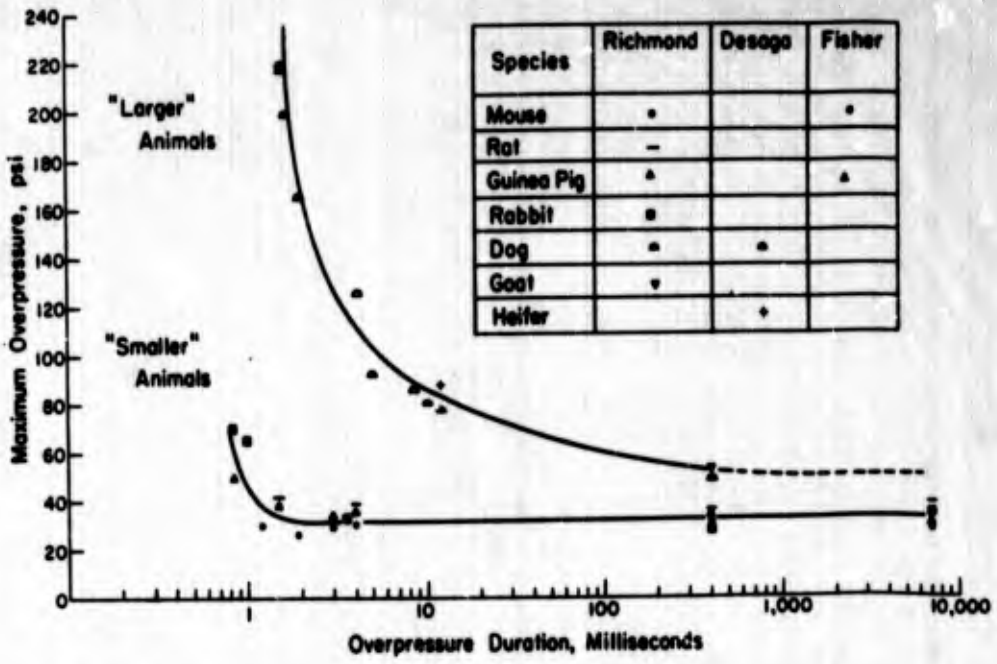


Figure 6. Pressure-duration relationship for 50-percent survival. (Modified from Reference 20; data from Richmond, References 21 and 22; Desaga, Reference 11; and Fisher, Reference 23.)

TAYLOR: Is there any appreciable difference between what happens, say, up to a tenth of a second, which is quite long on your scale, and a very long time like a minute?

WHITE: Not if the pressure stays...

TAYLOR: So these show that at 45 psi you get a tenth of a second. Then it changes.

WHITE: Yes. You are really pushing now, Ted, for an understanding. If one had a real square wave, or let's say one that decayed very slowly with time as occurs with very large yields, of course, the question is, does the animal "care" about the early or late part of the pulse or both? We know a little bit now about how much of the pulse is significant. The reason is because a transducer placed inside an animal exposed to a "fast"-rising pulse reveals that the pressure inside the chest very quickly overshoots the outside considerably. The cause of this is really simple. The body wall is vehemently hurled inward because the animal is immersed in a high-pressure wave. Energy of motion must be dissipated in some way. What stops the inward moving portions of the body is the buildup of

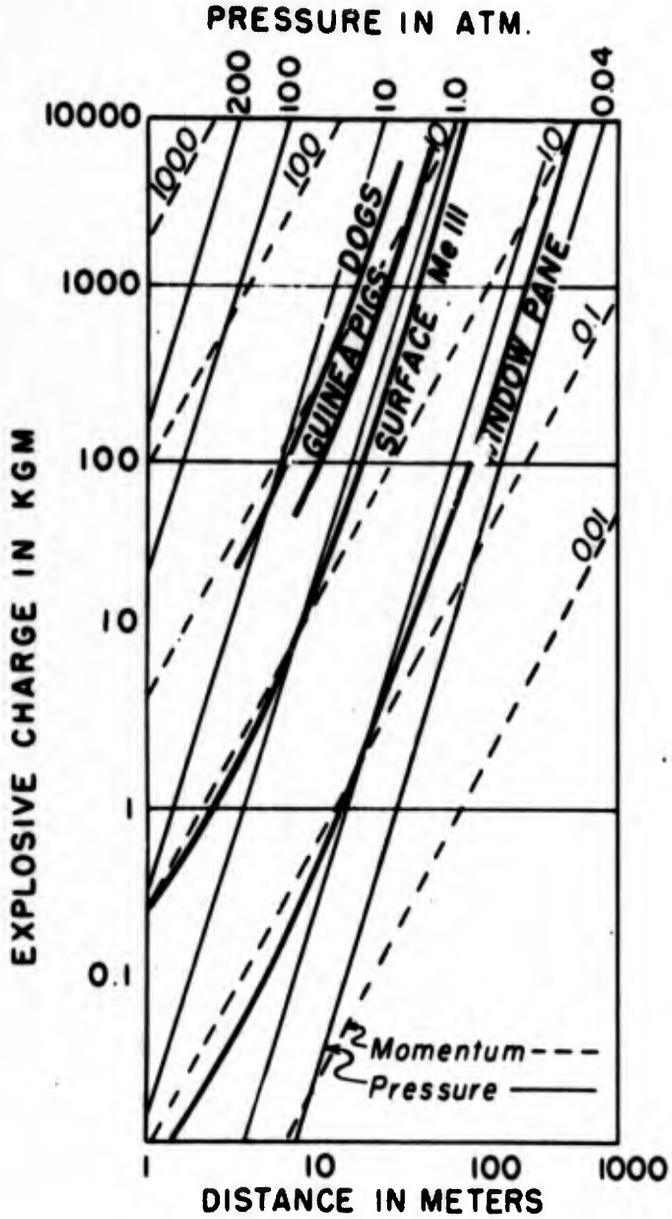


Figure 7. Characteristic lines of destruction for dogs, guinea pigs, surface of an aircraft, and a window pane in a charge-distance diagram. Solid and broken lines show pressure in atmospheres and momentum in atmospheres times seconds, respectively. (Modified from Schardin, Reference 24).

the pressure in the gas-containing portions inside (Reference 25). Over the time to reach peak pressure and a few oscillations after that, trauma that is critical to the animal has already occurred. What happens subsequently is a function of a lot of pathophysiological events that we just can't talk about here.

UPTON: I would like to pursue this just a bit further. You have spoken about mortality. Is it basically the same pathologic process that kills the animal irrespective of duration?

WHITE: Yes. This is because of this critical time relationship just noted.

Relevant data obtained by Dr. Don Richmond and collaborators (References 6–8 and 26)—and I should say that all I can do is speak for a group of people who have collaboratively done this kind of work for years—for 11 or 12 species of animals show some of the relevant intraspecies relationships in animals ranging in size from the horse to the steer (References 6, 8, 27, 28). Shock tubes from 6 inches to 6 feet in diameter are at hand and have been used. We have learned how to make wave forms of different kinds which I will try to show in a minute. We are struggling with the interspecies scaling problems now. One eventually must have enough data to elucidate generally what is going on. If this proves successful, effects referable to man as a mammal eventually will become clear.

TAYLOR: Do these differences between species get preserved as you go toward the...

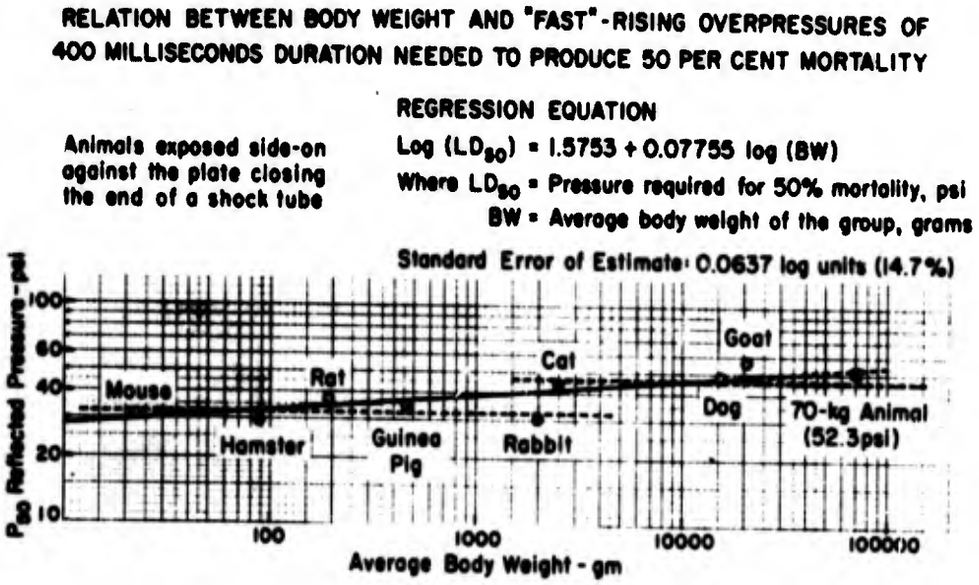
WHITE: Yes, they do, but they become less at the longer durations. The differences for 50-percent survival among mice, goats, and steers for long-duration pulses involve a spread of about 25 to perhaps 55 psi. For shorter duration waves the difference ranges from 200 to 250 to around 25 to 30 psi. Pulses of fractions of a millisecond are required before the mouse curve turns up (see Figure 6). The critical duration, the duration beyond which pressure is the controlling parameter, is a function of species size.

BUSTAD: Body size.

WHITE: Yes. And it looks like something else, Leo, species type, which I will discuss.

If one wants to try to extrapolate to a mammal the size of man, different possibilities dependent upon pulse durations exist, as a study of Figure 6 will suggest. The 50-percent survival curves will be very steep for the shorter durations and very flat for the longer pulse durations (References 6, 25, 26), a matter that is related to what Ted said.

One kind of exciting, not so amusing, but intellectually challenging thing that has occurred is related with the long-duration case. Points for different species can be used to plot a curve like the one shown in Figure 8. The curve can be extrapolated to predict tolerance for 160-pound individuals. When the curve was first drawn, even with five or six species like the rodents and the dogs and goats, the Albuquerque group was very aware that two curves like the dotted ones in Figure 8 also fit the data very well. Indeed, most now feel that the mammals



	MOUSE	HAMSTER	RAT	GUINEA PIG	RABBIT	CAT	DOG	GOAT
NC ANIMALS	140	110	164	96	104	48	35	30
MEAN WEIGHT*	22g±1.9	89g	192g±25	455g±37	1.97kg±0.26	2.48kg	15.1kg±3.1	20.5kg±3.6
P_{50} (PSI) +	30.7±0.56	28.6	36.6±0.61	34.5±0.64	29.6±0.90	43.6	47.8±1.06	53.0±2.79

* Figures represent mean and standard deviation.

\+ The ± figures represent the standard error of the mean.

Figure 8. Intraspecies extrapolation for "fast"-rising, "long"-duration overpressures. After White, et al. (Reference 16).

are of two varieties, although previous estimates for cattle (500- or 600-pound steers) were predicted with the sloping curve. Subsequent field tests indicated the prediction was probably high (Reference 27). Shocktube, laboratory studies by Damon, et al. (Reference 28), not feasible at that time because the equipment was not ready, have subsequently revealed that the cow has a 50-percent survival pressure of 43 psi best predicted by an extrapolation using the top dotted line in Figure 8. There are complications however. The steers used were only year-old animals. Plots of gain in weight for steers reveal it takes about two years before the rapid gain begins to level off (Reference 29). Thus the steers used by Damon, et al., were not young adults. No doubt the study will have to be repeated sometime with adult cattle. So there are interspecies scaling problems just to get the lethality or survival extrapolations for man.

The relationships noted for survival parallel those for threshold effects for the lung, with the latter however occurring at lower overpressures (References 6 and 8).

ABRAHAMSON: Sam, are you talking about sort of immediate lethality?

WHITE: Yes. Lethality within the first 24 hours and if you look at the lethality time curves for blasted animals (References 4, 20, 26), they rise very steeply. Most of what I've said is lethality in 24 hours. A few animals have now been followed for 20, 60, and 90 days (References 4 and 14).

ABRAHAMSON: Is this pertinent to long-range effects, though?

WHITE: No, but the events associated with what I have been talking about are the kind of events that leave their mark on the animal and they cause long-time effects.

AUXIER: Do you see the same patterns for mortality in small and large animals?

WHITE: Yes. I want to tell you something else first if you don't mind, Staff.

If one puts animals against a reflecting plate and moves them further and further away from the closed end of a shock tube—this might be like being exposed in front of a reflecting wall in a building—the tolerance of the animal changes. I can best illustrate this by asking

you to imagine exposures to about a 52-psi load composed of two steps: an incident pressure of 18 psi and a second one of 34 psi; that is, an incident wave of 18 psi "rolled down" a closed shock tube will reflect to 52 psi. An animal mounted away from the reflecting end plate will "see" the load in two steps, whereas one mounted against the end plate will receive the maximum overpressure practically at once.

In the latter case all subjects exposed suffer 100 percent lethality (see Figure 9). For a mouse exposed at 0.5 inches from the end plate, lethality is 60 percent; at 1 inch it is 30 percent; and at 2 inches it is zero. The time involved between the first and second pulses is about 100 μsec/in. Thus the mouse can "tell the difference" between two pulses that are 50 μsec apart. The animal does something, or something is done for him, rather, by the first pulse that makes it possible for him to survive the second pulse, which if given by itself, would be lethal.

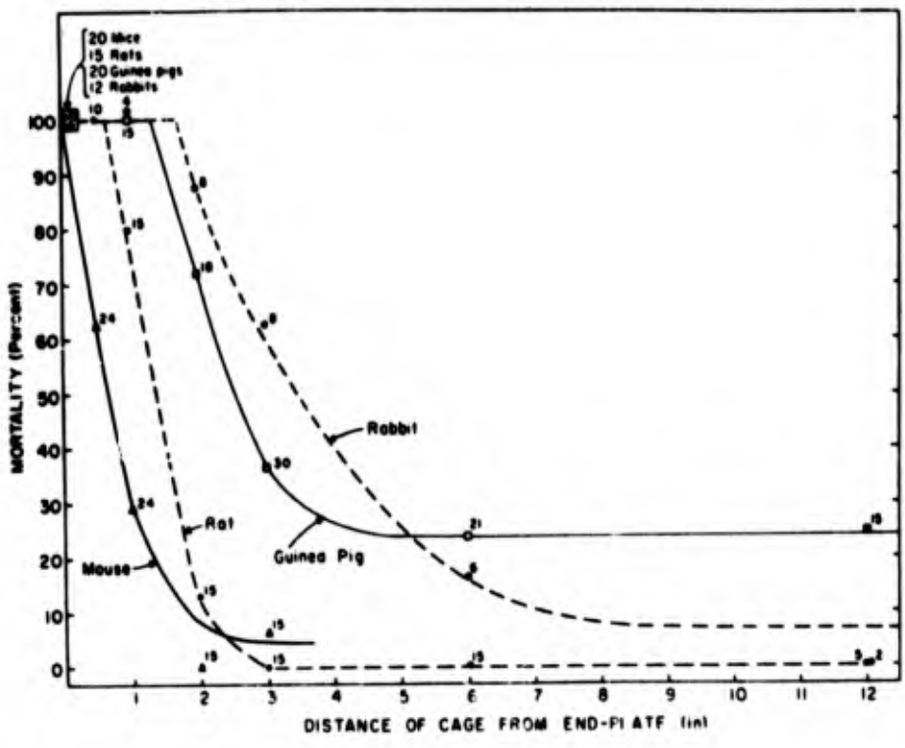


Figure 9. Mortality for small animals exposed to a reflected pressure of 52 psi when located at various distances in front of an end plate closing a shock tube. After Richmond, et al. (Reference 21); also see References 6 and 25.

FREMONT-SMITH: What is the time interval between the first and second?

WHITE: Fifty microseconds at 0.5 inch but longer for greater distances from the end plate. The larger the animal, the further the lethality curves move to the right as shown in Figure 9.

Any criteria that I referred to earlier were for "clean" wave forms, having almost instantaneous pressure rises. If the latter involves two steps, the animal's tolerance is almost double. If smooth- but "slow"-rising pressures are involved, tolerance may go up (as far as the p-max is concerned) by factors of 3 to 5 (References 4 and 16). There are no good criteria for chewed-up wave forms that can occur inside buildings having various geometries.

The increase in tolerance is an absolutely fascinating fact. What is the animal doing in such short times or what is happening to the animal that can explain this?

To follow up, imagine a given volume of gas inside the body, particularly the lungs, at a sea-level absolute pressure of about 15 psi. Also imagine a 15-psi square wave—absolute pressure of 30 psi—being imposed on the animal. This represents a factor of 2 difference between the ambient outside pressure and that inside the body.

If the square wave is of very long duration, the animal will start to get smaller, he is imploded, and then he will recoil to his normal size. Meanwhile the pressure inside will go to 15, 20, 25, and 30 psi, remaining there until the overpressure falls. If the animal has imploded and the absolute pressure inside is 30 psi, and I want to load the target by a factor of 2, I have to use an overpressure of 30 and not the 15 psi noted above. Thus the simple gas laws help our understanding of the implications of what the animal "does." In reality the implosive process is not slow, but rapid and the internal pressures overshoot the outside ones followed by damped oscillations. Consequently, the effects of a second pressure pulse will be influenced by what the internal pressure is at that time. Theory says there should be a rhythmic variation in tolerance. There are few data to support this view.

One might say that in response to an overpressure, an animal is making himself a "new" internal ambient pressure and that it is this that influences tolerance to the second wave. If true, then blast

tolerance should be a function of the ambient pressure at which exposure occurs. To test this, Damon, et al. (Reference 5), used an evacuated shock tube and one that was pressurized up to 42 psi. The Δp or overpressure associated with 50 percent survival when the ambient pressure of exposure changes over this range varies for the mouse from about 20 psi to almost 90 psi for ambient pressures of 22 and 42 psi, respectively. Thus ambient pressure has come to be known as an important factor in blast tolerance.

Already the situation has become so complicated that I really ought to sit down, but Staff asked me a question. What happens in the animal that is responsible for the very rapidly developing lethality-time curves? During the implosion event, air gets into the left heart and arterial portions of the vascular system (References 4, 9–12). At the same time hemorrhage begins in the lung. According to Chiffelle (Reference 14), the lung bleeds from two places: from small arteries, subendothelial in location in the higher airways; and from actual arteriovenous fistulas that have been seen in tissue sections. The air emboli can, if they go to large coronary vessels, stop the ticker just as though there were a massive occlusion (References 9–12). I have some beautiful pictures that Drs. Tom Chiffelle and Don Richmond have gotten of emboli in the coronary vessels in dogs. They also go to all the body organs. In sheep and cattle recently looked at 30 days after blast exposure, there were multiple infractions of the kidney. Some kidneys even at that time were contracted and half-normal size. There was evidence of multiple scarring of the heart with hypertrophy of the unaffected heart muscle.* Thus one now has a possible explanation for the blast hypertension that Blockers (Reference 30) and Ruskin (Reference 31) described in survivors of the Texas City disaster and noted even in some followup cases (Reference 32).

FREMONT-SMITH: How about the central nervous system?

WHITE: We have coronary problems and renal problems that can be intermediate and long-term effect ones. Softening in the central nervous system has been described by Rössle (Reference 12). It is probably more prevalent than is known, but the whole pathophysiology of air-embolic insult (References 9 and 33) particularly should surfactant material in the lungs get mixed up with the emboli and help prolong their life in the vascular system, is very little studied and understood.

Lungwise, multiple fibrotic spots become chronically apparent because repair of the traumatic emphysema fails to be complete. Interested people are referred to the work of Chiffelle for further details (Reference 14). There must be significant long-term variations in respiratory function. Technology at the Albuquerque blast site is only now up to beginning periodic respiratory function tests. But this is another illustration of fundamental research asking more questions than it answers. Getting the people, the equipment, the money, and the proper animals to work on are all problems that get to be an administrator's nightmare.

There are, therefore, intermediate- and long-term as well as very hazardous early primary effects from overpressures. If one exercises after exposure, the early effects are enhanced. Thus there's not much chance, judging from the available literature, of learning much from the Japanese information about these very early, highly lethal experiences from either whole body impact or from overpressures which might have occurred in buildings. People had to exercise after the experience and exercise in a lung that's been damaged this way is "bad news." I suspect that people seriously injured in this way expired early and just didn't get into medical channels in Japan.

AUXIER: Wouldn't this be true any place, though?

WHITE: No, not necessarily. Blast casualties did get into medical channels in World War II. The British have a nice literature on blast injury and some information on how to treat it. Leavell in this country has a nice, therapy-oriented article on blast lesions (Reference 13).

We know, from dropping animals from increasing heights, that the lethality-time curve from whole body impact is steep like primary blast (References 4 and 34). These kinds of trauma in combination ought to be very hazardous indeed.

Let me ask you to assume that the tentative criteria for assessing blast effects are reasonable. What can one do with them? One can do this kind of thing: Take the pressures that existed in, say, Hiroshima and Nagasaki, and scale them as function of range for the incident and for the reflected pressures and then overlay the threshold criteria for, like 5 psi, and for lung damage, around 10 to 12 psi, and for low and high lethality. These are hazards criteria wherein significant physical parameters are related to specified biological

responses. One can say what is potentially a "safe" and a "lethal" range and identify roughly a casualty range. Also one can scale translational effects for window glass and for people. Too, the thermal and ionizing radiation curves scaled as a function of range can be added to define the free-field challenges as they vary with distance from the explosive source. Figures 10 and 11 show such range effects curves.*

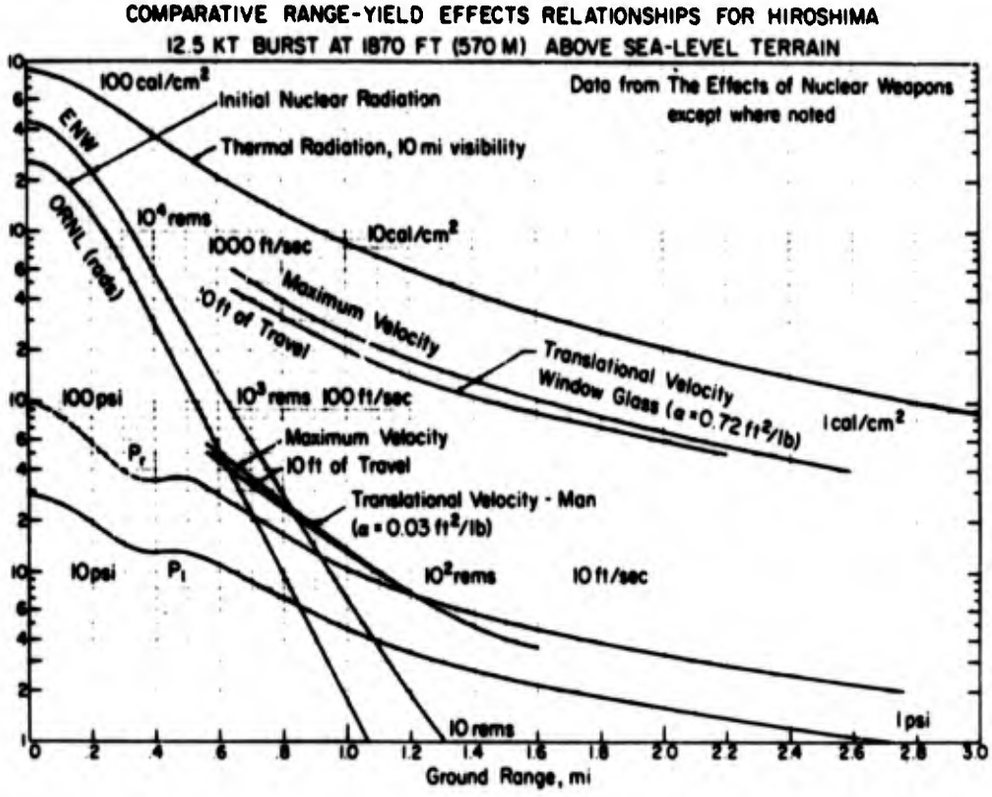


Figure 10. Scaled free-field effects data prepared by Bowen using data from Glasstone (ENW) and Auxier, et al. (Reference 35).

One can do little with the information except lay out areas of potential risk, areas inside which the hazard specified can occur. What is also needed is the shape of the casualty curve for each effect inside each of these areas. One cannot meaningfully assess that until the geometric factors I mentioned are understood along with the contribution the conditions of exposure make in determining differences between exposures in the open and exposures in wooden and in concrete buildings. In Hiroshima there were differences at the 85 percent survival level by factors of 3 between these curves;

*I. G. Bowen, Personal communication.

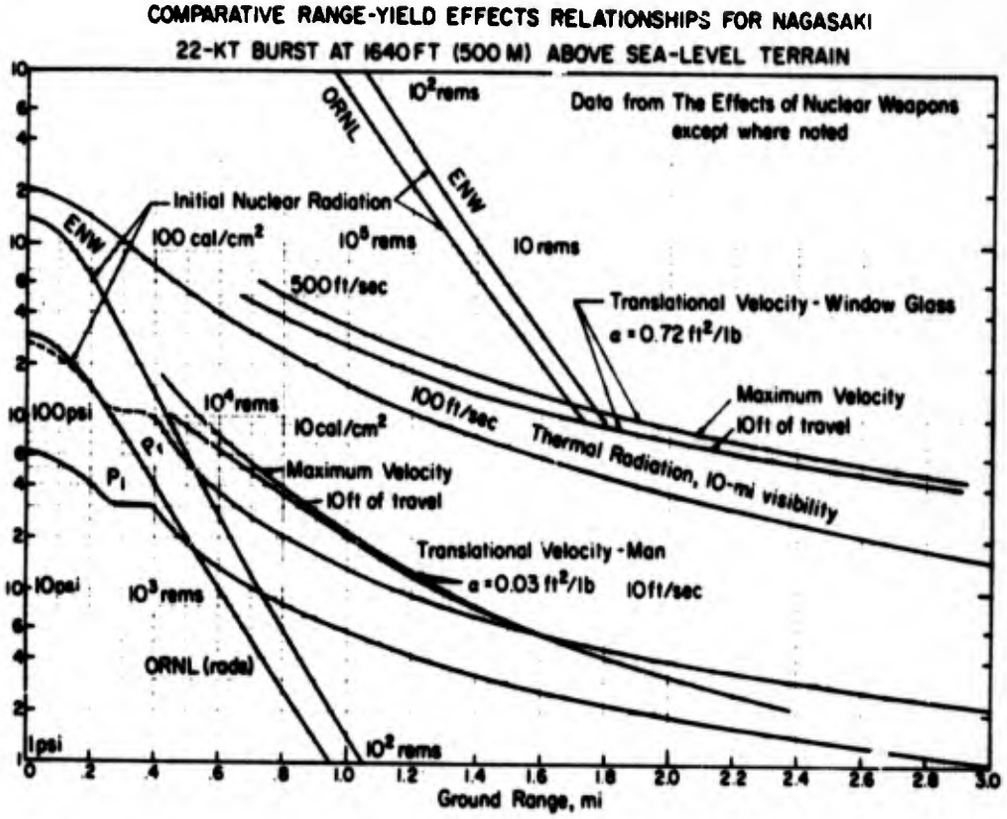


Figure 11. Scaled free-field effects data prepared by Bowen using data from Glasstone (ENW) and Auxier, et al. (Reference 35).

that is, about 1.6 miles for exposures in the open and about 0.3 mile in seismic concrete buildings (References 3 and 18). For inhabited buildings, what the environmental challenges are where exposures occurred are the things one must find out.

These problems are approachable today. If there are enough data in the Japanese experience about casualties in some of the heavy concrete buildings, one can model them and/or compute what the environmental challenges were inside where individuals were located. Perhaps such information can be used to help understand where man fits in the intraspecies data that are being accumulated throughout this country. There are extensive blast as well as radiation studies under way that involve several species of animals, as you know.

I should apologize for talking so long, but these are not simple problems being discussed here. If one just looks at the blast effect

situation and begins to dissect it, it is clear that while steady and solid progress has been made, most of the things I have discussed here really are relatively new in science. There is so much yet to do before one can get a definitive understanding of the factors involved and can think quantitatively with them, that I get sometimes both discouraged on the one hand and excited on the other about what tomorrow's work really has in store.

CASARETT: Sam, years ago we did a little work on animals which were sacrificed periodically after exposure to dynamite blasts, on four species: dogs, rabbits, rats, and mice. We noticed that one of the earliest changes was the occurrence of hemorrhagic bands that corresponded with the overlying ribs, and these seemed to be less severe in the dogs and in the rabbits and more severe in the mice and rats. These changes were followed by pulmonary edema. I wonder if you have any idea now whether the resistance to lethality from blast between species of different sizes, or perhaps between the young and old of the species, might be related to the rigidity of the rib cage?

WHITE: No. We just know in a few preliminary studies that in rats or guinea pigs age is a factor, and age has not been systematically studied.

FREMONT-SMITH: In which direction is the age a factor?

WHITE: The younger animal is more susceptible than the older animal. In the intraspecies scaling picture it seems that perhaps the ratio, the functional residual volume to the total body mass, is going to be a valuable scaling parameter. For example, the rodents, and large animals like dogs, show functional residual volumes plotted against body mass, that define different but parallel curves (Reference 25). The curve for rodents is below that for the larger animals. Rodents are "fast" breathers, that breathe 100 or 200 times per minute. Slow breathers represent the larger animal groups. Rodents avoid differences in alveolar oxygen and carbon dioxide by a "washout mechanism." For the slow breathers, a diffusion mechanism is at play. Each time the "small" animal breathes, he moves about half the air in his chest; for the slow breather the figure is about one-fifth to one-seventh. So the animals may differ in their responses to the implosion process. The damage to the lungs is more in the lower lateral portions of the thorax than it is in the upper parts where the chest is quite rigid.

So, by implication I can give you some response to the question, that wherever the body wall moves the most over the lung is where the lungs are most traumatized. However, if squeezing the animal continues after the maximum implosion has taken place, as in the case with a closed airway in a diving situation involving increasing depth, the body wall cannot move in much more but fluid flows into the lungs and compresses the still compressible gas to the organ. Blood vessels, of course, have to grow and they are easily ruptured. This may be one reason why very long-duration overpressures are relatively more hazardous. This kind of thing might occur, Ted, but in a very long-duration blast rather than the diving situation, the animal's airway is open. The problem may involve "long-term" gas exchange rather than the more acute events that I have been discussing.

Rib markings are very much a function of what moves, but they may not be caused by the ribs although they are called rib markings. Dr. Wright at the Royal Navy Physiological Laboratory at Portsmouth in England resected a rib, for example. After blast there was an H-shaped mark near where the rib was removed and the space previously occupied by the rib behaved like the intercostal spaces between the ribs.* So it is the inward moving intercostal tissues that traumatize the lung.

MILLER: I would like to say something about the frequency of chronic disabling effects in Hiroshima and Nagasaki. They appear not to be very great. In 1954 we studied about 4,000 children who had been within 3,500 meters of the hypocenter, and only 1 percent had disabling effects from the heat or blast (Reference 36).

WARREN: What kind of disabling effects from the blast?

MILLER: Contractures, chronic osteomyelitis, scars of the face, things of that sort.

WARREN: From fractures and cuts and things?

MILLER: Yes.

WHITE: How about hypertension and emphysema and asthma? Inside the buildings the pressure rise rates are slower and tolerance

*H. D. Wright, Personal communication.

goes up by factors of 3 to 5. In a building the pressure may be 100 or more psi and have nothing of biological significance because of the slow rate of pressure rise. "Slow," relatively smooth rising pressures have been imposed on dogs when the time maximum pressure was reached varied from 150 μsec to 90 to 60 to 30. Pressures as high as 90 to about 200 psi produced not at all the kinds of hemorrhagic lesions that are responsible for high lethality (Reference 37). They "pinch" the lung laterally in the sulcus, like local trauma will, but that's all. So this is not a surprising thing and it again helps to reemphasize the significance of knowing what happens to these events inside buildings. One must not assess the effects of nuclear war on free-field scaling. I forgot to emphasize how much I used to be frightened by looking at the distance the thermal pulse would travel, like 30 miles for a second degree burn; that is about half the distance between Albuquerque and Santa Fe. In Nevada, animals were recovered when the thermal flux outside the shelter entryway was as high as 600 cal/sq cm. Just being indoors changes a great deal, you see, the thermal load that reaches the target.

I don't know how to assess non-line-of-sight thermal hazards but under certain circumstances hot gas burns can occur inside open structures. This was seen in 1953 and 1955 and 1957 in certain positions (References 15 and 16), but not in other locations in the structures. One needs physically to know the attenuation criteria for thermal pulses to help assess how much thermal protection a building gives. There's a lot more work going on in radiation shielding than there is in blast and thermal shielding in this detailed regard, and it's a shame.

TAYLOR: I would like to underscore what Sam has been saying about the complexity of the problem, by pointing at not just free-field condition variability. I want to say just a little bit about that because I think it is important, although we are concentrating on Hiroshima and Nagasaki, to realize some of the differences between what happened there and what might happen if a war ever took place.

Everybody is familiar with the change in the character of the pressure pulse with yield, and Sam referred to it. There is one thing that is perhaps not quite so ...

FRANK: Excuse me. I am not familiar with that.

TAYLOR: As the yield goes up, the blast pressure is above the normal pressure for a longer time.

FRANK: Does it go up at the same rate, though?

TAYLOR: That changes, and I guess in a very complicated way.

What I wanted to refer to is, that as one goes up, not only in yield but in the altitude of the explosion—this is something that is coming up these days in connection with the question of defense—there is much more interest in the blast effects that take place from an extremely large yield at an extremely high altitude which produces a completely different set of phenomena on the ground from those at Nagasaki or Hiroshima. For example, 100 megatons, which is now compatible with what people have in the way of missiles, does things which are very strange if exploded at very high altitudes. The blast effects on the ground offhand sound small because one talks to people about pressures of only 5 or 10 psi and Sam has been talking about lethal pressures of ...

WARREN: 14 to 100.

TAYLOR: Yes. Up to 100 psi.

I want to emphasize that 10 or even 5 psi, lasting for a long, long time, is really like immersing the entire city in this relatively low overpressure for a long time and produces blast effects in a very different way. For example, it knocks down buildings by squashing them instead of by pushing them over.

WARREN: It gives the time for the kinetic energy to be transferred to the buildings, doesn't it?

TAYLOR: Yes. And so the energy goes to the building and then from the building presumably to the individuals.

WHITE: These are the secondary indirect effects.

TAYLOR: Exactly. It seems to me it gets extremely complicated in a situation like that, because what under some circumstances offers protection—the building—may actually do the opposite. A person may be much better off to be out in the open and feel this

relatively gentle and long 5-psi pulse than to be inside a building which has a roof, and is just squashed flat, not knocked over.

WHITE: People in the Texas City disaster were better off in the open than they were in buildings because of the debris problem.

In Hiroshima and Nagasaki, they were better off inside than they were outdoors, because by going indoors the thermal hazard was taken off their back and the hazards they assumed from the debris were relatively less in magnitude than the thermal. And looking at the changes that occur on the ground as a function of burst type, one has got to look at the free-field scaling information first and then assess the effectiveness of planning conditions of exposure to reduce hazards.

The most interesting, and very challenging, aspect about the influence that the conditions of exposure had on survival in Hiroshima and Nagasaki is the simple question, what if there had been a 5-minute warning and everybody outdoors just got inside some structure, or if people could have picked strong buildings? The numbers would have been different. What this really says, and the Nevada data say, is that survival can be enhanced remarkably if you plan the conditions of exposure properly. There is no question but that this is so.

FREMONT-SMITH: Could you add then that if the enemy plans the conditions properly, too, the opposite can take place?

WHITE: If you play this game, it might force him, I suppose, to use higher yields and more bombs.

There is another uncertainty that fascinates me about using the Hiroshima and Nagasaki information to help learn how it is with yields that Ted Taylor is talking about, or large yields.

If, as a function of range and yield, one scales various pressure parameters, as depicted in Figure 12, the larger the yield the greater the range that the iso-pressure lines reach. If one scales the thermal gain with the yield, the increase with range is greater. The scaled translational effect curve goes just about parallel with the thermal curve (see impact velocity curve in Figure 12). The radiation curve for 100 to 200 roentgens is flatter than all the other curves.

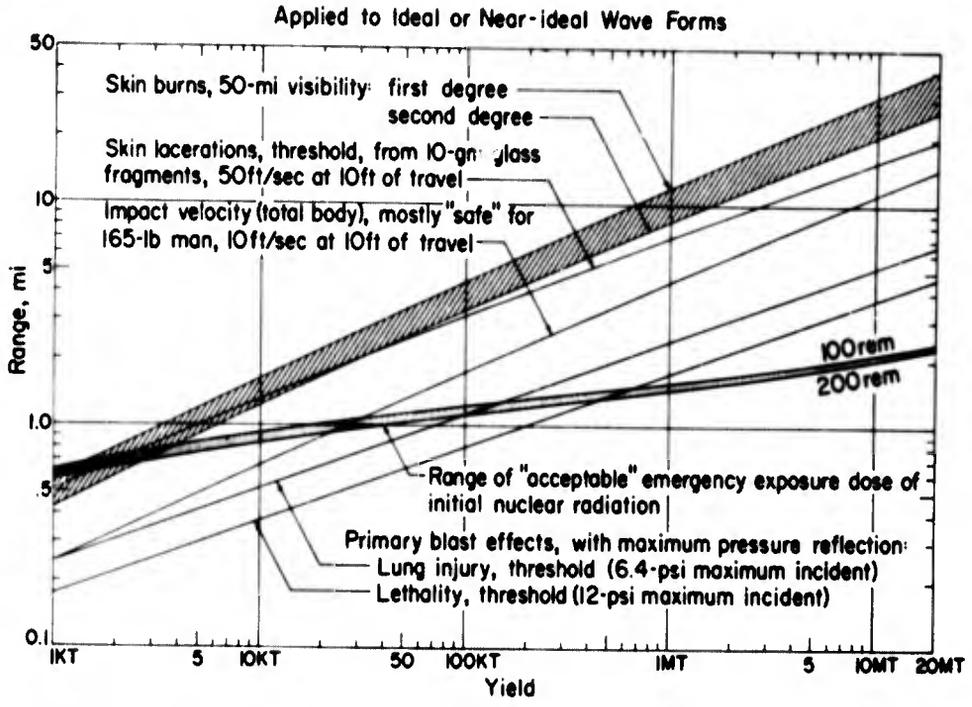


Figure 12. Comparative effects data showing ranges inside which indicated biological responses may occur for sea-level bursts (Reference 4).

What happened in Hiroshima and Nagasaki applies to one little portion of this range-yield effect spectrum; that is, that between 10 and 25 KT. If the first and second degree burn curves, the ionizing radiation curves, and the iso-pressure curves were all parallel, then moving to lower or higher yields would be a lot easier. But what happens is that the ratio of the free-field effects to one another changes. What occurred blastwise in Hiroshima that was complicated by immediate ionizing radiation will not help one understand what will happen blastwise at large yields uncomplicated by ionizing radiation. This is so simply because higher pressures occur at lower ionizing radiations for the higher yields. This is shown in Figure 13, a diagram of ionizing radiation on a log-log scale plotted against pounds per square inch with iso-yield and iso-range lines added. It is easy to see for the larger yields how much higher the overpressure is when the radiation stays the same. Do you follow?

The challenge is to know how to use what happened in the 10- to 25-KT part of the spectrum for the high-yield case. It is not easy

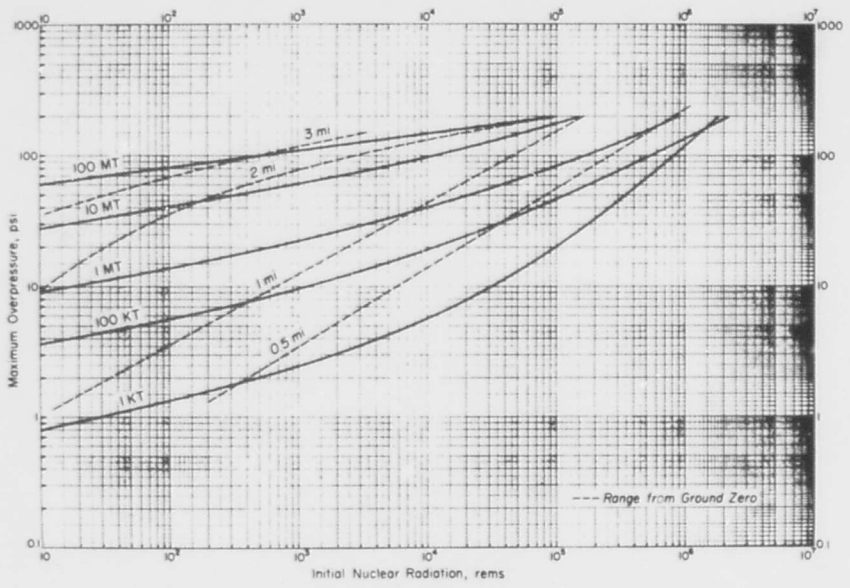


Figure 13. Relationship between maximum overpressure and initial nuclear radiation for surface burst at sea-level ambient pressure (Reference 4).

and, indeed, if I could have located anyone who knew how to do this well, I would have hired him a long time ago to educate our staff and to help in giving more precision to casualty estimations. Just think about this a little while, because this is one of the key problems in understanding nuclear effects.

AYRES: One difference I think should be mentioned, for the sake of the record, between the Nagasaki and Hiroshima cases and hypothetical nuclear war is that in the Nagasaki-Hiroshima cases, the bombs were really unexpected. People were doing pretty much what they would normally have been doing.

WARREN: No. There was an air alert and the people went in the shelters and then a lot of them came out.

LIFTON: There was an all-clear.

AYRES: There was only one plane.

WARREN: Yes, but people were not altogether unaware.

AYRES: Okay, they had an alert, but they weren't paying a great deal of attention to the alerts and there was only the one plane, whereas in a nuclear attack, presumably the warnings would be taken much more seriously.

WARREN: Yes. The situation would be quite different.

AYRES: Civil defense planning has been pretty much predicated on the idea of the surprise attack, but I don't know anybody who has really thought about it who really believes in that.

TAYLOR: I think there is another point of difference and that is that one bomb was dropped on each city. I think people are gradually coming around to the idea that this would not be the situation in a nuclear war if it broke out sometime in the future. There would be many bombs on the same city.

WARREN: That's right.

FREMONT-SMITH: And not necessarily all at the same time.

TAYLOR: Therefore the effects of the first explosions are really very different from the second time when many of the buildings would have been knocked down.

WARREN: The reason I referred quite so glibly to the alert is because I asked about this quite extensively and also because there were still lots of bodies in shelters because most people were probably goofing off and didn't come out.

LIFTON: It is true there was an air raid alert but Hiroshima had had many air raid alerts without ever having been bombed. Alerts had come from planes flying over to other cities, and people had got into the habit of ignoring air alerts. The great majority of people in Hiroshima were at home or on their way or otherwise getting ready to begin their jobs. There were people in the shelters, but a very small number, almost negligible, I should think.

AYRES: That was the point I wanted to make.

WARREN: I am glad to be corrected. The coffee hour is imminent, and I would like to summarize this. I have slightly different points of view from Sam [White]. They are not too bad, but I have on the board a list of injuries presumably that could occur, and, as Sam has pointed out, the lethality is almost a yes or no situation. If you are going to survive, you will live beyond 24 hours. If you are not going to survive, you are dead before, and sometimes within, a few minutes for different reasons.

WHITE: That has got to be assessed now. If clean wave forms for the pressures are involved and the latter are very high, the animals die quickly. At the 50 percent level, they don't die so quickly, and down in the lower end of the lethality curve is where one picks up significant numbers of survivors, of course.

WARREN: Yes. That is a typical survival curve.

WHITE: Yes.

WARREN: My difference is that I look at a nonrigid body as being approached by a sheet of pressure with a positive and negative component, and the speed of approach of this sheet is so rapid that as it passed over it, the body can receive a series of blows which implode it, but then the elasticity of the structure attempts to restore it to the former position aided by the negative pressure. However, in any case, the effect is a violent to-and-from movement which passes rapidly in a ripple-like fashion across the nonrigid body.

If the body is unrestrained, enough energy transfer may occur so that it can take off like a kicked football or become a missile. In the mouse facing the blast wave, high-speed movies show that the head end of the body folds into the thorax and the thorax into the abdomen as the wave front passes by (Reference 38). Then, presumably influenced by the negative component and the elasticity of the body, the head overextends, stretching the thorax and abdomen in the opposite direction. It resembles an accordian. There are extensive fractures of the vertebrae and pelvic bones and multiple internal hemorrhages.

As it passes across various organs, these organs are subjected to this violent to-and-from movement which is responsible for tears and hemorrhages. The defects that occur in the lung that Sam talks about are not simple. I am not convinced that it is the quantity of

air in the circulation that is the main factor in the lethality, because you can inject a lot of air without causing . . .

WHITE: Not on the arterial side, you can't.

WARREN: Yes, you can.

WHITE: You can if you do it tail-up. If you do it head-up, something like a fraction of a cubic centimeter will kill an animal.

WARREN: You can absorb a lot of air. I think it is the other things in addition—the tearing of the alveoli and the other organs and the disturbance of circulation and the bleeding—that all contribute. All I am saying is that it is not just a single factor, except in one condition which is similar to the automobile accident where the skull moves faster than the brain, which can result in a brain tear at the base of the pituitary stalk. This produces pulmonary edema (Reference 39). Sam and I have argued about this for many years, but this is one injury that can drown the animal in a plasma-like pulmonary edema within a few minutes. I can cite no evidence that this has occurred in humans except in automobile accidents and in other conditions where the head was translocated quickly.

You have, of course, concussion with fractures of the skull and other damage of other parts of the central nervous system. I think that you could have a direct trauma to the heart as this pulse goes across the thoracic cage. I bring this up because in automobile accidents, a steering wheel causes ruptures in the heart or damage that later results in infarcts (Reference 39). These may have been present in a small percentage of cases, although not identified in any research I have been able to see. But it is akin to the changes that Sam has mentioned, the chronic changes in the lungs.

AYRES: Could I ask, as a nonmedical man, what actually happens in the heart when a bubble of air arrives?

WARREN: You get a murmur that you can hear if it is in the ventricles, and after a while it is absorbed and disappears if it isn't pumped out (Reference 40).

WHITE: If it gets into the coronary vessels, that is something else. I have got whole series of slides here. You can see some of these things that Staff [Warren] is talking about if you want to.

WARREN: I have no doubt that he can demonstrate them but I did not want to leave the impression that air embolism was the main factor.

I can readily see why the kidney would suffer a similar tearing, and in many of the animals we had tearing of the large vessels with hemorrhage interperitoneally. It depends upon the situation. And there may be others.

Of course, you have got missiles; glass has been mentioned and the lumber in buildings. It is a very complex problem and most of the results that you have mentioned from studying the individuals later in Nagasaki have been secondary to fractures, with bad healing and infection and things of that sort.

It is difficult to visualize a blast wave front and what might happen to it under different conditions such as passing through an aperture or entering shelters. I would like to show you some spark photographic studies made of a three-room shelter model published by Cassen, et al., in 1950 (Reference 41).

The side walls of the model are of smooth brass. Plate glass is cemented on top and bottom to permit the light to pass vertically through the interior of the model to the photographic cassett beneath it. Refractive discontinuities in the air show sharply at exposures of 1.5 μsec. The blast wave front created by the shock tube has been calibrated at 14 psi. The flat blast wave front enters the square aperture at right angles to the walls of the model. The primary, secondary, and reflected waves show as sharp lines. Briefly, the wave travels down a corridor or antechamber passing through an open doorway into a long dead-end room. Near the dead end is an open doorway in the right wall leading into a larger second chamber or room, which in turn has a doorway in its right-hand wall near its dead end. This third room is the largest chamber and has only the one doorway mentioned. The doorways have simulated door frames only on the side walls so as to produce constriction of the aperture only on the sides for simplicity of patterns.

Figure 14 is the actual shadowgraph, taken 100 μsec after the blast wave front has entered the model. The wave has passed to the midpoint of the second doorway, leaving behind it a collection of vortices near the first doorway and many reflections. The right edge of the wave front has "slipped" through the doorway in the beginning

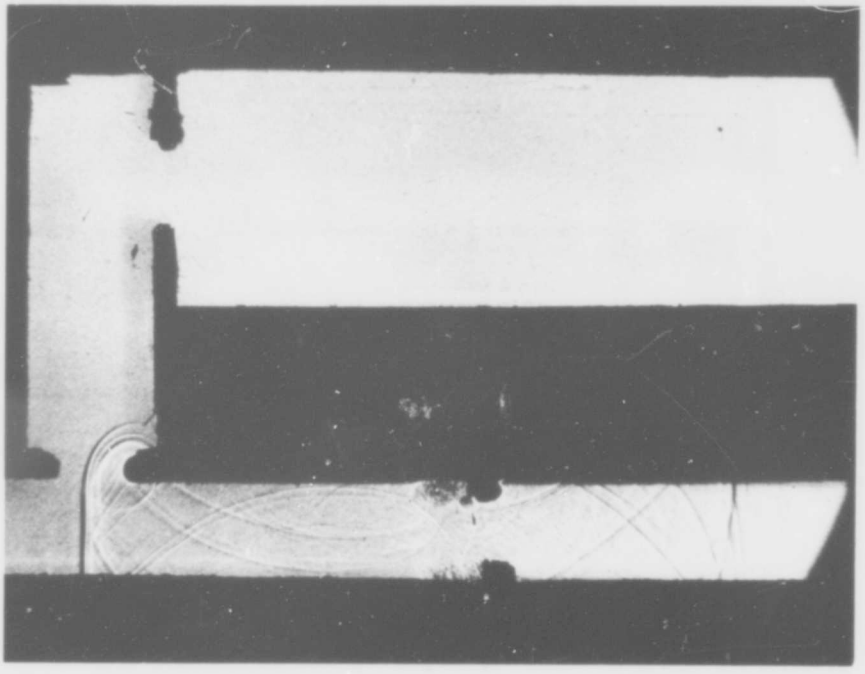


Figure 14. Spark shadowgraph (1.5-μsec exposure) of shelter model made 100 μsec after blast wave front entered the square aperture of model. The wave front has advanced to the middle of the second doorway where it has moved into the second room around the door frame. Several sizes of vortices remain around the first doorway and a large vortex is forming on the proximal door frame of no. 2 (see Figure 15).

of a turning motion. The dark hemispherical shadow of the vortex is already forming on the door frame. About one-third of the wave front has already turned into the second room, leaving two-thirds of the original wave front still perpendicular to the left-hand wall and passing toward the dead end. Since the details become more and more complex as the blast wave passes more doorways and enters the second and third rooms, the main features are shown by tracings of the shadowgraphs.

Several features may be identified (Figures 15 to 18):

1. After passing through the first door head on, it forms vortices at the door margins which follow it into the first room,

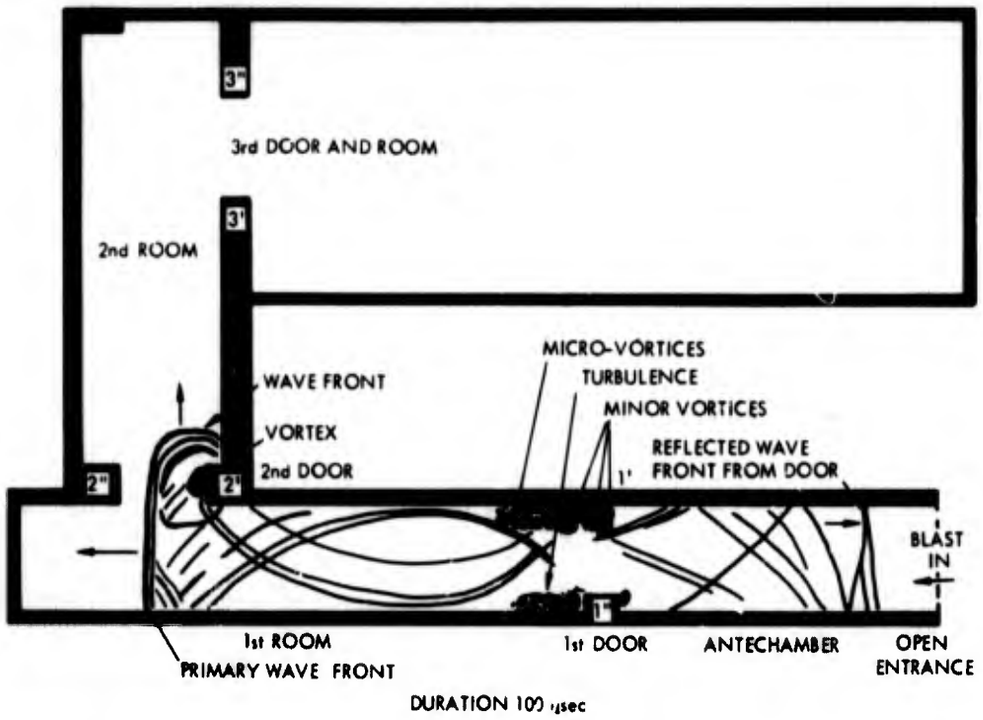


Figure 15. Tracing of Figure 14. The duration of the interval of travel from the entrance to the model to the position midway of the second doorway is 100 μsec. The pressure of the blast wave front is 14 psi. The door frames of the first doorway have small vortices clinging to them still, but there is a large area of microvortices and turbulence along both walls just inside the first room. Waves reflected from the doorway are moving back toward the entrance. The mechanism of vortex formation is demonstrated at the proximal door frame (2 ft) of the second doorway where the primary wave front slips around the corner.

but the vortices tend to break up into smaller vortices (microvortices) and turbulence which remain near the wall. They break up the wall side of the reflected waves returning to this first doorway (Figure 18).

2. A doorway in the side wall provides a side slip and turning movement as the wave front passes the opening (Figure 15) which is greater at the proximal door frame than at the distal frame. Thus the proximal vortices are larger (Figures 16 to 18).

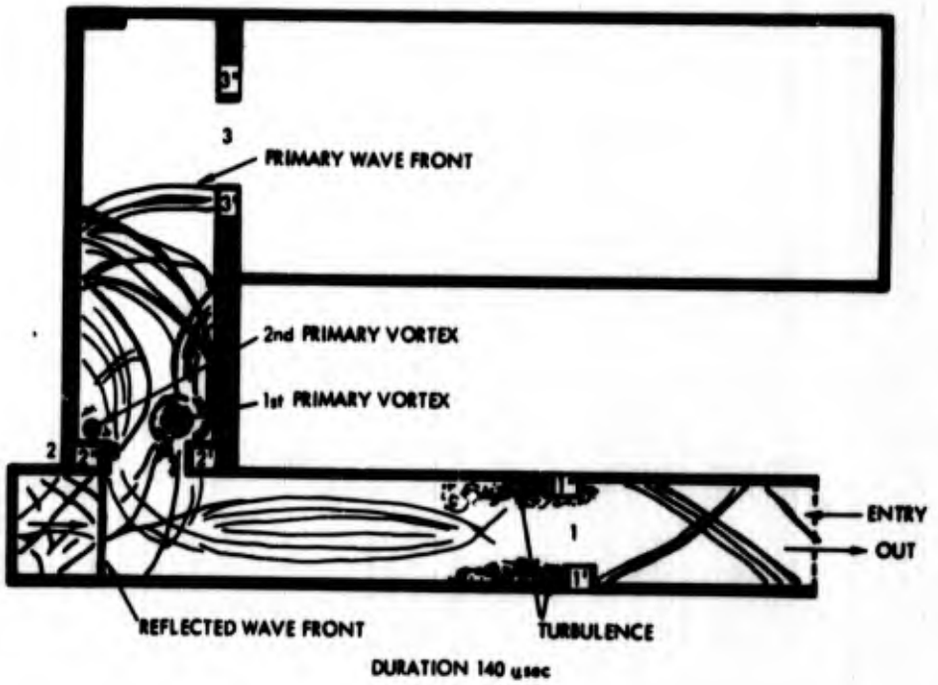


Figure 16. Tracing of shadowgraph taken at 140 μsec. The black disks are evidently vortical cylinders viewed in cross section and extend from floor to ceiling. The primary wave front continued forward for the most part and is shown reflected from the back wall to the distal door frame of door 2. There are many strong cross-reflections following it and it is presumably enhanced by its reflection. The portion refracting into the second room has advanced almost to the third door. Turbulence is still present distal to door 1.

3. The turbulence in the second and third chambers (Figures 17 and 18) is extremely complex. The vortices seem to follow the side walls as discrete entities for a considerable distance (Reference 2). Then they break up into what appears to be very small vortices and then just turbulence which effectively distorts reflected waves of some magnitude. This is typical of many situations illustrating the difficulty which Sam and others have had in actually establishing what happens in a shelter or a room exposed to blast waves of different forms and coming from different directions through a variety of apertures. Instrumentation tells you what occurs at that one place. Sam has found by instrumenting some of his animals and dummies in shelters that they have been subjected to tremendous pressures but I believe that these pressures were

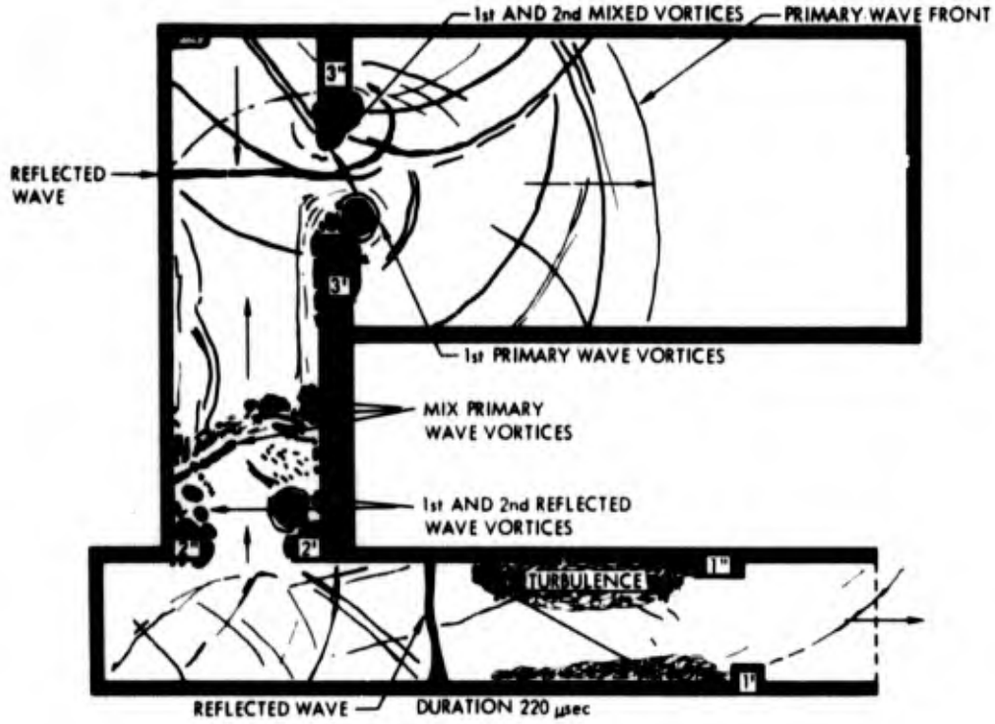


Figure 17. Tracing of shadowgraph taken at 220 μsec. The primary wave front has reached the middle of room 3. The primary wave form of Figure 16 has passed doorway 3 and been reflected back from the end wall of room 2 and has reached the midpoint of doorway 3. In room 1 the primary wave has been reflected from the end wall as in Figure 16, and has reached almost the midpoint of room 1. Turbulence is still prominent inside door 1. There are now two sets of vortices traveling forward from door 2, and door 3 has a primary set of vortices at the proximal frame and a mixture of primary and secondary vortices at the distal frame. The tendency of the vortices to creep along the walls is clearly evident.

not a simple wave form but a very complex series of waves. I think that these tracings explain why.

After reviewing Cassen's material, I reexamined the photograph of a Nagasaki elementary schoolroom (Figure 19) which I had taken because of the peculiar condition of the contents. The school building was about one-quarter of a mile almost west of zero. The blast wave front evidently struck the corner of the building and passed down each wall. The photograph was taken on the second floor from one

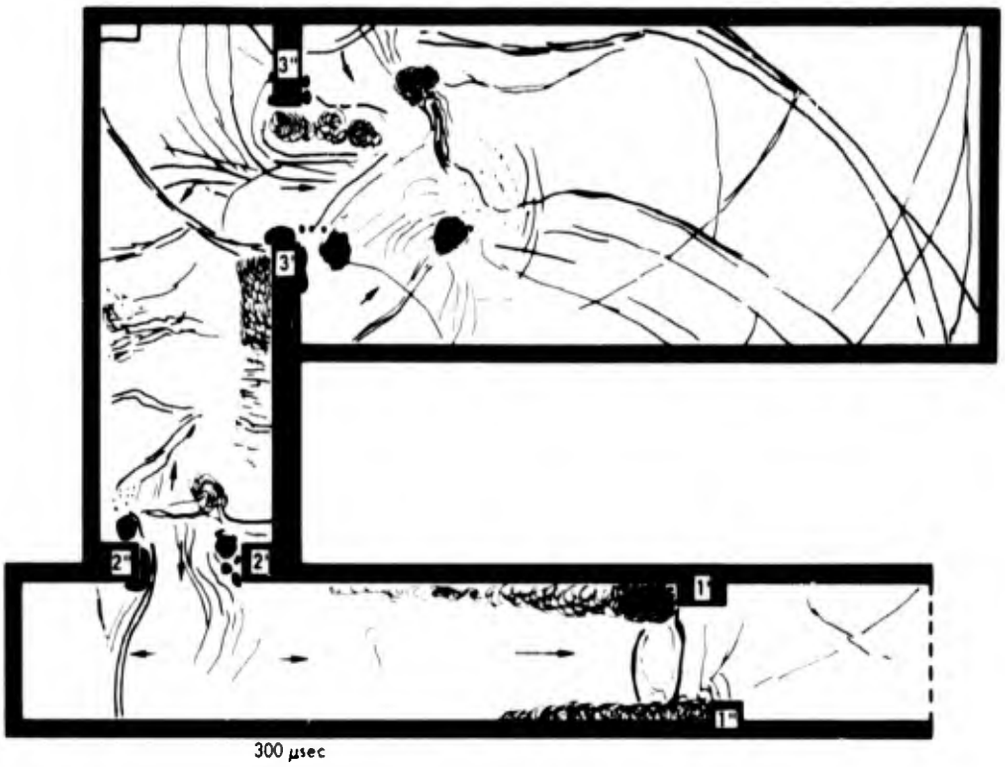


Figure 18. Tracing of shadowgraph taken at 300 μsec. The primary wave front has just about reached the back wall of the third room. The vortices produced by the two successive passes of the pressure front across door 3 are clearly evident in room 3. The reflected wave in room 2 is meeting resistance from the complex turbulence in the proximal two-thirds of room 3. A backflow through door 2 has produced a wave front in the direction of the back wall of room 1. The first reflected wave in room 1 has reached the region just inside door 1. It has been deformed at the edges by the turbulence and consists of three wave fronts, probably a result of additional reflections. (Adaptations of Dunne and Cassen, Reference 38.)

of the hall's two doorways looking directly at the corner of the room and thus directly toward zero. The contents were not yet disturbed. Apparently almost noone was in the building at the time of the blast. If one were to speculate that cylindrical vortices developed inside each window aperture and traveled along the walls, ceiling, and floor, an explanation is presented for the removal of the wainscoting, ceiling, soundproofing, and the tearing up of the floor near the windows. The larger lumber is the remains of benches and tables. The smaller probably represents the furring and wall panels. The

Figure 19. Nagasaki elementary school second floor corner facing blast about one-fourth mile from zero. Undisturbed contents in September 1945. It is suggested that cylindrical vortices formed on the four edges of each window aperture and crept along the walls and ceiling tearing off the wainscot and sound-proofing panels by their violent action. The debris was left in rough circular distribution on the floor probably by waves reflected from the walls and corner (see Figures 14 to 18). The large lumber was from benches and tables. The school was not occupied at the time. Lethal casualties would result from missiles, blast, and ionizing radiation at this location.

concrete is completely bare of any covering and the floor area below each window is essentially clear. All of this destruction must have occurred in a very short time.

The hallway behind the photographer was likewise stripped of door jambs, wainscoting, and ceiling covering by the whirling winds which destroyed the rooms across the hall in a somewhat less violent manner.

As you can see, the physical phenomena are very complex. To interpret the effects of blast alone from missiles, from translation of the body, from concussion, and from a variety of other effects is complex too.

AYRES: Why would this be difficult to instrument, aside from the large numbers of individual measuring devices required?

WARREN: Anything you put in there is going to disturb the pattern. Sam White has been able to instrument some of his animals successfully. The instruments tell only what happened at that spot. Large numbers would be required. As you can see, it is a very complex problem. There is still a lot to be learned, requiring perhaps as much as 10 years more work.

WHITE: Staff, didn't you tell me about an experience you had had in Japan, seeing a cow at the bottom of an airshaft?

WARREN: Yes, in the Nagasaki Medical School, an ox was dead, still standing, leaning against the wall of its stall. The hay in the manger was clean as was the straw on the floor. There was a window without a frame over the manger leading into a lightwell on the back of the building away from the blast.

There was what I took to be dried blood or blood and edema fluid on the nostrils and the floor beneath the head. It resembled the fluid which gushes out of the mouth and nostrils of the experimental animal when the pituitary stalk has been torn by the sudden excursion of the head from the blast wave. There were dried, somewhat formed, brown droppings but no evidence of diarrhea. From the looks of it, with the hay distributed in a circular fashion on the floor, I now believe that the ox had had the head sharply thrown against the wall and had had acute pulmonary edema resulting from the stalk tear.

WHITE: How far out was the medical school?

MC CULLOCH: 350 to 400 meters.

WARREN: Something like that.

UPTON: I would like to call again on Dr. Warren to pick up the thread.

THERMAL EFFECTS

WARREN: It is quite a complicated skein. I haven't much to say about thermal effects except the intensity of the infrared radiation, which was commented on a great deal, before the bomb was set off. This was in meetings immediately after the Alamogordo first test. Apparently in Alamogordo, even in the dry air, there was not enough ultraviolet to be of any significance physiologically and those of us at 9 miles who were out in the open had no sunburn. But the heat was quite apparent. It might be enough to set fires. Nobody thought about the fact that the blast wave might come along quickly enough to blow the incipient fires out. Some of you have seen the pictures of the building in The Effects of Nuclear Weapons (Reference 2). It begins to smolder and then it is blown out by the blast wave.

There was a lot of evidence of that in both Nagasaki and Hiroshima. Many structures were charred. There is also a picture (Reference 2) of roughly upholstered chairs that were sitting inside the schoolroom window and the backs of the chairs were charred to show the shape of the window aperture.

You all perhaps have seen roof tiles and pictures of the monuments that were partially melted on the surface to show the intensities at close-up locations.

While I am on it, the fires in the two Japanese cities started about a half hour afterward, presumably from the debris which fell on and around the hibachis. Many people were cooking breakfast, of course, at this time. In the medical school particularly the faculty had done just what a lot of us used to do when we blew a fuse. They inserted a metal coin, so the power didn't fail until the blast wave hit the substation, which was a little out of town. The electrical fixtures were suspended a couple of feet from the ceiling, particularly in the medical school. The ceiling soundproofing was made of compressed corn husk. When the blast wave went through the buildings the fixtures swung enough to short-circuit at the base and to set the soundproofing afire, which fell on the floor. The maple floor burned throughout the building. This was a secondary fire kind of thing.

LIFTON: You are not implying that all the fires in Nagasaki and Hiroshima emanated from the heat itself?

WARREN: No. There were a few fields of rice which were ripe enough to be brown, which had pretty well started but were not put out by the blast. But that is the only evidence that I can find from the people that came for interviews to show that the fire was initiated by the infrared.

UPTON: Would this be true of clothing, too?

WARREN: No. Darker clothing was charred. White clothing tends to reflect the radiation sufficiently so that the individual might not have skin burns underneath. Of course, this is all related to distance, for people close up would not be safeguarded in this way. They had to be out at a certain distance where the black configurations on the clothing would be a problem. Among the women there were a lot of shirtwaists, silk, I imagine, with barred patterns and there were a lot of patients that were in first-aid shelters with the pattern burned into the skin.

Now I will ask a question. Isn't it true that the main effects again are twofold: the acute ones where wide areas were burned and the individuals were apt to have serious illness and intoxication from it, large loss of fluid and minerals and plasma, and then it is nip and tuck, and the patient may die at some time subsequently? Then some burns get infected. The Japanese had some proclivity to produce keloids, but in comparing their recently healed burns with burns in our own armed forces it seemed that the number of keloids developed were not more numerous than ours for the same period.

I would like somebody to say something about keloids if they have any information. Dr. Brues, would you like to comment?

BRUES: I was in Japan with Drs. Henshaw and Neel* 18 months after the explosions, and the chief thing we observed was the large number of persons who had this reaction to burns.

Mr. Yamashita (see Figure 20) was a pick-and-shovel man. He was leaning back on his shovel, so his arm protected his side from

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* Dr. Paul S. Henshaw and Dr. James V. Neel were members of the first Atomic Bomb Casualty Commission (1946–47).

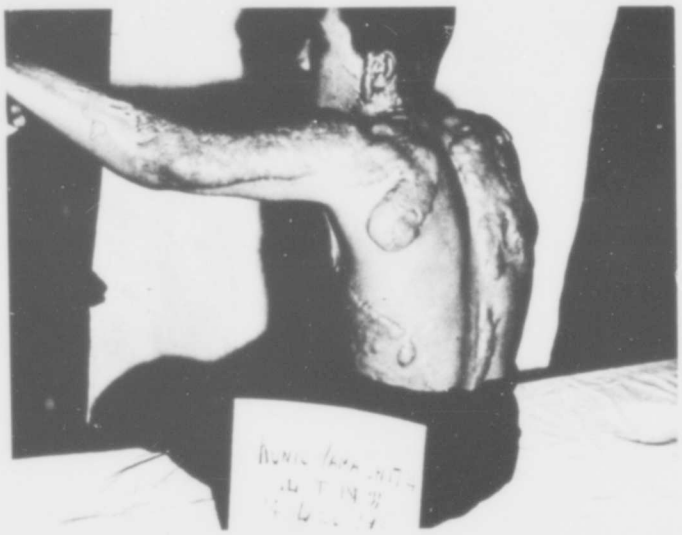


Figure 20. Keloids of a man burned by thermal radiation in Hiroshima. Photograph made in December 1946.

the heat. The only way in which one could distinguish this keloid from one following a flame burn was that in these cases the individuals could be arranged so that all the lesions were visible from one point, representing the point source of intense heat which impinged upon them. This sort of reaction appeared to be quite common, and we must have seen hundreds of persons in the Hiroshima area who had these. In fact, they didn't attract any attention on the street. That is about the only thing which was obvious in the population at that time.

I would like to raise a question on that. At the time, we speculated on the possibility that these lesions might lead to later complications such as skin cancer. I haven't seen any reports on that from the ABCC. Could anyone enlighten me as to what happened to these people?

JOHNSON: A study has just been concluded by Dr. Marie-Louise Johnson (Reference 42). She has examined over 10,000 subjects in the sample we examine every two years at the ABCC clinics, the Adult Health Study sample.

As to the burden of cosmetically disfiguring A-bomb scars, about 250 cases were found. Keloids were observed, but in addition keloids and hypertrophic scars following surgery or trauma, but unrelated to A-bomb injury, were more prevalent among persons exposed within 1,400 meters. In other words, the proximally exposed displayed a propensity to develop hypertrophied scars in circumstances following but not related to the A-bomb experience.

During the study period 1964–1966, no cases of carcinoma were observed in the survivors examined. Two cases occurred in the comparison groups: one was a Bowen's squamous cell carcinoma in a farmer; the other an early squamous cell carcinoma in skin severely damaged by therapeutic x ray.

Dr. Johnson has also noted some pigmentary disturbances, mostly around hair follicles, even in areas partially shielded by clothing.

LIFTON: In that disturbance there is no general darkening of the skin or pigmentation?

JOHNSON: No. There are areas of mottled hyper- and hypo-pigmentation around the A-bomb scars, even total depigmentation, but such changes are quite gross. There is another type of pigmentation, however, quite discrete, rather spotty, sometimes patterned although barely visible, and always peri-follicular.

LIFTON: I ask that because one of the many kinds of body mythology expressed later on was the belief that people who were exposed to the bomb became darkened, that their skin became generally darkened. I think the "kernel of truth" came from some of the burns that were witnessed at the beginning, but of course the belief also had psychological overtones.

JOHNSON: I believe Dr. Marie-Louise Johnson has noted no gross increase in generalized pigmentation. She has done some objective measurements with a reflectance meter, but these data are not yet available.

LIFTON: Another early belief was that the few Westerners who were in Hiroshima tended to burn much more readily than the Japanese. This was expressed in some of the very early records of interviews, but I would doubt that there is very much evidence of it.

JOHNSON: I would doubt this very much.

MILLER: When you spoke of hypertrophic scars, did you mean that the survivors with normal looking skin more easily developed scars when they were operated on?

JOHNSON: Yes. It is a finding which is difficult to evaluate. We have no real estimate of the tendency of the Japanese to form keloids. Similarly, it is difficult to delineate within the cases of keloid formation those persons who would have developed keloids as a result of other types of trauma.

ANGEVINE: Is there any indication that the Japanese have any more susceptibility to keloid than the Negro in this country? Another point of some importance, one would not expect to have carcinoma in this instance. If anything, one would expect a sarcomatous change.

SPEAR: As one of the nonmedical people here, may I ask, is there a simple way of defining keloid?

JOHNSON: Dr. Angevine can do that.

ANGEVINE: It could be defined as a non-neoplastic overproliferation of connective tissue.

FREMONT-SMITH: What we saw on the picture?

ANGEVINE: Yes.

UPTON: Exuberant scar.

FRANK: Is it implied that something about being exposed to radiation makes you subject to hypertrophic scars later?

JOHNSON: Yes.

FRANK: What is the link?

JOHNSON: I don't know.

MILLER: Dermatologists also look at the hair, and graying of the hair occurs in animals experimentally irradiated.

JOHNSON: Yes.

MILLER: What observations about the hair were made at ABCC?

WARREN: The hair was also burned off and frequently the rims of the ears were burned off below the cap level without any epilation being visible from ionizing radiation.

FREMONT-SMITH: Didn't the cattle that were exposed to Alamogordo also have gray streaks on their backs?

WARREN: Yes. It looked like powdered sugar on the side where the wind blew the fallout on their hide. They developed ulcers. Maybe somebody at Oak Ridge can correct me, but I don't know that any malignant tumors developed. Some of the hair grew back in the original color and some remained gray; isn't that right?

AUXIER: Right.

WARREN: This is presumably mostly beta.

CASARETT: I believe there's been one malignant tumor among the cattle so far.

FREMONT-SMITH: In the area on the skin?

CASARETT: Yes.

JOHNSON: The association of cancer developing in areas of the skin which have been radiated is well known.

UPTON: I think a distinction should be made here, though, in that in the Alamogordo cattle the lesions in question resulted from very extensive beta-ray burns associated with severe injury of the skin, ulcerating changes in many instances, whereas the thermal burns that we are talking about now were more superficial, of a more acute nature.

WARREN: That's right.

To summarize what Dr. Johnson found, you would say there was some evidence of summation between the gamma radiation, neutrons and the thermal radiation.

JOHNSON: Yes.

COURT BROWN: Isn't it a fact, going back to Dr. Angevine's remarks on keloids, that squamous tumors in the skin over keloids are

liable to arise if the scars are on the flexor or extensor aspects of joints?

ANGEVINE: I wouldn't think so, no.

COURT BROWN: I have always thought this so and certainly have seen it. Another point: I wondered to what extent it is fair to draw any conclusions at this time about the incidence of malignant change in keloids when it is possible that the proportions of those developing such changes may well have died. I certainly remember seeing a case in the Red Cross Hospital in Hiroshima in 1958 which was pointed out to me as a malignant change in keloid in an old man. There may well have been others.

JOHNSON: Richard Doll told me about this case.* The case I think you saw was a man who had a very extensive burn on the leg and a chronic skin ulcer. He was at one time treated with x ray but subsequently his leg was amputated. We have not been able to recover the histologic sections, but it seems unlikely that one could say with any degree of certainty that he had an A-bomb-induced skin cancer.

COURT BROWN: This really reflects the general problem of organizing tumor registries and one pertinent to the ABCC.

JOHNSON: I think you were going to say that not enough time had elapsed to be certain that tumors would not develop in the skin. I think this is true.

MILLER: There is one case in the literature of a tumor developing in a burn scar 60 years after the burn was sustained (Reference 43). Also you never got to answer the question about the hair.

JOHNSON: Dyeing of hair is commonly practiced in Japan by both women and men. It was possible, however, in this study to evaluate hair graying by close inspection and by direct questioning.

This study appears to be unique in furnishing some direct evidence supporting the concept of accelerated aging in the exposed. Hair graying occurred at earlier ages and was more prevalent in persons exposed within 1,400 meters of the hypocenters. In this

* R. Doll, Personal communication.

exposure group, other findings included functional vascular disturbances such as cutis marmorata and Raynaud's phenomenon. In addition, there were more benign neoplasms such as compound nevi, fibroepitheliomata which occurred more often in the exposed.

UPTON: Ken, do you relate these to thermal exposure or do you presuppose that these are actinic radiation lesions or others?

JOHNSON: I presuppose they are a combination of all things which happened to an individual at that time. They are not actinic in the sense that we have taken into consideration exposure to actinic rays related to occupation, as in farmers and fishermen.

UPTON: Is it possible to exclude the thermal exposures?

JOHNSON: No.

UPTON: On the basis of shielding factors?

JOHNSON: I don't know.

UPTON: Can one separate between thermal effects and effects from ionizing radiation in this population?

JOHNSON: I think you can separate on the basis of a third degree burn or a scar.

WARREN: You can also separate those that are from missiles, flying glass, or something else that would cause the scar.

JOHNSON: Yes.

ABRAHAMSON: Are there many chromosome aberrations noted in the tissues of these individuals?

JOHNSON: About 100 persons examined in the dermatology study also had cytogenetic study performed on peripheral lymphocytes by Bloom, et al. (Reference 44). We were interested in knowing if one could predict chromosomal aberrations of the lymphocyte from the severity of A-bomb scars. The 16 individuals in this group with significant A-bomb scars did not correlate strongly with the individuals who had chromosomal aberrations. Just as many without scars or without other dermal stigmata had chromosomal abnormalities. Is this clear?

ABRAHAMSON: Yes.

FREMONT-SMITH: Warren McCulloch has been trying to get in. Warren, speak up because nobody will let you otherwise.

MC CULLOCH: A question out of ignorance: What is the graying of the hair in these cases? Is it in the chelates or is it bubbles in the hair?

JOHNSON: It's just a loss of pigmentation of the hair shaft.

MC CULLOCH: Yes, but isn't that a lack of chelates, or is it the presence of air bubbles; what sort of thing is it?

JOHNSON: What produces graying?

MC CULLOCH: Yes.

WARREN: The cell neglects to produce enough pigment.

UPTON: It is injury of the melanocytes, which contribute pigment to the hair shaft that is being formed. These cells are damaged by radiation.

MILLER: How much grayer were they? Can you say that a person of 50 was as gray as a person normally would be at 60 or something like that?

JOHNSON: Those who were less than 20 years of age at the time of exposure, who would now be less than 42 years of age, had a significantly increased prevalence of grayness than the comparison group. I'm sorry I can't give you the actual numbers involved, but it was a significant difference.

WARREN: Was this from the thermal or gamma radiation?

JOHNSON: I have no idea.

FREMONT-SMITH: A sort of total combined effects, wasn't it?

JOHNSON: Yes.

MILLER: You do get graying in animals from radiation alone, so it is reasonable to suppose that that was the cause.

WARREN: Very often the hair comes back curly when it was straight before. You didn't see any of this, did you?

JOHNSON: In the 12,000 people, I think there were two or three people who had curly hair. Curly hair is almost unheard of in the Japanese.

WARREN: Yes, but gray hair. The gray was still straight?

JOHNSON: The hair grew back straight. As you know, when hair becomes gray, it also becomes more coarse.

BRUES: To go back to this question of the nature of the lesion, the graying of hair following radiation, as Dr. Upton said, is probably due to the destruction of the melanocytes or of their ability to produce pigment.

JOHNSON: Yes.

BRUES: That which occurs in aging is, I suspect, due to other causes, changes in the character of the hair fiber itself. Thus it is fair to question whether the suggestion that the graying of hair is in some way related to acceleration of the aging process, as this suggestion may be a red herring.

CASARETT: I think that there are two phases to radiation effect on the pigment of hair: one, the regrowth of depigmented hair after the acute effects of radiation, that is, after epilation; and second, the delayed graying of hair that has not been preceded by epilation. I think the distinction has to be made between these two. Is it possible to determine whether these people were originally epilated?

JOHNSON: From the subject's own history, which we have recorded, the occurrence of epilation can be ascertained. You are quite correct in reference to the acute effects stated by many survivors thusly: "My hair when it grew back was at first white and later became black again." But we are discussing now the pattern of graying, starting at the temple, the type of pattern usually attributed to aging.

WARREN: In this case, earlier.

JOHNSON: In this case, earlier. Also, the skin lesions which are related to aging occurred earlier in the group that was proximally

TAYLOR: Are the dose levels at which these two different effects produced very different from each other? Is there a difference of an order of magnitude in the total exposure necessary to produce a graying which is not associated with original acute damage?

CASARETT: The delayed effect is time dependent as well as dose dependent. In other words, the larger the dose, the earlier the delayed effect will appear and the smaller the dose the later it will appear.

TAYLOR: Is one talking in tens of r total effective or hundreds of r?

CASARETT: We're talking about many hundreds of r for the acute epilation effects.

TAYLOR: Yes, but how about the other?

CASARETT: And the other, of course, may occur some time after regrowth of hair following epilating doses or after smaller doses which do not cause epilation, with the latency being inversely related to size of dose.

TAYLOR: Is there any evidence that this is produced below, say, 100 r?

UPTON: We didn't see it in the LAF-1 mouse, which is normally brown, below 200 rad of gamma rays.

CASARETT: We've seen it in the hooded, partially black, Long-Evans strain rat with these doses. Toward the end of life, to be sure, but premature in the irradiated.

BUSTAD: With what kind of doses, George?

CASARETT: On the order of 100 r or greater.

MILLER: One biologic dosimeter is the small opacities produced in the posterior capsule of the lens of the eye, and I wonder what their relationship in those that were gray earlier was to other findings on the examination.

JOHNSON: I haven't the answer, and I would doubt that the relationship could be established. The useful matching of the relatively

small number of patients, prematurely gray, with ocular examinations demonstrating polychromatic sheer in the posterior capsule is unlikely.

WARREN: It was reported to me that there were a few cases who had been looking toward the detonation with their eyes open at the time who got severe burns of the cornea and maybe more. Any follow up on those?

FREMONT-SMITH: On the cornea, not the retina?

WARREN: Well, I don't know. I had no facilities to follow these up and these may have been acute ones.

JOHNSON: I do not recall any significant statement about keratitis in the recent review of ophthalmologic findings by Miller and Nefzger (Reference 45). The authors stated that it was difficult to know the total number of subjects examined by all ABCC ophthalmologists. Let us say it is approximately 7,000. There were only ten persons who were noted to have serious visual impairment secondary to radiation damage.

MILLER: That was due to cataracts.

JOHNSON: Yes.

WARREN: It is difficult to pin that down. Is there any other effect, chronic or acute?

UPTON: You mentioned burns, Staff. I wonder about retinal burns associated with thermal radiation.

FREMONT-SMITH: The total number of eye defects was low and most of these were cataracts. The opportunity for burns is very slight, isn't it, for retinal burns?

UPTON: But is this not a lesion that has been observed at least in animals? Is there someone who could contribute something on this effect?

WARREN: Maybe at the next meeting we can get somebody posted on this.

FREMONT-SMITH: Right.

TAYLOR: Isn't one point about that, that it is not clear that retinal burns would be observed unless they were extremely severe? As I understand it, a retinal burn that is considered very severe in one context is not observable in another. For example, a person perhaps will not know that he has a retinal burn until he tries to fire a rifle in a shooting contest.

WARREN: He can't see the sights.

TAYLOR: That's one of the difficulties. It's not an obvious thing.

FREMONT-SMITH: You mean it's a very small area and it might be in full view but it might not, depending upon what his position was the moment that that burst went up.

TAYLOR: Yes.

UPTON: A cataract is not a retinal burn. If one were to get a full burn of the retina, this would be irreparable.

FREMONT-SMITH: It might not be noticed.

TAYLOR: That is so, but the question is whether it has been noticed in the studies that were made in Hiroshima and Nagasaki.

FREMONT-SMITH: They might be obscured by the cataracts. There might be retinal burns behind some of these.

JOHNSON: "Radiation cataracts" are not cataracts in the usual sense; they do not resemble senile cataracts. More properly we should speak of certain ophthalmic changes, a migration of lenticular cells toward the posterior pole where they aggregate and reflect light on slit lamp examination. This reflex is called a polychromatic sheen on plaque. It is the most common finding related to radiation of the eye. Its appearance differs greatly from that of a cataract and it does not usually produce visual impairment.

MILLER: There were ten patients that the ophthalmologists classified as having Grade III or Grade IV cataracts, which is different.

FREMONT-SMITH: These were regular cataracts?

MILLER: Regular.

WARREN: Would this be a normal incidence in the population?

MILLER: No. These were people under 45 years of age. The other study related to epilation in 165 patients who had 90 percent epilation; 81 percent had small granular plaques on the posterior capsule of the lens, whereas the ordinary frequency was 10 percent (Reference 46).

WARREN: There was some speculation afterward—I don't know how verifiable it is now—that some people standing in shelters or in a crowd in a street car whose heads emerged above the mass of the crowd suffered cataracts, developing later presumably from neutron bombardment.

As long as we're on the eye, any evidence on that by you gentlemen who have looked recently?

MILLER: No.

JOHNSON: No.

MILLER: Apparently the effects in the American physicists were much more marked than those observed in Hiroshima and Nagasaki (Reference 47).

WARREN: Yes. With the neutron doses, we expected a lot more. But, of course, everything else masked it.

Anything else on thermal?

ANGEVINE: It was stated that these capsular or retrolental lesions are specifically associated with neutron radiation. Is this correct?

UPTON: I think one can get these with gamma rays. The work of Merriam and his colleagues at Columbia on patients exposed in the region of the eye has indicated that lesions of this kind are inducible by x rays and gamma rays, but the dosage required generally exceeds the lethal dose if given to the whole body (Reference 48).

WARREN: These are pituitary exposures?

UPTON: A variety of radiotherapy cases involving incidental exposure of the lens.

COURT BROWN: One can certainly get them; one has seen them. But the point is that the RBE is greatly different.

UPTON: Yes.

In animal experiments it takes about half as much total energy to produce a serious lesion with neutrons, densely ionizing particles, as it does with x rays or gamma rays which are sparsely ionizing.

COURT BROWN: For neutrons it's higher than that, isn't it?

UPTON: This would depend on the energy of the neutrons, the size of the lens, and other factors. For low-energy neutrons and exposures lasting only a few minutes, the RBE has been observed to be as high as 10 for induction of lesions in the rabbit lens, and if the radiation is given over a long period of time in many small exposures, the RBE may conceivably be as high as 20 or more (Reference 49).

FRANK: Would you mind telling us what the scale means?

UPTON: Let's say that it takes 20 times more energy deposited in the lens by x rays or gamma rays than by neutrons to produce a lesion of given severity when a dose is given over a period of weeks. In indicating that the lens can tolerate relatively large amounts of gamma rays when accumulated over long periods of time, and in indicating that this is not apparently true for densely ionizing radiations, such as neutrons, the data on experimental animals are consistent with those on man.

FRANK: The term "retinal burn" has me confused. Is this a burn of actinic rays or a burn through x rays or ionizing radiation?

WARREN: I thought that at first it was corneal injury from heat from the fires (infrared). We have mentioned scotoma earlier as being caused by the brilliant light of the detonation causing a temporary injury to the retina.

FRANK: If you look at an atomic burst, are the eyes damaged mainly from the heat or mainly by...

WARREN: Mainly from the light, I think, because of the distance. At 9 miles the heat is appreciable, but it is not damaging. But the light will produce quite a scotoma.

FRANK: But you distinguish that from a burn, you said, that is recoverable?

WARREN: Yes, a mild burn would be recoverable, too. The retina could be damaged irreparably if there was enough light or infrared radiation.

MILLER: In 1954 we examined 4,400 children in Hiroshima and their age at that time is shown on the slide, along with their age at the time of the bomb. We tested the visual acuity, as do pediatricians, by having the child cover each eye alternately and read a Snellen chart at a distance of 20 feet. Table 1 refers to the percentage of children with visual acuity loss not due to detectable ophthalmic abnormality, lesions revealed by examining the eye with an ophthalmoscope. We were not really looking for a radiation effect. We were wondering if the Japanese were, as is said, more often myopic than Americans were. But because we had the data available, we distributed them by distance from the hypocenter, and in the group that were 7 to 10 years at the time of the bomb, or 16 to 19 years at the time of examination (they had just passed through

Table 1. Percentage of Hiroshima children in 1954 with visual acuity loss: Snellen score of 20/70 or worse in at least one eye not due to organic disease (Reference 36).[a]

Age (years)		Distance from Hypocenter (meters)		
1945	1954	1,500 & Under	1,501–1,800	Over 1,800
0–2	8–11	7.1[b]	5.0	4.0
3–6	12–15	8.6	8.6	8.2
7–10	16–19	20.5[c]	21.1[c]	9.1

NOTES:
[a] Reproduced with the permission of the Journal of Chronic Diseases and of Pergamon Press.
[b] Differs significantly from group over 1,800 meters: $P < 0.05$.
[c] Differs significantly from group over 1,800 meters: $P < 0.001$.

adolescence), there was twice the frequency of visual acuity defect of 20/70 or worse in at least one eye among those who were within 1,800 meters than among those who were more distant from the bomb (References 36 and 50). In the youngest group there was some hint of a differential in the same direction, and in the middle group there was none. We don't know how to interpret this array of data. We thought that perhaps this impaired visual acuity was due to myopia, but we had already examined the children and no reexamination has been made.

UPTON: There were some data developed in monkeys exposed by the Air Force which also suggested impaired visual acuity.

MILLER: That is correct. We had thought that this observation in man was valuable because it would be less easily demonstrated in animals, for it is difficult to test their visual acuity. But the Air Force did such a study. Monkeys were trained to differentiate between a perfect circle and a broken circle in which the break was of varying length. The investigators showed that focal irradiation to the head did impair the monkey's ability to discriminate between the test objects (References 51 and 52).

WHITE: That is ionizing radiation, not thermal.

MILLER: Ionizing.

JOHNSON: One should mention a recent review of ophthalmologic findings by Miller and Nefzger (Reference 45). Except for Miller's study, there is no evidence of significant diminution in visual acuity among A-bomb survivors. It may be that the age groups examined differed.

MILLER: Correct.

JOHNSON: There may be a relationship to age.

SPEAR: Staff, I think that we went rather lightly over the possible physical damage resulting from thermal radiation as a primary cause of fire. I seem to recall that some years back, in some of the Nevada experiments, there was an attempt to isolate this and to find out what was susceptible to ignition from prompt thermal radiation. As I recall, if you had a large building, a frame house, you would get charring and then the blast wave would suppress the ignition.

WARREN: Yes.

SPEAR: But it was also true that if a highly combustible curtain material were hanging on the inside of a house, it was not extinguished by the blast; that if very dry weathered lumber, trash, paper—things that would be blown around by the blast, not rigid so that the blast would blow them out—were outside the house, they would start fires. I am a little surprised that in Hiroshima there would not have been more than the kind of rice field that you describe which sustained combustion through the blast wave.

AUXIER: There is really very little trash in Japanese communities and there were very little untended areas. In other words, there were no dry grassy lawns to speak of. They didn't exist. Also, the climate at that time of year resulted in lush, green growth, generally, except for the rice fields, and there was nothing dry enough to actually ignite.

Speaking of the Nevada experience, though, one of the things examined, and it turned out to be nearly indestructible, is the average automobile. You can turn it over a few times and make it look bad, but it will still run. The weakest point of the automobile was the interior; the windshield and the rear windows would remain intact, at reasonable overpressures, but, with the side windows open, the heat would ignite the interior which was protected from the blast and thus the fire was not blown out. Almost all of the cars would burn if they were exposed in this way.

For a typical Japanese house and with a nominal yield weapon, the ratio of the thermal to blast energy was appropriate, ignition occurred indoors, through windows which in some cases maintained their integrity, and a fire was possible.

WARREN: A very good point.

SPEAR: I think there is a marked difference in American cities as regards the absence of trash, which you mentioned. I recall that we did have a survey in one city, which estimated that there were approximately 10,000 ignition points per square mile in what was taken to be a typical American city, given the kind of lax housekeeping that we have in yards and similar areas. One of the positive steps that was suggested was trying to avoid this kind of disorder.

UPTON: There was indeed a fire storm, was there not?

WARREN: Yes.

UPTON: But this was an indirect effect and not a direct one?

WARREN: I think so. Products of combustion were furnished by all the broken lumber in these buildings. We had a good fire storm in Beverly Hills a few years ago. With a large enough area involved, you will have a cataclysmic, dynamic affair.

AUXIER: This happened, of course, in Japan, only in Hiroshima, and, as Dr. Warren pointed out, the fires actually became noticeable in 20 to 30 minutes and they grew fairly slowly. Thus the fire storm developed over a period of hours and as the heat grew, the rising column of air drew in fresh air from the sides such that the winds became appreciable and helped fan the fire. The temperature got to be extraordinarily high, comparable to those reached in ceramic kilns. This is what led to the totally desolate views of downtown Hiroshima seen in typical photographs.

UPTON: To what extent are the keloids that we have been discussing associated with the fire scar as opposed to the direct thermal radiation exposure? Is there any way to estimate what fraction of keloids might have resulted from one type of injury as opposed to the other?

JOHNSON: One way of estimating it would be a consideration of the pattern of keloids.

UPTON: The pattern?

JOHNSON: The pattern. From the topography of scars we have been able to reconstruct the physical attitude or position of the person in relation to the bomb's hypocenter. It is relatively easy to do when there is a distinctive pattern of scarring. Gregory and Johnson (Reference 53) were able to identify from the location of scars the quadrant of the body which was facing the epicenter. Their particular interest was the relation of shielding the spleen and survivorship. If the keloids or scars do not form a distinctive topographical pattern, then there is no way of differentiating the radiation effect from the secondary effects of trauma from falling timbers, fire, and infection.

WARREN: If you want to read in great detail of a fire storm, read The Night Hamburg Died (Reference 54).

There are two other items, as long as we are talking about fire. The fire can be so vigorous that it uses up the oxygen. Maybe Dr. Auxier can check me on this. I believe that there were quite a few deaths in other cities, as well as in these two, from lack of oxygen, particularly those that were in trench shelters beside the road.

AUXIER: Yes.

WARREN: The Japanese had a habit of putting water in all the hollow places for fire protection and in Yokohama at least there were quite a few illustrations of people trapped by fire all round them who lay down in these puddles and then were boiled because the intensity of the heat was so great.

I could conceive of people running by a fire in a narrow street who would get burned on one side. It would be hard to differentiate these but I don't think they were as numerous as the ones who were actually exposed to the thermal radiation flash from the detonation.

AUXIER: Also, I think that the shielding histories, in which I have much more confidence than a lot of people, even those in ABCC, provide the documentation of the location of the survivor at the time of the bombing and the history of his meandering experiences. These histories all have a designation as to whether the interviewer thought they were reliable. Those that the interviewers have marked as reliable, we have found to be very reliable. I think that from these you can tell whether it was a flash or secondary burn, because the interviewers always ask.

JABLON: This is not a direct answer to Dr. Upton's question, but the overwhelming majority, 90 percent, of the burns that are recorded by ABCC are flash burns.

LIFTON: I don't think this is the time, but since the issue of mortality rate in Hiroshima did come up, I have always found it a very confusing and difficult one and I would like to raise it again, really in terms of two things: the most accurate figure and the reliability of that figure. The initial estimates that I have read were anywhere from 65,000 to 78,000; there have been later estimates that have been as high as 350,000. The city of Hiroshima

estimates 200,000. Obviously many things enter into these estimates other than scientific exactitude. There are people in Hiroshima who feel that the official estimates, even if they were done very carefully, could not be reliable because of the nature of the counting and the chaos at the time. Those people say that if the statistics could have been accurate, they would be higher. Are there any other thoughts or impressions on this?

WHITE: One of the uncertainties as I remember in the discussion over the years that contributes to this is the applicability of the numbers available to the civilians in the city on the one hand...

LIFTON: And the military, yes.

WHITE: Others include the unknown number of military personnel.

LIFTON: There is the military personnel and also the commuting population, which is a sizable number. Actually, estimates of the overall number of people in the city at the time vary enormously, by several hundred thousand.

WHITE: Yes. I have looked at the Joint Commission report very carefully. I thought that if people, with the help of the Japanese, gave careful assessment as to the civilian situation, it seemed unlikely that anybody at some future time could do better. I personally put, from the figures I quoted earlier, plus or minus five or ten thousand to them, apply them only to civilians, and know that some of the long-term deaths or long-term sequelae have to be added to the totals compiled at the time the Joint Commission reports were written. Other than that I don't have any figures.

FREMONT-SMITH: What, then, is your best estimate of the figures?

WHITE: I quoted them earlier, and they are in Oughterson and Warren's textbook (Reference 18).

BRILL: The best current estimates are cited in a recent paper by Zeldis, et al. (Reference 55). Their figures were taken from Professor Masayama's estimates as well as from the Joint Commission data. For Hiroshima, the exposed population was 255,260 and the number of survivors was 187,020, whereas for Nagasaki the number of persons in the city exposed was 195,300 with 157,300 survivors.

WARREN: All of this is nicely complicated by the fact that the Japanese had taken no census and that in Hiroshima some x number of thousand—according to our people, between 10- and 30,000—people had come in to help evacuate the city the night before. And you could see on the little posts of the houses as far as 20 miles out of town little diamond-shaped cards which said, "Here lived a victim of Hiroshima," which kept us a little queasy for awhile. The military in their headquarters had a big hospital—I don't know how many beds, but reportedly around 500 or more—and something like 20,000 troops bivouacked, varying from day to day. So where are you? I was not satisfied with the first estimates because I thought that from the evidence we got from teachers, a few survivors of the Mitshubishi administration who came back and talked to us, and from appropriate civilians, it must be on the order of 100,000. But this is moot probably anyway. We don't need to strain on these figures. It was a large casualty figure.

UPTON: Dealing with the casualty figures, we have considered burns and blast effects. To what extent do casualties from ionizing radiation come in? Is it possible to estimate the number of deaths caused by exposure to ionizing radiation in the absence of the other injuries?

WARREN: This is a good time to make the transition to ionizing radiation, too, because I think we have gone full scale on these other two.

Has anybody any information they would like to give out on this new item?

WHITE: The National Academy of Sciences in Washington, D.C., 19-23 April 1965, had a symposium on protective construction. The program covered several days. The fire marshal of Hamburg, Mr. Herman Leutz, was there and he gave a very interesting discussion on fire storm (Reference 56). I highly recommend his presentation to this group. Mr. Leutz gave some quantitative information about the time it takes to get a fire storm going and the amount of combustible material per acre that it takes to sustain a fire storm. This was one of the few real sensible hard-headed analyses of conditions involving fire storm I have ever had the pleasure to listen to.

COURT BROWN: What period of time, Dr. Warren, do these casualty figures relate to? Are these casualties in the first day, the first week, the first month, the first year, or what?

WARREN: I think they are casualties in the acute period of, say, the first two or three weeks because the rate of death varied. I might tell you what was described to me most vividly for Nagasaki because the Urikami Valley was a rather discrete area, the majority of which was involved in the bomb detonation. The first recognizable relief came from the railroad trains which were backed in from the north about ten o'clock, which was roughly 2 hours afterward. They couldn't get south of the bridge, 1,000 meters north of zero, and yet people stormed onto that train. They had about 15 or 18 cars, a very long train, and the people were packed on it, standing on the roofs and holding onto the steps. The train moved out to the north about 3 or 4 miles to the first schoolhouse, at which point they tried to unload some casualties. They were astounded to find a lot of people dead standing up in the press of bodies. I could not explain this. I thought it must be a blast effect, but now I think that it was due to an ionizing radiation superexposure much like the experiments at Frenchman's Flats with Gerald McDonel's pigs and burros (Reference 57). Fairly close to the center, they got very large amounts of ionizing radiation which apparently affected the brain and caused a general state of acute shock, collapse, and death within hours.

That is the first part of the casualty group and nobody knows how many. As the train went north and finally emptied out, there were fewer and fewer of these dead. Then later on in the day, as you remember, more trains were filled and unloaded but there was no more of this particular type of case.

Then probably for the next few days, lasting for about a week or so, casualties developed a bloody diarrhea and died during the second to fourth weeks. The Japanese doctors identified it tentatively as some infection like cholera or acute typhoid. Since the flies at that time were numerous, it was thought that typhoid and cholera could be readily spread. Even six weeks after the bombing, when I got there, the flies were so numerous in Hiroshima that, looking at people coming along the street, you thought they had white clothes with polka dot patterns; but when they approached close enough, you saw that these were flies. The stench, of course, from the rotting bodies was like a pigpen odor. Flies were eliminated by the spraying of the area by the American Force with DDT, which was a great boon.

The Japanese found from autopsies during the first few weeks that the intestinal tract, particularly the colon, was injured, apparently

by the ionizing radiation. This was unexpected, for all of our prior experience had been with animals, chiefly dogs, where the small intestine was the main vulnerable organ during this acute stage.

The collapse of the bone marrow became dominant as a cause of death between the fourth and eighth weeks, during which time the anemias and the purpuras appeared. I arrived about the fifth week in Hiroshima and the sixth week in Nagasaki. Large numbers of patients with purpura and pallor were brought into the first-aid stations where they died within a day or so. The reason they were brought in was because the prefectural government in both places had the ability to deliver two balls of rice and a pinch of tea for each occupant of a shelter. The shelter was anything from a piece of corrugated iron which would keep the rain off to some part of a building that wasn't leaking. I saw almost no burns at this time. Then, in Hiroshima, almost to the day six weeks after the bombing, the large numbers of dying purpura cases stopped coming in. I suspected from earlier dog experiments (Reference 58) that this might happen. So I went to Nagasaki right away but was only able to get there three days later. The large-scale deaths with purpura were beginning to diminish there also.

There were many purpura cases after that, but not much mortality from this cause. I mention this because Shields Warren's party came in just after the large-scale deaths from purpura had ceased. They saw relatively few cases and were not as impressed by this finding as I had been. And with the lessened pressure on the first-aid stations, the farmers brought back the burn cases which they had been feeding and caring for. I think this is why the subsequent investigators were so much impressed by the number of burns. If I had not heard about them later, I would have thought that there were not as many burns as there were bone marrow injuries, and Shield's party was impressed the other way, that there weren't many bone marrow cases and there were lots of burns.

These casualties were the result of exposure to the prompt radiation and the radiation coming from the rising cloud of fission products. The fallout, which was one of our important assignments, was not involved. Although we could trace it in the Nagasaki areas all of the way to the east, to the sea, about 50 miles by roads and jeep, the intensity was definitely below any biological significance.

We didn't get enough instruments to Hiroshima to make as good a survey, but Lt. Col. Hymer Friedel traced the fallout to the west up

over the hill. He ran into a bamboo forest and then torrential rains came and his instrumentation failed. Anyway, it was time to come home so he pulled out before that was investigated very far. But in no case was there much more fallout evident than what could be detected with survey meters with occasional maxima of two to four times background. Close to zero, I would say out to maybe 1,000 meters or more, the radioactive silver and stainless steel fillings in human teeth from neutron bombardment could be identified. The radioactive sulfur in the sulfur insulators could be identified. Many of the surgical instruments in the prison infirmary and in the medical school were weakly radioactive. I brought some of these home and was asked the other day whether we could backtrack and figure what the neutron bombardment was. All I can say is that in one large brass coin about 3 inches in diameter there is a little cobalt contamination but its radioactivity is so near the background that our boys are unwilling to make a calculation from it. It came from a corner of the medical school building, apparently in a professor's office. But it is unfortunate that we can't do more in this respect.

AUXIER: This has been overcome now, Dr. Warren, by other study groups.

WARREN: Good.

AUXIER: In this case they are using cobalt, again. But it is taken from a known location in a building which still exists; in other words, we can still get cobalt identified and measured quantitatively. The difficulty comes in the calibration because this is thermal neutron-produced cobalt and the thermal neutron distribution is such a sensitive function of the environment. But, by getting enough samples from deep enough in concrete, two different study groups in Japan have been able to get enough data to at least assure themselves that they are in "ball park" agreement with the latest neutron distributions that we have. So it has been a useful verification of our backdoor approach to the whole problem.

WARREN: Good. Are you now in a position to give neutron contours of any sort?

AUXIER: Yes, sir. We can now and we have given them to ABCC. ABCC has, as a matter of fact, already processed them through the computer and they have new dose assignments for all but a few of the people for whom there were shielding histories. There are a few cases that are listed as impossible at the moment or that must be deferred. Some of these happen to be very interesting cases.

WHITE: I want to say something briefly about a problem that still is very fascinating. Consider the overall survival curve which you see dotted in Figure 21. The figure was drawn from the old Joint Commission data for Hiroshima, giving percent survival against range in miles (Reference 59). One looks at the photographs of Hiroshima and listens to the talk about the area of complete destruction; physically this extended out to 2 or 3 psi, a range of around 1.3 to 1.5 miles. However, the exact overpressures it takes to lay light Japanese houses down is something I am not too familiar with, but probably 3 psi is a reasonable number.

Biological survival at about 1.3 and 1.5 miles was over 90 percent as you see from Figure 21. To depress biological survival on the average to very low numbers, like 10 percent, one must move into around a tenth or two-tenths of a mile from the hypocenter. In attempting to understand what medical loads might be and how to estimate the need for caring for folks after a disaster like this, one

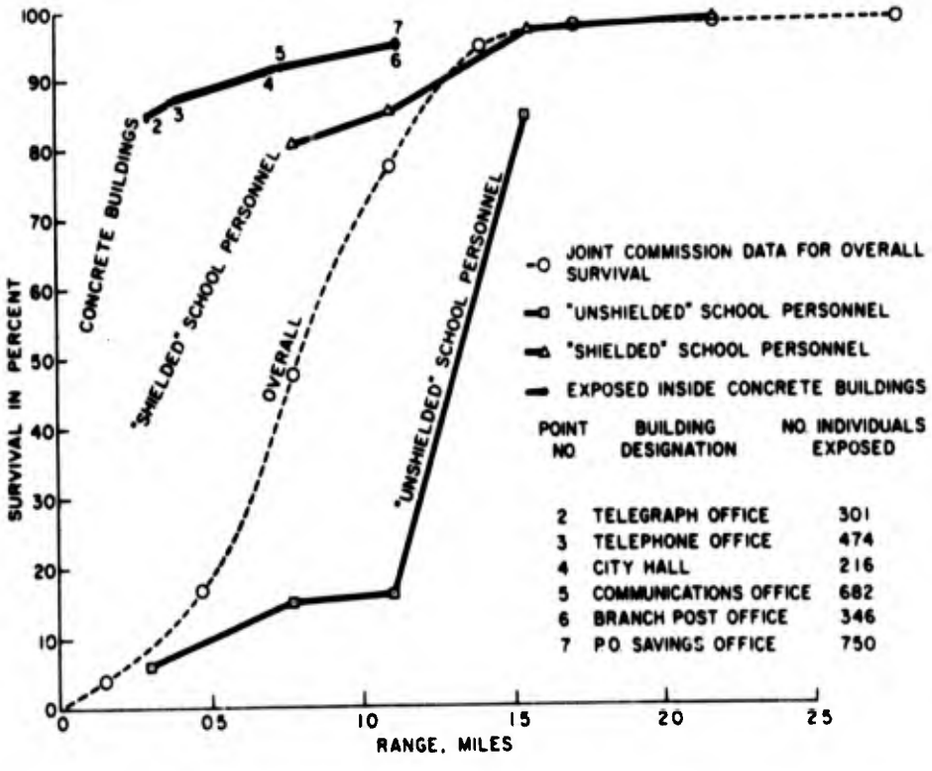


Figure 21. Percentage of survivors as a function of range from ground zero (Hiroshima). After White (Reference 4); data from Oughterson, et al. (Reference 59).

has got to distinguish very carefully the difference between physical and biological destruction.

Figure 21 stimulates talk about this in a definitive way. The relative contribution the destruction of or damage to buildings makes to protection or to survival, which was mentioned earlier, is something that is very much a matter of where people are and what kind of construction it is. I bring this up because there have been attempts to tie the injury incidence to the kind of structures in which individuals were exposed both in Hiroshima and Nagasaki by the Dikewood group (Reference 60) currently, and by them and others in the Texas City disaster.

Let me just leave this thought with you: Do not in your mind mix up physical and biological destruction, because they are certainly two different things.

WARREN: Does this include the summated effects, radiation as well?

WHITE: Yes. That overall survival curve is everything, Dr. Warren.

ACUTE RADIATION EFFECTS

BRUES: I thought in opening the session this afternoon I might show an illustration that is rather a classic. Figure 22 is from a Japanese newspaper. Perhaps someone can translate this headline announcing the...

FREMONT-SMITH: The atomic bomb.

BRUES: One notices in the headline that the typesetter has devised a special character for the B-29, which probably was used before in connection with raids. You will note it at the top of the second line from the right. Dr. Matsumoto, would you like to tell us what it is?

MATSUMOTO: The headline says that a new type of bomb hit Hiroshima. Small numbers of B-29's, I suppose, attacked with a large number of casualties. A very detailed survey of the survivors is being conducted.

BRUES: Thank you. That, I take it, is still the case!

BUSTAD: What was the date on that?

MATSUMOTO: I think it was on the top. 1945, August 8.

BUSTAD: That's two days later.

LIFTON: But it is not a Hiroshima paper; it's from Tokyo.

BRUES: Therefore the study started very soon after the occasion.

Dr. Warren, you asked a question concerning the relative numbers of casualties. I think this is something we need not get into, and I would like to explain, in a few words, why.

Figure 22. Front page of Tokyo newspaper published 8 August 1945.

The acute radiation effects are based mainly on the loss of and the necessity for regeneration of certain tissues which normally keep themselves going by rapid proliferation. The cells lining the duodenum and small intestine are worn off very rapidly and have to be replaced. The deficiency of lining cells after a couple of days has serious consequences, such as loss of fluid. Cell loss also occurs in the lymphatic system and as a result of this, probably, bacteria which reach the lymph nodes are passed more readily through this natural filtration system and reach the blood stream. The bone marrow, another proliferating tissue, is similarly damaged, and before recovery occurs there is a bleeding tendency from loss of platelets, and a reduction in resistance to circulating microorganisms as well.

All these effects are going to act in concert with blood loss from direct injury, with fluid loss from burns, with increased bacterial contamination of the environment and portals of bacterial invasion. Therefore it is not always possible to say whether in a given case the radiation exposure is an accessory factor for a person who has been injured or burned or the reverse. A great many of the casualties may be attributed to a combination of these things. Single causes of death, of course, are terms in which the laity likes to think.

In a case beyond the area of severe ionizing radiation exposure, say 2,000 miles away, an individual might receive a thermal burn without receiving injuries from missiles or falling objects or a significant dose of radiation. But only in such instances could we characterize the casualty in a single category of cause.

WARREN: In other words, you've got a series of concentric circles. The furthest out is probably the infrared burn effect and then the ionizing radiation plus the burns and then inside that the blast, thermal effects and ionizing radiation plus missiles and other secondary problesm. This is one of the troubles in trying to distinguish the cause of the mortality and morbidity by determining these radii of effects, and I don't know if anybody here has gotten the trend over there.

AUXIER: That's a good general summary, but on an individual basis there are a lot of exceptions. We hope that on the basis of these exceptions we can begin to separate some of the effects of combined injuries.

A particular instance is the case of the people close in, in heavy structures like Fukuya Department Store in Hiroshima, in which they were well shielded from blast except for, in a few cases, translational effects. Due to the geometry of the building there were a lot of people who, in subsequent interviews, indicated that they received no physical trauma and whose medical histories show very little other than radiation.

There were two department stores, Fukuya and Tenmaya, right next door to each other. So this gives you sort of a fiduciary in close. You have people whose major trauma was that associated with ionizing radiation and, of course, the situation you mentioned, in which, at the greatest distances, people had primary radiation burns and no other trauma.

UPTON: How accurately do you think you can estimate the radiation doses in the cases where good shielding histories are available?

AUXIER: That, too, is on an individual basis, but I'll make a few blanket statements first. For Nagasaki, for all except the heavily shielded cases, we will have the overall dose—pardon the expression, but the "air dose," the dose that a small sample of tissue exposed in the open received—to an accuracy of not less than ± 15 percent for one standard deviation. This is rather phenomenal, in our opinion, after all these years. In Hiroshima, at the moment, for the same conditions the accuracy is reduced to about ± 30 percent because we still have a built-in error of ± 25 percent for the yield. We can talk a little bit more about that later.

For unusual cases, particularly those in the heavily shielded configurations, we have to assume that the accuracy is less than this because each one has to be done on an individual basis. We have to make a detailed analysis for each one.

This requires about one man-week of effort for each one, but this isn't so bad because there are only between 100 and 200 people in this category that are of great medical significance.

UPTON: What do you mean by very heavy shielding?

AUXIER: Dr. Hachiya (Reference 61) put his finger on it very clearly in the early days, but it is only lately that we could check his calculations physically. Hachiya discovered, as early as 1945,

that the formed elements in the blood of the people exposed in the Fukuya Department Store were depressed by the same amount, on the average, as those at 1,200 meters from the hypocenter in Japanese houses. We have since gotten the shielding factors for Japanese houses and we have the air-dose curves and we can estimate, on the basis of Dr. Hachiya's data, a shielding factor for the building. If I remember correctly, it is 100 or so, on the average, for the people in the department store. These people were the closest to ground zero, and they were among the most heavily shielded. A calculation of a shielding factor of 2 orders of magnitude can probably be made to an accuracy of ± 25 percent if we are very lucky. We have been lucky on some of the others.

BRUES: I would like to add that Dr. Hachiya's book, Hiroshima Diary (Reference 61), is, in my opinion, the best literature that has been written on the subject. It is edited from a diary which he kept. He is a very sensitive and observing man, and anyone who wishes a fuller understanding of the situation should read it. It was translated and edited by Dr. Warner Wells, who, when he was with ABCC, spent a great deal of time with Hachiya. Dr. Hachiya was in charge of a large hospital and was himself injured. However, he recovered and has survived.

TAYLOR: Does anybody happen to know the population density at ground zero, people per square mile?

WARREN: It was higher than it was at Nagasaki because of the running track, the prison, and the other things.

TAYLOR: There is no simple number that would be available?

WARREN: No, and they had no census, so there isn't a good figure.

AUXIER: We actually have some very detailed photos from both cities now. It took a long time to dig these out of the archives, as you may know, and on the basis of these photos, we "reconstructed" blocks of the city on paper. We did a "census" for the areas where there were a significant number of survivors. One of the problems in getting the exact number of people involved, or at risk, in Hiroshima was the fact that everybody around ground zero was killed, of course, and there was nobody to interview to find out how many people were there. That is the very part of the town in which

the greatest uncertainty exists about how many of the people had arrived at work and how many did not. So I don't think you will ever find a precise answer as to the total number of people killed, but the tables that would be most readily accessible to you are at Dikewood Corporation.* These would give the best estimates available today.

TAYLOR: I was curious about a very rough number because Sam's [White] graph of the mortality percentages, as a function of distance, as I remember, gave the 90 percent distance as approximately one-tenth of a mile.

WHITE: That's right.

TAYLOR: If that is so, then, at least to me, it is surprising that the vast majority of the deaths in Hiroshima were in a region where the survivial probability was a lot higher than 10 percent.

WHITE: And the average of the 50 percent survival was eight-tenths of a mile. If you believe the old Glasstone figures (Reference 62) for Hiroshima and Nagasaki, they are around 35,000 and 65,000 per square mile, respectively. However, I think that those numbers are not right, because there was some confusion in the literature as to whether the areas used were total areas or populated areas. There were regions in the cities containing a lot of people and others where there weren't any. In any event, these are the numbers. I compared them once with the population densities for 30 American cities (Reference 63).

TAYLOR: Isn't the highest concentration in Manhattan under 35,000?

WHITE: No. Manhattan gets up very high. The average for New York City is about 25,000/sq mi, that is, the average if you put the five boroughs together. It gets up to 75,000/sq mi in Manhattan (Reference 64). Actually the figures for Manhattan were 87,397 for 1950 (Reference 64) and 76,163 for 1960 (Reference 65). Then one must ask, are you really talking about population densities day or night? I just don't know, you see. And anything done on a free-field

*Dikewood Corporation, 1009 Bradbury Drive, South East, Albuquerque, New Mexico. Contact L. Wayne Davis.

basis without defining how many people are outdoors or indoors produces not just a little, but a great deal of error.

WARREN: As an aside more or less, but of some interest, Dr. Tsuzuki,* told me that after the Japanese had gone into Manchuria, it was official policy that the duty of every woman of child-bearing age was to have a child every year. If the woman of the house couldn't produce, the brothers tried and the local gendarme tried, failing which she was put in a factory on reduced rations and another individual was brought in to take her place. And on this basis they estimated they could put one million men in the field 15 years after the Manchukuo episode, and that they could afford to lose, each year, one million men if they could produce uniforms, ammunition, food for them, and transport. So that the density, you see, could have been almost anything. But I don't think he was pulling my leg, either. Does anybody know anything about this?

LIFTON: I would be skeptical right now. It might have been one policy that was put forth at some time, but I doubt whether it was, in all the lurid detail you describe, acted upon.

WARREN: I am not so sure. This was a pretty brutal thing in the beginning, contrived in every way, and their culture was suitable for this kind of stricture.

ABRAHAMSON: With respect to Dr. Taylor's question, couldn't you get an estimate of population from an equivalent sized city of an equivalent area at that time?

FREMONT-SMITH: You mean a Japanese city?

ABRAHAMSON: Yes, a Japanese figure.

FOUNTAIN: Would that give you any better figures than the 30,000 to 65,000 range? I doubt that it would.

WHITE: There are a great many differences in this regard, I think, between Hiroshima and Nagasaki anyway. I think that if you want to think about this, you must do the population density for the specific population at risk. This is why American cities ought to be analyzed on an individual basis, and to some extent this is being done now.

*Masao Tsuzuki, late professor of surgery at Tokyo University.

SPEAR: In some of the early defense analyses we found it not uncommon for a metropolitan center to have a disparity of eight to one in the daytime-nighttime density, particularly in the central areas. Some were even higher. As I recall, downtown Manhattan was considerably higher.

WARREN: I think, too, that the working population was differently distributed than we might think, because so many dwellings had small metal parts in piles with files and other things spread around in the ashes of the remains of the buildings. You can see this all through Nagasaki in the Yurakami Valley. I don't recall it being so common in Hiroshima. But this would mean that they took advantage of the local dwellers, without having to move in a big population from outside.

Everything there, as we explore this, makes the difficulty a little worse and the plus or minus a little larger.

Let me get down to radiation now a little more. Does anybody have a yen to suggest any chronic effects from neutrons as one element? You heard earlier that they have a higher ionizing potentiality. They go around the corners by scattering, more so than gamma rays.

FRANK: For those of us who are not familiar with this field, would it be worthwhile to lay out what kind was caused by radiation and what was caused by neutrons?

AUXIER: Figure 23 is a typical air-dose distribution for gamma rays; the ordinate is the gamma-ray dose, times the square of the distance, and the abscissa is the distance. You see an exponential decrease of dose with slant distance. By using the word "slant," we indicate the separation of the point of detonation and the target, as opposed to the horizontal distance, because most nuclear devices, including the Hiroshima and Nagasaki weapons, were exploded well above the ground.

Now I want to talk just a little about why this curve looks like this (Figure 24). If you could take a point source and put it in an infinite vacuum, infinite in dimension, and if you measured the dose as a function of the separation from some appropriate detector, you would get the horizontal line labeled A. In other words, correcting for the square of the distance away, the dose would be invariant.

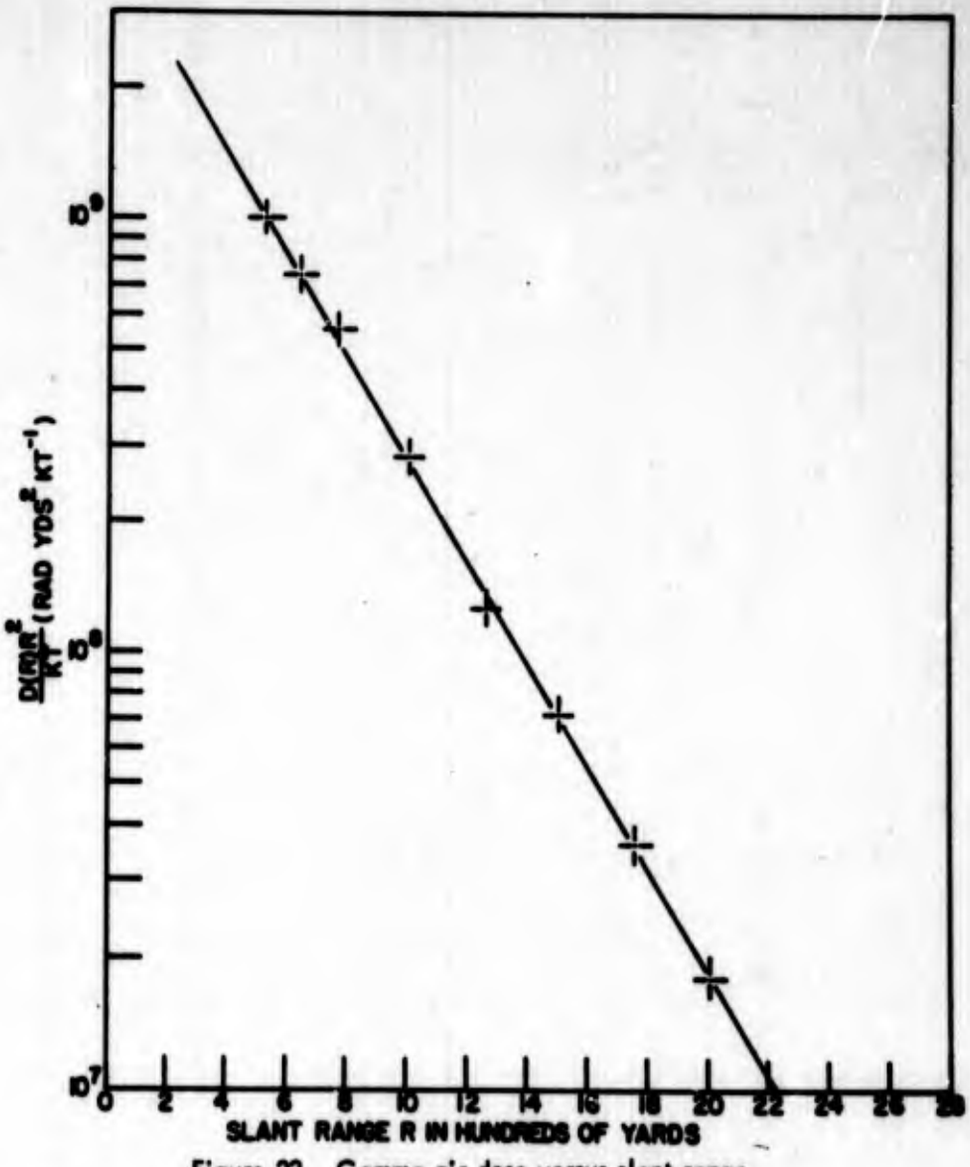


Figure 23. Gamma air dose versus slant range.

If, on the other hand, a detector is put at some fixed distance from the source with a narrow attenuator of some kind—it could be tissue, air, or whatnot—and plot the dose under the same terms as before, you get the curve labeled B. The ordinate here is logarithmic and you see the typical exponential attenuation with which I believe you are all familiar. It doesn't matter whether the radiation is neutrons or gamma rays. I started with gamma rays so that we could start with a single curve.

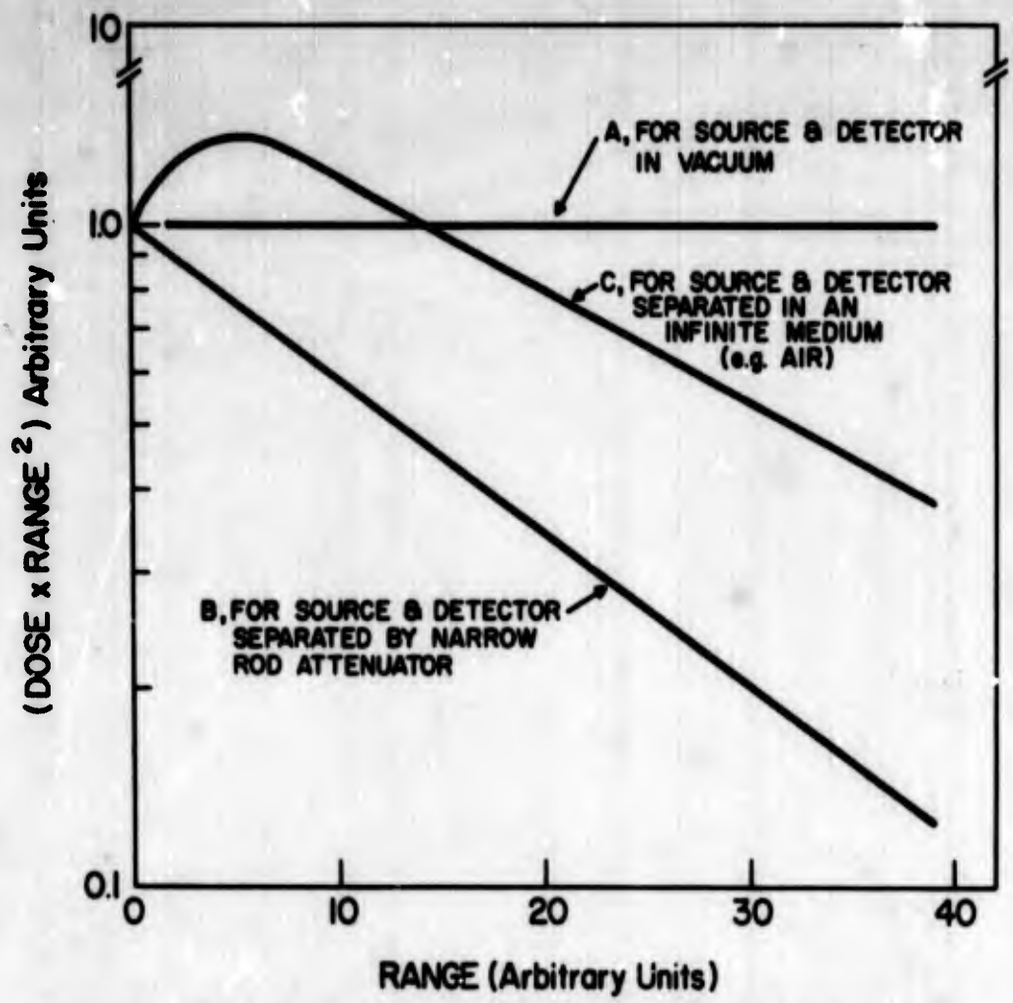


Figure 24. Effect of geometry on the dose as a function of separation range.

If this attenuator in between were an infinite medium of air or if it were a broad slab of any material, two changes are apparent. Radiation that starts off in a direction other than toward the detector would not, either in a vacuum or under the narrow beam (narrow attenuator) conditions, ever reach the detector. However, when the source is located in air or adjacent to a broad slab, radiation can conceivably be scattered so as to reach the detector, although it started in a different direction. This scatter enhances the radiation and results in a "buildup" which can amount to a factor of 4 or 5 and, as a consequence, curve C is now elevated relative to B at all points. The curve shows that there is an initial buildup of radiation in an infinite medium of air. This is not a negligible effect, but a significant increase in the dose at any given distance. This initial peak in the dose distribution occurs at a distance of ten's of meters to a few hundred meters as opposed to the big distances of interest in, for example, Hiroshima and Nagasaki.

Once this initial buildup is established, however, one is still not through with the effects of further scatter. The slope of curve C is always less than curve B. The apparent mean free path, or "relaxation length," is that distance on this type curve in which the radiation dose decreases by a factor of $1/\lambda$. If it were a mean free path, we would normally call it lambda, but in this case we normally call it L, to denote that distance observed as opposed to what would be calculated without consideration of buildup.

You might think that at this point we understand radiation propagation. However, the radiation picture is just about as muddled as the others, but because of the romance associated with radiation we have been able to get more attention on it and have done somewhat more with it, although not nearly enough.

Anyway, we are still not through. It turns out that if the source is lowered toward the ground, and a detector is placed near the ground and measurements are again made as a function of distance, one finds that close to the source, the radiation field is enhanced relative to the same separation without the ground nearby. The effect of the ground, or the air-ground interface effect, is analogous to a reflector. The radiation "albedo" is the ratio of the total radiation to the incident unscattered radiation. If the source is high in the air and the dosimeters are located close to ground zero, we find that curve C must be increased by about a factor of 2 to account for the ground scatter. But this factor decreases with increasing distance from the source, so that at some point the ground scatter buildup approaches unity, that is, the effect of the ground is negligible at this distance. However, as the dosimeter or detector is moved still further, that is, to greater distances, the radiation scattered near the source can get to the detector only by penetrating the ground for some distance; that is, it is attenuated because of the small grazing angle with the surface.

As the radiation scattered just under the surface has a small probability of getting out of the ground in the direction of the detector, the ground acts as a greater attenuator. Thus at distances of many hundreds of meters, the radiation field observed is lower than would be predicted by curve C; this factor also approaches 2.

This elementary discussion is intended to give an idea of why we were worried about such things as the exact height of the burst, the exact location of the hypocenter, etc., at Hiroshima and Nagasaki, and, of course, why we were interested in the yield of those devices. Assuming we know all about the curves given here, we still have to normalize them to the total source strength, and the total source strength is, of course, related to the kiloton yield.

We were not, from the radiation point of view, interested in the kilotonnage, per se. We were interested in the source term, and if there had been some other way, such as the use of cobalt activation in substances on the ground in the two cities, to have gotten the source term without the kiloton yield, we would have been pleased. However, it turns out that that seemed rather hopeless until recently. Thus, we had to take the long way around and determine the yield of the weapon and then the number of neutrons, for example, that got out of the weapon for each fission that took place in it. Then we had to know something about the energy spectra of the radiations so we could determine the dose-distribution curve.

BRUES: The yield of the weapon is expressed in kilotons, equivalent to such an amount of TNT. Is this then in the dimension of energy?

AUXIER: Yes. We're talking about 10^{12} cal/kiloton or 1.42×10^{23} fissions/kiloton.

There is one other thing we should say about radiation, though, as it is transported through the air. I talked about the fact that it is scattered in air, and that on the basis of the slope of curve C the attenuation is considerable after a distance of several relaxation lengths; the dose is down several orders of magnitude by attenuation alone. At distances of 1,000 meters or so from the source, only 5 percent of the radiation that arrives at that point has arrived there unscattered; that is, 95 percent of the radiation has been scattered and has arrived at the point of measurement from all angles of incidence relative to the direction to the source. So another very important parameter that we had to explore was the angular distribution of the radiation, that is, the direction of incidence of the radiation when it arrived at the target. This required that the detectors be put in a collimating device which could be pointed in many different directions. The dose distribution as a function of the angle of incidence will be shown a little later.

Another consideration is the energy distribution of the radiation. The interaction probabilities per atom, or the cross sections of the various radiation interactions, depend on the radiation energy in a very convenient way. In general the cross sections for these scattering reactions decrease with increasing energy.

UPTON: John, I think some people in the room are having a little trouble following the nomenclature. What do you mean by "cross section," for instance?

AUXIER: The cross section is simply the apparent area of the target for the projectile. On the microscopic scale we talk about the apparent area in square centimeters or, a handy unit to use is the barn, which is 10^{-24} sq cm. Thus the cross section is nothing more or less than an apparent size of the target as viewed by the projectile; it gives the probability of an interaction.

BRUES: The more effective a target is in slowing something down, the larger you say its cross section is?

AUXIER: Yes, the more interactions per unit mass, the larger the cross section.

WARREN: This is essentially the nucleus that is represented?

AUXIER: Yes. In this case we're talking about the nuclear cross sections. In the case of a photon, that is, a gamma ray or an x ray, the target is the electron complement of the atom, but again, the higher the photon energy, in general, the smaller the apparent target or the smaller the cross section. Thus once the photon or neutron undergoes an interaction, it has lost some energy in that interaction, so now it has less energy. Consequently its probability of undergoing further reactions is increased. This continues until the photon or neutron is annihilated or captured. The process is somewhat analogous to decay, namely, if you have a parent nucleus that has a longer half-life then does the daughter, the two soon come into equilibrium. In this case, the higher energies control the whole scatter-capture sequence due to the increased probability at the lower energies. With the exception that there are resonances, the cross sections decrease monotonically with increasing energy. At a distance of 500 or 600 meters from the source, these cross sections result in energy spectra which change very slowly with increasing distance, that is, energy equilibrium obtains.

I can demonstrate this for the neutrons because we were able to measure the neutron spectrum long before we could measure the gamma-ray spectrum. Figure 25 shows an approximation of a typical neutron spectrum. The lowest curve is the "dose curve." Each of the other lines represents a part of the energy spectrum as shown by the key; the lower curves are for the neutrons of highest energy, that is, those of greater than 2-1/2 Mev, the next higher curve for those above 1-1/2 Mev, and so forth.

These curves, as you can see, are very nearly parallel and, consequently, the spectrum must not be changing with distance over

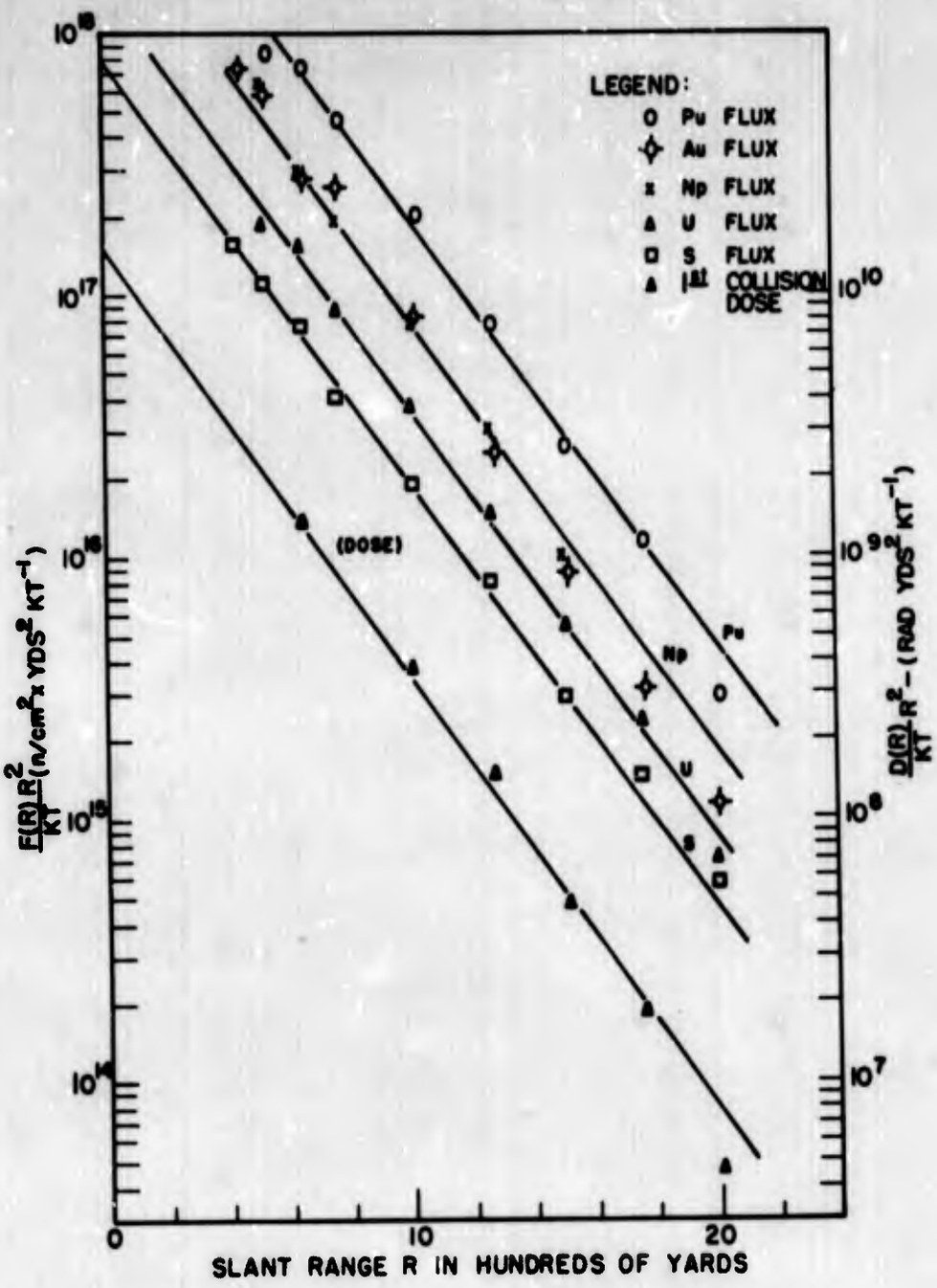


Figure 25. Neutron air dose and flux versus slant range.

quite a range of distance. Equilibrium in the spectrum makes the interpretation of the dose curves much simpler. In addition, it makes the shielding problems solvable. If a different spectrum obtained for every different distance or if a different angular distribution obtained for every distance, calculating the doses in the

Japanese houses would have been extraordinarily difficult.

Everybody has talked about flattened Japanese houses. I thought that I should show a picture of one that was still standing (Figure 26). This one was built in Nevada in 1957 for our initial studies of shielding by Japanese houses; you can see the typical construction, imported tile, the mud-oyster-shell-bamboo construction on a wood frame.

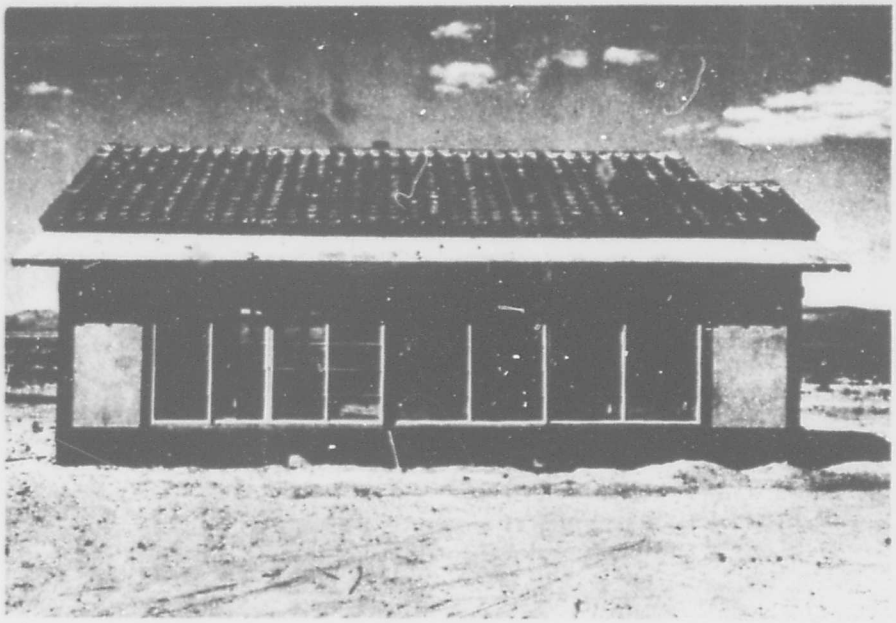


Figure 26. Typical Japanese house.

In Figure 27 you see an array of Japanese houses. These were used in the 1958 Hardtack experiments (References 35 and 66). We found that the typical Japanese construction was too expensive to permit our doing a good experiment with them, and they were very friable, about 2-psi overpressure and they were destroyed. So experiments were conducted in the laboratory using all of the domestic materials that were available in sheet form to determine the relative neutron and gamma-ray attenuation factors. We found that cement-asbestos board, a commercial product that goes up in sheets, had precisely the same shielding characteristics as the Japanese materials and, more fortuitously still, the material could be used for both roof and walls; only slightly different thicknesses were required.

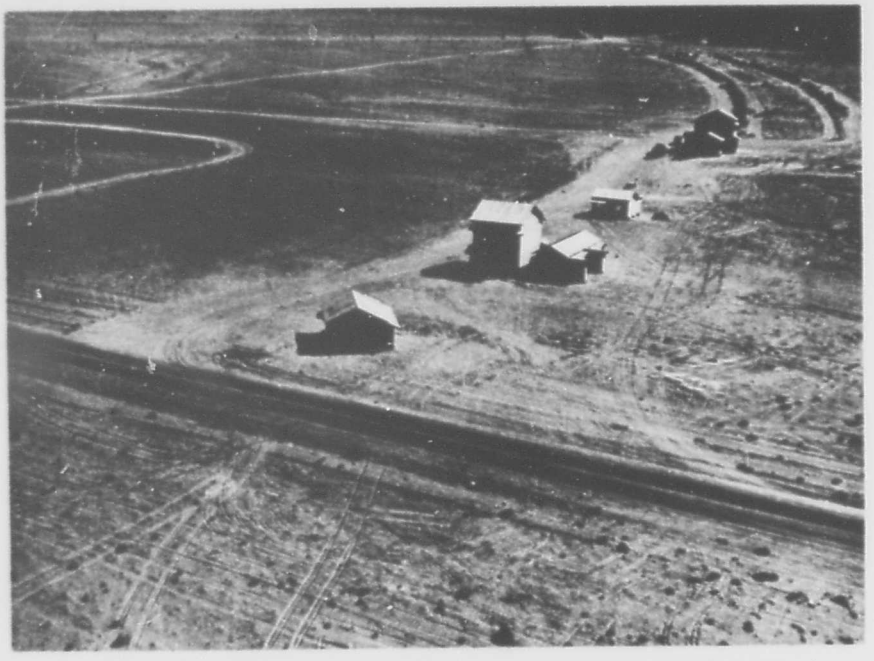
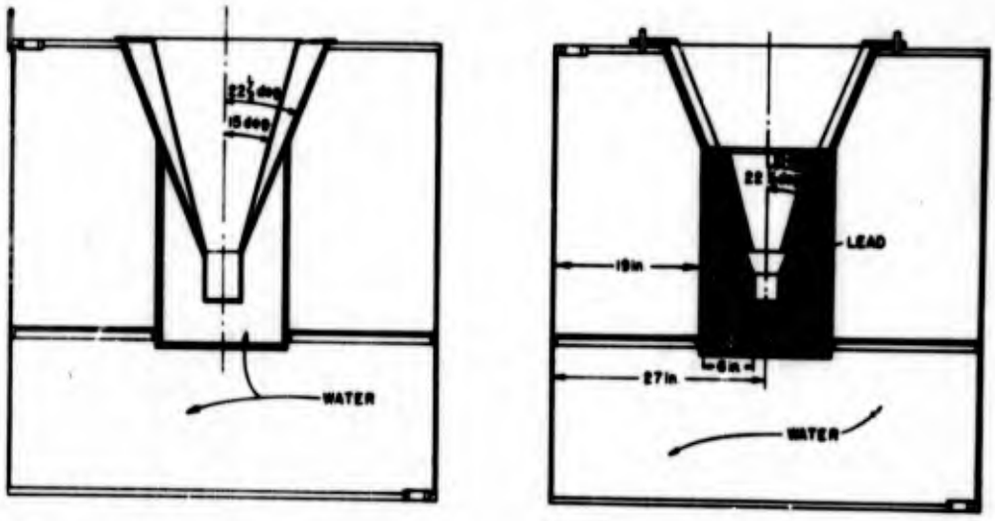


Figure 27. Field array of radiation analogs of Japanese houses.

Those rows of "cans" in the background are the collimators used for studying the angular distribution of the radiation.

Figure 28 is a cutaway view of the collimator in which the detectors are inserted. The collimator assemblies are "pointed" in a variety of directions to determine the quantity of radiation coming in from each direction. The collimator for gamma rays is just like the one for neutrons, in that they both have the same outer water tank, but a lead insert μinches thick is placed in the center of the gamma-ray collimator. If only the lead were used for gamma rays, an exceedingly great thickness would be required because extraneous gamma rays are generated by neutron scattering in the lead. These are "hard" or energetic gamma rays and are difficult to attenuate. Consequently, the water tank was used to protect the lead attenuator in order to make a good collimator.

Figure 29 is the result of the angular distribution measurements made inside the collimators. This graph is for neutrons; notice that these values were determined at a distance of 1,000 yards.



NEUTRON COLLIMATOR GAMMA COLLIMATOR

Figure 28. Cutaway views of collimators.

There is no appreciable difference in the distribution for ranges from about 750 to 2,000 meters. The angle of the cones plotted here represents the size of the particular opening of the collimator; this was about as small as was feasible in 1958. The value of dose from a zero direction is that for the collimator "looking" right at the bomb and is shown as 100 percent; at 30 degrees you see that the dose is reduced to about 75 percent with further decrease with increasing angle. But the solid angle subtended is of the order of 5 percent of the total solid angle. For any angle other than zero, the total solid angle is that obtained by rotating the cone about the zero direction by 360 degrees. Consequently, at a "polar" angle, shown on the graph, at which the dose is about half the zero direction value, the integrated value is greater than that from the zero direction cone. The total radiation coming in at angles that are greater than 30 degrees from the target is greater than half the total. In general, a zone of about 45 to 50 degrees to all sides about the zero direction contains half of the radiation; the other half comes from angles greater than 45 to 50 degrees.

BRILL: Would the head-on radiations be higher energy neutrons than those coming from the sides?

AUXIER: No. Even with this narrow collimation, a large fraction of the radiation has been scattered at least once at this distance.

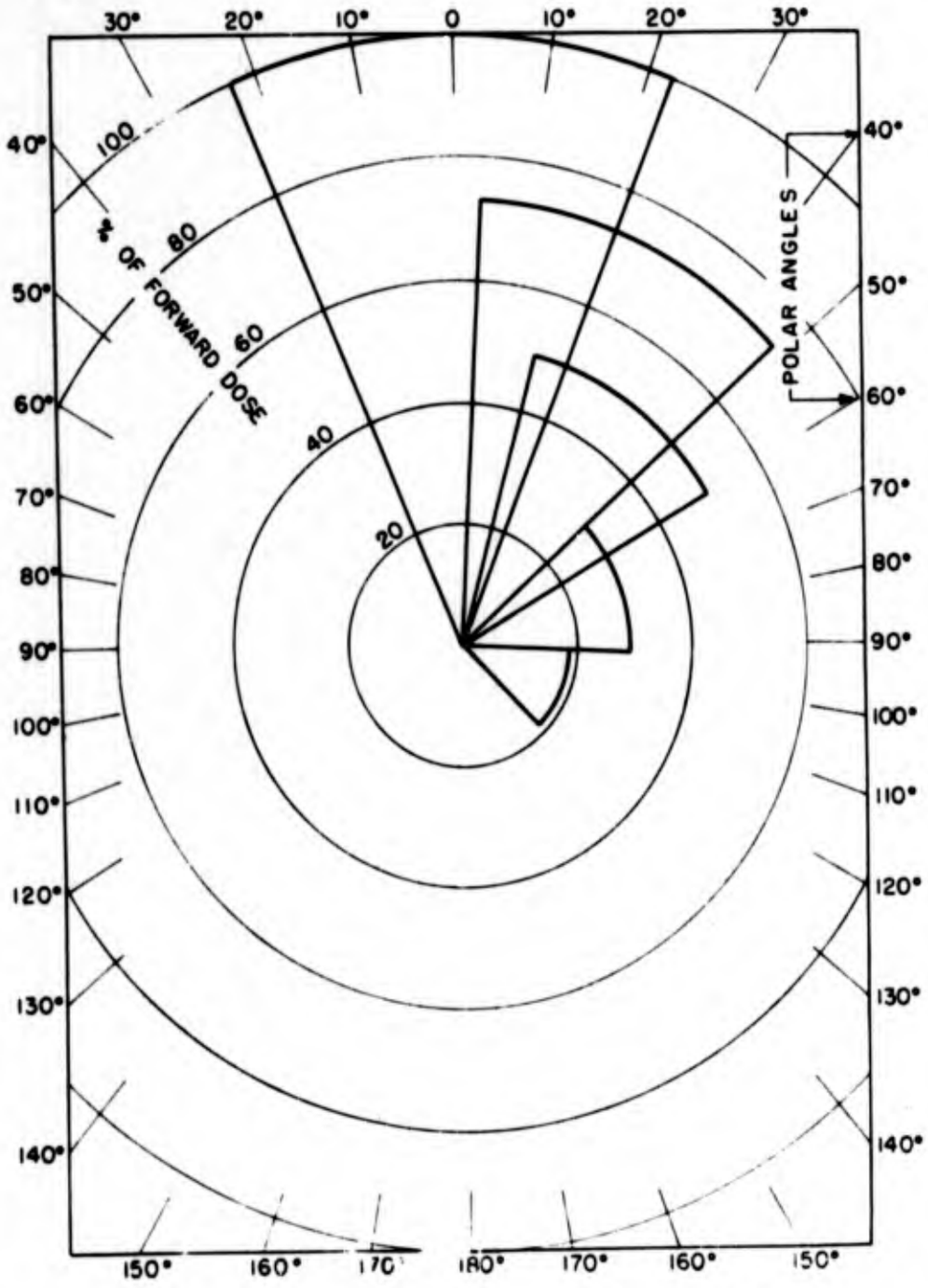


Figure 29. Angular distribution of neutron dose at 1,000 yards.

Figure 30 is a similar plot for the gamma rays. It shows more peaking in the forward direction. There are fewer of the total gamma rays scattered at any given distance. One factor is that the relaxation length, i. e., the attenuation distance, is greater. Another factor is that the variation in cross section with the energy is greater than for neutrons, that is, once they are degraded in energy by scattering, the probability of their being scattered again is increased greatly. So for the equilibrium distribution shown here the curve is more peaked in the forward direction. More of the dose comes in from the forward direction than in the case of neutron.

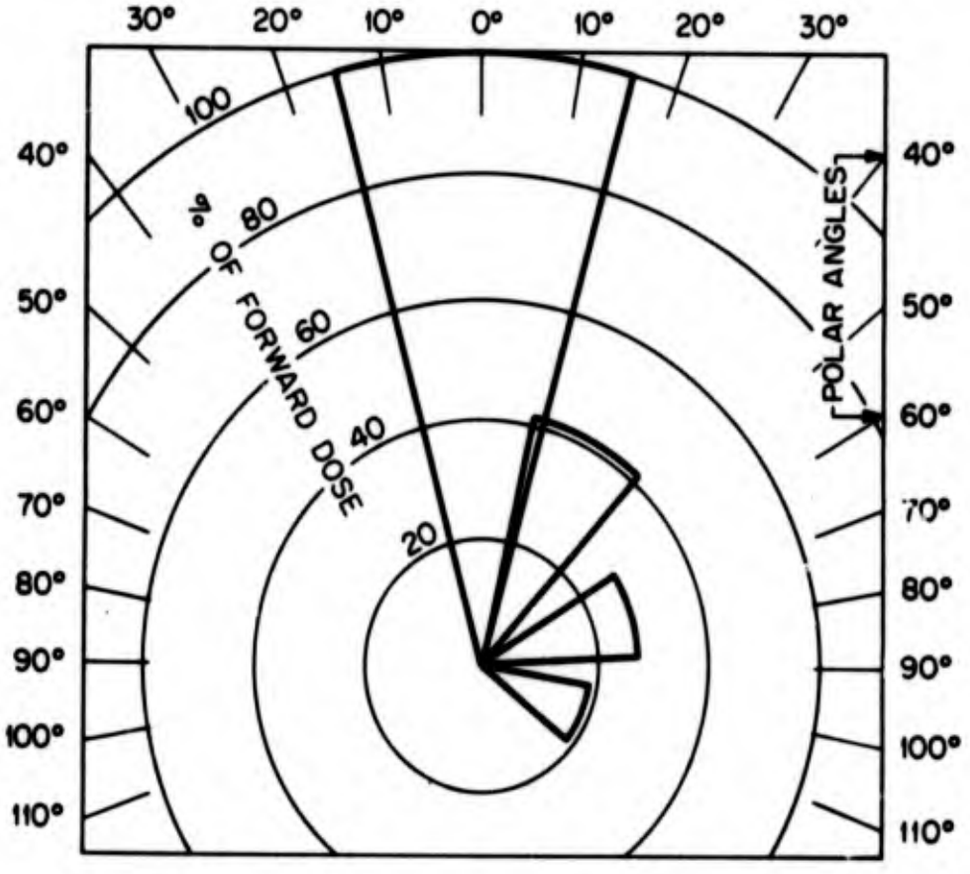


Figure 30. Angular distribution of gamma-ray dose at 1,000 yards from a typical nuclear weapon of normal size.

I must get in a plug for the field experiments which are still going on in Nevada. This is the 1,527-foot BREN tower at the Nevada Test

Site (NTS) (Figure 31). In 1962 we put a small bare reactor on this, the Health Physics Research Reactor. The reactor was put on the tower to provide a facsimile of a "slowed" bomb so that it could be studied with sensitive laboratory instruments. We wanted something which would permit a good look at the gamma ray and neutron spectra. The reactor was mounted in the aluminum car used at heights of from 27 to 1,500 feet above the ground; this permitted an analysis of the source, or "bomb," on the dose distribution at the surface. A lot of the early weapons data had shown anomolous results, or apparently anomolous results. There were available data on air-dose distribution, but it appeared to be spurious until after the tower experiments when definitive information on the effect of the air-ground interface became available. After these experiments, it was possible to make corrections for height, and much of the early data proved to be valuable.

At present we have on the same tower in Nevada a small 14-Mev source. It's small in physical dimensions, but it is the most intense 14-Mev neutron source in the world. With a new target, it provides an initi. yield of about 3×10^{13} neutrons/sec with a target half-life of 90 to 120 minutes. This is attained by having a half ampere of deuterons impinge on a big tritium target. The target, of course, under this bombardment doesn't last but a few hours, but provides an average 10^{13} neutrons/sec for a 4-hour period. This is about the minimum intensity needed for experimental work of this nature. The major advantage of using the 14-Mev source is that it provides fiduciary to the calculations. Of course, with fusion weapons there is a very important reason for being interested in the 14-Mev component.

FRANK: What is the important reason?

AUXIER: Because in the fusion process you get 14-Mev neutrons; with a pure fusion weapon, if one were available, you would have a 14-Mev source such as this. There are several things you can do with this if you also have data for a fission spectrum. You can "mix" the two spectra in various ways; for example, to the 14-Mev spectrum, a fission component could be added to the fusion component to get the combination for any conceivable weapon, the spectrum for any conceivable weapon.

FRANK: The right mixture? Is that the one that does the most damage?

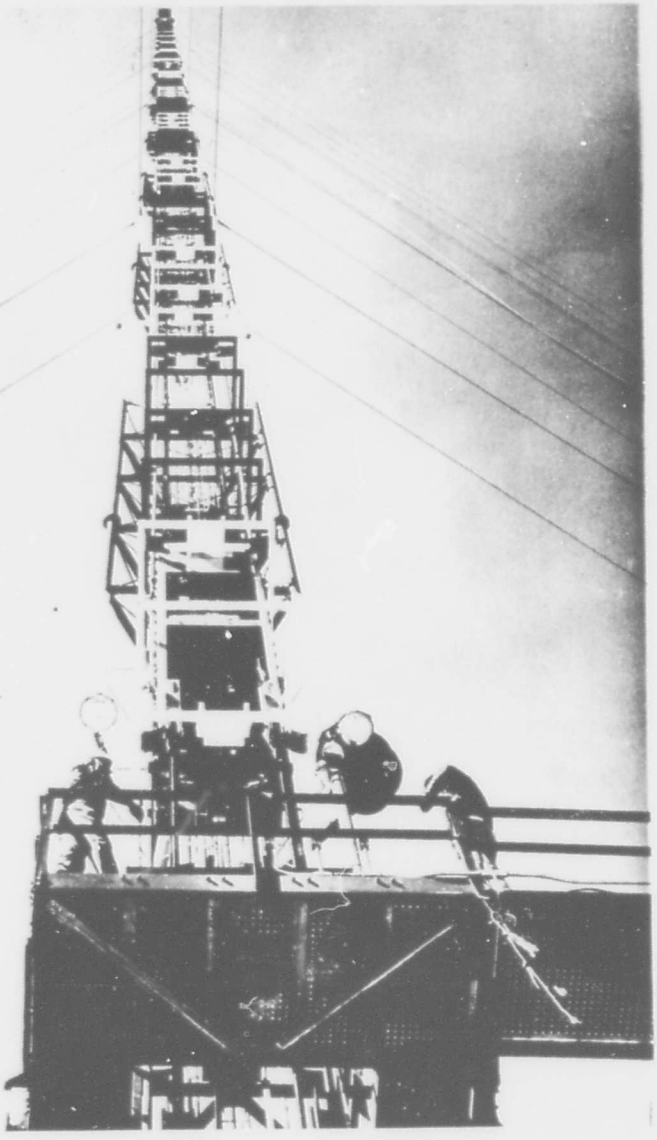


Figure 31. 1,527-foot BREN tower, Nevada Test Site.

AUXIER: If you had a weapon based on pure fusion, hypothetical in this case, and a pure fission device, you would have the two extremes. (Actually a highly moderated fission device such as was the case in Hiroshima would provide the softest neutron spectrum.) Any real weapon other than a pure fission one, would, in general, provide a spectrum which would show a mixture of the two spectra. The damage per unit dose would not vary greatly in either case, but the "harder" or more energetic spectra have longer relaxation lengths, that is, they cause damage at greater distances for a given yield.

This is the Health Physics Research Reactor (HPRR) (Figure 32). This is the core which is 22 cm in length and 20 cm in diameter. There is an aluminum screen around it, but the core you can see, vaguely, inside. It is highly enriched uranium metal alloyed with 10 percent by weight molybdenum for metallurgical reasons. The spectrum of neutrons that are emitted during operation closely approximates the spectrum from pure fission. For the purposes of this discussion, they are the same.

Figure 33 is the neutron spectrum for the HPRR, or for fission. The abscissa indicates the energy scale and the ordinate indicates the number density of neutrons for each energy increment. But one particular weapon, that used at Hiroshima, was different in many ways from any other weapon that has ever been built. Not only was a replica of the Hiroshima weapon never tested before or after it was detonated in Japan, but the particular configuration and design of the weapon resulted in an emitted neutron spectrum which was different than any weapon that we have studied. This curve indicates, for example, that at 3 Mev, the number of neutrons in the HPRR spectrum is 100 times greater than for the Hiroshima bomb; this presented many difficulties in interpreting the data for Hiroshima (Reference 67).

LIFTON: Is that called Ichiban?

AUXIER: No. Ichiban is a Japanese word for A-No. 1 or the foremost. We needed a code name for the project, but this one has haunted us since the beginning. When we commenced working with the Atomic Bomb Casualty Commission (ABCC), they thought that this would have the wrong implication to the Japanese community. Actually, I think most Japanese liked the connotation of importance placed on the project.

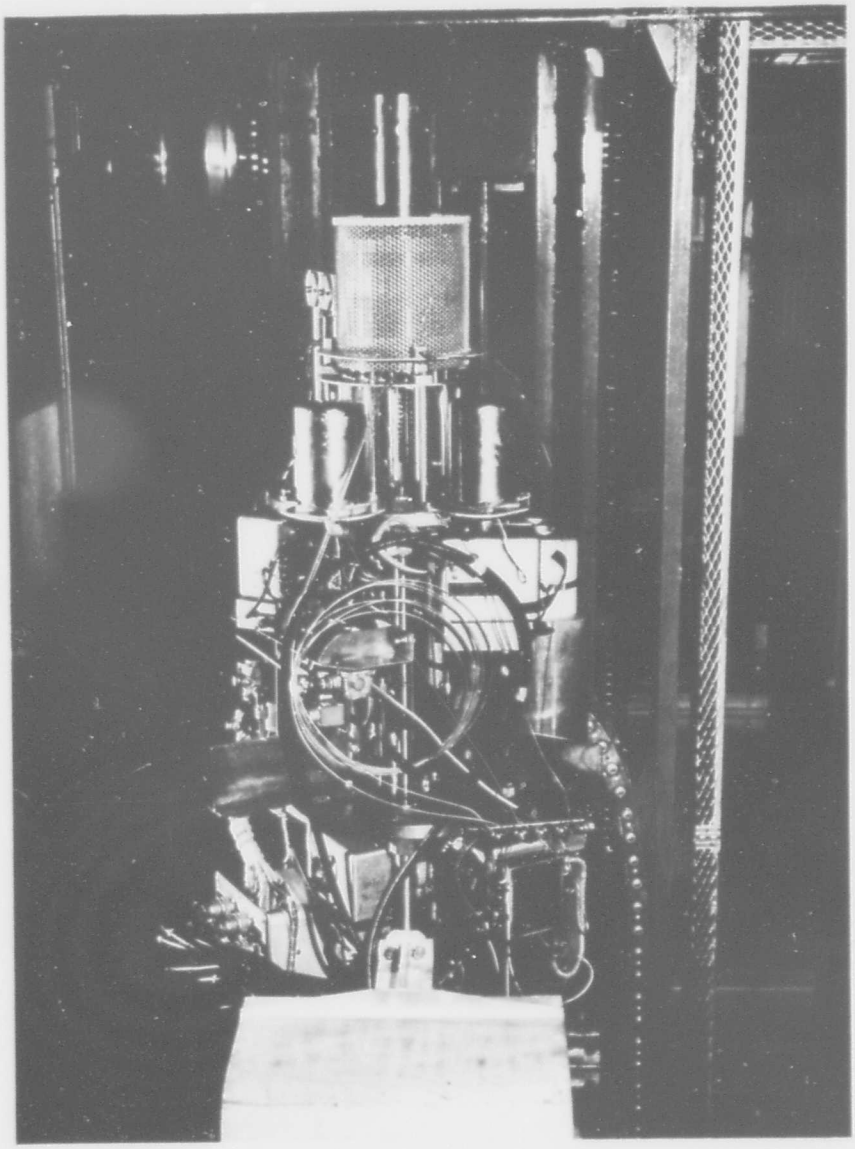


Figure 32. Health Physics Research Reactor.

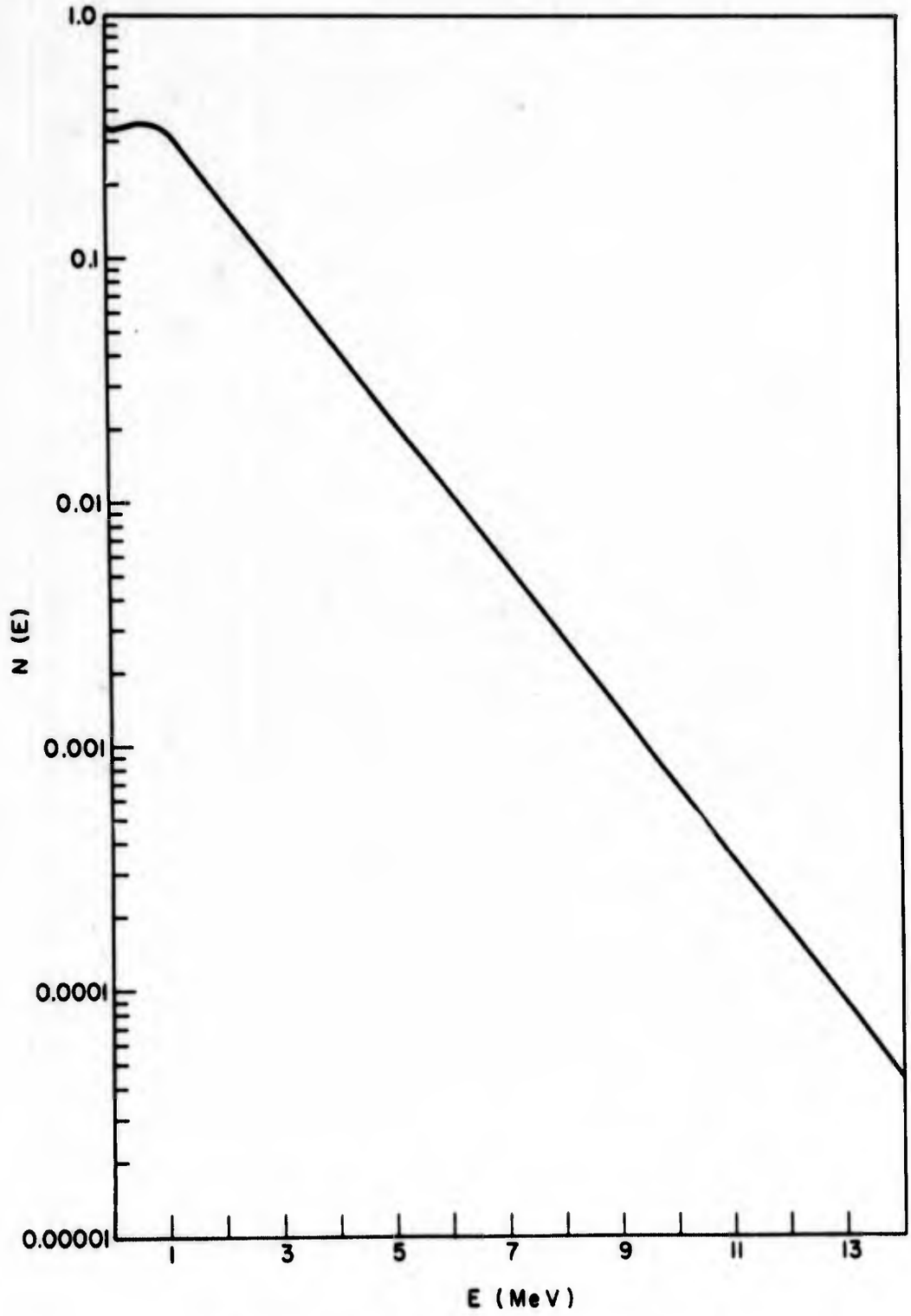


Figure 33. Health Physics Research Reactor fission spectrum.

This is the most important part of what I have to present, namely, the most recent air-dose curves for both Hiroshima and Nagasaki (Figure 34). These are published values, and you can get copies if you wish. These were completed within the last year, and reflect refined data resulting in an improvement over the curves calculated earlier. There is a considerable difference in some cases, as much as a factor of 2 at most distances for Hiroshima. The effect on the reported dose values for cases in the medical studies, on the average, were to lower the doses by about a factor of 2 over what they were before.

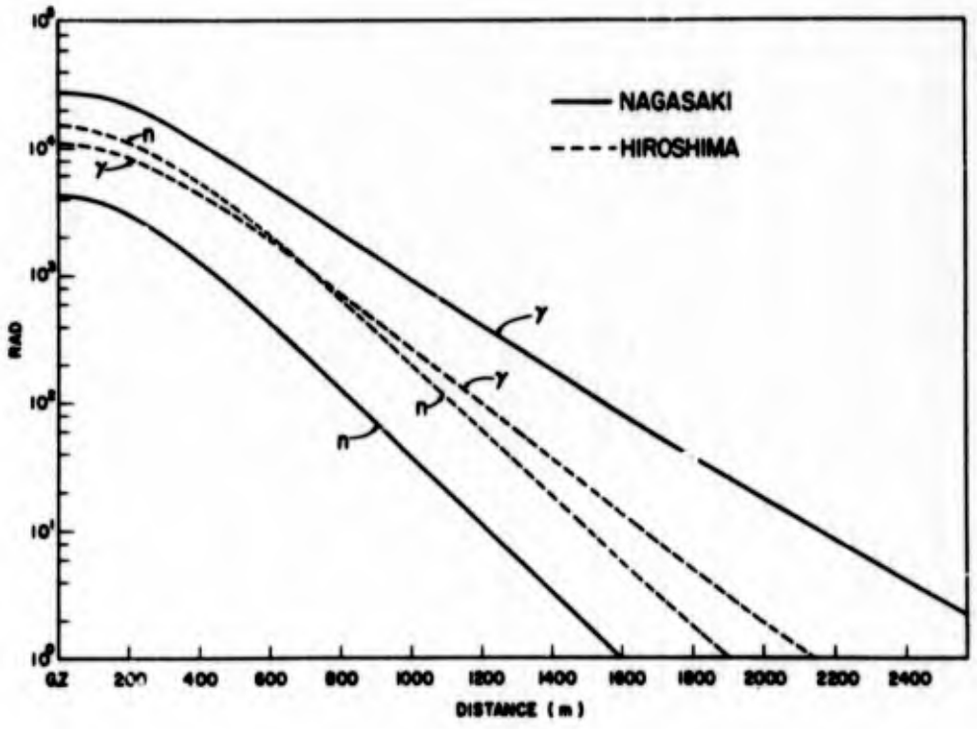


Figure 34. Air dose curves for Hiroshima and Nagasaki.

For Nagasaki, the differences between the old and the new are much less. There is a more uncertainty in the gamma-ray curve for Hiroshima and the neutron curve for Nagasaki than there are in the others, but they represent the less important part of the dose in each case. The uncertainty at great distances, near 2,000 meters for either of these curves, is probably ± 40 or 50 percent. In closein Nagasaki the gamma curve is the overwhelming part of the dose, but the opposite was true for Hiroshima. The major correction of the old curves represented by the new curves for Nagasaki was the

correction for the air-ground interface effect. The gamma-ray dose curve is based very largely on some of the data obtained in 1951 (Reference 68) and 1953 (Reference 69) for some of the early weapons. We were fortunate that the Nagasaki weapon was, for purposes of this discussion, identical with the ones that Dr. Warren talked about earlier, the Bikini series, and that up until the early 1950's data on weapons of the Nagasaki type were obtained.

ABRAHAMSON: That is the total dose received at that distance, isn't it?

AUXIER: Yes. This is the dose that you would have 3 feet above a level plane, or a level area, without any shielding matter around. Let us look at the shielding equations. In an earlier figure (Figure 27) you saw an array of Japanese houses used in one of the weapons tests. We had three such arrays during the weapons tests because the weapons yields were such that they did not destroy those houses and we were able to use them over and over. That was in 1958. Therefore, we obtained extensive data for dose distributions in these houses as a function of house size, orientation relative to the bomb, and shielding provided by neighboring houses.

Then in the BREN experiments (References 70 and 71) in 1962 we reconstructed these houses and explored the dose distribution inside as a function of the size of the house, its orientation relative to the source and relative to its neighbors, that is, in the same fashion as for actual weapons. A considerable reservoir of information was accumulated. The data from the later experiments only, that is, BREN, were used in a multiple regression analysis (Reference 70). From the analysis came a set of empirical equations which fitted all these data. The precision of the measurements had been good and the equations provided a fit to the data of better than 5 percent for one standard deviation.

The basic equation is quite simple. It consists entirely of multiplicative and exponential terms, for example, ratio of the dose in the house to the dose outside in the air

$$D_H/D_{Air} = A_0 + A_1 e^{G_1} + A_2 G_2 + \ldots A_7 G_7$$

to a maximum of eight terms. Of course, there are two equations for gammas for each city and an equation for neutrons for each city, or a total of six equations. Nevertheless, this simple equation,

where the As are constants and the Gs are geometrical terms, such as the distance from the front wall, the distance above the floor, the distance from a window of a certain area, etc., provides an accurate result from simple categories of input information such that the Japanese shielding technician can use it without extensive training. Once the equation was developed, all of the data from the weapons series were put into it as a rigorous test. The result was that one standard deviation of the result was ± 6 percent. This was success greater than had been expected. Consequently, the equations have been all programmed in Japan now, and I believe numbers are available for all the cases in which shielding histories have been made. The equations cannot be applied to the 20 to 25 percent of the survivors exposed in "heavy" buildings and other shields, but progress is being made in evaluating these separately.

WARREN: I think someone would like to know what is meant by clean bomb.

AUXIER: We were just talking about the Chinese bomb a while ago. Some of you know the last Chinese detonation resulted, 14 days after firing, in contamination of the West Coast and Nevada, in places, 0.5 to 2.5 milliroentgen/hour. This is a lot, considering the 10,000 miles and 14 days. So you have to assume, on the basis of our earlier tests, that this is what you would call a dirty bomb. We used to increase the yield of early "hydrogen" bombs by putting uranium around the assembly. This was a relatively cheap way to increase the yield. This was one way of making a very dirty bomb. Another way would be to put cobalt-59 around the bomb and generate cobalt-60. Almost anything placed around the weapon would increase the amount of local and worldwide fallout much more than it increases the yield of the bomb. It's one way of getting yield if weight is no problem, but a very efficient way of getting fallout.

BRUES: So in getting rid of the uranium that you want to get rid of, you are converting it into something worse.

AUXIER: Yes.

FOUNTAIN: As far as the Chinese are concerned, it might not be something worse. They're increasing the yield of their devices.

AUXIER: Yes.

FOUNTAIN: To them it's a plus factor and they are not concerned with worldwide contamination. They already have a poor image! [Laughter]

AUXIER: It could be a developmental phase or it could be deliberate, for that matter.

FOUNTAIN: Cobalt wouldn't increase the yield, though; it would just increase the dirtiness.

FRANK: What was the distinction between yield and fallout? Yield is the TNT equivalent?

AUXIER: Yes. We're talking about the yield in kilotons. The fallout is the contamination which falls over a wide area, in general, and is a function of yield, weapons design, burst conditions, etc.

FOUNTAIN: This is also related to tonnage of fallout of fission material.

AUXIER: Yes. Actually in the case of fallout we usually switch to megacuries or megacuries per square mile sort of thing.

JABLON: Do you happen to recall about what is the effective shielding of a Japanese house under this new scheme?

AUXIER: Yes.

JABLON: Under the old scheme it turned out that for most people in a Japanese house the dose came to something like 50 to 80 percent of the air dose at that point.

AUXIER: Yes. Now it runs from about 30 to 100 percent.

BRILL: It used to be 30 to 70, wasn't it, Seymour?

AUXIER: It varies, but in some cases it goes below 30 for neutrons, but it's unusual. The average gamma radiation attenuation is much less.

WARREN: Could you also talk about the failure of the inverse square laws we once used in measuring radium needles when a large area is contaminated by fission products? It goes back to your angle of scatter.

AUXIER: What Dr. Warren is interested in goes back to the point detector-point source discussion. If you had a point source of radiation and the detector at some distance from an isotropic source, the probability of a given quantum of radiation, or particle of radiation, reaching the detector is directly proportional to the solid angle subtended by the detector. As the distance between source and detector increases, the solid angle decreases as the square of the distance—attenuation by the air is exponential. But in the case of a large plane, the inverse square relationship is no longer valid because the source is no longer a "point." In the case of fallout the decrease with height is more complicated because the source is not even a plane isotropic source.

If you have radioactive material right on the surface, a smooth even surface, you would have, effectively, a plane isotropic source. Hence, by simple calculations you could show that the dose rate above the plane would fall off about at 1/R, where R is the height. But in the case of fallout it is never really a smooth or uniform surface; irregularities in the surface, even small ones, have a pronounced effect on the angular distribution of the radiation. Such things as ground roughness plus the effects of weathering tend to change what would be an isotropic source to one with an enhanced vertical component, which, in turn, causes a slower decrease in dose rate with increasing height than would be the case for the "smooth" plane.

WARREN: I discovered this by flying over the green grass area in Alamogordo before we went to Bikini, and the change with altitude was much less than was expected and, of course, this influenced our problems in Bikini. I was almost court-martialled because our Geiger counter men used various distances from the deck for making the measurements, some went knee-high, some went hip-high, and the officers, all engineers, charged me in this open meeting that any fool knows the inverse square law works and the measurements could not be correct. So I took some of them out on a contaminated ship and proved it to them.

AUXIER: Of course, in your case, depending on the kind of counter they had, the relationship was much more complicated by the fact that the beta-to-gamma dose ratio at the surface was very high. It was further dependent on the response characteristics of the instrument. A few feet could make a marked difference.

WARREN: Would you like to talk about the instrumentation and the measurement of betas and alphas and gammas? Neutrons I imagine would be only a matter of theoretical importance to the defense and probably would be measured by fixed installations?

AUXIER: Yes. Most of the emphasis, and I believe this is true throughout the military and the civilian organizations that are interested in these dosimetry problems, is now on the kind of instrumentation which is beginning to find universal application for nuclear accident dosimetry. For this application, a fixed installation, generally an integrating device, that is, a passive device, is used which is attached in the area in which an excursion might occur, or which would be carried into the area of interest in the case of a bomb. Besides these instrument systems for the case of the nuclear installations, such as a criticality accident dosimeter, another system has been used to integrate the dose, and I believe that, whether the military plans on it or not, the fiduciary for individual doses would be the sodium-24 in the blood. The hair on the body and the sodium in the blood are two detectors that have proved to be most useful for the detection of neutron dose; also they are the only two detectors that people have not found some way to lose at just the wrong time. So those two detectors are likely to be important in any unplanned exposure.

For the gamma rays, the same passive integrating system is still useful. The neutron-to-gamma ratio is measured with those passive systems; one can get an indication of the individual doses from these and from the sodium activation. From a combination of these two it is possible to calculate a person's dose.

ABRAHAMSON: When you say you could calculate his dose pretty well, are you also plugging into that dose calculation the RBE value? In other words, if you have a certain neutron component and a certain gamma, are you multiplying it by a relative biologic effectiveness?

AUXIER: As a physicist, I don't. As a health physicist who at times must respond to people who ask, "Well, join them together, putting the RBE or the quality factor as used in applied work and tell us what the total radiation quantity for risk purposes is," I would perhaps attempt it. In this case, if the interest is that of health physics records only, we are guided by the appropriate international and national committees to take the fast neutron dose and multiply by a factor of 10 and add the gamma dose to get the "rem" values (References 72 and 74).

However, in reality, many of you, if you are interested in that specific problem, are familiar with NBS Handbook (Reference 63), which gives Snyder and Neufeld's calculations of depth dose that take account of the specific energy losses at the microscopic scale in tissue, in other words, the LET values which, for the physicists and biologists is simply DE/DX, or the energy loss per unit track length by the charged particle. Of course, for neutrons, on the average, the LET is much higher than for gamma rays. The neutrons interact by colliding with atoms, particularly hydrogen in tissue, and producing protons which lose energy at a rate of up to 100 kev/μ that is, 100 kev of energy/μ of distance traveled by the proton in tissue. For oxygen, nitrogen, and carbon recoil nuclei, the energy loss is up to 800 or 1,000 kev/μ. If you relate damage to energy absorbed, on the basis of recommendations of the I.C.R.P., to its energy loss functions, that is, LET, then the RBE for neutrons can vary from near unity to a maximum of about 20 for the higher energies. Those recommendations are, of course, purely legislative, but they are based on the best judgment of a distinguished group of radiobiologists. However, if you take an even more specific case, for example, the genetic data of Russell (Reference 75) for mice, then for this particular end point, you find RBEs for neutrons relative to gamma rays that range to 30 or even greater; there are dose rate considerations which also bear heavily on these high RBE values.

If you were interested in the radiation of seeds of plants and in particular, the inhibition of growth, the data from the AEC-University of Tennessee experiments indicate RBEs of greater than 100 for fast neutrons, relative to the gamma rays from cobalt-60.

To get back to your question concerning Hiroshima and Nagasaki, we are reporting the doses in rad. We're making the calculations of the LET distributions and by working backward from the biological data, we would like to find what the RBEs are; based on the different neutron-to-gamma dose ratios for the two cities, this is possible, conceivably.

WHITE: This, of course, goes to the point of what is the physical parameter that is important biologically. One may have to work hard to find out what it is, as I noted this morning. Have you got any idea of what this is likely to be in the radiation field, John?

AUXIER: If I started to answer that it would be like you starting your three-day talk. I don't think I should start at this time.

WARREN: Haven't we got enough now to go on with the chronic effects of it?

BUSTAD: John, I'm afraid of what's going to happen tomorrow and the next day. I mean if the geneticists and psychosocial scientists start talking like you've been talking, I'm not going to understand them, if you know what I mean.

FREMONT-SMITH: You will be among friends!

BUSTAD: I'm wondering if we took that last chart that you have up there to give some dose-effect relationship, that is, what does or what did 50 r do, or what would 100 r do, what would 200 r do? I believe this would be helpful to several people here.

AUXIER: Surely. This is the level of radiation to which the people at risk were exposed (see Figure 34), unperturbed by the shields, which, of course, decreases the doses in all cases.

WHITE: If I'm going to let you talk about what the target dose is and discuss dose effects biologically when you're trying to relate this back to weapons, then you must distinguish between a scaled dose in air free-field or a dose where the target is. The former might be 100,000 rad, and if there is a shielding factor of 1,000 in the building, then the latter comes down to what? One hundred rad, you see. You have to distinguish them.

WARREN: Regardless of that, we are really only interested in the chronic effects in those who were at some location which enabled them to receive from 200 to 1,000 r.

BUSTAD: That's the point I'm getting at.

WARREN: The other calculations are for other purposes, but for our purposes the chronic effects are only derived in this range.

WHITE: That is that dose where a person is, but not necessarily a free-field dose.

WARREN: No.

WHITE: The free-field dose was what was on that chart.

WARREN: I think we can make the jump.

AUXIER: Let me clarify one thing. When I'm talking about absorbed doses, unless I slip, I'm always talking in rad, and that's 100 ergs/gram of tissue regardless of the type of radiation we're talking about, and for the purposes of this discussion all those numbers on the chart, those are rad as opposed to r and so they can be used directly.

FRANK: These terms are coming up over and over again. I've heard them defined, but what is rad and what is r?

AUXIER: When 100 ergs of energy are absorbed in 1 gram of tissue, we have 1 rad.

FRANK: What is r that you talked about?

AUXIER: r is a term which originated back in the 1920s and is actually equivalent to about 93 ergs/gram, usually, when the radiation which produces 1 r is incident on tissue; it depends upon the spectrum, but approximately 93 ergs/gram. An r is a unit which is based upon the ionization produced in air. When we relate it to tissue, it produces almost a rad, you see, just 7 percent or so less.

FRANK: Does it make any difference whether it is gamma or beta or neutrons? The rad is the unit for all three of them?

AUXIER: A rad is purely energy per unit mass. It doesn't matter from what type of radiation.

FRANK: But does it matter which kind of radiation it is or is all that matters the energy?

AUXIER: No. That was Sam's [White] question a minute ago. It doesn't matter what type of radiation it is. Everybody, I think, will agree to the following: The total energy absorbed, or dose in rad, is probably important. It is probably the most important single parameter, but the microscopic distribution of that energy is certainly of great importance. If you put into tissue 100 ergs/gram with just a few heavy ionizing particles that look like big bullets going through, you get a lot more damage in general than if you put the same amount of energy in by electrons that yield up to 3-1/2 kev/μ

of distance traveled. This results in the RBE effect. At least it is assumed to be generally related to this energy loss function. There are other things that happen, too, if you're not careful, and that is related to the fact that the human body is rather large compared to the range of most radiations, even neutrons. Therefore, in addition to these microscopic distributions you have to worry about the gross macroscopic distribution of dose throughout.

Undoubtedly, one of the important parameters is dose rate. If 100 rad is delivered to a biological target in seconds, it will, depending upon the end point, produce an effect that will differ in degree from the result of the same dose delivered over a period of weeks, or perhaps even days or hours.

BUSTAD: If we were exposed to 100 r from now until we die, we probably would not able to tell it. That is the good part of it, at least at my age.

COURT BROWN: I want to know why Dr. Warren said we were only interested in the chronic effects of doses of 200 rad or more? Aren't we interested in the effects of doses under 200 rad?

WARREN: They are pretty hard to demonstrate and they are probably of less significance in a discussion of this sort because we have to take rather maximal effects with survival to indicate where the area of damage is.

COURT BROWN: I disagree with you entirely. They are of enormous significance.

TAYLOR: We've had more public discussion, Ithink, about the genetic effects at very low levels.

WARREN: Yes, and very low levels. I'm only the instigator. Go right ahead with the argument.

BRUES: I would think the answer to that question is one that had best unfold in the next day or so rather than be brought out now.

WARREN: Yes. I'm not setting any limits; I'm just putting the parameter down to simplify our approach.

LIFTON: I would like to hear about these chronic effects now. We have very little time and they are very important.

FOUNTAIN: I would like to know what dosages some of the survivors were exposed to. They haven't been mentioned yet. I think this is important if we're going to talk about what happened to them.

AUXIER: We have a large number of survivors, that is, a large total number as opposed to the percentage that had exposures in the middle lethal range; the doses ranged from "zero" at distances of 2 miles or more to the supralethal near ground zero. On the last trip I made to Japan there was concern because one of the Japanese scientists had withdrawn from the files, based on the old numbers, all of the cases that showed doses in excess of 1,000 rad. This totaled, out of 15,000 cases he surveyed, 150 cases, and this excited some people when they took a cursory look at it.

FREMONT-SMITH: You say he had withdrawn it?

AUXIER: He scanned the whole 15,000 cases or so and he found 150 cases where it was apparent that the people had more than 1,000 rad. However, there was one serious thing wrong with this; he was not taking advantage of all the information available. I pointed out yesterday, those shielding histories are much more valuable documents than they are sometimes thought to be. The man withdrew these 150 histories from the files and recorded the dose values only, and these from the old curves. But, by inspection, without calculations, just inspection, I eliminated 140 cases as having something wrong; that is, for example, this case of a man who said he was exposed 800 meters from ground zero in the open, with nothing around him, but he had no burns. He had reported no burns; he had evidenced no burns. But you don't have a man exposed 800 meters from the Hiroshima weapon, in the open, and have him survive, especially with no flash burns. This was ample evidence that there was something wrong; the man was either another half-mile away or behind something. This sort of analysis was sufficient to indicate that most of the 150 cases should be discarded. The other cases all showed that the shielding interviewer had rated the reliability of the interviewed person very low.

There may well have been a few people, as you all know, who survived doses near 1,000 rad. When you have this many people at risk and whether you assume some normal distribution of survivors or some skewed distribution, there may be a person or a few people that take a dose of close to 1,000 rad and survive; I don't know. But there are people in the files with more than 500 rad, certainly, and many people with doses in the 200-, 300-, and 400-rad range.

MILLER: Are these reliable estimates now?

AUXIER: Yes, these are considered to be reliable estimates.

TAYLOR: How long would you say the doses have corresponded to observable effects which are still persisting?

AUXIER: That would depend. Dr. Johnson could answer that better, but before he does I would like to interject one thing before I forget it. That is, Dr. Johnson observed that there was no correlation between those people who had chromosome aberrations, of any and all types, I suppose, and the burns on individuals, and to me this is significant. You would not expect, on the basis of what we know about aberrations and burns and radiation, that there necessarily would be; a person could have had no burns but high doses and vice versa.

COURT BROWN: I don't think you can put any value on that statement at all because I don't think you can correlate chromosome aberrations observed in lymphocytes with damage observed in epithelium.

AUXIER: That's the point. You, too, said there need be no correlation. I think this is good, because if you look at it from the radiation point alone and forget the burns, there may well be correlation between absorbed doses and aberrations; this might then provide one of the keys for separating combined effects. If you have a chromosome aberration of a given type, you might relate it to high LET radiation, neutrons in particular, such as Gooch and Bender do (Reference 76), and which they can observe for long periods of time after irradiation. If you see aberrations still but you see no evidence of physical trauma, such as blast or burn effects, this in itself may be one of the tools that can be used in the combined injuries analysis.

JOHNSON: What I said was that a limited number of people had been looked at.

AUXIER: Yes, very few as a matter of fact.

JOHNSON: A total of 94 persons, exposed and controls. Perhaps 16 had significant bomb scars. In this small group of scarred people, 6 of the 16 had chromosomal aberrations; there were 10

persons with aberrations in a group of 36 nonscarred exposed persons. The frequencies of aberrations in the scarred and nonscarred group are similar. My point was that scars were poor predictors of finding chromosomal aberrations. This is not to say there is no correlation between A-bomb scars and chromosomal aberrations.

AUXIER: Pardon my misinterpretation. My point was that it might be that this is another approach to combined injuries analysis. So far there has been nothing except a cursory look, but maybe there is something there.

COURT BROWN: Before you try to declare too much from these types of observation, I think you should realize that there are technical limitations to the demonstration of chromosome abnormalities which tend to minimize their frequency. This is a point to be discussed tomorrow.

CHRONIC RADIATION EFFECTS

WARREN: Could we go on now to a more definitive discussion of the chronic effects? I don't know whether you will bear with me in the position that the bone marrow probably represents a sensitive organ that could be badly damaged by the radiation with survival and repair.

With that rather flat-footed, oversimplified statement I would like to ask Dr. Upton if he would discuss the bone marrow, the chronic situation as he sees it after the acute damage is past.

UPTON: It's a big order, Dr. Warren. I would say by way of introduction that, as has been mentioned earlier, the marrow continuously has to replace short-lived cells that circulate in the blood. Hence after a dose of radiation to the marrow large enough to destroy such progenitor cells, the marrow will become depleted and, depending on the severity of depletion, recovery may take a matter of weeks, or longer.

In general, if the damage is not too severe, the marrow will restore itself ultimately. Only now, however, are techniques being devised in the laboratory to measure residual damage to marrow function, that is, to determine whether the functional capacity of the marrow does in effect restore itself fully. We can't really say very much yet about the extent to which recovery is less than complete, based on functional tests. There is some evidence, however, that recovery is not fully complete, since ultimately the risk of cancer of marrow cells is increased systematically as a function of radiation exposure to the marrow and other blood-forming tissues. These cancers are known as leukemias.

This relation between leukemia incidence and radiation dose is one of the most significant findings which has come out of the studies of the Japanese A-bomb survivors and correlates nicely with observations that Court Brown and Doll (Reference 77) have made on patients treated with large doses of radiation in the treatment of rheumatism of the spine.

I think that Dr. Court Brown himself would be better qualified to discuss this, or Dr. Brill, Dr. Johnson, or others who have worked recently with data from the Japanese studies. It is primarily this particular effect on blood-forming tissues which constitutes the best evidence we have for residual damage of these organs.

Before going into a discussion of the incidence of leukemia and the distribution of the cases in relation to dose and time, I think it may be of interest if someone could summarize briefly for us something about the numbers of individuals that are known to have been exposed at various distances and to have survived, that is, what populations do we have on which to try to study the incidence of long-term effects on the marrow? Seymour, could you or someone else here quickly summarize the figures for us?

JABLON: I can give you some numbers quickly. The numbers are for the population of what is called the Life Span Study, a group of approximately 100,000 persons in both cities, being studied by the Atomic Bomb Casualty Commission for mortality from 1950 forward. As Dr. Johnson mentioned earlier, the sample for the Adult Health Study is a portion of this larger group.

The population includes 28,000 persons (as of 1950) who were within 2,000 meters of the hypocenter. About 9,000 of the 28,000 were within 1,400 meters.

The figure 1,400 meters is significant because this distance has often been used as a dividing point for the comparison of groups. According to the older (York) air-dose curves and the so-called T-57 dosimetry, almost all of those within 1,400 meters received at least 100 rad, and those beyond 1,400 meters had less than 100 rad. With the new air-dose curves and dosimetry, for Hiroshima at least, these dose figures require revision downward. Roughly speaking, therefore, there are about 9,000 persons with doses of 100 rad or more (T-57); about 19,000 with doses in the range 2 to 100 rad; nearly 45,000 present in the cities but beyond 2,000 meters who received little, if any, prompt radiation; and about 27,000 who were not in the cities at the time, but came in afterward. The last two groups are used as "comparison groups" since they are not, of course, really controls. The numbers are shown in Table 2, which is taken from Jablon, et al.(Reference 78).

The numbers are shown by age and by sex. There are a few points that require comment. The age distribution for males is

Table 2. JNIH-ABCC life span study sample by age at time of bomb, and distance from hypocenter, and by sex (Hiroshima and Nagasaki).

Age	Total	Distance from Hypocenter (meters)			Sex	
		<2,000	2,000+	Not in City	Male	Female
Total	99,389	28,142	44,673	26,574	41,704	57,685
0–19	40,712	11,327	18,380	11,005	19,006	21,706
20–39	27,572	7,828	11,874	7,870	8,410	19,162
40–59	25,047	7,280	11,508	6,259	11,768	13,279
60+	6,058	1,707	2,911	1,440	2,520	3,538

quite abnormal in that there are so few in the range 20 to 39 years. The age distribution for females is not remarkable. The total number of females is greater than the number of males by a ratio of nearly three to two because of the deficit of young adult males. The abnormality in the male age distribution posts a warning signal: that the sample of young adult males may be quite peculiar. Those included were almost all men who had not been taken into military service, and this at a time when Japan's manpower reserves were quite low. While no doubt in some instances essentiality of occupation was the reason for not taking men into the military, the group also contains all those who were rejected for service for reasons of health. We have, in fact, observed a very high tuberculosis mortality rate in just these men, and we wonder whether some of the higher mortality rates seen in this group during the 1950s may not be, at least in part, a consequence of ill health already present in 1945, which kept them out of service in the first place. Unfortunately, we have no solid information on this subject.

TAYLOR: Has any study been made of some similar group of people in a city that was not bombed but which was comparable to Hiroshima?

JABLON: No, sir, except that we can compare with Japanese vital statistics.

TAYLOR: I mean that particular segment of the Japanese population that was young males left home during the war but from another city. Is there any way of going back through their records?

JABLON: I don't know.

UPTON: I think this kind of question sets the stage very nicely for a general consideration of late effects. We have some question here whether there are, as a result of radiation, specific changes in certain cells, in the marrow of blood-forming organs or other tissues which predispose to specific diseases, leukemia, and other cancers; and whether there are non-specific changes of a sort which create a general deterioration, a reduction in vigor associated with susceptibility to infections, tuberculosis, more general susceptibility to disease, to shortening of the life-span, to acceleration of these deteriorations we otherwise associate with neutral aging.

UPTON: Before starting to talk about late effects, I was asked to review very quickly what we mean by the dose-effect relation and what kinds of effects there are. At the risk of generating arguments, I shall summarize briefly a few dose-effect relationships to put our discussion in perspective.

If we were to deliver a very large dose of radiation in a short period of time (10^7 rad), all living cells would be killed. We could use a dose of this size to sterilize meat, since even the most resistant bacterial spores would be destroyed.

If we were to drop the dose to 10^6 rad, no animals would survive such an exposure, but some of the more resistant bacterial cells would survive. Furthermore, this dose delivered instantaneously would be almost immediately lethal to mammals from damage to the brain. Going down further, to 10^5 rad, all mammals exposed at this level would die at least within a day or so, again through devastating effects on the brain. At 10^4 rad, radiation might take a little longer to kill, but this dose delivered to the whole body or even to the head would kill within a couple of days from brain damage. At 10^3 rad, a dose range is entered in which some mammals would survive, but all men would probably be killed if it were delivered to the whole body, although there is some argument about this.

FREMONT-SMITH: How about women?

UPTON: Any human would probably fail to survive a dose of 10^3 rad delivered instantaneously to the whole body. Death would generally result from damage to the bone marrow, and one might guess that this dose could be survived if blood cell formation were restored by marrow replacement. I don't think there is good evidence on this point for man.

WARREN: Not yet.

UPTON: Not yet. Certainly, one can rescue animals from 1 000 rad by giving bone marrow or by shielding bone marrow.

At 100 rad it is doubtful whether any human beings would die. Certainly, however, many cells in the body are killed by 100 rad. One would expect, in general, the dose required to kill half the human population to lie between 100 and 1,000 rad. There may, however, be highly sensitive people whom 100 rad would shove over the brink. At 10 rad, we can be quite sure that no individual would die, although some cells would be killed even at this dose level.

The question is, are there going to be, as a result of killing certain cells or causing chromosomal aberrations in cells, long-term effects? We can feel sure that at 100 rad there probably are long-term effects. I think it will turn out from our discussion that the incidence of certain tumors has been shown to be increased at a level of about 100 rad.

JABLON: Or more.

UPTON: Or more, right. The question is, can one extrapolate down and see a smaller effect at a level of 10 rad?

At a dose of 1 rad some very sensitive cells are found to be killed. We do find cells that contain lethal kinds of injury, such as bilobed lymphocytes circulating in the blood, in individuals exposed at this level. The question is, what does this mean? Are we dealing with subtle effects on certain cells that can ultimately have some significant consequences for the life expectancy or health of the individual?

Finally, at a tenth of a rad we are dealing with a level of radiation which exists normally in the environment, that is, 0.1 rad spread out over a year. And as we go down to a still lower level, a tenth of that, we are down to the level at which fallout is encountered.

FRANK: Are these all instantaneous doses? There's nothing cumulative about it?

UPTON: Yes, down to the level of 0.1 rad. Here we're speaking about a dose spread out over a year. A tenth of a rad over a year is

the level characteristic of the natural radiation to which we are all exposed. It includes radiation from cosmic sources, radiation in our bodies occurring from radioactive isotopes, and radiation from rocks in the earth's crust.

What we're going to be concerned about now are the observations that can be made on the Japanese survivors, say at the level of 1,000 rad and down; those who survived the acute effects. What do they manifest in relation to the long-term outlook?

SCHULL: Art, what dose is required to induce permanent sterility?

UPTON: I am probably less well qualified to answer this than others here. It would be my impression that a dose of 300 to 500 rad delivered to the female in a single treatment or a couple of closely staged treatments, but I may be wrong.

SCHULL: Isn't it higher than that?

UPTON: Court Brown is probably the one to answer this.

FREMONT SMITH: Are you talking about total body?

UPTON: No. Dose to the ovaries.

WHITE: Or to the testes.

SCHULL: Yes, both.

UPTON: In man, the testes must tolerate more than 300 to 500 rad. I'm not aware that men who have survived exposures at this particular level have been found to be sterilized.

COURT BROWN: It is probably dependent upon age.

WARREN: Dr. Court Brown, go ahead, please.

COURT BROWN: I don't know very much about it, that's the trouble.

FREMONT-SMITH: Naturally, but tell us.

COURT BROWN: One knows not so many women, but one knows of cases of women who have had, let us say, 600 rad delivered to the midline of the pelvis and who have subsequently resumed menstruation and conceived. But the point here is that these were women in their early 30s. The same dose given to a woman in her early 40s may be sterilizing. So it is age dependent.

FREMONT-SMITH: And younger, she would be safer?

COURT BROWN: I don't think it's very safe! [Laughter]

FREMONT-SMITH: I mean but more likely to resume.

COURT BROWN: Yes, I think so.

CASARETT: There are radiation therapy data on this subject wherein the ovaries were sterilized deliberately or incidental to other treatment. I reviewed these data a few years ago. It seems that a dose of 600 r localized to the ovaries will sterilize most women, but there are young women who require larger doses. There is a paucity of data for the young women, but doses of the order of 900 or 1,000 r may be required to sterilize effectively most young women.

When you go down to a range of 150 or 200 r you can cause cessation of menses temporarily, for a year or two. I think this gives a range of effectiveness as far as the clinical material is concerned for adults. There is, as Dr. Court Brown indicated, a dependency on age. Perhaps this is owing to the presence in the young woman of many more follicles than in the older women whose follicle populations have undergone a considerable amount of atresia.

ABRAHAMSON: What about the male, George? Is it in the same range?

CASARETT: The single total-body dose required to cause complete and permanent sterility in the male is greater than the acute lethal dose. Survivors of total-body irradiation would tend to recover after a temporary sterilizing effect. However, I would like to point out that I have found certain modes of dose fractionation that can cause complete and permanent sterility without causing acute death or illness.

I was interested in the efficiency of fractionated radiation, that is, in reducing the wastage of radiation, in causing sterility, and worked this out on dogs with some preliminary experiments on rats. The idea was based on the observation that some of the spermatogonia (Type B and some of the Type A) have a short interphase period and divide frequently, but some of the Type A spermatogonia have a longer interphase period associated with the duration of each of the four cycles in the spermatogenic wave. My hypothesis for efficient fractionation was as follows: one of the most efficient dose-time relationships for the killing of cells would be a fractionated schedule in which a small intensive dose fraction was combined with an interfraction time interval which would take greatest advantage of the mitosis-linked death of injured cells and of the cell division kinetics. This work has been in progress some dozen years now, and I have found so far that a dose of 3 r/day, given in 10 minutes each day (about 15 r/week), up to a total of 475 r, causes complete and permanent sterilization of the dog testis. By "permanent," I mean we followed them for life, 10 years or more, without detecting recovery of a single sperm. We've tried to find the single dose that will do this and we are limited by serious acute lesions of the testis. We've gone up to 2,000 r, and this does not completely sterilize or permanently sterilize the dog testis. A dose of 2,000 r permits some recovery in some of the dogs. We now have a factor of about 4 for greater efficiency of this particular mode of fractionation in causing complete and permanent sterility in the dog.

BUSTAD: At what age did you start the dog?

CASARETT: Twenty-one or twenty-two months of age, as young adults. When we have given this fractionated scheme total-body, there is no sign of acute illness or death, and very little hematopoietic change as determined by the hematological measurements. So I would say that we can't assume anymore that all modes of irradiation in nuclear warfare are not seriously damaging to the reproductive apparatus of the male unless they are in the lethal range.

UPTON: You mean fallout.

CASARETT: Yes.

UPTON: Is there any indication, though, that in the survivors at Hiroshima or Nagasaki there has been permanent infertility?

JABLON: Seigel published a study (Reference 79) not very long ago and reported that he could find no effects on reproductive performance, in either males or females, in the 10-year period after 1950.

UPTON: This would be consistent, as I take it, with your acute studies.

CASARETT: Yes. The problem is that these dose-time relationships are likely to be critical in that if you change the daily dose without changing the interfraction time interval, or if you change the time between doses without changing the dose fraction, you may decrease the effectiveness. I'm talking about a critical dose-time relationship.

BUSTAD: I am interested in the early effect, because in our laboratory I think Dr. Andersen's* dogs, which survived a dose of acute radiation that was lethal to some when exposed at 10 to 12 months of age, manifested little or no sterility. When younger females were exposed to similar acute doses, a decrement in reproductive function occurred.

UPTON: I hate to be arbitrary, but I would like to propose, since we're going to be getting into genetics later and reproductive performance will figure very importantly in that discussion, that we leave this question momentarily and settle down on the leukemia problem.

JOHNSON: I will summarize briefly the ABCC experience in detecting leukemia in the survivors of Hiroshima and Nagasaki during 1946 to 1964 (References 80 and 81) recognizing the presence of Dr. Brill, who preceded me at ABCC and who can add much to the interpretation of dose-response.

We can think of radiation leukemia response in three time periods: the early period (1950–54), the transitional period (1955–59), and finally the late period (1960–present).

The early period brought the first and greatest peak in the incidence of leukemia. The persons who developed leukemia were for the most part below 30 years of age at the time of the bombings. The

* A.C. Andersen, Radiobiology Laboratory, University of California, Davis, Personal communication.

unusual type of leukemia seen in these young people was chronic granulocytic leukemia, a type usually seen in older persons. During the early period, acute lymphocytic leukemia occurred at a high rate. In Western countries, acute lymphocytic leukemia is the rather common type of leukemia in childhood, but not so in Japan where children are most apt to develop acute granulocytic leukemia.

In the transitional period (1955-59) the rates of leukemia generally diminished in all persons exposed within 1,500 meters of the hypocenters, but we see in this period another interesting phenomenon. A smaller peak in the incidence curve developed in 1958 and consisted mainly of cases of acute leukemia in persons who were over 30 years of age in 1945. Thus we have seen a different period of latency as a function of age. Age is a co-variable of dose. In the late period there is an absolute decline in the rate of leukemia, but still, even to the present, the rate of leukemia among the exposed persists five to six times higher than expected.

UPTON: I'm wondering if it would not be helpful to those who do not have the data well in mind to say something about the magnitudes of these effects. I think this is a very important question in relation to long-term implications.

JOHNSON: Although the incidence of leukemia in all Japan has been increasing since 1950, a demographic phenomenon not related to A-bomb exposure, we can estimate a rate of spontaneous leukemia yearly incidence of 30 cases per million. In those persons exposed to the A-bomb within 1,500 meters, we found the yearly incidence of chronic granulocytic leukemia, for example, during the years 1950-54 to be 48 cases per 100,000 or 480 cases per million.

FRANK: Is this in the Hiroshima group or all of them?

JOHNSON: Nagasaki and Hiroshima combined.

UPTON: These are all distances?

JOHNSON: No. The rate I quoted was for persons within 1,500 meters.

UPTON: How many cases is the sample based on? Is this a highly significant difference. Do you have a figure there, Dr. Miller?

MILLER: I think Dr. Brill might have it.

UPTON: Do you have a slide, Randy?

BRILL: Yes, I think I have one of the ones for the 1950-58 period if you would like to show it.

JOHNSON: I would like to add that the calculation of incidence is based on approximately 90 cases of leukemia in a defined sample of 100,000 persons, survivors and controls, known as the ABCC-JNIH Life Span Study sample.

FREMONT-SMITH: Ninety cases?

JOHNSON: Yes, sir.

UPTON: The 90 are not shown here, are they, or some of them are?

BRILL: There were 76 leukemia cases (Table 3) which were diagnosed between 1950 and 1958 in the lightly shielded portion of the master sample displayed really as a function of distance, but the distance groups were converted to rad based upon estimated shielding attenuation factors and the estimates of air dose that were available in 1960.

You can see that in the most distally exposed groups in Hiroshima and Nagasaki, the rates were 22 and 41 cases per million over this time period per year. In the most closely exposed groups, you can see estimates of 1,560 cases per million per year in Hiroshima and in Nagasaki it was 2,200, or a multiplicity of about 50-fold between the incidence in the most closely exposed and the distally exposed (which is very close to other estimates of the spontaneous frequency of leukemia in Japan).

This table really could be quite controversial. The reason we put it together originally with numerical estimates of dose, in rad, was related to the feeling that if we didn't do the analysis in this way, others with less access to the basic data would do so with even less validity.

When we first came to Japan, the controversy concerning linearity hypotheses growing out of Lewis' article and Duchesne's provocative editorial in the same issue was raging. Clearly, there was more in the way of emotion than facts with respect to the magnitude of: (1) the

Table 3. Average incidence of confirmed leukemia in master sample proper plus reserve (1950–58) light shielding by radiation dose (Hiroshima and Nagasaki)(Reference 82).

Dose (rad)	Hiroshima			Nagasaki		
	Man-year at Risk 1950–58	Leukemia Cases[a]	Rate[b]	Man-year at Risk 1950–58	Leukemia Cases	Rate[b]
1,281 and over	3,204	5	1,561	387	0	—
641–1,280	9,999	10	1,000	1,341	3	2,237
321–640	7,623	5	656	2,043	2	979
161–320	21,888	7	320	6,408	4	624
81–160	37,278	7	188	12,681	6	473
41–80	48,798	3	61	11,565	0	—
21–40	48,402	2	41	9,981	1	100
0–20[c]	547,839	12	22	217,782	9	41
Total	725,031	51	70	262,188	25	95

NOTES:
[a] Includes four cases of leukemia with onset in 1958 not included in TR-02-59.
[b] Per million person-years at risk.
[c] Includes A-bomb survivors exposed between 2 and 10 km.

doses and (2) the incidence of leukemia that could be documented in the Japanese. So what we did was put together a case listing of all of the cases that had been seen up until that point, documenting the type of leukemia, whatever else was known about it along with relevant populations in the hope that any interested investigator might be able to put these together in different ways to test various hypotheses.

WHITE: How much does the rad dose go down according to the figures now available?

BRILL: Perhaps by a factor of 2 as I understand it.

AUXIER: A factor of 2 plus the shielding effects which may add another factor of 2.

JABLON: With the new dose estimates, the Hiroshima numbers will move down one line.

BRILL: Which is fine, because in Figure 35 you will see that the Hiroshima and Nagasaki data will come to lie almost one on top of the other. With the data as shown in the figure, if one calculates a neutron RBE, it comes out negative, that is, a little bit of neutrons are good for you. The revised data should bring this into line.

UPTON: What you're saying, Randy, is that these early dose estimates were too high by a factor of 2 and hence these points would have been displaced on the graph?

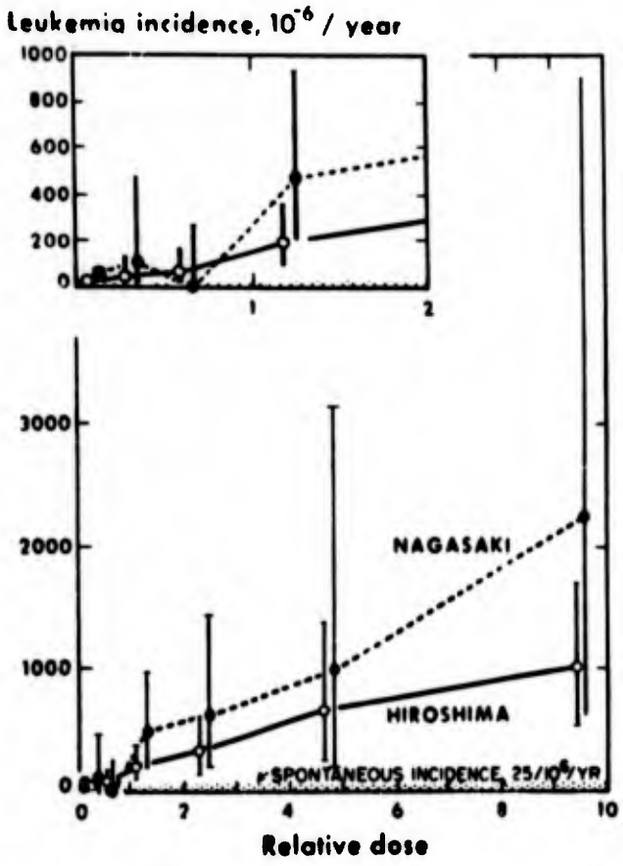


Figure 35. Average incidence of confirmed leukemia in the master sample, proper plus reserve, 1950–58, light shielding, by relative radiation dose (Reference 82).

BRILL: To the left.

UPTON: And the curves would then superimpose?

BRILL: Right.

CASARETT: If you unitize, that's an RBE of 1?

BRILL: If that was a rem dose, it would be based upon dose. In that case it's equivalent to an RBE of 1.

ANGEVINE: Randy, what would be the incidence of leukemia in the United States, at the same time, when compared with your figures in Hiroshima and Nagasaki?

BRILL: In the white population of the United States, age adjusted to the standard population in Japan (1950), the rates are 44 and 60 cases per million per year in females and males, respectively (Reference 83).

ANGEVINE: About threefold?

BRILL: About twofold.

MILLER: Dr. Johnson described this graph, which is his graph, revised (Figure 36). The black bars indicate acute leukemia, with

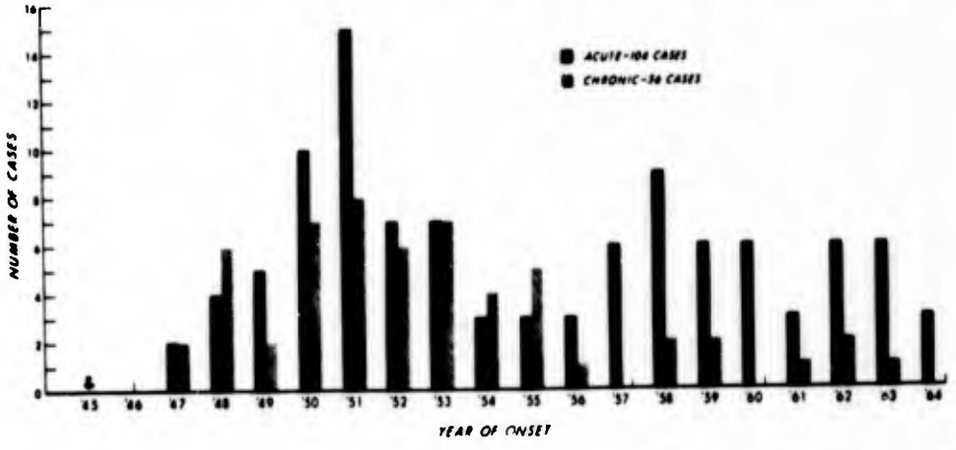


Figure 36. Leukemia in Hiroshima and Nagasaki among persons within 1,500 meters of the hypocenter (data from Atomic Bomb Casualty Commission).

year of onset from 1947 through 1964; and, as he said, there was a peak in 1951 and perhaps a second peak in 1958 for acute leukemia. Chronic leukemia had a broader peak centered about the same year, 1951, but the rates have since declined to near normal (Reference 80).

UPTON: It's still elevated, I take it, Dr. Johnson?

JOHNSON: Yes, it's still five to six times the expected rate.

ABRAHAMSON: May I ask what this would mean as a per rad risk figure? Does it compare with the estimate of E. B. Lewis (Reference 84) of two cases per million rad?

BRILL: The estimates for Hiroshima at that time were between 1 and 1.3 cases per million per year per rad and in Nagasaki they were between 0.8 and 1.2. Now, presumably the Hiroshima figures will be closer to the 0.8 to 1.2 cases per million per year per rad averaged over that time period.

As you extend the time base to the present, and include a period in which the incidence has been lower, the average incidence is going to come down. As one follows these patients for an increasingly long period of time, the average estimate of the leukemogenic effects of radiation in a lifetime will become smaller and smaller with the passage of time.

ABRAHAMSON: Assuming no other peaks coming up.

BRILL: Right.

WHITE: Was that portion of the graph, Dr. Miller, the control group?

MILLER: No. That was the chronic group as contrasted with the acute group.

WARREN: Does that mean that the cases that are going to develop leukemia are dying off and there are fewer in the sample remaining?

JOHNSON: We have so interpreted it!

UPTON: Is there a relationship between distance and time?

JOHNSON: There is a relationship certainly with distance, as a function of dose. The first and maximal peak of leukemia occurred during the 1950-54 period. Taking age in 1945 as a starting point, the average incubation period for leukemia in those persons exposed within 1,500 meters was approximately 8.3 years.* In general, males had higher rates of leukemia, expecially chronic granulocytic leukemia. We can now add sex to age at exposure, another covariable of distance.*

CASARETT: As I understand it, the leukemia incidence in Japan has been steadily going up at this time.

JOHNSON: Yes.

CASARETT: Since we're getting a decline in the increase in the radiated population, doesn't it seem likely that on an absolute lifetime incidence basis, if we extrapolated a rising normal incidence, that the total increase in incidence might be less than has been expected; that some of the cases might be simply displaced in time while the absolute increase may be less than that which would be indicated on an age specific basis during these years. I know that some people have calculated this, using earlier normal rates, to see what kind of excess there would be throughout life, but I should think one should extrapolate the normal incidence line and get the absolute increase in incidence on that basis.

BRILL: Table 4 illustrates that point, except it's a little old. It shows the expected lifetime incidence based upon the rates that were current in Japan between 1950 and 1958 for Hiroshima and Nagasaki. The total number of cases is shown at the bottom, 6.14 for Hiroshima and 2.64 for Nagasaki; a total of 8.78 cases thus was expected in the survivors in the sample exposed under 1,500 meters in their entire lifetime.

If the rates changed from 25 cases per million per year, as were experienced in those times, up to 100, that would be a fourfold increase which would raise it to 32 cases.

In this small period of time to which has been added additional cases, there were already 48 cases. So I don't think that aging alone will account for the entire rate.

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* R. Doll, Personal communication.

Table 4. Confirmed leukemia cases observed in master sample proper group exposed under 1,500 meters only for 1950–1958 and number expected in lifetime of group by city and age at time of bomb (Reference 82).

Age ATB	Hiroshima		Nagasaki	
	Observed Cases[a] in 1950–1958	Expected Number of Leukemia Cases	Observed Cases in 1950–1958	Expected Number of Leukemia Cases
Males				
0–9	4	1.16	6	0.37
10–19	3	0.71	1	0.53
20–39	7	0.55	1	0.29
40–59	1	0.57	3[b]	0.19
60 and over	2	0.05	0	0.01
Total	17	3.04	11	1.39
Females				
0–9	3	0.89	1	0.26
10–19	1	0.65	3	0.56
20–39	7	0.89	0	0.32
40–59	5	0.63	0	0.10
60 and over	-0	0.04	0	0.01
Total	16	3.10	4	1.25
Both sexes	33	6.14	15	2.64

NOTES:
[a] Includes three cases of leukemia with onset in 1958 not listed in TR-02-59.
[b] Includes one case with unknown date of onset, diagnosed in 1954.

CASARETT: No, but do you think that it would say in the final analysis that there was a certain increase in absolute incidence and that the rest was displacement in time to an earlier time?

BRILL: There certainly could be a superimposition of both accelerated onset plus induction of new cases that would not have ordinarily been expected in this particular population.

CASARETT: Conceivably, that radiated population incidence line could even cross and go below the normal incidence line if you had exhausted the susceptible population in the irradiated group.

BRILL: I think that with continuing follow-up such as is going on, this would be detected if it occurs and, of course, will be of great interest if true.

CASARETT: The point I wanted to make was that perhaps we think that many more cases are new cases of leukemia in this population than is actually so or will be true when we see the lifetime figures.

FREMONT-SMITH: You mean they are accelerated rather than new?

CASARETT: Yes.

ABRAHAMSON: Do you find this in an experimental population of animals, that you have a total number of cases being essentially the same but they are pushed forward in time?

CASARETT: In life shortening with total-body radiation, this is a large effect, that is, a shift in time.

ABRAHAMSON: I'm not referring to life shortening; I'm referring to some kind of leukemia.

CASARETT: I say when total-body irradiation has shortened life, there is a large tendency for diseases to occur earlier, with relatively little increase in incidence of many of these diseases on a lifetime basis, which is essentially a basis for the life shortening.

JABLON: How do you calculate incidence? Are you talking about the proportion of animals who ever develop a certain disease or age-specific incidence rates, or what?

CASARETT: The total percentage of incidence in whatever numbers you want to use.

JABLON: I contend that it is not reasonable to analyze the experience in terms of the percentage of animals that develop a disease. This is a problem that some refer to as "competing risks"; that is, if an animal is subjected to a procedure that kills it while it is still

young, that animal has no chance to manifest any disease which is characteristically a disease of old age. In such a situation, it would not be very informative to say, for example, that mice sacrificed at two months have a lowered frequency of senile changes. The most informative way of examining these questions is to study the age-specific incidence rate for the disease in question. I'm not too familiar with all of the animal work, but I have never seen any results of animal experiments which showed, following radiation, an increased age-specific incidence at younger ages and a lowered incidence at more advanced ages. This is what you would expect if the effect of radiation were merely to move forward in time the occurrence of disease that was due to strike at an advanced age.

UPTON: We have done this sort of thing (see Figure 37). In the mouse, which has been studied more widely from the standpoint of leukemia than any other species except man, the predominent form of leukemia, using leukemia in a broad sense, arises in the thymus as a tumor of thymus lymphocyte-like cells (Reference 85). We call this a thymic lymphoma. If we plot the age-specific death rate against time after irradiation, when radiation is administered early in life, the rate rises earlier in irradiated mice, in relation to the dose (Figure 37). After exposure to 450 rad, the peak probability of this disease is reached early in life, within six to eight months after irradiation, followed by a diminished probability. At a lower dose, the peak is not so high, and the incidence remains elevated. So that at 200 r, there is a higher probability of the disease essentially throughout life than in the controls.

BRILL: Are those significant inversions?

UPTON: Yes. One clearly is.

BRILL: I mean, at the next portion, is the 200 curve significantly higher than the 450 curve?

UPTON: I believe so. I would not be able to answer categorically. I believe it is.

AUXIER: Art, in following up Seymour's observation, though, does the experimental curve ever cross the control curve? Is it ever lower?

UPTON: In our experience this is not definitely true with the thymic lymphoma. It is not at all the case with granulocytic leukemia,

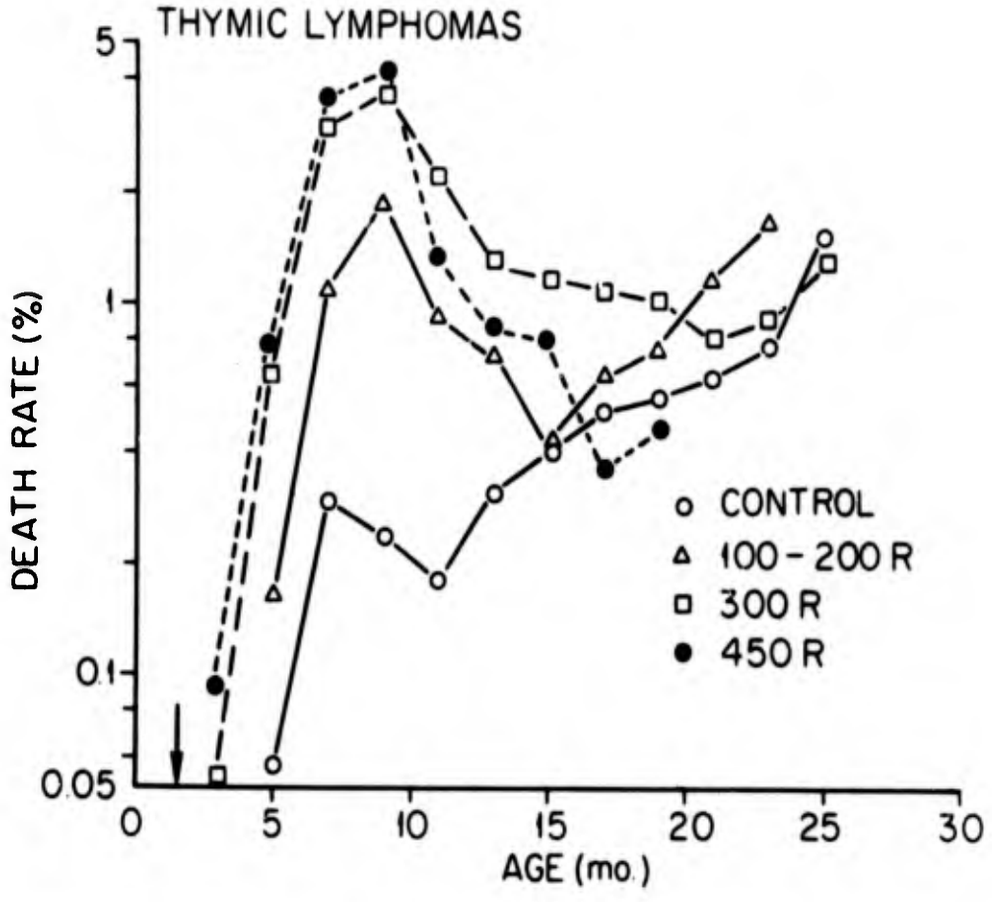

Figure 37. Age-specific death rate from Thymic lymphomas in RF male mice, as influenced by whole-body x-irradiation (Reference 85). Arrow denotes age at exposure.

which is a different kind of disease. The latter is a neoplasm of granulocytes, which are formed in the marrow. Again, our control data suggest a biphasic pattern, but the 450-r curve stays up, 200-r stays up, and 150-r does likewise (Figure 38). So that in these cases the incidence is clearly well above the control incidence throughout life.

CASARETT: These diseases, one of them, and possibly both, have a viral pathogenesis, do they not?

UPTON: Yes, at least many cases of these leukemias can be transmitted with cell-free filtrates.

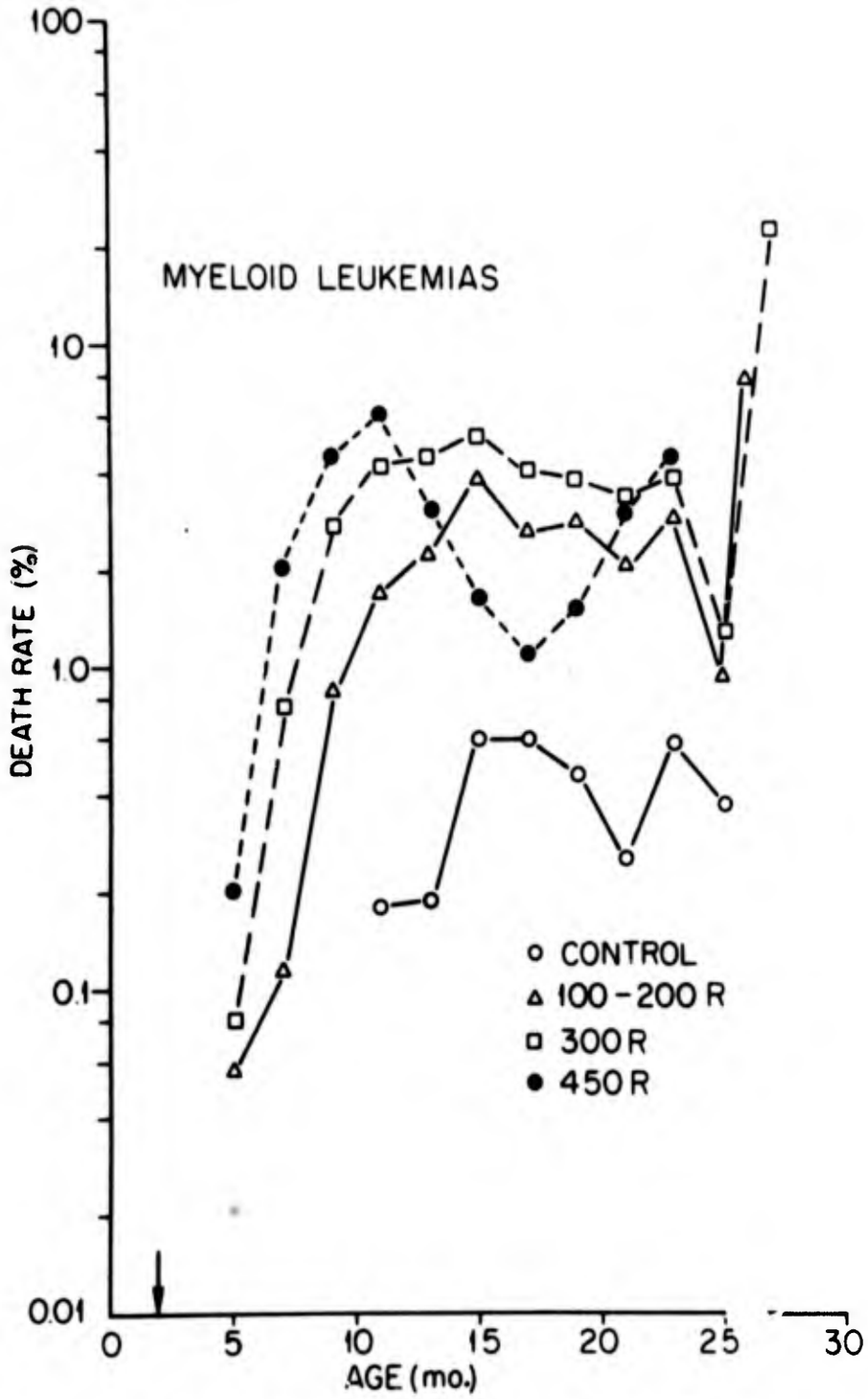

Figure 38. Age-specific death rate from myeloid leukemias in RF male mice, as influenced by whole-body x-irradiation (Reference 85). Arrow denotes age at exposure.

WARREN: Are you saying that if you have 1,000 controls, you have 900 die of leukemia and if you have 1,000 each in the other three samples, you still have 900 out of 1,000 die of leukemia, but at different intervals?

UPTON: I think in the case of the granulocytic leukemias, the curves of which are shown now, if we had 1,000 animals, we would lose 40 to 50 with granulocytic leukemia in the non-irradiated population. In the irradiated population, we would lose up to 30 percent, 300 or 400, from leukemia. The incidence in the 450-r group drops late in life below that of the 200-r group but not back down into the control range.

WARREN: Because you don't have enough left over, or what?

UPTON: No. This type of incidence is corrected for tne number that fail to survive. This denotes the death rate from leukemia among survivors at various ages. Beyond a certain point in time there is a higher probability that an animal surviving 200 r will develop the disease than would an animal surviving 450 r.

MILLER: I wonder if George can argue that you are bringing into the animal's life-span leukemia that is normally beyond his life-span; that if man lived to be 120 years of age, then that's how many cases of leukemia would have occurred. [Laughter]

CASARETT: This might be argued. I think my main point was that the absolute incidence in a lifetime is of some importance in relation to the question that Dr. Upton brought up at the outset of this meeting, namely, the distinction between nonspecific deterioration that shifts most diseases to the left and accounts for life-span shortening without any particular cause of death as compared with a true induction of specific causes of death, such as might be identified as a high incidence of leukemia. I think both are important if you wish to identify them. There is no reason to think that radiation can induce disease but cannot move it to an earlier time.

COURT BROWN: You are accepting this hypothesis of aging as an established fact. I don't think there's any evidence.

CASARETT: I'm talking about the well-known shift in time of disease without necessarily an increase in lifetime incidence in diseases. Dr. Upton put up a particular type of disease as a case in

point, but there are other instances from the Greenhouse experiment (Reference 86) and in the Lindop and Rotblat studies (Reference 87).

COURT BROWN: I think they are suspect.

UPTON: I suppose really the question we are trying to decide is how to evaluate the observations in the Japanese population. Have we seen essentially the bulk of the induced leukemias? I remember very well about 10 years ago when the first peak had passed, many people argued that this was the end of it, that in a couple of years the incidence would come back into the normal range. The second peak came as a surprise to these individuals. One may ask the question now: Will there be another peak or will the incidence persist in its elevation throughout the lives of those who were exposed? What do the data in fact mean?

BRILL: This is always a nasty question. We were concerned as to whether the rise between 1957 and 1959 was due to the fact that we were working so hard to find cases again and were actively pursuing the leukemia inquiry. This next peak comes again when people are hard at work. I really don't know that it has anything to do with it, but one is always concerned about the adequacy of case finding in this kind of circumstance, the concern being the small parts rather than the high parts.

MILLER: There may be other factors involved besides radiation, and when these occur again, another increase will take place.

What was the lowest dosage at which an increase in leukemia was clearly apparent?

BRILL: In the Hiroshima-Nagasaki data, it is approximately 80 rad.

MILLER: So it is well below 200?

BRILL: Yes.

UPTON: Incidentally, wondering about Dr. Court Brown's observations on the spondylitics, is it true in your experience that there is no longer a detectable increase in leukemia incidence after 15 or 20 years?

COURT BROWN: That's perfectly true, yes. It certainly looks very much as if after 15 years one is back down on to the natural risk level.

JOHNSON: Latency in your study was five years?

COURT BROWN: The peak risk period we found among the spondylitics, from about five to seven years, was not very different from the findings in the Japanese survivors. There are differences, however, between our spondylitic data and the Japanese data. One important difference is that in comparing adults with adults we found evidence that the risk of developing leukemia was, for a given dose, greater in older persons than in younger persons. This is not apparent in the Japanese.

I don't know the explanation, but I have always wondered about what degree of bias there may be in the Japanese data due to the fact that very large numbers of exposed survivors died within a year or so of exposure. One wonders whether there were selection factors operating at this period in such a way that many of those who would have developed leukemia later died at this time, and perhaps preferentially older people.

JOHNSON: Don't you think that you are limited by the age at which people develop spondylitis? In other words, they were well into their 20s or 30s before they developed a disease for which they received treatment? So that you do not have a sample which is less than 30 or 26 years of age?

COURT BROWN: Yes.

JOHNSON: So that you don't know what would happen at a younger age.

COURT BROWN: We know what happens at an older age. This is the point. When you take men of 45 or 50, they appear, for a given dose, to have a much higher risk of developing leukemia.

JOHNSON: Yes, and spontaneously they would have a higher risk of developing leukemia.

COURT BROWN: Yes.

JOHNSON: And radiation has been added.

COURT BROWN: Yes, but this doesn't appear in the Japanese data.

UPTON: I looked at the data that Randy published and got the distinct impression that if one analyzed the rate of leukemia by hematological type, the incidence in the radiated population was clearly related to age at exposure (Reference 82), very much in the same way as was the total incidence in your series. Thus I had the impression that radiation was not inducing a given number of cases irrespective of age but rather multiplying the natural age-dependent rate by a given amount, which was essentially constant throughout all ages.

BRILL: Except in the younger groups, which perhaps had the highest increase.

UPTON: Yes. There was the suggestion that they were somewhat higher, but in general the rate seemed to parallel the natural rate.

BRILL: But the thing that stands out to me in the ankylosing spondylitis data is that they had a remarkably increased rate of induction in the very oldest groups, which we didn't see. It seems to me that it is in this group that they had a disproportionately large increase in incidence.

COURT BROWN: You had a relatively low increase! [Laughter]

MILLER: There is an interesting thought in regard to radiation leukemogenesis. Radiation can induce several types of leukemia—acute lymphocytic, acute granulocytic, or chronic granulocytic—but it notably cannot induce, so far as we know, chronic lymphocytic leukemia. It has not been seen in Japan and it has not been seen in Dr. Court Brown's study of ankylosing spondylitis treated with x-irradiation.

COURT BROWN: I think perhaps one still should be cautious about this topic. I agree that there is no evidence that radiation can cause chronic lymphatic leukemia in man, but we are now learning that the lymphocyte is a much different cell from what it was once thought to be, certainly in terms of its mean life-span.

WARREN: I've heard mention of the thymus, but has anybody mentioned the other phases? What about aplastic anemia and

thrombocytopenia in the Japanese population? Has there been any indication that thymic cells have been damaged and have long-term chronic effects developed?

JOHNSON: There is indication of an increase in lymphoma and myelofibrosis (Reference 88).

MILLER: But not aplastic anemia?

JOHNSON: Aplastic anemia is a very broad, poorly defined term.

WARREN: How about anemias generally?

JABLON: In Nagasaki, a significantly increased number of deaths were attributed to anemia on death certificates. These were usually called aplastic anemia, or sometimes "A-bomb" anemia on the certificates. When we reviewed these deaths, it turned out, in almost every instance, that if the patient had been seen at ABCC, a diagnosis of leukemia had been assigned. The conclusion was that the reports from Nagasaki of an increase in aplastic anemia were caused by diagnostic confusion.

BRUES: The differentiation between acute leukemia and aplastic anemia is a difficult one without more study than a great many people get. That's the point. Figure 39 almost bears on one of your points; it shows that elsewhere than in Japan leukemia has been going up in the past couple of decades. The figure is from Loutit's book (Reference 89*) and shows the age-specific incidence of acute leukemia in the United Kingdom. †

JABLON: Acute?

BRUES: Yes; the same is true for the chronic leukemias.

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*Reproduced from an earlier publication by Court Brown and Doll (Reference 90).

†Subsequently, Dr. Brues commented, "My attention has been called to a later publication on the same subject: W. M. Court Brown, R. Doll, and I. D. Hill, 1964. Leukemia in Britain and Scandinavia. Pathol. Microbiol. (Basel) 27: 644-654."

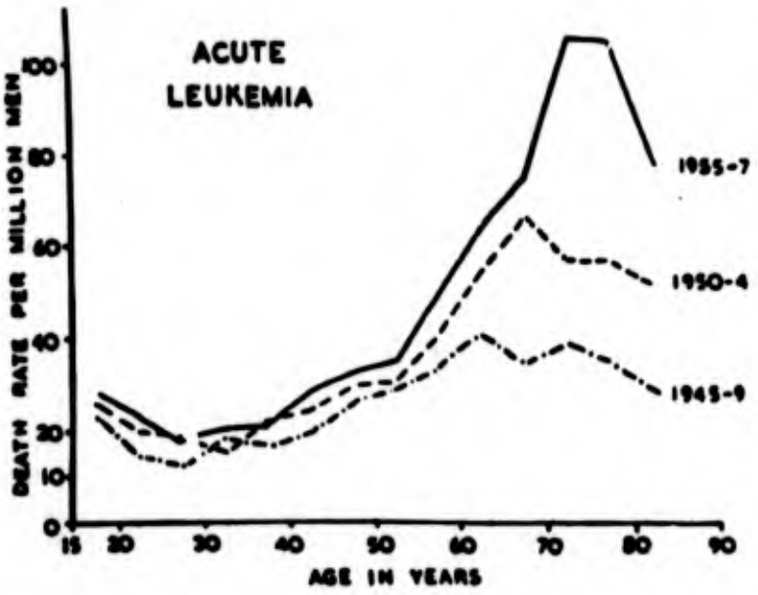

Figure 39. Graph of death rates from acute leukemia in adults in the United Kingdom, distributed by age. The three lines illustrate the increase in death rate during period between 1945 and 1957. From Loutit (Reference 89). (Reproduced with permission from the British Medical Journal.)

MILLER: May I show a figure about a period that you missed in Figure 39; that is, Figure 39 began at 15 years of age, I think, or at least you grouped them that way.

BRUES: Yes, the earlier leukemias are not shown here.

MILLER: Figure 40 concerns U. S. leukemia mortality rates in children, and the interesting feature with respect to radiation-induced leukemia is that among the white children in the U. S., 1950–1959, there was a tremendous peak at four years of age, but for the nonwhite children in the U. S., in the same time period, there was no hint of a peak (Reference 91). The peak among white children was apparently due to acute lymphocytic leukemia; the rates for the granulocytic form of the disease were about constant throughout the early years of childhood. This peak was not always present. It has emerged since 1940 in the United States. In Great Britain, Drs. Doll and Court Brown have pointed out that it emerged about 20 years before

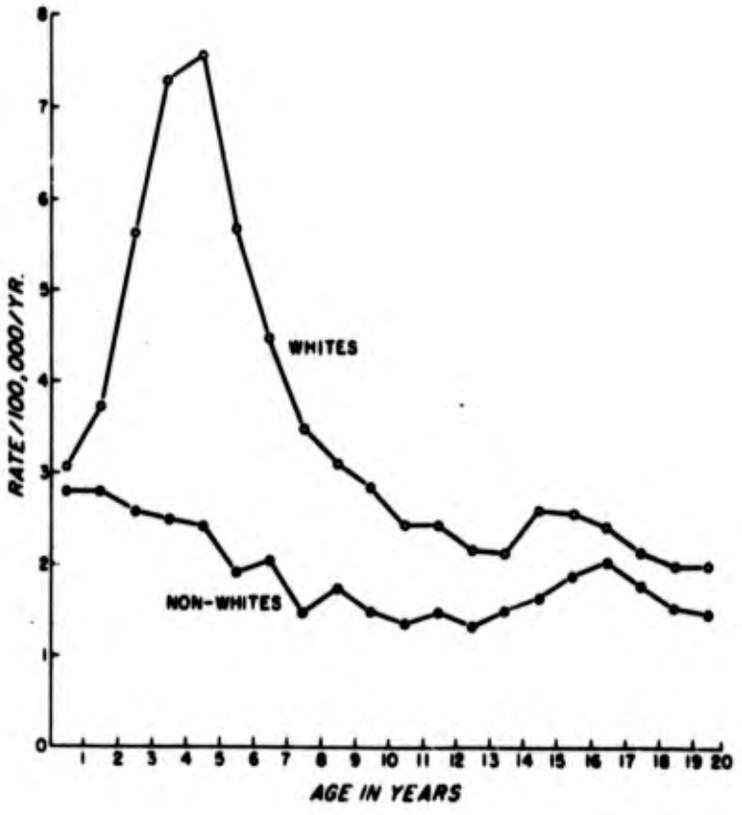

Figure 40. U.S. childrooh-leukemia mortality rates, 1950–1950, white versus non-whites. (Reproduced with permission of the Yale Journal of Biology and Medicine.)

that (Reference 92). It has been supposed that the United States white children were either exposed or susceptible to some leukemogen to which the nonwhite children were either not exposed or not susceptible. It is of interest that the peak, which resembles that which occurred following atomic bomb exposure, occurred about five years from the time that the child was in utero.

FRANK: Could that include pelvimetry?

MILLER: It seems unlikely, because everybody in the country would have to have had pelvimetry in order to produce a peak of this magnitude, because pelvimetry only doubles the rate of leukemia at most.

COURT BROWN: We have done a detailed analysis of leukemia mortality in Britain. What has consistently emerged is that the crude mortality is increasing, and a good deal of this increase is due to an increase in recorded deaths in the elderly from chronic lymphatic leukemia. This, however, could be due to more efficient case recognition. But when we come to look at what is happening in the middle part of life, let us say from 30 to about 55 years of age, we also find an increase with time, but in this instance the increase appears solely in the deaths from acute leukemia and chronic myloid leukemia. I suspect that there are many factors in our environment which are leukemogenic like radiations, and it may be the operation of these agents which is responsible for these changes in mortality seen in middle life.

FRANK: I have heard rumors, though, that other diseases are going up now in the population in Hiroshima, other types of cancer. Since immunity mechanisms must be depressed for awhile after radiation, is there any evidence that chronic infectious diseases are going up? You mentioned that leukemia may have a virus component.

UPTON: Dr. Johnson, would you like to tackle this one?

JOHNSON: Mr. Jablon has already mentioned the high rate of mortality from tuberculosis in our sample during the years prior to current antituberculosis therapy.

FRANK: Was that within the women as well as the men? I thought it was just the group of men.

JOHNSON: More men than women.

As to the role of radiation in inducing altered immunologic mechanisms, there is no direct evidence at hand. This is not to say that the immunologic mechanism was not damaged, but simply that we are not aware of its expression in our sample.

There is one bit of evidence in the work of Kanamitsu (Reference 93). Persons who were very young when exposed to the A-bomb should

have had antibodies to influenza A during the pandemic of A1 influenza in 1945. When challenged with a different type influenza vaccine, they did not respond anamnestically—in the manner of the doctrine of original antigenic sin (Reference 94) as did control subjects.

In reference to neoplasms we now have good evidence that thyroid cancer, especially in women exposed within 1,400 meters, has developed excessively. This was noted previously by Hollingsworth (Reference 95), Socolow (Reference 96), and most recently by Wood (Reference 97). Dr. C.K. Wanebo (Reference 98) had preliminary data indicating that there is an excess of carcinoma of the breast and lung in survivors exposed within 1,400 meters.

BRILL: Do you have equal confidence in the thyroid cancer, and the lung and female cancers being increased? It seems to me there was very good evidence with respect to the female genital cancers and perhaps with respiratory cancers, whereas I've never been completely happy with the thyroid cancer story. Are you now?

JOHNSON: Yes.

BRILL: Have there been more cases than initially appeared?

JOHNSON: Yes.

ANGEVINE: I think the type of cancer should be stressed. In the series we analyzed (Reference 99), 20 out of 21 did not kill the patient. Many were incidental small sclerosing carcinomas somewhat analogous to occult carcinoma of the prostate and for these carcinomas, in large series of cases that have been carefully studied, there is a very favorable prognosis. Pathologists seeing them for the first time wonder whether they are truly malignant tumors. The problem is somewhat analogous to sclerosing adenosis of the breast. I think this should be recorded because out of this group of 21, I think there was only one with widespread metastases. The follow-up, of course, may be different.

JOHNSON: Yes. We now have many more cases. As to pathologic type, there is a great variety: papillary carcinoma, follicular, occult sclerosing. Several have shown evidence of vascular invasion or invasion of the capsule. One had metastasized to local lymph nodes.

I think the situation now is much more clear than when you were last reviewing the first dozen cases. I have confidence that cancer of the

thyroid has occurred excessively in survivors and that the larger collection of cases has given the pathologists less difficulty in interpretation.

WARREN: You seem to have a lot of tumors. How about the gastrointestinal tract?

BRILL: Before we leave the thyroid, just for a second, are the cases cited in the Life-Span sample? Are they from autopsies or biopsies?

JOHNSON: These cases are in the ME-200 sample and have all been examined most recently by Dr. Wood. Diagnosis is based on histologic evaluation.

UPTON: Those who are not initiated into this terminology might be confused about the samples you're talking about. Could you define these for us?

JOHNSON: I'm sorry; Dr. Brill is an ABCC alumnus. The ME-200 is the portion, the 20,000 persons, of the Life Span Study, examined biennially in the ABCC clinics.

AYRES: Could I ask an immunological question? Have you been keeping track of hepatitis?

JOHNSON: Yes, we have. Hepatitis is a very common disease in our clinic sample, affecting about 6 percent, according to a recent, yet unpublished, study by Gregory, et al. As yet we have drawn no association between hepatitis and exposure status.

WARREN: Is this an infectious hepatitis?

JOHNSON: It is a peculiar type of hepatitis seen in Japan, Taiwan, and other places in Asia. Frank jaundice is usually absent and it is referred to as anicteric hepatitis. We suspect its presence when we detect liver tenderness and elevated serum transaminase levels. It produces easy fatiguability but is not a serious cause of morbidity.

LIFTON: Could you say more about what you mentioned before—the preliminary data on cancer of the breast, ovary, and lung? That is of great concern among the Hiroshima population now.

UPTON: I would like to exercise the chairman's prerogative, if I may. We have almost reached adjournment time, and I think there is undoubtedly a desire to pursue the discussion of tumors other than leukemia. I wonder if it would not be helpful, before we stop now, to explain a little more clearly what the Adult Health Sample is.

JABLON: The entire closed sample consists of about 100,000 persons as of October, 1950, the date of the Japanese national census, from which the bulk of the selection was made. About 75 percent of the 100,000 are Hiroshima cases and 25 percent are from Nagasaki. The Life Span Study, which is a study of mortality and survival based on procurement of death certificates through the regular Japanese vital statistics procedures, uses the whole closed sample of 100,000, and this group is also the basis for the autopsy studies in which an attempt is made to obtain an autopsy on every person among these 100,000 who dies, no matter where. Dr. Matsumoto can probably tell us what the procurement rate is at the present time.

MATSUMOTO: Around 40 percent.

JABLON: The basis for the Adult Health Study to which Dr. Johnson referred is a subsample of about 20,000 persons selected from the 100,000 (References 78 and 100). The relation of the part to the whole, by distance, is shown in Table 5. The sample members

Table 5. Relation between sample for JNIH-ABCC Adult Health and Life Span Studies (Hiroshima and Nagasaki).

Distance from Hypocenter (meters)	Life Span Study Total	In Adult Health Study	Not in Adult Health Study
Total	99,389	19,962	79,427
Less than 2,000	28,142	9,980	18,162
With acute symptoms	4,993	4,993	—
Without acute symptoms	23,149	4,987	18,162
2,000–2,500	16,663	—	16,663
2,500 and over	28,010	4,990	23,020
Not-in-city	26,574	4,992	21,582

of the Adult Health Study are invited to come for examinations every two years. As you can see from Table 5, the subsample was not selected at random from the larger group, but was more heavily concentrated among persons who were within 2,000 meters of the hypocenter. Within the Adult Health Study, about 14,000 are Hiroshima subjects and about 6,000 are from Nagasaki.

BRUES: Can you say then that the Adult Health Study is one in which the data are the best but the numbers are smaller than in the larger series, where the data are not as complete nor as precise as to the pathology?

JABLON: It is true that autopsy procurement is more successful among the Adult Health Study subjects, but pathological examinations extend to the whole sample. Probably procurement is better in the Adult Health Study group because of their continuing contact with ABCC, but the procurement rate is pretty good for the others also. What you do have for the Adult Health Study group, and not for the others, is a physical examination every two years, and a knowledge of the diagnoses that a physician in the clinic could assign.

BRUES: Don't you also have a tumor registry? Is that still something else?

JABLON: The tumor registry is something else; it is an attempt to learn of every tumor diagnosed within the cities of Hiroshima and Nagasaki. Subsequently, one would determine for each notified tumor whether the subject was a member of the samples being studied, but the initial attempt is to register them all.

WARREN: From this you would get your controls?

JABLON: No, sir. Controls are included within the samples.

WARREN: They are in there already?

JABLON: Yes, sir.

FRANK: Within 2,000 meters or beyond 2,000 meters, what was the control?

JABLON: We have two control groups. The study samples themselves were thought of as persons who were within 2,000 meters of the hypocenter at the time of the bomb, because beyond that, distance

radiation was then estimated to be about 5 rad or less and is now thought to be even smaller. Those who were between 2,000 and 2,500 meters are not included at all in the Adult Health Study and were originally thought to represent a marginal group for the Life Span Study. The control groups are those persons beyond 2,500 meters (actually 3,000 for the Adult Health Study) and additionally those who were not in the cities at all at the time of the bombing. These controls are all parts of the sample as shown in Table 5.

COURT BROWN: I would like, if I may, to say something more about the mortality experience of the spondylitics, because I think that's very relevant and that one ought not to just consider the survivor data in isolation but it should be considered in relation to data from other radiated populations.

We have a population of about 14,000 of these men who are defined as men who were treated with x rays for spondylitis at any one of the British radiotherapy departments between 1935 and 1954. Unfortunately we do not have the most adequate control for these men, that is, men not treated with x rays. This is because x-ray treatment has been so popular in the past that few men escaped it.

The latest follow-up data indicate that out of the 14,000 men, we would have expected, on the basis of the relevant national mortality data, some 866 deaths. In fact we found nearly 1,600 deaths, that is, near double the expected number.

In assessing these, we have to try to take into account the effect of the disease itself and this, of course, is one of the great problems of working with patients suffering from a particular disease. We have to try to take account of peculiar mortality experiences which may be associated with the disease. Quite a proportion of our deaths is recorded as due to ankylosing spondylitis and arthritis and other forms of rheumatism. In fact, there was a 100-fold increase over what we would expect in deaths ascribed to these causes.

Then there's another lot of deaths which are due to conditions which we believe to be associated with the disease; for instance amyloid disease, ulcerative colitis, nephritis, one or two heart conditions, and so on. Deaths from these conditions, associated with spondylitis, were increased about threefold.

Then we come to the very interesting group of deaths from leukemia and aplastic anemia. Deaths from leukemia were increased 10-fold

in our population; deaths from aplastic anemia or deaths ascribed to aplastic anemia increased nearly 30-fold but many of these were misdiagnosed cases of leukemia.

The problem of other conditions has now to be considered, and here the spondylitics are becoming very informative in regard to deaths from cancers other than leukemia. The main method of treatment in our population was irradiation of the spinal axis, and in discussing cancer deaths we can talk about cancers in heavily irradiated volumes of tissue and those in lightly irradiated volumes.

Our total deaths from cancer are considerably increased. Taking deaths from all types of cancer but excluding deaths from leukemia, we expected 180 deaths during the period of observation up to 1960, but found 260. Considering tumors from heavily irradiated sites only, we expected 127 deaths and found 200, while there was no important difference between the expected and observed numbers of deaths from cancers arising in lightly irradiated sites—53 expected and 60 observed. The increased number of deaths in the heavily irradiated tissues were contributed to by such common tumors as those of bronchus, stomach, and pancreas.

BRUES: Any bone tumors?

COURT BROWN: Yes. We got an increase in osteogenic sarcoma occurring in the spinal column; not very many. You don't need many cases to get an increase in this.

BRUES: What was the highest dose?

COURT BROWN: Offhand I can't remember.

WARREN: A couple of hundred r?

COURT BROWN: Far more than that. We are dealing with very much higher doses.

UPTON: Could you give a range, please, on the dose?

MILLER: I can remember; it was 400 to 2,500 r.

COURT BROWN: This is a very high dose.

BRILL: That is for the people who received only one course of therapy, isn't it?

COURT BROWN: No. These might range from people at one course to people who had multiple courses of treatment. We estimated the mean spinal dose to range from about 400 to 2,500 rad. Actually about 1 percent of the latter group was dying per annum from leukemia, a very big increase.

To come back to the problem of other types of tumors, I think the indications are quite strong from the spondylitis experience that certainly irradiation to these levels of doses with medium kilovoltage x rays is probably increasing the mortality from all types of cancer. Also, there is a sufficient indication to show that, as one would expect, the latent period distribution is going to be different from that of leukemia. Whereas the peak risk period for leukemia is around five to seven years after exposure, the special mortality risks for other forms of cancer are not seen until about seven years after exposure, and are still increased after about 20 or more years.

BUSTAD: To keep the record straight, you had some that were over 2,750 r. I know you had five cases reported some time ago and that group are over 2,750 r.

COURT BROWN: Yes. What we were talking about were mean spinal marrow doses and 2,500 r is an enormous accumulation of spinal marrow.

BUSTAD: Spread over many years?

COURT BROWN: Spread over many years, several years.

BRILL: One of the things that's striking is the close agreement in the rates of leukemia production per rad in the ankylosing spondylitis patients and in the Japanese experience, despite the very wide disparities in dose rates involved in the two exposures.

COURT BROWN: You can show the same sort of risk per rad for both the spondylitics and the bomb survivors.

BRILL: Yes.

COURT BROWN: But I am a bit dubious about that type of calculation. It certainly maximizes the pessimistic side of this situation

and is very valuable from the point of view of thinking in terms of radiation protection, but it may have not too much meaning, perhaps, in a biological sense.

BRUES: I would like to raise a point about the use of the term "dose rate." The instantaneous "dose rate" at which radiation is given at any sitting may be high, while the average "dose rate" over a period of years may be low.

COURT BROWN: Yes.

BRILL: I meant to preserve the distinction between fractionation and dose rate.

BRUES: Yes.

BRILL: I seems to me that the A-bomb exposures involved many thousands of rad per second as opposed to rad per minute from conventional radiotherapy.

COURT BROWN: Yes. We're talking about something of the order of 60 rad/minute on the skin's surface.

MILLER: The similarities between your study and the ABCC's were that the peaks occurred at about the same time, there was a dose-response relationship, and the same types of leukemia were induced; but in your study you are finding other cancers in excess of normal expectation in the field of radiation, a consequence not demonstrated at ABCC.

COURT BROWN: Yes, this is true and there was a difference in terms of age and risk.

LIFTON: We started before to answer those questions about the preliminary data at the ABCC, and I'm still interested in hearing about them. In other words, how important is it? How definite is it?

JOHNSON: Our experience is quite similar to Dr. Court Brown's and Dr. Doll's in that pulmonary cancer, for example, has developed in the survivors who were within 1,500 meters, 2.48 times more in men and 2.02 times more in women than in the control groups. These ratios are based on Dr. Wanebo's collection of 54 cases which have occurred in the Adult Health Study sample.

UPTON: Could you describe these rates for us, please? I'm not sure I have it straight.

JOHNSON: For the period 1958-65, the case rate per thousand persons per year would be for males exposed within 1,500 meters about 2.8, and about 1.4 for males exposed at greater distances and not in either city in 1945.

UPTON: What's the significance? Do you have an idea?

JOHNSON: The significance?

UPTON: The level of statistical significance.

JOHNSON: We have't gone quite so far, but I think the differences will be significant at least at the 5 percent level.

BRUES: It will be necessary to continue for a short while to pick up some loose ends in connection with the somatic effects of the nuclear weapons at Hiroshima and Nagasaki.

I would like to pick up at least one loose end, in connection with our discussion of neutrons, gamma rays, relative biological effectiveness, linear energy transfer, etc., because I think some of the group may have been left a little bit behind in that discussion and it is worth understanding.

First I would like to show a concept of an atomic bomb as it was presented in an early picture in a Japanese magazine (Figure 41). This is a U-235 weapon and illustrates how, by implosion, the fragments are brought together. These things give off both gamma rays and neutrons and depending on the design, they may give out different proportions of the two, as Dr. Auxier mentioned yesterday. The basic point is that the gamma ray moves with the speed of light. It is a photon and it leaves bits of energy very dispersed. A neutron, as Dr. Auxier said, displaces a proton with a great deal of recoil energy and the proton, having mass, moves slowly and deposits its energy in a dense short track. If a small amount of energy, like that expended in a single ionization, is enough to produce an effect, it doesn't matter whether the ionizations are dispersed or close together. But, if it takes more than one in a small region to produce the effect, the dense short track is more efficient. For such an effect, the relative biological efficiency of neutrons as related to gamma rays is high.

Figure 41. Diagram of U-235 weapon as imagined by Japanese artist. Picture from an unidentified popular Japanese magazine.

The breaking of a chromosome, which would require more than one area to be hit in the same region, is therefore something which is much more efficiently done by neutrons. As to the instantaneous dose rate, if this is high, the dispersed ionizations will be more effective than if it is low, but with dense ionization tracks it makes less difference. This is because the "multihit" effects require that the hits be close together in time as well as in space; otherwise small lesions may have time to heal. An extreme case of the large, dense track is that of heavy cosmic ray particles at high altitudes, where mice with pigmented hair develop visible patches of white because a large area of hair follicles is affected by such a particle.

WARREN: Excuse me. How about writing down RBE and LET, and defining them again for us?

BRUES: RBE is relative biological effectiveness, and is often a misused term, for when you say that a neutron or an alpha ray has a certain relative effectiveness, you must say relative to what. There are differences in the effectiveness of the ordinary voltages of x rays and of gamma rays on some systems, so one has to specify which. Also the RBE differs, depending on the biological effect.

ABRAHAMSON: There is good evidence for certain systems that x rays are more efficient than gamma rays with respect to inducing genetic disorders.

WARREN: It has a lower RBE.

BRUES: Yes. The gamma-ray track is not quite so concentrated as the x-ray track if we refer to the gamma rays that we usually encounter, which are of high energy, and the x rays ordinarily used in experimental or diagnostic work which are of lower energy.

The biological effect may be very important. For reasons we don't understand, the dense ones are much more effective in producing cataracts.

For producing acute lethality, where it depends on damage to the bone marrow or intestine, and probably also in the production of tumors, the RBE factor between fast neutrons and gamma rays is between 2 and 4. For cataracts it may be around 8 or 10.

FREMONT-SMITH: Do I gather then that RBE used by itself is a meaningless term and it only becomes meaningful when you define it with respect to a particular situation, in which case you might just as well define the particular situation and leave RBE out? Am I right?

BRUES: Yes, except that RBE has been useful in protection work where one must be arbitrary and choose a suitable factor. In this case there is a tendency to choose a factor which would cover all the effects.

FREMONT-SMITH: You choose it for convenience, but not for reality?

BRUES: Yes.

LET is the linear energy transfer and is defined as the amount of ionizing energy per micron length of track. This is a precise definition, although it may be difficult to ascertain with precision for a given situation, and LET is related to the RBE in a known but not precise way.

As you gathered from the discussion, the Hiroshima weapon delivered comparable doses of neutrons and gamma rays; the Nagasaki weapon delivered much less effective neutron energy than gamma-ray energy because of the difference in the design of these weapons. This might suggest the possibility of determining some differences in the effects. I don't think this has taken place as yet, although there have been some differences in the reported results coming out of the Hiroshima experience and the Nagasaki experience.

In Hiroshima the hypocenter of the bomb was in the congested, highly urbanized "downtown" of the city, including the area of restaurants and entertainment. In Nagasaki the hypocenter was over a combination of residential suburbs and heavy industry, about 2 miles away from the center of the city. There was therefore a possible difference in the environmental differences which are a function of urban geography. Therefore, it may be quite difficult for epidemiologists to detect differences in neutron exposures from the two weapons.

FRANK: Two more terms that keep going around. What was Mev?

BRUES: That means million electron volts.

FRANK: Someone threw beta radiation at me yesterday.

BRUES: A beta ray is an accelerated electron. It penetrates tissue a matter of millimeters or less. Its LET is intermediate between the others I was speaking of, and it ordinarily falls in the same RBE range as the gamma rays.

Alpha rays are helium nuclei which penetrate only a matter of microns, with a high LET. They are not important as external sources but only when an alpha-ray source such as radium or plutonium is deposited in the organism. The neutron is a special case, because it passes easily through tissue, like an x ray, until it runs into a proton and dislodges it; so it is penetrating and yet has a high RBE because of the high LET of the proton to which it gives its energy.

WARREN: Nobody has brought it up yet, but you might as well describe body burden while you're at it. You mentioned alpha particles.

BRUES: The body burden is the amount of a radioelement which is carried in the body. This is not supposed to exceed a certain value to conform to the rules of safety. Will that do?

WARREN: Yes.

BRUES: In the case of body burdens and radiation levels, it is now accepted that a worker with radiation may be exposed to a somewhat larger amount than the general population. This raises some complicated questions. For example, if there were such a thing as a permissible dose of cigarettes, that philosophy would say that a man who makes his living by smoking cigarettes on television commercials might smoke ten times as many as the general population.

COURT BROWN: One point I think worth making in relation to the problem of radiation and tumors in general is, that only does this very low dose of radiation appear to increase the risk of developing leukemia in children, but it would also appear to increase the risk of developing those other tumors which children develop. I think this is another piece of information which has to be considered in relation to the risk of developing other tumors in radiation.

LIFTON: What other tumors are these?

COURT BROWN: I think I'm right in saying, am I not, Dr. Miller, that the main childhood tumors are leukemia and the central nervous system tumors plus tumors of the kidney.

FREMONT-SMITH: Thyroid doesn't come in there?

COURT BROWN: No, no as far as I know.

WARREN: No, but you get there pretty quickly, and that's the point.

COURT BROWN: I think you're begging the question on etiology here.

ABRAHAMSON: I think there was one difference between Dr. Court Brown's data and data that were being presented on the Hiroshima situation.

One of the things that occurred to me was that while there were very high doses being given to the ankylosing spondylitis patients, these were not whole-body doses; they were restricted in general to the spinal cord area. The same individuals, had they received that dose as a whole body dose, would probably have been killed. Therefore if is possible that we did not see these cases, because these individuals never survived to produce this type of tumor.

COURT BROWN: The only other point that I wanted to make was that not every survey of a radiated population has shown up an increase in leukemia and there is one very interesting population which is being studied by MacMahon and his colleagues. It's a very big survey on an international basis of women treated for cancer of the cervix, I think by combined radium and x-ray techniques. This very extensive population, so far as I understand it, failed to yield any evidence of increased leukemia, and this is extremely interesting, a fact which runs counter to all other knowledge.

WARREN: There's not much of the bone marrow exposed in the cervix cases.

COURT BROWN: No. One has to set against this the fact that we have a survey of our own, Dr. Doll and I, which is about to come out, in which we studied a group of some 2,000 women who had been treated with x ray for what is described as benign menopausal bleeding, and various disturbances of menstruation which occur at or around the menopause. Undoubtedly this has thrown up an increase in leukemia and we have to try to relate this to the negative findings in the carcinoma of the cervix group.

FREMONT-SMITH: Isn't that closer to the ovary than to the cervix and would that make a difference?

COURT BROWN: No. They were treated by a system which irradiates a central block in the pelvis including the cervix and the uterus.

FREMONT-SMITH: In both cases comparable radiation?

COURT BROWN: No, they are not comparable doses. They are doses that are greatly different and the distribution of the dose I think is very different. In the menopause patients the midline dose to the pelvis was 800 rad of x rays or a bit less, depending on thickness, given is a single exposure.

FREMONT-SMITH: I was wondering whether the difference could be accounted for by the different bottom in dosage and the locality rather than just being a discrepancy?

COURT BROWN: I think the difference is essentially that there is probably quite a small fraction of the target tissue raised to a dose which is not lethal in the cervix treatment. So effectively the risk is not very high.

UPTON: I think that what Dr. Court Brown may have been hinting at is that we really don't know the answer to the question that was developed yesterday, namely, whether there is a threshold for the induction of leukemia. The data that exist are compatible with a linear relationship between overall incidence of the disease in all age groups and dose, as one can derive dose from distance.

WARREN: Do you want to speculate about the fact that, if the radiation of the Japanese population had occurred at another time when there was not presumably an epidemic of leukemia rising anywhere, we might have had a different incidence?

UPTON: I think we simply can't make a decision as yet about the relationship between incidence and dose because of the statistical variables which exist, although taking the data from the spondylitics, one get a reinforcement of the Japanese situation.

I think another aspect of the problem is that if we look at the incidence of leukemia in infants exposed in utero, we see an indication of a greater incidence for a given dose. The doses were smaller but the ostensible effects are even larger. This then brings into the picture again the question of the relationship of age at the time of irradiation and susceptibility. It would seem, as we mentioned, that radiation probably is inducing a given increment in the natural rate of the disease rather than a given number of cases irrespective of the age at exposure.

The next question, of course, is whether leukemia induction is a unique situation. Is it the only neoplasm to be expected? And we know the answer already. It is not. There are data suggesting that other neoplasms are also induced in the irradiated population, both in the spondylitics and in the Japanese. Again, however, we really con't know how general this situation is. Solid tumors have a longer latency than leukemia. It may be too soon to decide that the incidence of some tumors is not going to be increased.

WARREN: Although the breast cancers have come up pretty sharply, haven't they?

JABLON: I don't know. We haven't seen those data.

WARREN: I thought that came out yesterday.

UPTON: I would like to ask now if we can summarize again those kinds of neoplasms, the incidence of which clearly seems to be increased in the irradiated populations, and if we can derive from these data anything in the way of a quantitative assessment, or is the information still largely qualitative?

COURT BROWN: I think that you passed a little glibly over the problem of dose-response relationships for leukemia, and I think that both the Japanese data and the spondylitic data, on which these so-called dose-response relationships depend, have imperfections. I think the Japanese data are in many ways the more useful set of data on which to at least guess at some sort of a dose-response relationship, because here one is dealing with a single exposure and one can follow the irradiated population in time and relate the number of cases of leukemia observed to the population and risk in the different exposure groups. But I think there is a considerable element of doubt about individual doses.

Second, there is this problem that one may have lost an appreciable number of cases who would have developed leukemia because of the early mortality. I think the spondylitic data are rather difficult to interpret. The trouble about the spondylitic data is that a fraction of our 14,000 men, quite a sizable fraction, has had more than one course of treatment. A man may be treated one year and then a couple of years later get another course of treatment for another area, and so on. This posed considerable problems in trying to build a dose-response relationship and we could only get around this

by making what may have been a somewhat artificial postulate at the time, namely, that radiation acted as a mutagen, that is, it produced a single nonrecoverable event. It has to be remembered that all this was 12 or 13 years ago. So that one could, in making this assumption, devise a system for dealing with a situation in which one had repeated doses. What we came up with, as Dr. Brues will remember, is something which was compatible with our assumption, but I suspect that our assumption was wrong. Therefore one has to be a little guarded in deriving too much or trying to derive too much with respect to what may be the effect of low levels of doses from the experience of spondylitics.

The idea originally was that if we could describe a dose-response effect for the levels of dose which these men were getting, then we could extrapolate this to low levels of dose and obtain some indication of the risk of low levels of dose. I think that while the Japanese and spondylitic data are compatible in terms of making a guess at the effect of 1 rad, this is a somewhat artificial guess, and that we have no reason to believe that 1 rad in fact had the effect that we would guess it had.

UPTON: I would like to clarify what I said, since I would not argue at all with Dr. Court Brown's assessment. The point I was trying to make is that from the two sets of data we can no longer feel confident that there are not effects down below 100 rad, and the data from studies on the association between exposures before birth and subsequent incidence of cancer reinforce the belief that there may be effects below 100 rad. I don't think we are in a position, from the data as they exist, to close the matter conclusively.

BRUES: Figure 42 shows what I think is an anomaly. It is derived from some data about 10 years ago by Dr. Eugene Cronkite (Reference 101) showing leukemia rates recorded at that time by distance from the hypocenter and also according to whether the individuals had or did not have acute radiation complaints, consisting, I believe, of diarrhea, nausea and vomiting, petechial hemorrhages under the skin, and epilation.

The anomaly that I find interesting is in the area around 2,000 meters where the radiation doses were too small to be expected to produce these acute radiation complaints; yet most of the persons who later got leukemia seem to have reported these complaints. This makes me wonder whether these people had forgotten where

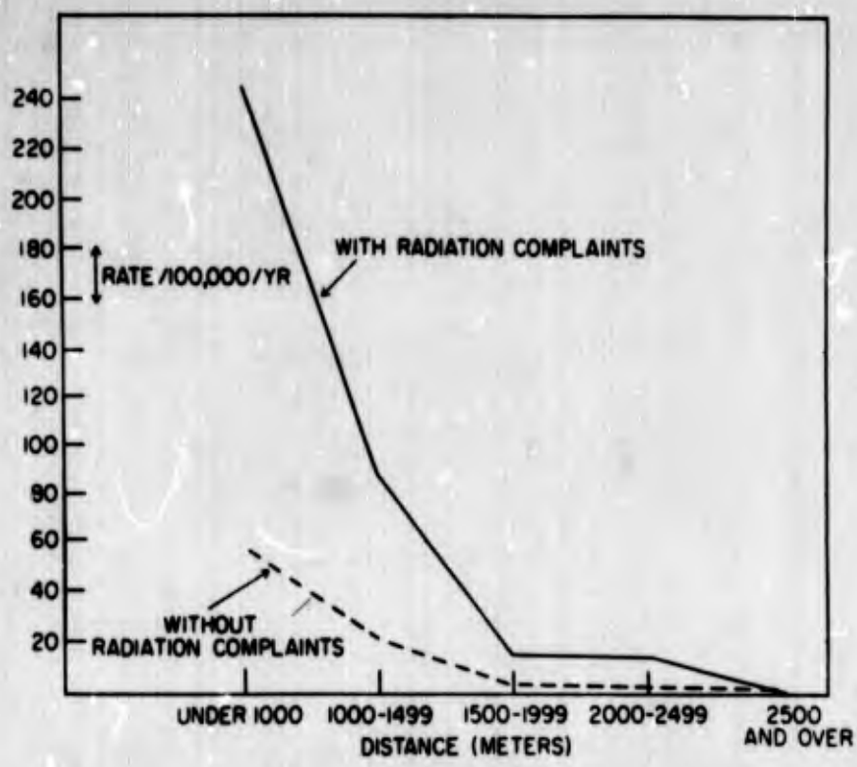

Figure 42. Death rate from leukemia in Hiroshima and Nagasaki in relation to distance from the hypocenter in the presence or absence of acute radiation symptoms.

they actually were, or whether it represents a specially susceptible population to both acute complaints and leukemia, or perhaps that some form of dysentery might be related to the leukemia developing later. At any rate I have wondered about this, since it might raise questions about the validity of the leukemia data in this low radiation area.

MILLER: The question has been raised, what is the relationship between intrauterine radiation exposure and the occurrence of childhood leukemia? The history is that in 1956 Dr. Alice Stewart and her associates published a preliminary report (Reference 102) and later, in 1958, a definitive report (Reference 103) which indicated that diagnostic radiation exposure (very low dose levels) of the maternal abdomen during pregnancy resulted in a 92 percent increase in childhood leukemia and in other childhood tumors taken as a group. The study was made by interviewing mothers of children with leukemia, mothers of children with other cancers, and mothers of children with

no cancer. The possibility existed that the mothers of children with leukemia or other cancers might remember or report differently their histories as compared with the control mother. Thus the difference that was observed might be due to the methods used rather than to a true difference in the consequences of diagnostic radiation exposure.

Dr. Brian MacMahon at Harvard got around this problem (Reference 104); instead of asking the mothers for their histories he ascertained among children born in the New England area, 1947–54, those who died by 1960 of leukemia (N = 304) or other cancer (N = 252). He then went to the mother's obstetrics records to determine whether radiation had been recorded during the pregnancy. Comparison was made with data from the records of mothers of children who did not have cancer—a 1 percent sample of all children born in the area. The results showed 44 percent more intrauterine diagnostic radiation exposures of children with leukemia and children with other cancers. One can no longer attribute the difference between cases and controls to maternal memory bias.

There is still a problem, however, in interpreting the results. Not only leukemia, but also all other childhood cancers taken as a group were related to maternal diagnostic radiation exposure of the abdomen during pregnancy. One would have to postulate that doses of x ray in the homeopathic range can induce various cancers in childhood. We have no other evidence consistent with this notion.

Mothers who received diagnostic radiation exposures were probably different from other mothers and that is why the x-ray examination was made. Perhaps that difference, that abnormality or suspicion of abnormality which led the physician to recommend the diagnostic x-ray procedure, was primarily related to the occurrence of cancer of the child, and the x ray was a concomitant variable which merely indicated this abnormality.

In Hiroshima there were only 98 children heavily exposed in utero, and in a 20-year follow-up study, Dr. Johnson and his associates have found seven deaths, none attributed to leukemia (Reference 105). It should be noted that an increase would have to be 30 or more times greater than normal to have been detected in such a small sample. So the ABCC study of leukemia following intrauterine exposure is not a very sensitive one.

One of the deaths that occurred was due to primary carcinoma of the liver (Reference 36), a very rare tumor of children but more frequent in Japan than it is in the United States. Another child in the group had a psammoma of the meninges, which is a benign sand-filled tumor of the covering of the brain. In both these children the tumors may have occurred by chance rather than as a result of the radiation exposure.

BUSTAD: Before you go on, are you going to state the radiation dose received from diagnostic exposures of the abdomen during pregnancy? Do you want to put some limits on it?

MILLER: I think the radiologists would be better able to say. I was under the impression that it was something like 2 to 4 r.

BUSTAD: You are sure that there were no cases of additional exposure from x ray?

MILLER: Correct.

I should mention a study made by Saxon Graham and a group of extremely able epidemiologists at Johns Hopkins, Roswell Park Memorial Institute, and the University of Minnesota (Reference 106). They made an exquisitely detailed retrospective study concerning the history of diagnostic x-ray exposure of the mother or father before conception of the child and they found a relationship between childhood leukemia and a history of preconception radiation up to and even greater than 10 years before. In other words, some abnormality was produced by a very low dose of x ray many years before conception of the child—an abnormality sufficient to raise the risk of leukemia in the offspring by 30 to 100.

One has to think what would be the mechanism, and I have no suggestion. The results are not consistent with those from Hiroshima and Nagasaki where a study has been made of almost 18,000 children, about 50 percent of whom had at least one parent exposed within 2,000 meters of the bomb.

FREMONT-SMITH: Prior to conception?

MILLER: Right. And a comparison was made of the frequency of death from childhood leukemia according to parental atomic bomb exposure up to 16 years before. In this instance the radiation doses were much higher and the sample size much larger than they were

in the retrospective study of diagnostic intrauterine exposures. No relationship could be shown between leukemia in the offspring and parental radiation exposure category (References 107 and 108).

BRILL: Bob, do you feel confident in the historical data in that first population?

MILLER: The investigators made every effort to check and recheck the hospital records of the mothers and the fathers. The trouble was that the histories given by the parents disclosed something less than all of the pertinent hospital records. But the investigators traveled through New York State, Minnesota, and the area around Baltimore, looking for the records in the most exhaustive search possible for evidence of medical radiation exposures.

BRILL: To what did they compare these?

MILLER: Data on 319 leukemic children were compared with 884 control children selected at random.

LIFTON: Did they separate the influence of maternal and paternal exposure?

MILLER: Yes, they did.

LIFTON: So that each one was a significant variable?

MILLER: Correct.

TAYLOR: Anyone who has one chest x ray is in there?

MILLER: As I recall it.

AUXIER: One comment there. Until very recently at least, the doses you mentioned, or someone mentioned 2 to 5 r, were typical of any radiographic examination, whether G. I. tract, pelvic, or what not, and a lot of the examinations carried out, especially by obstetricians 20 years ago, involved a fluroscopic examination.

MILLER: Correct.

AUXIER: And the doses there frequently ran in the 50-r range in some of the old machines we have examined. So the doses could have been quite a bit higher than 2 to 5 r.

MILLER: Not in the intrauterine studies. The contribution, if any, from fluoroscopy was small. MacMahon's cases were born between 1947 and 1954 (Reference 104) and those in the study by Alice Stewart and her associates were born between 1953 and 1955 (Reference 103).

AUXIER: You said they looked at a lot of things. So I assume that if there had been an incident of this kind, that they would have made special note of it.

MILLER: Yes, they did.

WHITE: Did they try to look at something else beside the diagnostic x-ray exposure, like the incidence of infectious disease and the use of any number of antibiotics?

MILLER: Yes. Dr. Stewart did do that (Reference 103). There was an excess frequency, not very great, of spotting during the first trimester of pregnancy, reported by mothers of leukemic children, and there was some slight excess of genitourinary tract infections among these mothers. There were also a few more viral infections during the early months of pregnancy.

WARREN: With the large employment of diagnostic radiology throughout the country and the world, why isn't there more leukemia then? This is a carefully selected series of leukemic children whose parents were then investigated.

MILLER: Correct.

WARREN: And it is logical to expect that almost no parents would exist among that group who had not had diagnostic radiation of some sort, a chest film or even, I suppose, dental radiation. That's quite an appreciable exposure even though only to the head.

This study has bothered me all along and I just wondered about the special selection and then the special emphasis on one factor in that selection. If all the leukemic parents who lived on the north side of the street had been selected, maybe there would have been a differentiation there. I just wonder if this is an appropriate way of handling these kinds of data. Do you have doubts yourself on it? I noticed you weren't too enthusiastic.

MILLER: I have difficulty reconciling the fact that the frequencies of all cancers were increased, not just leukemia, and we know that, say, a Wilms' a tumor of the kidney, has very different epidemiologic characteristics from leukemia (Reference 109). We have no basis for suspecting at the moment that radiation induces Wilms' tumors.

ABRAHAMSON: Chronic myeloid leukemia is the one that has the chromosome abnormality associated with it. In these studies were any of these broken down into the type of leukemia?

MILLER: Not in these studies, but chronic myeloid leukemia is very rare in children.

COURT BROWN: Do you want me to say something about chronic myeloid leukemia?

ABRAHAMSON: I would be happy to have you say something about it, if you will.

COURT BROWN: I'm talking about adult leukemia. As Dr. Miller says, it is a very rare phenomenon in childhood. In adult cases of chronic myeloid leukemia around 90 percent of the cases have in every bone marrow cell a very distinct and defined marker chromosome which is an abnormally small acrocentric chromosome. But there are a proportion of cases apparently without any change in this particular chromosome and not very much difference has been found between the cases with and without the chromosome in terms of their behavior.

The very interesting thing about chronic myeloid leukemia is that there is now very reliable evidence that the characteristic marker chromosome—the Philadelphia chromosome—is present in the bone marrow in red cell precursors and megakaryocytes as well as in the cells of the myeloid series. This finding is important in relation to discussions on the monophylitic origin of blood cells.

FREMONT-SMITH: Is it carried way back into very early embryonic life? Is that right?

COURT BROWN: No. I should say that this chromosome is associated with the leukemic cells and is not associated with normal cells in the individual. It is a feature of the tumor process. Just where it fits in the tumor process is completely unknown.

FREMONT-SMITH: Would it come when the progenitor cells of this whole series are first differentiated?

COURT BROWN: It looks as if it is something which arises as possibly part of the etiological mechanism of the tumor in a stem cell which is capable of producing not only granulocytes but red cell precursors and the megakaryocytes.

BRILL: One of the unusual portions of the ABCC leukemia experience was the fact that in the early 1950's there were five children in the closest exposed group who had a picture that was indistinguishable from chronic granulocytic leukemia, which is extremely rare in childhood and not seen in the more distally exposed survivors. And, as Dr. Johnson pointed out, chronic granulocytic leukemia was the type of leukemia that was most increased in the most closely exposed survivors. The point I was going to make yesterday was, it seems to me that the more recent experience with acute types of leukemia, in contrast to the earlier chronic granulocytic leukemia excess, reflects a progressive waning character but that there has been a progressive waning of this chronic granulocytic leukemia increase in the most closely exposed survivors. In the earliest of the three periods that he had put on the board, there was 30-fold increase in CGL among the most closely exposed in contrast to more distally exposed. In the next period this was 18-fold and in the most recent it remains approximately an 8-fold increase. This is in contrast to the acute leukemias which were 22-, 9-, and 6-fold increased. So that today there continues to be a greater increase of the chronic forms of granulocytic leukemia than of the acute leukemias above expectancy. The acute leukemias are becoming proportionately more important as the chronic granulocytic leukemia incidences wane.

COURT BROWN: I think there is one more very important point to make in relation to radiation regarding chronic granulocytic leukemia. While admittedly the numbers of cases studied have been small, nevertheless, cytogenetic studies on cases of chronic myeloid leukemia having a high probability of being radiation-induced have shown nothing that is notably different from studies on cases with a low probability of radiation induction. Here I am referring to the distribution of cases between the various cytological subgroups that can be recognized.

JOHNSON: Isn't there an association between treatment and the appearance and disappearance of the Philadelphia chromosome in the granulocyte?

COURT BROWN: If you look at the proportion of cells in blood culturing which contain a Philadelphia chromosome, this proportion changes with therapy. As the blood count comes down under therapy—x-ray therapy or therapy with myleran—then the proportion of these so-called Phl-positive cells drops, and in fact one can virtually get it to disappear.

What is perhaps more interesting from the actual therapeutic point of view is that there is just a suggestion that if one really pushes treatment to the point that one is producing bone marrow failure, then one can in fact begin to drop the proportion of cells in the bone marrow which are Phl positive. In the ordinary course of circumstances every cell looked at in the bone marrow is Phl positive and the ordinary course of treatment does not affect this at all. If you really push treatment to the point that the individual goes into marrow failure, then there is just a suggestion that possibly some cases survive with a reduced proportion of Phl-positive cells in the marrow. This may be associated with a much longer remission of the clinical evidence of the disease. We know of one case in which this may have occurred and who has now survived for many years in good health with only a small proportion of Phl-positive cells in the marrow.

BRILL: How was she treated?

COURT BROWN: With myleran.

CASARETT: I find it easiest to accommodate the various sets of data with respect to the dose-response relationship and the time of occurrence of leukemias on the basis that in a heterogeneous population, and assuming that we're dealing with a multivent mechanism in leukemogenesis, there is a wide spectrum of predisposition and susceptibility to leukemia that is due to two general factors: one, the predisposition as a result of the genetic characteristics in the individual, and the other contribution, nonradiation pathogenic auxiliary mechanisms. Radiation injury can add to these factors of mechanism, and the amount of radiation that is required to precipitate a leukemia is therefore different according to the amount of predisposition and additive factor in the individual. This also determines the time at which leukemia might occur. Therefore, we may be dealing here, in the in utero studies, with a temporal advancement of cases that had a high degree of predisposition on a genetic basis. There has been no follow-up to find out, as far as I know, the relation of in utero irradiation or genetic predisposition without in utero radiation to the occurrence of

leukemia after childhood. There may be a precipitation of leukemias in people with a high genetic predisposition in early childhood as a result of small doses of radiation; and cases occurring later in association with large doses may be occurring among persons with a lesser predisposition from nonradiation factors. I still think we have to consider shifts to the left in time associated with additive factors, as well as the absolute induction of leukemia which is represented in my mind by the absolute increase in incidence throughout the lifetime.

AYRES: Nobody has mentioned the irradiated population of radiologists, and I am just curious whether someone would care to summarize that experience or explain why it is irrelevant.

MILLER: It is relevant; it is historically relevant, not so much of great importance in understanding leukemogenesis. In 1944, March (Reference 110) and soon after (Ulrich, Reference 111), before the era of big grants (they didn't need any money; they merely went to the library and looked at the Journal of American Medical Association for the death notices and compared the causes of death among radiologists with other physicians), I found a ninefold excess of leukemia among the radiologists. But they failed to correct for age, and when this was done several years later by Dr. Gilliam at the Nation Cancer Institute, he found that there was still good evidence for at least a threefold excess.* The early studies of leukemia among American radiologists led to the immediate search for leukemia among atomic bomb survivors.

One could argue, perhaps not very effectively, that radiologists may differ from other physicians with respect to variables other than occupational exposure to radiation. One cannot argue that differences other than radiation in Hiroshima and Nagasaki account for the leukemia frequencies experienced there. So a great value of the ABCC study is its relative freedom from concomitant variables to which leukemia might be attributed.

AYRES: On the other hand, Dr. Court Brown said there are some reasons why the ABCC data are obscure in certain respects, whereas the radiologist data may not be ambiguous in precisely the same respect. You have a log of potential leukemia cases from other experiences, for example.

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*A. G. Gilliam, Unpublished data. (Dr. Gilliam, who died a few years ago, did this work while at the National Cancer Institute.)

COURT BROWN: You don't get dose-effect relationships out of the radiologists' data. This is just impossible.

AYRES: Because the doses are so uncertain?

COURT BROWN: Unknown. It seems to me that we're beginning to point up some of the limitations of epidemiology in trying to understand what the risks are or trying to determine the dose. I think Arthur Upton brought this up. When he implied that all we had learned so far is that doses of perhaps 100 or 200 rad are undoubtedly associated with the risk of developing leukemia over and above the risk one normally incurs, the burning question is what happens below these levels of dose? I think it is here that epidemiology becomes too blunt an instrument to give any insight into the problems, and I think that it is here that one really wants to get down to cellular mechanisms. I don't think that one can possibly make any effective statements on the effects of low levels of doses until one really understands or gets some better insight than we have currently got into the mechanisms of tumor induction.

AYRES: For the purposes of this group, which is to look at the possible aftereffects of a nuclear attack, it might be more interesting to ask what happens between 100 and 200 rad. How does the dose-response picture look there? If it is highly nonlinear, for example, in that range, it would be quite a significant piece of information. Do we have any information that would suggest one thing or the other?

BRILL: I think the dose-response curve which we do have from the Japanese data is extremely gross. A number of assumptions were made with respect to the shielding factors in calculating doses that were received in the intervals and groups studied. Within the next few years one should, on the basis of the new dose calculations from Oak Ridge and Japan, get a much better estimate of the shape and magnitude of the dose-response curve. Nonetheless, the information is so limited that one will be unable, in all likelihood, to determine the relationship in the detail desired from these data. It is most unlikely that one will be able to look at different types of leukemia separately, and to break it further by age and sex. The one thing that I would hope for, however, is that when people do the kinds of model building which will be necessary to apply new knowledge of the mechanisms of carcinogenesis and to extrapolate on the basis of predictions into the lowest dose regions, their estimates will be

consistent with the available data. Philip Burch (Reference 112), for example, did a thoughtful mathematical analysis of radiation leukemogenesis, but the way in which he used the Japanese data doesn't square with the findings. He used an exponent of 2 on the dose and yet, based upon everything we know, this isn't the case, since the Japanese data which he was fitting are best fit by an exponent of 1 or less. It seems to me that the extrapolations will have to be consistent with what we learn epidemiologically but we will not learn how to extrapolate on the basis of the epidemiological data.

COURT BROWN: Any mechanism has simply got to take into account the epidemiological facts.

TAYLOR: Can I raise a question which I think is fundamental, from something that Dr. Court Brown just mentioned? Is it possible that there are differences, because of the mechanisms for carcinogenesis, between atomic bomb data and some of the clinical data that depend on the dose-rate dependence of all of these effects? The exposures at Hiroshima and Nagasaki were, at least at high doses, in times of the order of milliseconds. Judging from the almost infinitesimal amount that I know about this subject, some of the things that affect what happens on the cellular scale are affected by the differences in the exposure time, or the comparison between the exposure time and some other free radical, recombination times, which range from microseconds to milliseconds to hours, and, I gather, in some cases even to days.

The question is whether one is leaving out something important in comparing exposures that have taken place in a pulse with exposures that may have taken place in a series of long exposures over a number of years or months of treatment? Does anybody have any feeling about whether these things may be important?

Let me just say one thing more, and that is some gross effects I think have been clearly demonstrated to be dose-rate dependent. The number of r delivered in a few milliseconds produces different effects from the same number of r delivered in seconds or delivered in hours. I think there is some mass of data that displays that some observable effects, not permanent but observable biological effects, are produced by pulses of radiation below 1 r that are unobservable in 1-r exposures that are made over minutes. So that is something there. Whether it has anything to do with leukemia whatsoever I have no idea.

UPTON: I would say that experimental work in laboratory animals establishes beyond question a dose-rate dependency in the effectiveness of low LET radiation, such as x rays and gamma rays, for the induction of tumors of various kinds, including leukemia.

TAYLOR: That has been observed?

UPTON: Yes. There is no question about this. Radiation at very low dose rates is less effective by a factor of 3 or more than radiation at high dose rates.

TAYLOR: Is there anything quantitative there? Are you talking about the difference between milliseconds and minutes or between minutes and days or what?

UPTON: Unfortunately, the relationship between the effectiveness of a given dose and the dose rate has not been defined precisely. So we don't really know the shape of the curve. We do know that radiation given at rates of 20 rad/minute or higher, so that an exposure to a dose of 100 rad takes only a few minutes, is several times more effective than radiation given at a dose rate of 1 rad/hour, let's say, so that the irradiation would take several days.

WHITE: Only in recent years has the technology for giving these high dose rates that simulate actual detonations become available. This is the reason why nobody can really answer your question.

UPTON: We don't see the same kind of dose-rate dependency for high LET radiations. This would suggest, as Dr. Brues pointed out at the beginning, that the fundamental target, whatever it is, is probably... I'm getting onto thin ice here. What I'm trying to say is that whatever kind of repair goes on is probably not able to cope with the injury produced by a densely ionizing particle.

TAYLOR: Haven't there been some observations of leukemias with just the gross LD50 dose? I don't know.

UPTON: Yes.

TAYLOR: Isn't it true that under some conditions, a lower dose is more fatal, that the curve of the LD50 dose as a function of the exposure time is not a monotonic function of time but has some maxima or minima?

BUSTAD: The data that George Casarett brought up yesterday about the gonads of dogs might pertain to this; another case is Henry Kaplan's (Reference 113) early data when he showed that, if the dose is fractionated in a certain pattern, the incidence of leukemias in mice can be increased.

UPTON: Yes. This may be getting into the discussion that will follow tomorrow. I think one can visualize that radiation at low rates may be more effective than radiation at high rates under certain conditions, at very high levels of dose.

WARREN: May I ask Dr. Court Brown one question? Has the spondylitis series continued to be treated or was this a popular phase which waxed and waned or was it the leukemia incidence that reduced the popularity of it?

COURT BROWN: No. They continue to treat spondylities with x rays. It is a chronic crippling disease; it produces chronic pain in the back, which is a very disturbing phenomenon.

WARREN: It's improved by the therapy?

COURT BROWN: I believe it is relieved by it. But there has been a distinct change in the pattern of behavior of radiologists toward these patients. They have dropped the doses. They are unwilling to repeat courses of treatment.

WARREN: Because of the leukemia risk?

COURT BROWN: Because of the leukemia risk, and now the other tumor risk has been giving them a lot more food for thought, and I personally feel that they could really reconsider their methods of treatment. I think it comes back to what Dr. Upton was saying. It is probably safer to give these people one single dose of treatment—it may kill all the cells in the marrow in the irradiated area—than to give them a series of fractionated treatment which simply raises a whole lot of cells up to a state in which a leukemic change is possible.

WARREN: We can drop that now and we can go on to aging if you like, because the time is getting short.

LIFTON: I just wanted to add one more idea about dosage in leukemia, which is probably the most controversial of all, but since

we're encouraged to speculate, I feel I should do this. Probably some of you know the work of William Greene (Reference 114) at Rochester, who is trained in both medicine and psychiatry, and who feels he has strong evidence that the onset of leukemia is influenced by experience of severe loss—either actual loss of someone very close or symbolic loss involving a fundamental change or a threat to a life pattern.

I've talked with him about this and he believes there is at least a possibility that, in some of ABCC data, the general Hiroshima data, the experience of loss and of other of the psychic experience that I will talk about tomorrow, could have influenced at least the time of onset of leukemia. If this is true, this would, of course, fit in with other work being put forward, some of it also from Rochester by George Engel, on the so-called unitary theory of disease. This is not a new idea, but an inclusive concept of health and disease in relationship to a general equilibrium which can be disturbed at any level—anatomical, physiological, psychical, or social. It would then perhaps suggest that the doseage of radiation contributing to leukemia and influencing its onset is a relative factor. And keeping in mind this principle of multiple causation, it may be possible to go only so far in correlating radiation dosage and incidence or timing of onset.

MILLER: Dr. Court Brown has already alluded to the difficulties in performing satisfactory epidemiologic studies, but Dr. Greene (Reference 114a) has complicated his life, and I think ours even more, by refusing to have any controls to compare his observations with.

COURT BROWN: One has always felt that psychiatry needs some scientific underpinning! [Laughter]

BRILL: Actually you could do the same thing with any disease that had a high incidence in the middle 40's and 50's of life. At this time in life one expects to lose significant parental figures, and the etiological significance of this event with respect to leukemogenesis is difficult for me to accept.

LIFTON: In other words, the principle applies to all diseases. In fact, if the theory is at all true, it would have to be relevant for all diseases.

BRILL: I think so.

JOHNSON: I thought Greene was thinking more in his term of breaking the love link. The example may be the child with leukemia whose father died one year before the onset of leukemia. There is no support for this hypothesis in the ABCC data. Seven or eight years elapsed before leukemia appeared in great numbers.

JABLON: A lot of problems have been mentioned, but being a simple-minded person, I'm impressed with some of the essential simplicities. It seems remarkable, considering the differences between the Japanese and spondylitic experiences—different races, different dose rates, and so on—that the results are so imilar. To be sure, there are some differences, notably the slope of the age curve, but in both instances, over the range that can be studied, the response seems to be approximately linear, and if you calculate for each how many cases are induced per person per year per rad, you come up with about the same number.

WARREN: That's pretty low.

JABLON: It's of the order of one per million, and one could argue whether its 0.7 or 0.8 or 0.9. But I think it is remarkable that the two experiences are so close.

WARREN: Yes.

JABLON: Maybe the process is really not so complicated as might be thought. Given the differences between the two populations, the similarity of the results may be a good reason for thinking that the process is a fairly simple and fundamental one, and maybe the safest thing to do until we know better is to assume that the response is linear all the way down to zero dose.

WARREN: In other words, would you be willing to oversimplify it by saying the little virus was sitting there all the time and it just took this little trigger to set it off eight years later or some period later? There is quite a bit of thinking on this, I think, in the tumor field on some of these other viruses. Has anybody else got something urgent to say about tumors and leukemia?

TAYLOR: Is it really clear that everyone agrees that there is no question about the direct statistical correlation between the appearance of leukemia and exposure to nuclear radiation?

MILLER: In sufficient dose?

TAYLOR: In sufficient dose.

BRUES: Do you mean by that, to ask if there is any doubt as to whether radiation is a leukemogenic agent?

TAYLOR: Is there any doubt in anybody's mind?

MILLER: There are three features that indicate that it is definite. One is that there is a dose-response relationship; second, there is no other variable known to which the effects can be attributed; and third, that the results are consistent with studies elsewhere. So it all adds up.

WARREN: Does someone have information about the accelerated aging of the population as a result of exposure to nuclear radiation?

UPTON: Could we consider this, Staff, first in the context of overall mortality rates as a function of distance?

WARREN: Who has the information? Dr. Jablon?

JABLON: In Japan, the people who were within 1,400 meters of the hypocenters in the two cities, that is, people who had doses ranging upward from 50 to 100 rad, depending on which air-dose curves are used, seemed to have accumulated mortality about 10 percent greater than expectation over the 12 years from 1950 to 1962. Having said that, I have to add immediately that, as Dr. Brues has pointed out, these experiences were not experiments. There was not the randomization that enables one with some confidence to ascribe observed differences in outcome to experimental differences in treatment. Differences of a socioeconomic nature distinguish various distance groups, so that whether differences in observed mortality rates result in whole or in part from irradiation is not at all clear.

UPTON: If one subtracts from the group that shows the higher overall mortality rate those individuals who died with leukemia, does this eliminate the difference?

JABLON: No, sir.

WHITE: Or all tumors?

JABLON: If you start to get specific about causes, and I have some information on particular causes, the material starts to fall apart. In fact, it starts to look very peculiar, and, more than that, it starts to suggest that perhaps the differences are more directly attributable to sociological variation than anything else. Here are a few numbers (Reference 78) and you will see why I'm so grey! I'll talk about "elevation" or "depression" and will always refer to the mortality rates in persons who were within 1,400 meters as contrasted with those who were further away or not in the cities at all.

For tuberculosis, Hiroshima males had an elevation of 50 percent but Hiroshima females were depressed by 7 percent. The elevation in males is highly significant, but the decrease in females is not significant. In Nagasaki males, the increase was 12 percent, in females there was a decrease of 26 percent. In both cities, therefore, the males had elevated tuberculosis mortality, the females depressed mortality. I'm not sure what to make of this.

FRANK: Didn't you mention earlier that these males were probably excluded from the military service?

JABLON: Yes, sir. That may well be the answer. Here is another pattern. All malignant neoplasms excluding leukemia: in Hiroshima, for males an elevation of 7.5 percent (not significant); for females an elevation of 53 percent (highly significant).

TAYLOR: Was there a control group here?

JABLON: There are two essentially persons who were in the cities but at larger distances, who had a small dose; and, as a separate group, people who were not in the city at all, who came in later. Continuing with the malignancies other than leukemia, Nagasaki males were elevated by 5 percent but Nagasaki females by 21 percent. So, in both the cities, the females had fairly large increases but the males only small increases. I don't know how to explain that.

Dr. Frank asked yesterday about infective diseases other than tuberculosis. For the whole class of infective and parasitic diseases, excluding tuberculosis, Hiroshima males who were within 1,400 meters had a decreased mortality of 40 percent and Nagasaki males a decrease of 38 percent. On the other hand, Hiroshima females had an increase of 109 percent and Nagasaki females, an increase of 67 percent. So for this class of diseases, the females in both cities had high mortality, but the males had decreased mortality.

SPEAR: What kind of whole numbers are we talking about here in the deaths?

JABLON: The numbers of deaths were, in Hiroshima, 54 in males, 49 in females, and in Nagasaki, 16 in each sex group. For Nagasaki, the numbers are not very large, but in Hiroshima they are large enough to be reasonably stable.

If we take all causes of death in both cities in all distance groups, including leukemia, we have about 8,600 deaths, so you can see that leukemia will not offset the overall rate by much. For the total, we find elevated mortality in those under 1,400 meters in all four sex-city groups: Hiroshima males, 14 percent; Hiroshima females, 19 percent—both statistically significant. In Nagasaki, the elevations are 11 percent in males and 17 percent in females. But, when you analyze it by cause, the excesses come from a lot of different places, that is, in different ways, and I don't claim to know what it means. It seems to me to be at least likely that the excesses result from differences in the kinds of people at different distances. But I can't tell you that it is not radiation; I don't know.

TAYLOR: Have you made any comparisons in which you just arbitrarily take all the people on one side of Ohta River and the people on the other side? I mean is it true that 11 percent is statistically important when you compare one group with another group where there are describable differences in the environment of the group, such as one is in a poor neighborhood and the other one is not?

JABLON: We have only begun to cope with the problem you mention. The early studies have all been what we can call "ring" studies; that is, circles of various sizes are drawn around the hypocenter point, and then the central circle is compared with the various rings created. This is superficially attractive, since the comparison group, coming from a ring of the city, might be thought to include all kinds of people. When you look at a map you find that this actually is not so. A ring may be empty in various places because it goes out into the bay, or up into the mountains, or into the rice paddies; and most of the people included in the ring may be concentrated in one particular densely populated area.

WHITE: And these early studies were just correlated with range, not with dose.

JABLON: Yes.

WHITE: So the difference between free-field dose and the real dose is not included in those at all.

TAYLOR: The Yucantal, the north part, will include a population which must have a substantially higher death rate than the one to the west.

SCHULL: There are many lines of evidence which suggest that individuals who were close to ground zero at the time of the atomic bombing were and still are in a socioeconomically less favored position than those survivors who were farther away. It seems unlikely, therefore, that socioeconomic factors would obscure a radiation effect; if anything, they might lead to a spurious one.

FRANK: I understood from the earlier discussion that it was true of the socioeconomic group in Hiroshima, but is this also true of Nagasaki?

SCHULL: Yes.

UPTON: It is claimed by the Japanese.

LIFTON: The socioeconomic factor may apply in that way but insofar as it has itself been affected by the bomb and is a relevant factor in aging and mortality, then it would not be merely a "contaminating factor." Rather, it would be a bomb result of a nonradiation kind which contributes to disease and has significance in that sense.

BRUES: It might also be sex-linked, might it not to the females?

WARREN: You mean they are tougher?

LIFTON: I think the things he described are confusing for any interpretation.

CASARETT: I was just going to say that one of the problems is that we don't have enough age-adjusted data as yet because of the paucity of deaths. Much of the interpretation has to wait until we can get age adjustments.

JABLON: I beg your pardon, sir. These data have all been age adjusted.

CASARETT: What are the years, sir?

JABLON: This is 1950 through 1960.

CASARETT: I have heard you give an overall mortality. You have given overall mortality during this period but not the ages.

JABLON: These are all ages, and the numbers that I have been citing are ratios of the observed deaths to expected deaths, and expected deaths are calculated by applying the proper age distribution for the population under study to rates for the whole population.

CASARETT: It was a 10-year age group then?

JABLON: Ten years.

CASARETT: All the people in that group were aged within plus or minus five years, that is, they are all within the same age bracket?

WARREN: They are younger?

JABLON: I can describe the calculation. The data were tabulated simultaneously by two-year periods of calendar time, by ten-year groupings on age at observation, by a large number of cause-of-death categories, by city, by sex, and finally by distance. The same was done for dose.

CASARETT: I'm sure that there have been age adjustment studies on the data that you just gave, but are the data for all age ranges or do they apply to...

JABLON: These particular ones are all ages.

CASARETT: That's my point. In terms of manifestations or changes I want to relate changes to the chronologic age to determine whether there has been any physiologic aging that is premature, and that is what I wasn't clear about.

WARREN: Were they younger when they died than the average? That's one significant evidence of aging, too.

JABLON: If mortality rates are increased, as they are, age at death will be younger. This follows as a straightforward mathematical consequence. However, it is true that if you look at the data by specific ages, there are some peculiar differences. One concerns children who were less than five years old at the time of the bombing.

Mortality rates in children over five are very small, as you know, and there aren't very many deaths. But, for what they are worth, at observed ages five through nine, among those within 1,400 meters, in both cities, there were ten deaths from disease. The expected number is 1.9.

MILLER: Five of those deaths were due to leukemia.

JABLON: Five of those deaths were due to leukemia. We're still left with five deaths.

MILLER: Right.

JABLON: Against an expectation of the order of one and a half. When you look at the individual cases there's nothing about them that seems remarkable. They die of the things that children die of or their deaths are attributed on the certificates to the things that children die of: diarrheal diseases and things of that kind. But it does seem a remarkable thing.

LIFTON: Is it possible that there is an age-related defect that is responsible, that is, having a nonspecific nature which might express itself in any disease? Is that a feasible interpretation?

JABLON: It might be.

JOHNSON: I think you can note a secular trend in the data. Ciocco (Reference 115), in his analysis of mortality in the Life Span Study sample, 1950-55, computed ratios of death in two groups, within 1,400 and beyond 1,400 meters and not in the city. He found that there is the advantage of being female in that women had a ratio close to unity in almost every age group except those who were less than 20 years of age at the time of exposure. The males, however, had a ratio of 2.8 during the 1950-55 period which fell to 2.0 for the entire period 1950-64.

Looking at the entire period 1950-64, one gets the impression that the early period (1950-55) was a period of increased or accelerated mortality. This is of interest because it suggests a latency period, similar to the experience with a more specific disease, leukemia.

JABLON: Was this all mortality, Ken?

JOHNSON: Yes.

JABLON: You see, for all mortality, though, there's a special problem you have to look out for; that is, in the early years tuberculosis was just a tremendous portion of total mortality. And, as in many countries, the tuberculosis death rate in Japan has been going down very quickly. So that secular comparisons are going to move somehow because of the drastic changes in tuberculosis.

SESSION III

GENETIC EFFECTS

Chairman: Arthur C. Upton
Initiator: William J. Schull

INTRODUCTION

UPTON: In turning from the somatic effects problems to genetic effects problems I would like to thank Dr. Warren for the marvelous job he did in guiding the discussion of the first topic.

We are equally fortunate in having Dr. Schull with us, who, with Dr. Neel, has looked very extensively into the genetic effects in the exposed populations. Dr. Schull, would you take over?

SCHULL: My charge at the moment is to set the stage for those individuals who, along with myself, will be trying to direct the remainder of the day's discussion. The first will be Bob Miller who will help us make the transition from a delineation of somatic effects to an enumeration of the genetic ones. Specifically he will concern himself with findings, primarily from Hiroshima and Nagasaki, in the pediatric age group. I am sure that most of you are aware that the Atomic Bomb Casualty Commission no longer maintains a pediatric program as such, although one did exist in the past. Bob will summarize the findings of this latter program. He will be followed by Dr. Court Brown, who will bring us up to date on what is known about the relationship of ionizing radiation to chromosomal changes. And, finally, Seymour Abrahamson and I will concern ourselves principally with gene changes, "point mutations" if you will.

I think it is important to recognize at the outset of this discussion that we are perpetuating a fiction when we treat somatic effects as though they were distinctly different from genetic ones. There now exists a very large body of evidence suggesting that the somatic response to irradiation is under a fair measure of genetic control. An outstanding illustration of the nature of this evidence is a study by Douglas Grahn (Reference 116), who has shown that in mice the LD^{50} varies as a function of the genetic strain of animals which one uses and that within limits one can control the LD^{50} by appropriate crossing. It is not our purpose in introducing this thought to try to enlarge the genetic horizons at the expense of other disciplines, for we

have enough problems as it is. But I believe it important to realize that this dichotomy of effects which we are trying to maintain has no firm biological basis.

It is our aim in the presentation today to give you some insight into where our knowledge stands at the present time, but particularly we hope to emphasize a "forward look" and not merely survey material which is already in the literature and which, if you were prepared to take the time, you could undoubtedly summarize as well as we.

FREMONT-SMITH: You will bring out the dilemmas as well as where we stand, won't you?

SCHULL: That's right. We want to use the data as a springboard to a discussion of what we can and should be doing in the future. We shall try to focus the discussion upon two broad questions which are related to what John Platt (Reference 117) has termed "strong inference." First, we are going to ask ourselves, "What reasonable genetic hypotheses can we exclude with the information which we have thus far been able to collect?" Patently if we cannot exclude some hypotheses we have made little progress. Then we shall ask, on the basis of the hypotheses which we must still entertain, "What kinds of experiments, and I use the word 'experiment' in the broadest possible context, can we envisage which are feasible and would lead to a further exclusion of hypotheses?"

EFFECTS ON CHILDREN OF HIROSHIMA AND NAGASAKI

MILLER: First I would like to make clear in discussing the intrauterine effects of radiation, that they are not genetic but a maldevelopment that occurs during embryogenesis. The pertinent history and the development of the hypothesis which could be tested in Hiroshima were as follows: Dr. Douglas P. Murphy (Reference 118), at the University of Pennsylvania in 1928, published an account of 14 case reports culled from the literature concerning women given pelvic radiotherapy early in pregnancy whose children at birth had small head circumferences and mental retardation. By writing to about 1,700 obstetricians throughout the United States, Goldstein and Murphy (Reference 119) were able to accumulate 16 additional cases which they reported in the following year. Laboratory experimentation at about the same time showed that exposure of pregnant animals to radiation produced a wide variety of congenital defects even when the dosage was relatively low (Reference 120). The anomalies included microphthalmia (small eyes) and exencephaly (bulging of the brain through a defect in the skull). From their report, Goldstein and Murphy showed only what appeared to be an excess of microcephaly with mental retardation, in contrast to the diversity of defects produced in experimental animals.

So with this information already in the literature at the time of the bomb, it stood to reason that people working at ABCC should look for the same effect in children who were in utero at the time of atomic bomb exposure. In summary, there were 15 cases found in Hiroshima, 13 of whom were born of mothers who last menstruated 7 to 15 weeks before the bomb (Reference 36).

This is a very sharply limited period during pregnancy. The results indicate that the human embryo is susceptible to radiation effects only in this narrow interval. The mothers reported their last menstrual periods to have been 7 to 15 weeks before the bomb. If one assumes that there was a two-week interval between the last menstrual period and conception, the period at the time of maximum susceptibility would be between the 5th and 13th week of gestation.

The head circumference of the children exposed in utero could be classified by the number of standard deviations below normal for the age and sex. There were two categories to which I would like to call your attention. The first concerns children whose head circumferences were much smaller than normal for their age and sex (three or more standard deviations (S. D.) below normal) and who were mentally retarded. Under 1,200 meters there were eight mothers exposed during the susceptible period of pregnancy. All eight had children who were retarded in intelligence and whose head circumferences were much smaller than normal. Thus it has been possible to identify an incapacitating delayed effect of radiation which besets all members of a subgroup within the exposed population.

In the next 300-meter interval from the hypocenter (1,201 to 1,500 meters), there were 22 embryos at risk and four had microcephaly and mental retardation. In the next 300-meter interval, there were 18 exposed and one was affected; in the next 400 meters there were 19 exposed and none were affected. One can see a dose-response relationship as distance from the hypocenter increases; 8 out of 8, 4 out of 22, 1 out of 18, and 0 out of 19.

In the intermediate distance categories, from 1,201 to 1,800 meters, there were 10 children whose head circumferences were smaller than normal (-2 to -3 S. D. below normal for age and sex), but not so small as those whose intelligence was retarded. These children in the intermediate distance categories had a transitional limitation of their brain growth (and of the skull surrounding it), but not so much as to cause them to be retarded mentally.

FREMONT-SMITH: Was there any hydrocephalus in this group?

MILLER: No. That is an important point. There was no other anomaly that occurred in numbers which exceeded normal expectation. There were four children out of 95 within 1,500 meters of the hypocenter who had congenital defects, but these were not all of the same type and probably occurred by chance (Reference 36).

ABRAHAMSON: The ten normal children are now old enough to have reproduced. Have any of them reproduced and have you followed any of their offspring?

MILLER: That suggests a hypothesis that comes from the recent literature on animal experimentation. Dr. Bustad referred to it the

other day. Dr. Roberts Rugh (Reference 121) at Columbia University was the first one to report that male rodents exposed in utero were sterile when they reached the mouse-bearing age. Work at the Radiation Biology Laboratories of Texas A&M and elsewhere has shown similar effects.* The children exposed in utero are now 21 years of age, and the time is just arriving when the presence or absence of sterility can be observed.

JOHNSON: When I last looked at marriages in the group as of 1965, there were very few and of short duration. We do not have information at this point relating to reproductive performance.

SCHULL: Bob, are you telling us that the distribution of head sizes has been shifted to the left, that is, toward smaller sizes, and that the evidence for this is the absence of individuals with abnormally larger heads?

MILLER: I said there was one with a large head.

SCHULL: That's right. But there is no conspicuous increase at that end of the distribution, and since mean head size is diminishing with irradiation, presumably the whole distribution has been moved to the left.

MILLER: The brain didn't grow as much as usual and in consequence, the skull surrounding it did not grow.

UPTON: I wonder if you could explain a little more as to what the significance is of this particular period during gestational development. There may be some confusion as to what the pathogenesis of the small head is here as you see it. You hint that it is not an effect on the germ plasm.

MILLER: No.

UPTON: Why do you not think so and why do you think there is a certain age involved here?

MILLER: Injury to the germ plasm is an event which affects the individual before conception. Among those exposed in utero, injury

*Krise, G.M., H.B. Pace, and E.W. Hupp. Unpublished data from the Radiobiological Laboratory of Texas A&M University.

took place 7 to 15 weeks after the last menstrual period. So this was an injury to the embryo; something went wrong, and I can't suggest what, with the development of the brain in particular. I don't know of any postmortem examinations of the brains from these patients to determine what the abnormality was.

UPTON: Isn't this the period of major organogenesis, the time in embryological development when many tissues of the body are undergoing their rudimentary formation, and injury at that stage would be expected to be most likely to cause malformation?

MILLER: Right. It is interesting that in this instance the malformation was induced by a physical agent, ionizing radiation. German measles produces anomalies at about the same time in gestation: microcephaly, cataracts, congenital heart disease, and hearing defects in particular (Reference 122). Thalidomide in this interval produces short limbs (phocomelia) (Reference 123). In this instance the injury is due to a chemical. In Japan, near Minamata Bay, the people ate fish from the Bay into which a factory dumped mercury as an industrial waste. (Reference 124). The fish concentrated the mercury, and the people who ate the fish experienced an epidemic of a severe neurologic disorder. There was at the same time in the same area an epidemic of children born with cerebral palsy. So there are chemicals, viruses, and ionizing radiation which produce congenital malformations in man, but each agent produces a different type of congenital defect.

FREMONT-SMITH: Aubrey Smith has frozen hamsters and there's a critical period in the gestation period when the hamsters come out with a variety of congenital malformations if they have been frozen during pregnancy and then resuscitated, whereas at a slightly later stage these congenital malformations do not occur. So this is another physical agent.

MILLER: Right.

FREMONT-SMITH: Another agent operating in somewhat the same way with a peculiar, a special period of vulnerability.

ABRAHAMSON: Let me rephrase the question I asked you before. In the parents who did not have fetuses at the time, females or males in this case, who received exposures under 1,200 meters distance, did the head sizes of their children show any reduction in circumference?

MILLER: Preconception radiation? Is that what you're referring to?

ABRAHAMSON: Yes.

SCHULL: We were unable to demonstrate a reduction in head size among children conceived subsequently to exposure to the atomic bombings (Reference 125).

ABRAHAMSON: Then I would not expect this to follow up.

ANGEVINE: Is there any indication that there is a delay in the development of the skeleton?

MILLER: Yes. They were shorter as is characteristic in mental deficiency generally.

TAYLOR: Is it true that the German measles-induced deformities are never propagated to the next generation?

MILLER: There's no reason to expect that they would be.

WHITE: They are not.

MILLER: But the benefit of this study in Japan and of the previous studies before it is that it has gone a long way, I think, toward helping in the epidemologic identification of etiologic agents which produce congenital malformations. This is one of earliest studies that added to this literature and thinking.

ABRAHAMSON: There's one point I would like to mention with respect to the question Dr. Taylor just asked. There have been several articles (References 126, 127) published in recent years which suggest that a correlation exists between the outbreak of virus epidemics prior to, or around the time of, conception and increased incidence of chromosomally associated mongolism some nine months later.

TAYLOR: In the next generation or the one that was fetal at the time?

ABRAHAMSON: In offspring of people who were exposed, as I understand it. Am I right on that?

MILLER: Yes. There are two reports. The first, by Dr. Irene Uchida, appeared in Lancet a few years ago (Reference 128). She made a retrospective study which is bedeviled by the same problem that was discussed earlier today with regard to diagnostic intrauterine or preconception radiation in relation to leukemia. By questioning mothers of children with mongolism, she found that there was an excess frequency of maternal radiation prior to conception as compared with the histories given by mothers of children without mongolism. Dr. Lilienfeld and his group at Johns Hopkins (Reference 129) reported a sophisticated study in which a similar relationship was described. The question once again is, did the radiation contribute to the occurrence of mongolism, or did it merely indicate that the mother was different from normal?

SCHULL: We have been unable to demonstrate an increase in the frequency of mongolism in Hiroshima and Nagasaki which can be unequivocally related either to maternal or to paternal exposure (Reference 130). One cannot conclude from this, however, that ionizing radiation does not affect the frequency of chromosomal changes, for our data may fail to reveal an effect simply because the exposures and the numbers of individuals at risk are too small. While I personally believe that ionizing radiation will increase the frequency of chromosomal changes, the effects found by Uchida (Reference 128) and by Lilienfeld and his group (Reference 129) seem to me much too large if the exposures, etc., are taken at face value.

ABRAHAMSON: I spoke with Dr. Irene Uchida just a few days before the meeting and she supplied me with some unpublished data from a study that she has been carrying out over the past four years. The gist of her study was that there were nine chromosomally abnormal children (8 mongoloids and 1 chromosome 18 trisomy) born to women who had received abdominal irradiation prior to conception. There were a little over 1,000 women sampled in this group. In an almost equivalently sized sample of women receiving no abdominal irradiation prior to conception, only one case of mongolism (Down's syndrome) was observed.

UPTON: What are the doses involved?

ABRAHAMSON: In this study the doses were straight diagnostic irradiation to the abdominal regions.

SPEAR: Did I understand, Dr. Schull, that there may be a slight increase in the frequency of Down's syndrome among the exposed offspring that is not statistically significant?

SCHULL: No. Not only is there not a significant effect, but the "heavily exposed" parents have actually had fewer children with Down's syndrome than the "lightly exposed" or the nonexposed.

ABRAHAMSON: Does the Japanese population have a lower mongolism frequency than the population at large?

SCHULL: The present consensus is that the frequency of Down's syndrome among the Japanese is very similar to that in western Europeans.

ABRAHAMSON: The second question: Is mongolism as readily observed in the Japanese population as it is in others?

MILLER: It's very easy.

LIFTON: Why do you say it doesn't show up in the Hiroshima data if you think that it does probably exist?

SCHULL: Simply the number of cases that are at risk.

MILLER: But the doses were very large in Hiroshima.

ABRAHAMSON: And very low in Uchida's (Reference 128) studies. The Sigler-Lilienfeld (Reference 129) studies, however, did show that when therapeutic radiation was applied as opposed to diagnostic, the risk became much greater for mongolism.

MILLER: I don't remember that.

ABRAHAMSON: I have the paper here; I read it as I was coming out. I think this is true, that the therapeutic doses gave a much greater risk for mongolism.

MILLER: The findings from the Hiroshima survivors, to summarize, showed an embryologic effect which was proportionate to dose, and the findings were in accord with the results of animal experimentation and previous clinical observations.

Fetal loss following intrauterine exposure to the Nagasaki atomic bomb was described by Yamasaki et al. (Reference 131) in 1954. The pregnancies involved took place six years before the study was done. This investigation was made through histories given by the mothers. The women were classified with respect to their distance from the bomb and the presence or absence of major radiation signs. Of the 30 pregnancies which occurred among women within 2,000 meters of the hypocenter and with major signs of acute radiation sickness, 13 (43 percent) terminated in fetal wastage (abortion, stillbirth, neonatal death, or infant death). In the same distance category, but without major radiation signs, there were 68 pregnant women who were subsequently questioned, and 9 percent reported fetal wastage. The controls, who were too far distant to have received significant radiation (4,000 to 5,000 meters from the hypocenter), described fetal loss amounting to 6.2 percent.

Thus may the intrauterine effects to date be summarized. The suggestion has already been made that it will be of importance to study the frequency of sterility among those exposed in utero. Experimentally, in the study of oncogenesis, cancers are generally much more easily induced when the oncogenic agent is given early in life. Although the number of children heavily exposed in utero was small, it will nevertheless be of interest to determine, when they do die, what the frequency was of cancer as a cause of death.

SCHULL: May I make two observations here, Bob. First, there is another body of evidence relating mongolism in the Japanese to ionizing radiation which has not been mentioned as yet; the data stem from a study by Slavin et al. (Reference 132). They did cytological studies on all of the institutionalized cases of Down's syndrome in Hiroshima and Nagasaki, and then attempted to determine what proportion was the offspring of irradiated parents and what proportion was not. The proportions they found did not differ significantly from the general populations of these two cities.

The second observation concerns the Marshallese experience from which there has grown a body of data as large as or larger than many which have been published. There are now—I've forgotten the exact figure—possible 50 children who received exposures of as great as 150 r or so. To my knowledge there has emerged no conspicuous genetic or somatic effect among the younger people. But the details can be found in a recent paper by Conard and Hicking (Reference 133).

MILLER: Very few of the women were pregnant at the time of their exposures. There were no malformations among the children born (Reference 134).

I wanted to describe very briefly one other set of measurements among atomic bomb survivors. These concern not the in utero group, but people who were young, under 19 years of age, at the time of exposure. A study was initiated by Dr. Earl Reynolds to evaluate the growth and development of Hiroshima children, 1951–53; the results were eventually analyzed and published as a doctoral thesis and in the open literature by Dr. John Nehemius (Reference 135). The numbers of children studied, exposed as compared with nonexposed, are given in Table 6. The 12 measurements are height, weight, sitting height, span, shoulder width, hip width, head width, head length, head circumference, calf circumference, chest circumference and abdominal circumference.

Table 6. Growth study of Hiroshima children 1951–53 (Reference 135).[a]

Year	Exposed		Non Exposed	
	No.	%	No.	%
1951	2,422	100	2,238	100
1952	1,869	77	1,774	79
1953	1,293	53	1,182	53

[a] Twelve measurements: See text above.

The radiation-exposed sample consisted of 2,422 children. In the second year there were 1,869, which was 77 percent of 2,422.

JABLON: Reexamined?

MILLER: Reexamined, right. 53 percent had a third examination.

The results of multivariate analysis showed that as radiation dose increased, there were small but statistically significant decreases in the body measurements at all age levels, and in the growth rate

and in the change of growth rate following puberty. The differences in growth and maturation could be demonstrated by statistical tests but were of little consequence to the survivors for the differences were so very small (Reference 135).

BRILL: Do they conform to the earlier estimates of about 3 percent diminution in measurements/100 rad of exposure?

MILLER: Jack, can you answer that?

JOHNSON: That would be the approximate estimate from a recent study by Wood et al. (Reference 136).

MILLER: There is a possibility that nutritional differences between cases and controls could account for the differences in body measurements observed.

Finally, a word about mortality among atomic bomb survivors under 20 years of age at the time of death. By the time the data collection system was established, the youngest children exposed to the bomb were five years of age. In Hiroshima and Nagasaki 10 children who had been exposed within 1,400 meters of the bomb died between five and nine years of age between 1950 and 1960. Five of the deaths were attributed to leukemia. The other five may be compared with 1.9 deaths expected in this age group and time interval. There was an excess of three deaths observed. In the next age group, 10 to 19 years of age, there were 14 deaths, seven of them due to leukemia. There were seven other deaths as compared with 9.1 expected. It appears, then, that except for the deaths from leukemia, there is not much evidence of an excess mortality among children from exposures within 1,400 meters of the atomic bombs. Beyond 1,400 meters, there were 11 deaths among children five to nine years of age as compared with 16.5 expected, and among children 10 to 19 years of age there were 87 deaths as compared with 80 expected.

With regard to thyroid carcinoma, Socolow and his associates (Reference 96) reported 14 cases within 1,400 meters of the hypocenter in the two cities between 1958 and 1961. Six of the cases occurred among persons exposed to the bombs between 6 and 16 years of age.

BUSTAD: I have a question on that. I talked some with Ken and Seymour about it. But I was surprised that you had about 900 who

were exposed within 1,500 to 2,000 meters and there have been only two thyroid cancers in this group in the age group less than nine years of age. I looked at a special subgroup in Hempelmann's (Reference 137) study where the children were exposed very early in life to x ray in the thymic region. In a special subgroup where the thyroid was definitely in the field, there was about one thyroid neoplasm per 13 exposed or one cancer per 26, or something like this. I would like your opinion as to the reason for this apparent discrepancy. Is it because of the difference in ages of the children or the size of the dose?

JOHNSON: We have accumulated more cases in this age group, although I do not have rates. In the group within 1,400 meters who were zero to nine years of age at the time of the bombings, Dr. Wood (Reference 97) now has four cases, compared with two.

BUSTAD: Of cancer?

JOHNSON: Of cancer. Three in females, one in a male. In the age group 10 to 19, eleven cases, six females, five males. At age 20 to 29, three cases, female; 30 to 39 years of age, three cases, two males, one female. There were four in the 40 to 44 year age group and six in the 50 to 59 age group. The peak number of cases is in the 10 to 19 year age group.

BRILL: In what way did you ascertain those cases? In which samples were they encountered?

JOHNSON: These are all from Dr. Wood's examination of the Adult Health Study sample. This is the only sample where we can be sure that the thyroid gland was palpated without the knowledge of the examiner in the patient's exposure status. We do have a peak prevalence in those who were 10 to 19 years of age at the time of the bomb, but the experience of the 0- to 9-year group is not much different from other age groups.

UPTON: Does "peak prevalence" mean the largest number of cases or the largest of rates?

JOHNSON: I should say the largest number of cases; I can figure prevalence when I have a denominator.

MILLER: Were your cases examined the same way that Dr. Hempelmann's were detected? His were by careful clinical examination of his defined sample. Did you have an examination that was comparable?

JOHNSON: Yes. These patients are members of the Adult Health Study who have been to ABCC for examination every two years; usually, there are several examinations for each case. There are many biopsies.

MILLER: If there was no difference in the methods, then what accounts for the difference in the results?

JOHNSON: The difference may be related to the relative paucity of cases in the zero- to nine-year age group of our sample.

MILLER: But Dr. Bustad said there was one in twelve that ...

BUSTAD: Had thyroid neoplasm.

WHITE: What was the dose, Leo?

BUSTAD: I think that the dose in Hempelmann's (Reference 137) group was probably a little higher, around 200 r, but just to the neck region; maybe there were a few up to 600, or 900 possibly.

CASARETT: There were two series in Hempelmann's study.

BUSTAD: I'm talking about Subgroup C.

CASARETT: Yes. That was the high-dose group with anterior and posterior irradiation.

BUSTAD: That's right. 268.

CASARETT: That particular group would include doses of the order of 300 to 600 r, but series 1, which included the C group particularly, had an average dose of 326 r. To give you an idea of the comparison between series 1 and series 2, which had an average dose of 126 r, there were risk estimates made, respectively, of about 0.9 and 0.7 per million per year per rad.

MILLER: Was that strictly localized to the thyroid with no irradiation at all to the other areas?

CASARETT: There were a good many cases which probably included (by reconstruction, that is) the pituitary, but in series 2 the pituitary was more effectively kept out of the main beam because the exposure was anterior. The hypothalamus may also be involved.

BUSTAD: In the group I am discussing, which is Subgroup C, Hempelmann (Reference 137) is quite sure the pituitary was in it; isn't that right, in the anterior and posterior exposures?

CASARETT: Yes. One of the things that is important here is that most of these children in Hempelmann's study were infants.

BUSTAD: Yes.

MILLER: They also had large thymuses, which was the reason they received radiation.

CASARETT: Yes. This was the indication for irradiation, that is, in most of them it was a diagnosis of enlarged thymus.

BRILL: Didn't something over 90 or 95 percent of the cases come out of this one subgroup in the series in which the radiation doses are almost unknown? I think a 300- to 600-rad dose interval is a good broad range, but I wouldn't be surprised if it went even higher.

CASARETT: Yes, it could.

BRILL: The radiologist, as I recall, had died, his records had been lost or destroyed, and his nurse described from memory how she used to lay out the fields. I think the quantitation of these exposures is open to serious doubt.

CASARETT: Most of these cases of thyroid cancer came from the series of one physician, I believe.

I think you might be interested in what I consider something of a control for this, and that is the Conti work (Reference 138), which is also a study of a couple of thousand children.

BUSTAD: Yes.

CASARETT: In this study the average dose was 168 rad, and here the radiation was given prophylactically, that is, not upon indication or diagnosis of enlarged thymus. There was a long follow-up and no cases of thyroid cancer were observed.

BUSTAD: Yes, but he used a very small port and the thyroid was probably never in the field.

CASARETT: The thyroid could have been in the field, in some cases, but not the pituitary, and the thyroid was more often out of the field than in, in Hempelmann's study. But there were instances among these where the port size was big enough to include the thyroid.

I think that what we have to remember is that in all cases of data from therapy situations, one has to try to determine the effect of the disease being treated on the end result as well as the effect of the radiation, and there could very well be endocrine disturbances associated with, say, enlarged thymus and with enlarged adenoids and with hypertrophy or hyperplasia of lymphatic tissue generally. If this were so, that is, if the indication for irradiation were some manifestation of an endocrine disturbance, the sample population is a special population, and it is questionable whether one can relate these data to a more heterogeneous population.

FREMONT-SMITH: Isn't it very likely, though, that the clinical diagnosis of enlarged thymus was a very hazardous one, and in their day most of those cases would not be so diagnosed? Thus, it seems to me, the implication of an endocrine disturbance is very unlikely to be valid.

CASARETT: We don't know this. We question the diagnosis. That's why I said the indication was a diagnosis of this but I would not say that the thymus is necessarily enlarged.

FREMONT-SMITH: No, but if the thymus was not enlarged, if the diagnosis was wrong, and if the probability that enlarged thymus has anything to do with a general endocrine disturbance is doubtful, the whole suggestion that what we are dealing with is a special population of an endocrine disturbance becomes doubtful.

CASARETT: Right. What I would say is that there was something wrong in the area that was diagnosed, perhaps mistakenly. If the lymphatic tissue was hyperplastic, we know, for instance, that adrenal

cortical insufficiency can result in a hyperplasia of all lymphatic tissue, including thymus. However, this is speculative in regard to the cases in question.

FREMONT-SMITH: I think that one should cast a high degree of doubt over the whole business...

CASARETT: I do.

FREMONT-SMITH: On whether these children had anything the matter with their thymuses. I say this with some degree of feeling. One of my own children had an x ray of the thymus because the nurse noticed on the very day after he was born that he was choking slightly, and he was rushed to the hospital and had an x ray. This is the kind of basis on which in those days, because of the idea that sudden death in infants was due to enlarged thymus, the diagnosis was made. Anyway, I'm just showing my prejudice.

UPTON: I would like to ask Dr. Miller, who put the thyroid into the discussion, if he could sum up for us what he thinks the data do, in fact, mean.

BUSTAD: Can he include my next question in his discussion, and that is, your group of irradiated adults are the only ones reported to show an increase in thryoid tumors; there is no other group of adults I know of that have been irradiated in the thyroid region that show an increase in thyroid cancer. If you know of cases other than the Japanese, I would like to know about them.

MILLER: I haven't done the work myself.

FREMONT-SMITH: You said it is the only group. But there was the Nevada group, wasn't there?

BUSTAD: No. You're talking radioiodine; I'm talking x ray. There's no proven cancer of the thyroid due to radioiodine alone.

FREMONT-SMITH: No. There are nodules, are there not?

BUSTAD: There are some adenomas.

FREMONT-SMITH: And not unproved cancer yet.

BUSTAD: There is a lady among the Marshallese (Reference 139) and a child in Sheline's group (Reference 140), but there is a question whether it is definitely a cancer.

UPTON: Bob, could you try to put this into focus for us? Where are we going now on the thyroid study?

MILLER: The presentation that I made was intended to describe radiation effects among those in utero or during childhood, and for completeness I included information on thyroid cancer. Six of those who developed this neoplasm were exposed as children. The thyroid was mentioned here also to give Dr. Bustad a chance to finish his discussion pertaining to this organ.

UPTON: As background, there are data from other studies pointing to an association between irradiation of the thyroid gland in infancy and the subsequent development of thyroid nodules or, in some instances, thyroid cancer. The Japanese data, I think, come in because of the earlier information. The question then is, can one identify in the Japanese a particularly high susceptibility among those irradiated early in life, which seems to be characteristic in the other literature?

JOHNSON: The prevalence rates I have obtained from Dr. Wood's data are as follows: for males under 20 years of age at the time of the bombing and exposed within 1,400 meters of the hypocenter the observed prevalence is 9.8/1,000 persons.

I have more complete prevalence data for females (Table 7). The rate for those within 1,400 meters and under 20 years of age in 1945 is now 10.7/1,000 persons; 1,400 to 1,999 meters, 3.1/1,000; 3,000 to 3,900, 1.0; and not-in-the-city 1.4/1,000. The rate is also high in those persons who were within 1,400 meters and more than 40 years of age; it is 8.6/1,000 persons examined.

BRILL: Ken, do you have an under age 15 as well as age 20?

JOHNSON: No. The number of cases is small and does not allow many age subgroupings.

BRILL: I ask this because, in Western population, at least, thyroid cancer is exquisitely rare below puberty. Furthermore, in patients irradiated for cervical lymphadenopathy it looks like those

Table 7. Prevalence of thyroid carcinoma in ABCC Adult Health Study sample (Reference 47).

(Rate per 1,000 persons examined; Hiroshima and Nagasaki)

Age at exposure	Exposure category			
	< 1,4000 meters	1,400–1,999 meters	3,000–3,999 meters	Not exposed
Females				
< 20	10.7	3.1	0.0	1.4
40–59	4.4	3.7	3.6	3.8
≥ 60	8.6	2.2	2.0	0.0
Males				
< 20	9.8			

irradiated in childhood have a higher incidence of thyroid cancer than adolescents, who in turn have higher rates than adults with rather long latent periods. It would be very interesting to look at the ABCC thyroid cancer data in children under age 15 as opposed to the 15 to 20 age group.

JOHNSON: Remember that we are talking about age at the time of the bombings. If you want the age at the time of detection, you should add 20 years.

Part of our difficulty in speaking of thyroid cancer as rare or common is the uncertainty of its detection. It is a benign disorder not contributing in our sample to morbidity or mortality. If you do not look expertly for it, it will escape detection. One gets the impression that thyroid carcinoma is more prevalent that we realize in every population.

MCCULLOCH: Can you give us a feel for the dose in those range groups?

JOHNSON: I think Mr. Auxier can do that.

BUSTAD: Is that 1,400 to 1,999 and then you go to 3,000?

JOHNSON: It is as stated. We skipped.

AUXIER: It's a pretty reasonable thing to assume that all those beyond 1,400 had 50 rad or less, considering the dose curve and the shielding data.

UPTON: For those under 14 you are including those well within the lethal range; I mean at the tail end of the survival curve? From this we can't see any definitive indication of a difference of susceptibility with age? Is that fair to say?

JOHNSON: Yes.

BRILL: I don't see that. I look at 10.7 versus 0 or 1.4. I don't know what the proper comparison basis is, but that is a whopping big difference as opposed to 4.4 versus 3.6 or 8.6 versus 2.0. It depends heavily upon what rate for the nonexposed you accept.

UPTON: If we're comparing ages, we compare the numbers vertically; is that right?

BRILL: In respect to the nonexposed.

UPTON: If we compare doses, we're comparing horizontally.

BRUES: The point is that 10.7 represents some number of individuals, and in assessing the significance I would like to know whether that is less than 10 or more than 10 individuals.

JOHNSON: I do not have all the data on hand, but I remember it as being close to 10, and at least 9.* In any case, prevalence is based on cases observed divided by the total number of persons examined in the particular exposure age and sex category.

What I thought you implied, Arthur, was this: The prevalence of 10.7 in females under 20 years of age in 1945 and 8.6 in females over 60 years of age, both groups within 1,400 meters, seems to indicate that age at time of exposure was not as large a factor as it was, for example, in leukemia.

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*Actually, 13 cases.

UPTON: We don't have standard deviations, and we can't really compare one number with another, but I would infer, likewise, that looking at the incidence in the irradiated populations, there is no definite age dependency.

CASARETT: Did you get any impression of a big sex difference in the older age group that might relate to menopause?

JOHNSON: I have not. Offhand I couldn't answer that. Most of the cases are females except in the under 20 years of age at the time of exposure. The point is that we lose sex differential in those who were exposed young in life but after that most of the cases are females.

CASARETT: The interesting thing here is that both sexes undergo pubescence but only one undergoes the menopause, that is, a second cataclysmic endocrine disturbance, and this might be a biphasic situation with respect to the female but not to the male.

BRILL: It seems to me that I would be concerned as to whether there was a suggestion of an irradiation effect that was different from the youngest person to the oldest population. One could be very certain that there was an effect in the first place. The problem of diagnosis in thyroid cancer is a very serious one and subject to great differences in opinion. When you have very few cases and when there are disputed diagnoses in that small group, then I become a little bit concerned about drawing conclusions, either positively or negatively. When you look at the Mayo Clinic series, for example, the first go-round on the series they found a prevalence of thyroid cancer in their a.. opsy experience of less than 1 percent. After several reviewers worked over the slides on these patients, and multiple new sections were made, the prevalence rose to approximately 5 percent. Thus, the frequency with which you diagnose thyroid cancer depends heavily upon the intensity with which you pursue this diagnosis.

UPTON: Are you comparing across the board horizontally?

BRILL: Right.

UPTON: Asking whether these numbers are really different?

BRILL: Yes. If one is to document an excess of thyroid cancer in the most closely exposed, the best comparison group is probably the prevalence in those persons exposed between 3.0 and 4.0 km, since

the nonexposed are in many ways nonrepresentative. Thus, for a radiation hypothesis one is comparing a properly weighed sum of the prevalence in the under 1.4-km survivors of all ages to the 3.0- to 4.0-km group. On the other hand, if one is looking for an age effect, one looks at the ratio of incidence in the near to far groups as a function of age, that is, 10.7/0 in the under 20 females, is to be compared to 8.6/2.0 in the survivors over 40 years of age. Since the numbers in each cell are small, this is a hazardous comparison, but it is in line with an age effect. Although I don't think you can say that it demonstrates the presence of an age effect, conversely I feel strongly that one should not state that the data do not show an age effect, as I thought was being implied.

UPTON: There's a radiation effect?

BRILL: There is a suggestion that there is a radiation effect and an age effect with the youngest being most sensitive. However, I don't place much confidence in the findings at this point.

BRUES: I happened to bring along a statistical table (Reference 141) of Poisson probabilities because I thought it might be useful. From it I find that if there were nine cases in that group where the rate is 10.7, there is a 5 percent probability that nine cases might have been drawn from a population at which the expectation would be 4.7 cases or less. Relating that to the figure of 10.7, that means a 5 percent probability of a population as low as 5, a rate of about 5.8 from which this might have been drawn. These rates on the right are so sufficiently lower than that, that I think they are, on the face of it, significantly different assuming that they are unbiased.

UPTON: Murray, the question has been raised about the significance of the diagnosis. You have seen many of these slides. Would you like to comment on this aspect of the question?

ANGEVINE: I think now there is probably fairly good agreement, but there are several types of carcinoma, as I indicated yesterday. Many of them are microcarcinomas, which means that they are very small. There is often a question as to whether they have invaded the lymph nodes. The incidence of thyroid tests in lymph nodes has yet to be answered.

I think that one of our difficulties may be due to the fact that we have looked at this area a bit too closely and have found some things

that need further study. I suggested to Dr. Gen Niwayama that he should do such studies while he was at Roswell Park. I said, "Why don't you remove the thyroid with the adjacent lymph nodes on a series of cases in Buffalo as meticulously as you did in Japan?" We don't really have a good baseline for comparative studies and we need one badly. A recent report (Reference 142) is helpful.

I would not quarrel with the diagnoses. I agree with the way that Dr. Johnson has presented them, but of course many papillary lesions may remain localized and never extend any further. This is one of the difficult areas of diagnosis in pathology and I would venture to say that it will probably take time and greater numbers of follow-up cases before we can answer the question definitively.

SCHULL: Bob Miller wishes to make a few remarks about leukemia which may serve as a preamble to Court Brown's presentation.

MILLER: I think it is important to consider where the findings on radiation-induced leukemia stand in regard to other factors related to leukemogenesis. First, let us consider groups at exceptionally high risk of the disease. MacMahon and Levy (Reference 143) have reported that when an identical twin develops leukemia in childhood the probability that the co-twin will develop the disease is one in five, usually within weeks or months of the time that the first twin is affected. This is the group at highest risk of leukemia of any so far known—one in five, usually within weeks or months.

COURT BROWN: Could I make the point that we've been unable to confirm this finding in Britain when we studied this?

MILLER: Yes. This is a United States finding. It cannot be confirmed in Great Britain, but we have seen subsequent cases in the United States beyond those that have been described by Dr. McMahon. The same is not true of fraternal twins; they are not at that increased risk.

MCCULLOCH: Which kind of leukemia is that?

MILLER: At risk of leukemia; the kind is not given.

MCCULLOCH: Which was in the identical twins, do you know?

MILLER: I don't remember if the types of leukemia were concordant or if the types were known.

Among persons with polycythemia vera treated with x ray and P-32, the risk of leukemia was one in ten, but over about eight years (Reference 144). In Down's syndrome, the risk is about one in 75 over 10 years (Reference 145). Among Hiroshima survivors within 1,000 meters of the hypocenter, the rate was about one in 100 over eight years (Reference 82), quite different from the others except for the Down's syndrome. Sibs of leukemic children show a leukemia rate of one in 625 over 10 years (Reference 146), and among United States white children, in general, the rate is one in 2,500 over 10 years.

In Down's syndrome there is an extra chromosome of the G group, and the risk of leukemia among these children is 20 or more times greater than usual (Reference 145). Presumably the increased risk is in some related to the extra chromosome which occurs due to an error in division before conception of the child. Leukemia in Down's syndrome, therefore, is presumably related to an event that occurs before conception.

With regard to Klinefelter's syndrome, in which there is an extra sex chromosome (XXY), there has been no epidemiologic study, but only case reports, suggesting that this congenital defect may predispose to leukemia (Reference 109).

In D-trisomy there is an extra chromosome in the D group; again there has been no epidemiologic study concerning the occurrence of leukemia with this congenital anomaly. D-trisomy is a rare disease. The affected children do not live very long, so they do not have much time in which to develop leukemia. Yet two cases have been reported of leukemia in children with D-trisomy (Reference 147, 148).

When chromosomes are studied of children with leukemia in remission, abnormalities have been detected that would not otherwise have been suspected by physical examination of the child; that is, malformations of the child either escaped notice or were not present. Borges and his colleagues (Reference 149) have studied 25 children in this way; three of the 25 had extra chromosomes demonstrable in both the skin and blood, a circumstance which signifies that the cytogenetic abnormalities antedated the leukemia and were not a result of the disease. In one child the extra chromosome occurred in the F group. The mother had a minor clone of the same chromosomal abnormality. Another child had probably an XYY sex-chromosome pattern of the skin and blood. The third had Klinefelter's syndrome, which had been overlooked on initial physical examination, but which

was clearly apparent when the child was reexamined following discovery of the abnormal sex chromosome complement.

Thus far I have been talking about extra chromosomes. Our attention now shifts to translocation of a piece of one chromosome onto another. There have been two cases reported which concern a D/D translocation, one in a woman who was phenotypically normal but developed chronic myelogenous leukemia (her son was a carrier for the same chromosomal abnormality, see Reference 150), and the other case was a translocation in a child with mongolism who had congenital myeloctic leukemia (Reference 151).

There are two inherited syndromes, rare diseases—Bloom's syndrome and Fanconi's aplastic anemia—which are characterized by excessive chromosomal breakage in tissue culture. There are 23 cases of Bloom's syndrome known to date. Three have developed leukemia. Three out of 23. A fourth case has developed cancer of the tongue (Reference 152). Among persons with Fanconi's aplastic anemia there are two cases known with acute monocytic leukemia, a relatively rare form of the disease (Reference 153).

Ionizing radiation breaks chromosomes and definitely carries an increased risk of leukemia (Reference 44). Benzene also breaks chromosomes and very probably is associated with a greater normal frequency of leukemia (References 154, 155). Certain viruses also break chromosomes, but as yet have not been implicated in leukemogenesis. Thus, leukemia has been associated with pre-existing chromosome abnormalities which may be either inherited or acquired.

By contrast, genetic disorders in which there is a severe immunologic deficiency predispose to lymphoma or acute lymphocytic leukemia, but thus far not to other forms of leukemia (reviewed by Miller in Reference 109). There are three such genetic diseases. In congenital agammaglobulinemia, of 24 cases reported by Page and his associates, one developed leukemia and another lymphocytic leukemia. A third case, from Zuelzer's group in Detroit, had leukemia. Other similar cases have been described with congenital hypogammaglobulinemia, secondary to underdevelopment of the thymus. One developed reticulum cell sarcoma, and another was found at autopsy to have a leukemic stem cell line.

A second genetic disease with severe immunologic deficiency, ataxiatelangiectasia, causes children to stagger and have dilatation

of the blood vessels, particularly in the conjunctivae. The number of case reports of solid tumors of the lymph system among children with this disease has been growing rapidly in the past year.

The third genetic disorder in this category, Wiskott-Aldrich syndrome, characterized by eczema and thrombocytopenia, has also been the subject of a recent spate of case reports concerning the occurrence of solid lymphoid tumors in children affected with this congenital anomaly.

One can summarize schematically these relationships (see Figure 43). Certain syndromes with chromosomal abnormalities are associated with leukemia. This relationship is independent of the genetic abnormalities with immune deficiency which are associated with lymphoma.

ABRAHAMSON: I have seen two groups of disorders and associated fundamental groups of cancers of one sort or another. Would you not be willing to extend this statement to something that George was trying to imply before, that there is a great deal of genetic heterogeneity in the population, and based on this fact, there may be lots of what we might consider normal individuals running around who would have the same proclivity toward one or another kind of cancer under the exposure of irradiation because of this heterogeneity?

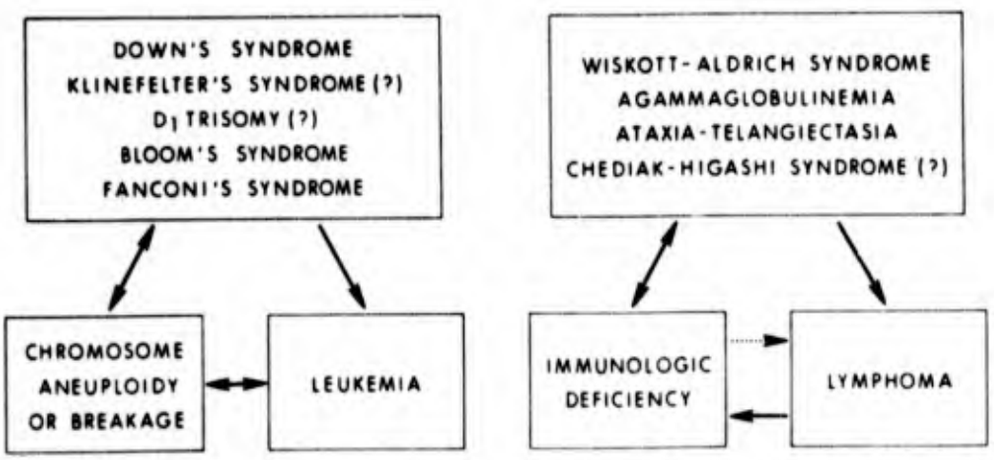

Figure 43. Relation between genetic diseases and leukemia or lymphoma. (This figure to be published in J. Nat. Cancer Inst. April, 1967, by J.R. Fraumeni and R.W. Miller.

MILLER: Yes. You mean that some people are genetically prone to develop leukemia given the right stimulus.

ABRAHAMSON: And x rays may act as a stimulus. This is summarizing one of the points you were making before.

MILLER: Yes.

FREMONT-SMITH: There's another way of saying it: that a genetic change only takes place when the appropriate environment is available; right?

MILLER: Yes.

IONIZING RADIATION AND CHROMOSOMAL CHANGES

COURT BROWN: I would like to start by saying that the time has come, I think, in this meeting, to turn our attention to what ought to be done in the future to answer some of the perplexities which only too obviously have arisen from the observations made so far on irradiated populations. At the risk of being provocative, I think that, to a large extent, clinical medicine has had its day in looking at irradiated populations. I think, to a large extent, classical pathology has had its day, and I think that, as I've indicated, certainly when you are worrying about the effects of low dosages of radiations, there are important limitations to epidemiology.

So the point is, now we must decide what we are going to do in the future, and one important bridge to the future is now recognized to be the study of human cytogenetics and the cytogenetics of irradiated populations.

Much of this will be somewhat unfamiliar or perhaps totally unfamiliar to a number of the people present. So I am going to present the findings in irradiated populations as simply as possible. These are now at a very interesting stage of development.

As probably many of you know, there has been a considerable explosion in the study of human cytogenetics, which was triggered off in 1956 when Tjio and Levan, as a result of bringing together a number of techniques, were able for the first time to produce preparations of cells in which the human chromosomes could be counted accurately and sorted out in terms of their size, and so on, that is, analyzed, to use the term which is current among cytogeneticists.

Here we are talking, of course, about the chromosome complement as seen in mitotic cells. It was not until 1959 that the first fruits of the work of Tjio and Levan began to appear, and these were the original publications pointing to aberrations of chromosome number in a number of conditions, congenital malformations, like mongolism, or conditions in which there were deficiencies in sexual maturation, like the so-called Klinefelter's syndrome.

In 1960, a very important thing happened, and this was the description by the Philadelphia group—Hungerford, Nowell, and collaborators—the blood culture technique. Up to that time work had been done on cultures of marrow cells, developed in Britain, and cultures of fibroblasts, developed by Lejeune in Paris. But these techniques have disadvantages. The tissues are not all that accessible, and a big advance was made when it became possible to culture white cells from the blood and make preparations in which there were many cells available for the counting and analysis of chromosomes.

In that same year it was recognized for the first time that if one applied the blood culture technique to an individual or individuals who had had very large doses of radiation, one could easily see the aberrations induced by radiations. Since that time there has been a steady buildup of interest in the subject of radiation-induced chromosome aberrations in man, and I think in the future this will merge into the more general subject of environmental agents producing chromosome damage, of which radiations are only one agent.

In the blood culture technique a sample of blood is taken and the red cells are separated from the white cells. The white cells are set up in a fluid medium which contains the necessary requirements for the growth of these cells together with a substance known as phytohaemagglutinin, derived from the American navy bean. This substance has the property of stimulating the lymphocytes in the culture to divide. It is generally agreed that the cells which do divide in these cultures are lymphocytes. The technique of making preparations for the study of chromosomes involves a deliberate interruption of cell division at metaphase, and the subsequent handling of the cells so that the chromosomes come to be spread out as far as possible, completely separated from one another.

First of all the chromosomes are counted and then they are sorted into groups. There are limitations at present to the extent to which we can sort them, but we can recognize without much difficulty some individual pairs, for example, pair No. 1, 2, 3, and 16. This sorting is done on the basis of length and centromere position. In many instances, however, we can't distinguish between individual members of a group whose morphology is very comparable; for example, the B group containing pairs 4 and 5.

If one takes a sample of the population and does a blood culture on every individual, then the individuals can be divided into two broad

categories. There is a small proportion all or many of whose cells show a constant abnormality of the chromosome complement, while the great majority have a normal complement. Among the latter, however, it is usual to find a few abnormal cells, many of these being preparative artefacts but some possibly caused by environmental agents or related to the aging process.

Broadly speaking, we may take it that with the current techniques we can recognize that about 1 percent of liveborn babies have an abnormality of the chomosome complement detectable in mitotic cells. These can be broken down into approximately as follows. About 0.25 percent have an abnormal sex chromosome complement; about 0.25 percent have an extra autosome, mostly mongols; while in about 0.5 percent we can recognize in mitotic cells that there has been a rearrangement of material, most commonly a reciprocal exchange of material between two chromosomes, that is, a reciprocal translocation. I don't want to take this particular aspect any further, except to say quite clearly that many of us would be very interested indeed to see what in fact was the frequency of individuals in the F_1 generation in the Japanese with structural rearrangements.

BRUES: These figures are based on unselected material? These are not people that were complaining?

COURT BROWN: This is a reasonable approximation to what you would get. I could spend the rest of the day discussing how one got these figures. I should say this, however, that they are underestimates. They do not approach the true frequency, and we don't know by how much they fail to approach it. I shall leave this aspect of cytogenetics and go on to the problem of radiation-induced structural rearrangements in mitotic chromosomes.

The period of time spent by a cell between successive divisions is divided into three phases. These are known successively as G_1, S, and G_2. During S the chromosomes are synthesizing DNA in preparation for the next division, and during this phase they become visibly double-stranded, as they can be seen to be made up of two chromatids, each of which will become a chromosome in a daughter cell.

The type of aberration produced by radiation is dependent on whether the chromosomes are irradiated in G_1, in the single-stranded phase, or when they are in the double-stranded phase. As far as we're concerned, in making observations on cultured lymphocytes,

we may take it that all lymphocytes circulating in vivo may be regarded as effectively in G_1. So that when breaks are produced, the breaks involve whole chromosomes, and when these breaks interact to produce rearrangements these rearrangements involve whole chromosomes. If, however, irradiation occurs in S or in G_2, then the breaks involve chromatids and the situation becomes much more complex. But fortunately, as far as we're concerned, we may regard all lymphocytes as being in G_1. There isn't any evidence at all from blood culture studies that in vivo irradiation or the in vitro irradiation of human lymphocytes leads to any increase in the frequency of chromatid aberrations following breaks in chromatids. The increase in frequency in aberrations is always in those of the chromosome type following the production of breaks G_1.

Radiation through complex physicochemical processes produces breaks in the continuity of the chromosomes. The broken ends are described as "sticky" because they have the facility to adhere to other broken ends. If you imagine a situation in which two different chromosomes lie side by side and a break produced by irradiation occurs in each chromosome at the same level, then, provided the distance separating the two chromosomes is not too great, there is the possibility of the interaction of the broken ends of one chromosome with the broken ends of the other chromosome. This distance is known as the rejoining distance, and currently it is thought that interaction of broken ends can occur if the rejoining distance is on average about 0.1 of a micron.

There are really three possibilities in the situation I have described. One, of course, is that the breaks can heal and restore the continuity of each chromosome. This is known as restitution. Second, the ends proximal to the breaks may join to produce a chromosome with two centromeres, or a dicentric chromosome. As the ends distal to the points of breakage also join, there will be a fragment of chromosome material produced in addition, that is, an acentric fragment. Third, the broken ends can interact to produce two monocentric chromosomes, the morphology of which can be, but not always is, different from the morphology of other members of the karyotype.

This brings me to an important point. Because each of the products of the third possibility has a centromere, they are able to align themselves in the mitotic spindle, and they behave normally at mitotic division. In contrast, chromosomes with two centromeres are likely

to get into difficulties at division. Because of this latter probability, this type of rearrangement has come to be known as an unstable rearrangement, and the cells carrying it known as unstable cells. In other words unstable cells are those that contain rearrangements, the nature of which increases the likelihood of the cell failing to survive division, or if it does, there is an increased likelihood of the death of one or both daughter cells. So unstable cells have a high probability of failing to survive division, and this is supported by work from plant and other material.

In contrast, the type of rearrangement producing two monocentric chromosomes is associated with stability at division. Consequently cells carrying stable rearrangements are known as stable cells. I have illustrated the unstable cell as one with a dicentric chromosome and an acentric fragment, but we also include in this category cells with ring chromosomes, with or without fragments, and cells with only fragments.

I should make the point that there are a number of rearrangements of the stable type which we can't detect in mitotic cells. In fact, if equal amounts of material are exchanged between two chromosomes, one cannot detect morphological changes in the product chromosomes in mitotic cells. Nor can one detect paracentric inversions or pericentric inversions if in the latter the points of breakage have been equidistant from the centromere. So there are important limitations to the extent to which we can detect stable chromosome aberrations. But we believe that the very great majority of unstable aberrations are detectable, and most of the work that is being done in radiation is based on the scoring of cells with unstable aberrations. In relation to this meeting, however, it is possible that the cells with unstable aberrations are not so significant in regard to long-term radiation effects. If abnormalities are important in relation, for example, to the development of tumors, then it may be that it is the stable abnormalities that require most consideration, just, in fact, those that we have the greater difficulty in identifying.

Before going on to describe what I think is probably, certainly in qualitative terms, the characteristic response to an acute dose of radiation in terms of chromosome aberrations recognized in lymphocytes, there are one or two important technical problems to mention.

The first concerns the question of the sheer labor of counting and analyzing chromosomes in a cell. This is done on the microscope

and it takes our people about 4 minutes per cell, but they can't keep up this rate of scoring for long. One can't ask an individual to count and analyze more than 50 cells a day. So that really we have a considerable problem here in that we are restricted in terms of the number of cells we can process with the current techniques. When you get down to the problem of scoring small increases in the frequency of aberrant cells in various situations, as in people in occupations exposed to radiation and so on, one has to count and analyze very large numbers of cells indeed to limit random variation, and we're thinking in terms of counting and analyzing, let's say, 500 cells per culture. This is an immense task, and it is in fact a task which no one at present undertakes.

Alternatively, forgetting about radiation problems for the moment, to get really good population data one has to examine very large numbers of people, and again this means a very large number of cells to be processed. So that several groups have decided to try to develop automated techniques for doing this. There is a group, for instance, in Philadelphia headed by Mendelsohn, and there is Wald and his group in Pittsburgh, while we are also developing this technique in Edinburgh. With the advent of automated techniques it will be possible to develop the study of radiation damage much more than has hitherto been practicable.

Another important technical feature is the problem of the conditions of culture, particularly in respect to the time cells are kept in culture before they are harvested and the final preparations are made. This is especially important in relation to the cells with unstable aberrations because the more divisions these attempt to go through, the more likely they are to get lost. So it is desirable that the technique should be so standardized that cells are examined when they are in their first division in culture. A consensus of opinion is developing that the optimum time is after 40 to 50 hours in culture, although it is fair to say that Bender and his colleagues from Oak Ridge disagree with this. However, good evidence to support this was originally produced in 1964 by Buckton and Pike, and more recent support has come from the work of Heddle, Evans, and others.

FREMONT-SMITH: Do I get the implication that the unstable cells are not significant because they are going to drop out of the situation?

COURT BROWN: We'll come back to that. I think that probably unstable cells are not particularly significant in terms of long-range harmful somatic effects, and they are likely to be quickly eliminated from tissues with a rapid cell turnover.

What I want to go on to now is what happens after a high dose of radiation. This has been worked out for a period of time up to 10 years or so on the individuals given a single course of x rays for ankylosing spondylitis. We must first of all consider the control preirradiation values for unstable and stable cells.

These control values are based on a very large number of cells, nearly 2,600 cells, counted and analyzed from a substantial number of spondylitics prior to any form of x-ray therapy. These do not differ significantly from the sort of values that are obtained in any ordinary members of the population.

MC CULLOCH: Excuse me. Have these people ever received any radiation?

COURT BROWN: They've received diagnostic radiation but they have never received any therapeutic irradiation. These are values obtained prior to getting therapeutic radiation.

Cells with unstable operations run about 0.5 percent of cells examined, as do also cells with stable aberrations. In these spondylitics one can find marginal increases in the number of cells with dicentric chromosomes, possibly the result of diagnostic x-ray exposure.

Now we come to the behavior of unstable cells and stable cells with time after radiation, starting scoring at time zero with the completion of x-ray treatment. For the record, all these individuals were given a dose of 1,500 rad to the skin over the spinal column and over the sacroiliac joints in 10 equal fractions in a fortnight, and this is medium-kilovoltage x-irradiation with a half-value layer of about 2.6 mm of copper.

Some very interesting things happen. The first thing to note is the very big increase at time zero in the numbers of unstable cells, from about 0.5 percent up to about 30 percent or more. There is also a big increase in stable cells, but the increase noted is not as great as in unstable cells and runs at about 10 percent. However, it must be remembered that we can detect only a proportion of the stable abnormalities produced.

The unstable cells expressed as a percentage of the total cells examined start to fall quickly as from the end of exposure. This

rapid fall is maintained for about two to three years, but thereafter the rate of disappearance of these cells slows up considerably, so much so that ten years or even more after exposure the level of unstable cells is still significantly increased above the control level. The behavior of stable cells is very interesting also. Ten years after exposure the percentage of stable cells is not significantly different from the percentage found at time zero.

Remember that we are dealing with lymphocytes and it is important, therefore, to look at the changes occurring over the same period in the peripheral blood lymphocyte count. Immediately after treatment the level is found to have between 500 and 600 lymphocytes per mm^3 as opposed to a preirradiation mean value of between 2,000 and 3,000 per mm^3. Full recovery to the preirradiation control levels does not occur until somewhere about four years after the end of exposure, and the much slower rate of disappearance of the unstable cells is associated with observations made subsequent to the time in which the lymphocyte level had returned to control levels. The recovery of the lymphocyte count must be a very complex process indeed, and we simply don't understand anything about it.

BRUES: You might say it appears to be very much longer in man than it is in mice and some of the small animals.

COURT BROWN: Yes.

The question has been raised of what is happening to the unstable cells and why are they remaining in the circulation for quite a long period of time. As soon as people started to observe individuals previously exposed to acute doses of radiation, for example, the work of Bender and Gooch following the Y-12 criticality accident or our own work on patients treated with x rays, it became clear that cells were being found at long periods after exposure which contained aberrations consistent with these cells being in their first division since irradiation when they were examined; for example, cells with a dicentric chromosome and an acentric fragment. These findings were puzzling in the light of the views then held that lymphocytes were cells which were turning over rapidly and had a mean life measured in days rather than in months or even in years. In fact these findings were only compatible with the mean life of the lymphocyte being much longer than had hitherto been thought possible.

From these findings there has evolved an attempt to measure the mean life span of the lymphocyte from an examination of the rate of

disappearance of unstable cells. This has been done in the United States by Amos and his colleagues from an examination of women treated with radiation for cancer of the uterus, and they came up with an estimate of a mean life span of about 530 days. From our own data, and taking our observations as from the time the lymphocyte count returns to preirradiation levels, we have an estimate for the mean life span of about 1,574 days with 95 percent confidence limits of 891 and 6,743 days.

The point is that evidence is now accumulating for the mean life span of the lymphocyte being much longer than anyone had ever considered, and these findings are important in the general problem of the nature of immunological memory. They are also important to the present discussion and in relation to the effects of chronic radiation exposure. For example, in a chronically exposed population, such as atomic energy plant workers, the proportion of cells observed with unstable aberrations at any one time will be determined by the rate of annual accumulation of dose, the amount of the dose per annum, and the annual loss of unstable cells.

We imagine that unstable lymphocytes will be lost when they are stimulated to divide in the course of their function as immunologically competent cells, and when they meet with the appropriate antigens to which they are sensitized to respond by transformation and division. A consideration in the spondylitic of the mean number of breaks per unstable cell estimated to account for the observed damage shows that this number does not change with the lapse of time after exposure, certainly up to 10 years. Nor does the proportion of such cells estimated likely to be in their first division when examined since the infliction of radiation damage. These cells are known as X_1 cells and in our data run pretty consistently at about 80 percent of the total unstable cells examined. We feel, therefore, that the evidence so far is in keeping with the idea that the amount of contained radiation damage is not influencing the ability of the cell to respond to the stimulus to synthesize DNA and then divide.

BUSTAD: Can I ask you a question here. I might have misunderstood something in your explanation. When did you start? Your results are over a period of 10 years, that is 10 years are shown, but you started this...

COURT BROWN: We're backtracking in time. We're picking up people who've been irradiated at some time in the past.

BUSTAD: That's what I mean, yes.

COURT BROWN: These data are composed of people whom we have examined ourselves, who have been irradiated, and whom we have watched all the time from irradiation onward, plus people who've been irradiated with the same dose but sometime previously, but all the people have only had the one form of exposure.

BUSTAD: Yes, but you have repeats on quite a few.

COURT BROWN: Surely.

BUSTAD: How many times has the same individual been checked? Do you get what I mean? In other words, did you start this in 1963 or 1964?

COURT BROWN: We started in 1960.

BUSTAD: I see.

COURT BROWN: To be precise, our data are based on the examination of 129 blood samples from 66 men at periods ranging from day zero up to 3,700, plus the control observations on about 50 men.

BUSTAD: That's actually what I was interested in.

COURT BROWN: The trouble is that the number of patients available for this sort of investigation is not large, because although many spondylitics are or have been treated, they tend to get retreated fairly quickly and, of course, you can't use these.

UPTON: Do you intend to say something more about the steeper exponential that you see early?

COURT BROWN: We just don't know what is happening during the first few years when recovery is taking place in the lymphocyte system. It must be complex enough in the circumstances of whole body exposure and even more so following partial body irradiation. In our estimates of life span we have assumed that once the blood lymphocyte count has returned to the preirradiation levels, then the whole lymphopoietic system is once again in a stable state.

FREMONT-SMITH: Would you say that, if an individual were exposed to a series of antigens as opposed to one that had been protected

from antigens, in the first case the lymphocytes would have a much shorter life because they would have divided?

COURT BROWN: Nowell of Philadelphia has some rather interesting preliminary data along these lines. I don't think that he claims that this is as yet conclusive, but it is interesting. He has taken irradiated individuals and studied the unstable cells in cultures to which phytohemagglutinin has been used as a mitogenic agent, and in cultures in which the antigen tuberculin has been used. After about six months following exposure the yield of unstable cells in tuberculin-stimulated cultures virtually disappears, if I remember correctly, whereas the yield of cells from PHA-stimulated cultures is not materially changed. This can be looked at from the point of view that tuberculin is a commonly encountered antigen.

To sum up so far, I believe that what I have described of the behavior of unstable and stable cells after radiotherapy does, in qualitative terms, represent the standard response to an acute dose of radiation. There will, of course, be quantitative differences depending on the dose, the type of radiation, and so on. But I think the qualitative picture will be much the same whether exposure is to a nuclear weapon or to an x-ray tube. The only data to come out of the ABCC so far, recently published by Bloom and his colleagues on the observations made some 19 years now after exposure, certainly show an increased frequency of stable cells and a small but suggestively increased number of cells with unstable abberations. However, we really can't compare Bloom's data with any of the other data because there are important differences in technique; for instance, he used a 72-hour culture time. But the findings certainly are not inconsistent with what I have been talking about.

A point that I should have made earlier on it is that in reporting these findings one can express them in two ways. The first is the classical method of relating the total number of aberrations of a given type to the total number of cells counted and analyzed and obtaining an aberration frequency per cell. Or one can express them, as we have done, in terms of the frequency of cells carrying either unstable or stable aberrations. Our particular method has been subjected to much unjustified criticism. It is perfectly valid, and in fact takes more account of the dynamic situation in vivo than does the classical method. One has to remember that in dealing with an animal like man and in dealing with lymphocytes, one is studying a

dynamic system of cells, and one is interested in the effects of radiation on the system as a whole. At a recent international symposium on human radiation cytogenetics at Edinburgh, it was decided that it would be desirable that investigators should, if possible, report their results both ways.

LIFTON: Should this radiation treatment go on if you can observe this aberration so quickly?

COURT BROWN: This is the point we have got to argue the toss about. What do these changes mean in terms of long-range harmful effects, if anything?

LIFTON: I see.

COURT BROWN: The next problem is the effect of chronic exposure, and this has been examined in a number of places—by Amos and his group in California, by Wald in Pittsburgh, by Visfeldt in Copenhagen, and by my unit in Edinburgh. The subjects chosen are usually workers in atomic energy establishments, or medical workers such as nurses or radiographers.

There is now no doubt that one can detect the effects of, say, an accumulated dose of about 15 rad and upward, and again our powers of detection will be improved by computer techniques. The situation is interesting because in chronic exposure we have to take account of the dose received per annum, the number of years over which a dose is accumulated, and, in scoring unstable aberrations, the half-life of the lymphocyte. What we have found is that nonexposed controls show about 0.5 percent of cells with unstable aberrations, but those with an accumulated dose of about 25 rad show between 2 and 3 percent of unstable cells. The problem of the life of the lymphocyte may be instanced by quoting the results of examining two physicists, each of whom up to shortly before the war had accumulated over 300 rad of exposure from penetrating radiation; yet neither of whom showed in 1965 more than a marginal increase in the percentage of unstable cells. The inference is that most of the cells with unstable aberrations will have disappeared during the years since the occupational exposure ceased.

The final point I wish to make is that, as far as human experience is concerned, more recent work is turning to the problem of the identification of clones of lymphocytes in irradiated persons, characterized by a structural rearrangement and the significance, if any, of these in considering long-term effects.

If we find, in counting and analyzing 100 cells, say three or more cells to have apparently the same structural aberration, then we have to consider whether these abnormalities were independently produced or whether the cells are the offspring of a common progenitor cell in which the abnormality was produced; that is, they are members of a clone. If the aberrations are complex, then we can be reasonably certain that we are dealing with members of a clone and not with independently produced aberrations. In our own work we are certainly finding evidence for these clones in some heavily exposed subjects. Having found this evidence we then have to consider whether, in the light of limited numbers of cells we can examine, unequivocal evidence for the existence of a clone implies that the cells of the clone may have some biological advantage; and, if they do, is this advantage disadvantageous to the host. All these problems remain to be evaluated in the future.

ABRAHAMSON: Is it possible that the "clonal" cells were being repressed earlier and then as the person ages the other populations of cells have to some extent been lost, giving the "clonal" cells an opportunity to now show up with some selective advantage?

COURT BROWN: Yes, this is possible.

MILLER: Why do you think radiation induces leukemia more than other forms of cancer?

COURT BROWN: I just don't know.

UPTON: You probably know of the work by Joneja and Stich (Reference 136), who found the appearance of clones of cells in the mouse thymus after irradiation. These were chromosomally abnormal cells, and there is evidence that these clones do in fact represent the emergence of morphologically transformed cells destined to become neoplastic.

COURT BROWN: Once you start knocking around with a genotype of these cells and once they are given some sort of biological advantage, perhaps bearing in mind the other suggestion, but they are able to multiply, then I think all kinds of things become possible.

WHITE: Is there any difference in the incidence of these chromosomal abnormalities in those individuals whose tumors have emerged now from the irradiated block of tissue?

COURT BROWN: I don't know.

WHITE: I was thinking, you see, if the emergence of the tumors means a change in the immunological competence of the host so that they can't fight the tumor cell, the lymphocyte may have something to do with this and if indeed there is a hint here, it would be very helpful.

COURT BROWN: This is an interesting point, and I think one should look at this.

BRILL: Have you looked at any patients who don't have this kind of splenic involvement?

COURT BROWN: I forgot the radium dial painters. It was done at a time before we truly appreciated the significance of standardizing on early culture, so that to some extent the work isn't as good as it might be. We found in a group of women formerly employed in the use of radioactive paint that the percentage of cells with chromosome aberrations was increased in blood cultures and this correlated with the body radium content. All these women had, on the average, stopped luminescing nearly 20 years before their study, and it is puzzling why so long after we continue to find abnormalities.

FREMONT-SMITH: You are assuming that these unstable cells are being formed throughout this whole period?

COURT BROWN: One wonders whether lymphocytes can accumulate breaks over a period of years which remain unrepaired, and these interact just before DNA synthesis occurs. This could be an explanation, for the lymphocytes would be circulating over this long period of time in a raised radiation field, although the intensity of dose in the field must be low.

BRUES: A cell that is not dividing will accumulate breaks, this is true.

UPTON: Can you get at this question by giving advanced lung cancer patients divided doses, 25 r today and 25 r a week from today?

COURT BROWN: No, you can't. Ethically it is a very difficult situation. It is one dose and that's that.

TAYLOR: Is there any hope of the technique developing to the point where you can actually follow an individual cell that has chromosomal abnormalities through the whole process?

COURT BROWN: Through a division process?

TAYLOR: Yes.

COURT BROWN: This has been done with plant material.

TAYLOR: Can you track a particular chromosome through the process or a particular pair?

COURT BROWN: No, I don't think you can track a particular chromosome through the process, but you can certainly look at the division of cells in plant material which are carrying dicentric chromosomes and you can track the behavior of the dicentric chromosomes. You can show the formation of the bridge.

TAYLOR: So at least there is some hope of seeing whether there is a tendency to propagate?

ABRAHAMSON: Dr. Morris has tracked one particular chromosome in the human cell. That is the sex chromosome which shows us the famous bar body and he has followed it through two or three cell cycles continuously.

TAYLOR: Then can't one imagine eventually settling questions like the extent to which the buildup of these abnormal cells is due to a propagation through division?

COURT BROWN: I'm sorry, I'm not quite certain of your problem.

TAYLOR: That's the question. Is there any possibility of seeing the clones actually build up in the individual cell, watching it under a microscope?

COURT BROWN: You can study the buildup of clones in vitro, and, in theory, if a clone is at an advantage in vivo, one ought to be able to study its buildup.

ABRAHAMSON: You must remember, Dr. Court Brown, that the normal nonchromosomally disturbed cell is also building up in clones as well.

COURT BROWN: Surely.

ABRAHAMSON: So you have got the two populations of cells or many populations simultaneously going through a number of divisions, and what Dr. Court Brown is recognizing, because he has a marker chromosome, is this one progenitor cell which is given rise by numbers of divisions to this abnormal line. That is what he means by the clone. But simultaneously the nonaberrant cells are also multiplying and cloning as well.

TAYLOR: But you wouldn't have to watch those; that is, if you had a technique for it, you would not.

ABRAHAMSON: If you could single that one out, yes?

TAYLOR: Single that one out. That is, you hunt one out until you find one of these abundant cells and watch that. Is that completely out of reason as far as the technique is concerned?

WHITE: Your chances of finding that one in any moment in time is about one in ten million.

TAYLOR: Once you find it, can you hold it and watch it? That's the question.

COURT BROWN: I don't think so. What we now have to do is to find out if a clone can be disadvantageous to the host organism, you see, and what they mean, if they do.

TAYLOR: So you want to watch their interaction with the rest of it.

COURT BROWN: Yes.

SCHULL: I wish to voice one small demurrer here, namely, that those chromosome breaks of which you speak represent some weighted function of radiation and chemically induced changes, and that at the present time we know so very little about the chemical mutagens to which individuals are normally exposed, much less to which they may be exposed if ill, it may be very difficult to relate chromosomal changes to low levels of exposure to ionizing radiation. At the higher levels of exposure the component ascribable to chemical mutagens is probably overwhelmed by the radiation component, but at low levels

of exposure this is debatable. Under the best of circumstances chemical mutagens might be randomly distributed with respect to radiation dose and would then pose no particular problem. But I wonder how likely this is.

COURT BROWN: This is a fair enough objection.

WHITE: Can you trace the antigen required, the stimulus required to make the small lymphocytes grow to the large lymphocytes and test this against the rate of increase, cell numbers, in some way?

COURT BROWN: I just am uncertain whether anyone has really tried this yet.

WHITE: When you start a search, you look for some marker, for some technique that is easier than this laborious thing that you described and the necessity to use machines. If there is another way, a simpler way to get this, it would be better.

COURT BROWN: Actually I have described a fairly laborious technique and we have always pursued the policy of analyzing all the cells we count in order to get the maximum amount of information we can out of every cell. But if you want to do radiation work on a greater scale, you can sacrifice some information and, for example, simply score dicentrics, as some do.

GENETIC STUDIES: *DROSOPHILA*, MOUSE, AND MAN

SCHULL: Seymour Abrahamson and I hope to summarize for you the present status of radiation genetics as it applies to man and as it applies to other experimental organisms, particularly to *Drosophila* and the mouse.

As a point of departure, I would like to go back to 1946, after the atomic devices were detonated, and give you some idea of what the radiation dogma, as revealed by *Drosophila*, was in that year. It was against this background of information, it will be recalled, that the studies in Hiroshima and Nagasaki, the largest yet on man, were formulated. At that time, we knew three things, or we thought we knew three things.

First, all of the evidence then available suggested that the yield in mutations following exposure was linearly related to dose. The evidence on which this belief rested stemmed largely from *Drosophila* and from doses extending roughly from 50 r at the lower limit to 10,000 or so at the upper limit (for an account of these data see Reference 157). For those of you who may not be aware, *Drosophila* is reasonably radioresistant. The LD^{50} is not precisely known, but it appears to lie between 40,000 and 45,000 r whole body. This is so far above the levels of irradiation at which experiments are generally conducted that no appreciable killing is associated with most radiation genetic experiments in the adult *Drosophila*.

The yield of mutations in these studies was measured in one of two ways. One way has been referred to as a specific locus mutation study, or alternatively, the specific locus method of measuring mutation rates. In this instance, one attempts to determine the frequency with which some specific phenotype, some specific appearance of the fly is induced as a function of ionizing radiation. The locus associated with the particular phenotype is assumed to be reasonably well localizable in a particular *Drosophila* chromosome. I should like to emphasize that the 1946 data were primarily concerned with what happens to the X chromosome of *Drosophila* upon

irradiation, that is, most of the studies involved X-linked genetic loci. The specific locus mutation rates which were obtained involved such loci as that responsible for white eye, for example. In any event, in this method one literally counts the number of mutations which occur at a specific genetic locus and relates this number to the amount of ionizing radiation which the flies have received.

The data available in 1946—and these figures still hold—suggested that the average specific locus mutation rate was about 1- to 2 $\times 10^{-8}$ mutations per locus per generation per roentgen. That is, the probability of inducing a mutation at a particular genetic locus upon application of 1 r of irradiation to an adult, male Drosophila is about 1- to 2 $\times 10^{-8}$.

ABRAHAMSON: What is the natural rate then, Jack?

SCHULL: We are going to talk about that a little later.

The other type of study involves measuring the frequency of some event presumably directly relatable to the genetic constitution of the animal but not to a specific locus. These studies generally are directed toward the estimation of the frequency of induction of sex-linked lethals. The frequency which one measures represents a summation of events at all of those genetic loci associated with the X chromosome where a genetic change can be induced which is incompatible with the fly's survival. One does not know, of course, at how many loci these events may be occurring. The upper limit is presumably the number of loci associated with the X chromosome, and the lower limit is obviously 1. The true number lies someplace between 1 and possibly 2,000 or so, where the latter number is one estimate of the number of X-linked loci in Drosophila. There is obviously not the same degree of genetic specificity in this latter approach—I shall term it the "population characteristics" method—that one finds in specific locus mutation studies.

Taken in the aggregate, studies of the rate of induction of sex-linked recessive lethals suggested that the rate increased about 3 percent with each additional 1,000 r of exposure. I don't recall, now, the rate of occurrence of sex-linked recessive lethals at zero dose, but I am sure Seymour does.

ABRAHAMSON: 0.26 percent.

SCHULL: Clearly this is an appreciable increase, but let us return to the dogma.

The second element asserted that the yield in mutations was independent of the intensity of the radiation. Alternatively stated, one could fractionate dose in any way consistent with the survival and reproduction of the fly and the number of mutations which could be induced would be directly related to the total exposure of the fly and not the way in which the dose was fractionated. Much of the evidence on which this assertion was based stemmed from the work of Timofeef-Ressovsky, one of the earlier radiation geneticists.

The third element of the dogma as it existed in 1946 stated that the yield in mutations was inversely related to ionizing density. There is the cryptic assumption that the same amount of absorbed energy is involved in all cases. Thus, alpha particles, slow neutrons, and the like are less efficient inducers of mutation than gamma radiation for the same amount of energy delivered to the fly. All of these aspects of the dogma are, it should be borne in mind, directed toward the occurrence of gene or so-called point mutations.

Basically, the dogma as it existed in 1946 represented three "working" hypotheses; at least one of these, intensity independence, is now known to be less general than was initially assumed (Reference 158).

There was another important bit of information known in 1946; it rested not so much upon Drosophila as upon plants, and concerned chromosomal aberrations. It was known even then that the frequency of chromosomal defects increased with exposure to ionizing radiation but not linearly. Insofar as gross structural changes induced in Drosophila sperm by x-irradiation were concerned, the yield increased approximately as the 3/2 power of the dose (Reference 157). It was also known at that time that one of the kinds of mutational events at the chromosome level which radiation induced in Drosophila was nondisjunction (Reference 159). This is of interest, for presumably nondisjunction represents at least one of the mechanisms whereby Down's syndrome, for example, arises in man.

FREMONT-SMITH: What is nondisjunction?

SCHULL: A pair of chromosomes; instead of one going to each of the daughter cells in the course of division, both go to the same cell. In the germ line, this may result either in a gamete with one too many chromosomes or a gamete with one too few.

To return again to the dogma, it was against the background just described that the Atomic Bomb Casualty Commission studies were formulated.

FREMONT-SMITH: Is that dogma consistent within itself?

SCHULL: In a sense the dogma merely represented three observations, and the issue then and now was the generality of these observations. We assumed, for example, that linearity in yield held for doses of less than 50 r—we now know this to be the case for Drosophila—or that extrapolations to other beasts, including man, were meaningful.

AUXIER: Jack, is it fair to say that, on the basis of what we know now, items one and three would appear to be inconsistent?

SCHULL: No, primarily because there may be other phenomena such as cell selection which are confounded. You can "overkill" a cell so to speak, and I would presume that so many mutations could be induced in a cell that it could not survive. But obviously one can measure mutation rates only within those limits of dose and ionizing density which are compatible with some element of survival and reproduction.

FRANK: Would you mind explaining again the difference between the terms dose and ionizing density.

SCHULL: In this context, a substance with high ionizing density produces a great many ionizations in a relatively small amount of tissue, whereas a substance with low ionizing density which produces in toto the same number of ionizations in the exposed organism produces fewer in the same small amount of tissue.

AUXIER: In the context we were talking about earlier, this means a high LET or a high DE/DX; when he says density he is also saying a high number of kev per micron.

SCHULL: I should like to turn now to a consideration of the data which are available on man.

There have been published the results of some dozen or so studies which have attempted to measure some aspect of man's genetic radiosensitivity. These studies, interestingly enough, date from as early

as 1928. The very first published account was work by Douglas Murphy (Reference 160) in a study analogous to the one Bob Miller has reported. Murphy was interested in the effect of radium radiotherapy on the conceptions of a woman subsequent to the radiotherapy itself. His study involved only a few hundred children and there were no control observations. He attempted to use the United States vital statistics as a means of ascertaining whether the frequency of congenital defect and mortality was elevated among the children born to mothers who had received radiotherapy as contrasted with the general population of the United States. Similar studies were subsequently undertaken in Germany (Reference 160a) and possibly other countries as well. In addition to the two which I have already cited there are published accounts of studies in Canada, the United States, Brazil, East Germany, Czechoslovakia, Bulgaria, France, Holland, Italy, and, of course, Japan (for specific citations see Reference 161). The latter are not solely investigations of the Atomic Bomb Casualty Commission. There are other studies on the populations of Hiroshima and Nagasaki as well as efforts to determine the consequence of exposure to ionizing radiation on radiologists and radiological technicians.

The sizes of the groups studied vary enormously. They range from observations on 90 or so children to observations on upward of 140,000 children. Obviously one simply cannot give to all of the studies equal weight in any overall evaluation of the radiation hazard for this reason if for none other. Exposures also vary greatly. There is one study, for example, by Musil (Reference 162) in which the exposed group must have received no more than a few roentgens at most of x-irradiation. It was Musil's aim to determine whether an increased frequency of congenital defect could be demonstrated among the children born to women whose radiation exposure stemmed from repeated diagnostic x rays in the course of TB therapy. Other studies (for example, see Reference 163) involve doses alleged to be as high as 1,500 r or thereabouts. Clearly the gonadal dose must have been less, for exposures of this magnitude would undoubtedly sterilize a substantial number of individuals.

The nature of the exposed individuals ranges from persons with overt disease in which radiotherapy or diagnostic radiation is associated either with the treatment or the recognition of the disease to persons whose radiation exposure represents that which accumulates from living in an area of the world which happens to have a higher background of natural radiation than most other areas. The latter

persons are, needless to say, chronically exposed to very, very low levels of radiation. Doses are generally stated in terms of exposures accumulated in the first 30 years of life. This is a convenient yardstick and represents approximately the midpoint in the reproductive span of the female. The differences between the so-called exposed and nonexposed groups in studies of this variety are usually quite small. For example, in the study by Gentry, et al. (Reference 164), in upstate New York, the difference between "low" and "high" areas of background radiation was roughly a factor of 2. Individuals in the "high" area had accumulated exposures of about 6 or 7 r in 30 years, whereas those in the "low" area had accumulated exposures of 4 r in the same time. Perhaps I should state that this study contrasted the frequency of congenital defects, as determined by birth certificates, in two areas of dissimilar background radiation. Studies of a similar kind in Italy (Reference 165) involve larger differences in background radiation, if memory serves me correctly.

Possibly the best area for a study of this type is, as many of you know, the southwest and southern coasts of India where radioactive sands occur in which ambient radiation is of the order of 1.5 r/year, and exposures accumulated in 30 years may reach 45 r or so (see WHO Technical Report No. 166, 1959, Reference 166).

Perhaps this is the place to introduce, for the first time, some of the difficulties in working with human beings. The Chavara-Neendakara and Manavalakurichi areas of India to which I have been referring provide an excellent case study of the problems. The indigenous people are primarily fishermen who spend the bulk of their day at sea where background radiation must be extremely low. They come home in the evening to sleep with their spouses on the sands, I presume. The indigeneous men and women receive whatever exposures they obtain through this form of contact with the soil or through merely being present in a "field" of ambient radiation of this magnitude. Clearly, since the women spend more time in this "field" than the men, their exposures must be the greater. There is, however, a large transient population which comes in following the monsoons each year to help in the harvest of the radioactive sands which are constantly being replenished by the sea. These individuals remain in the area only a few months and then return to those portions of the surrounding region from which they originally came. Clearly, these events complicate the definition of a comparison population as well as the exposures of given individuals.

An added complication arises from the fact that some years ago, the Norwegians established a very excellent hospital in the region and this had led to a level of medical care much higher than exists in many of the surrounding areas. This, of course, results in a confounding of level of medical practice with level of exposure.

There is still another complication which relates to changing social practices in India. Until very recently, and in many areas still, consanguineous marriages, that is, marriages involving persons who are biologically related to each other, have been extremely common. In some areas 25 percent or more of marriages are between uncles and nieces, and this was true of this particular region. But seemingly overnight, the attitude toward these marriages has changed, and now most young people prefer to marry someone other than a relative. A genetic situation has been created, therefore, in which it will be extremely difficult to obtain accurate and meaningful estimates of induced mutation rates. For every population that I could cite there exist comparable problems. Fortunately, in a number of instances these complications are of a kind we believe we can handle.

To return to the main theme, the prospects of a successful specific locus mutation rate study in man still seem to be too small to warrant the undertaking. For such a study to be feasible one must be able to either construct a laboratory situation such as obtains in Drosophila or in the mouse, or one must have a large number of loci which can be marked in a population. At the time when the ABCC studies were being contemplated not more than 10 markers, in the broadest sense of the word, were known in man. At present, there are not more than 100. This still seems too few for reasons which we shall advance later.

All of the published human studies are, then, of the population characteristics kind, that is, they are all studies wherein the investigator seeks to learn whether the value of some particular but generally common parameter of the population has been increased or decreased by ionizing radiation. Let me enumerate some of the parameters which have been studied.

The one which has unquestionably received the greatest attention and has the greatest emotional impact on most populations is congenital defect. In the sense in which I shall be using these words, a congenital defect or malformation might be defined as any departure

from normality which is either incompatible with survival or seriously impinges upon an individual's ability to perform normally in the society of which he is a member. An anencephalic child would represent one extreme under this definition and a child with polydactylism might represent the other. Obviously the latter malformation is compatible with survival in most, if not all cultures, but it imposes a stigma nonetheless upon the individual which makes effective functioning less probable.

Another yardstick has been mortality, including fetal, infantile, and, in some instances, juvenile mortality. There are more studies of fetal and infantile mortality than of juvenile mortality, however.

Still another parameter is the sex ratio. Here we ask whether the proportion of male births has been altered as a function of the exposure of the parents. Exposed fathers and exposed mothers are treated separately for reasons which we shall describe a little later. Attention has also been directed to such continuous variables as birth weight and length of gestation. We have also been interested in variables to which I shall refer as measures of growth and development. These represent efforts usually in terms of a series of simple anthropometrics to ascertain whether the children of exposed parents are developing or have developed in the normal manner and at the normal rate.

Unfortunately, as I am sure you are all very much aware, each of these variables, including the sex ratio, is known to be influenced by a number of variables other than ionizing radiation. So obviously a study is ultimately no better than its ability to control extraneous sources of variation. This has been the rub and will undoubtedly continue to be the rub in most studies. It is difficult, indeed, to satisfy oneself that one has removed all important sources of extraneous variability which might either obscure a radiation effect or create a spurious one. At this point, perhaps I should state that we geneticists no longer operate on the supposition that it is necessary to demonstrate that ionizing radiation is mutagenic. We accept this as an article of faith. What we are interested in is the best possible estimate of the radiosensitivity of human genes. This is quite a different question. And for it we obviously have to be able to remove all those variables which might bias the estimate of interest to us.

I shall describe briefly now the various ABCC studies and attempt to indicate when each was initiated, how long it continued, and what

the number of individuals in the study was or is. I shall then try to summarize the status of our knowledge with respect to all existing published data, not restricting myself to the data from Japan.

The first study to be undertaken in Japan has gone under a variety of names, but in the historical record of ABCC it is usually referred to as the GE-3 Program (a complete description will be found in Reference 125). It came into being in February, 1948, and was terminated, for practical purposes in December, 1953. During this period, in both Hiroshima and Nagasaki, and for a period of three years in Kure, an effort was made to examine the product or products of every pregnancy registered in any one of the cities—registration was contingent upon the pregnancy persisting for a minimum of 20 weeks.

At that time there was a ration law in Japan which permitted a pregnant woman, upon registration of her pregnancy either by herself or an agent acting in her behalf, access to supplemental rations of value to herself and expected child. The economic circumstances in Japan during this period were sufficiently stringent that virtually all women took advantage of this opportunity. Our best estimate is that during 1948-53, we actually saw the products of about 93 percent of all pregnancies in Hiroshima and Nagasaki which met the constraints I have just enumerated.

At the time of registration, historical information relating to the present as well as past pregnancies was obtained. The study was prospective in the sense that the radiation data, etc., were gathered before the woman or the father of the child was aware of the nature of the termination of her pregnancy. The fact of the termination of a pregnancy reached us through the midwives of the cities. In those days, some 90 percent or more of births in Japan were attended by a midwife, and very few occurred in hospitals save in the very largest of cities. This is not the case now. Although the bulk of pregnancy terminations does not yet occur in hospitals, a very substantial proportion does.

When a midwife notified us of the termination of a particular pregnancy, a physician and a public health nurse visited the home to ascertain the child's status of health and to record the measurements previously mentioned. In the event of no notification, a house call was made a reasonable period of time after the expected date of confinement obtained at the time of registration. There was, as a part

of this general program, a second examination of the child sometime between eight and ten months postpartum. The purposes of this second examination were essentially threefold: It gave us a longer period of risk of death to measure among these children. It also afforded an opportunity to diagnose those congenital defects which could not be readily recognized in a newborn infant examined at home under circumstances which were admittedly less than the best. Finally, it provided an opportunity to gather growth and development data.

Between 1948 and 1953, approximately 75,000 children were examined. Of this number about half were seen in Hiroshima and about half in Nagasaki. To divide the number another way—and these proportions do not hold in the cities separately—about half had one or both parents exposed and the other half did not. This gives you some idea, I hope, of the sample which was available for analysis.

The next study to be initiated was the so-called sex ratio program (References 167, 168). This came into being in 1954 and still continues. This study does not involve clinical observations; it is tied directly to the vital statistical systems of the cities of Hiroshima and Nagasaki. It represents an effort to relate the sex of every child born in these two cities to the exposure of their parents. It was begun for reasons which stem both from the 1948–53 data and from theoretical arguments which suggested that the sex ratio might be an unusually sensitive yardstick of radiation damage. In the interval between 1954 and 1962 about 65,000 children were added to the total data as a consequence of this program.

The third study (Reference 107) is called the F-1 Mortality Study. Conceptually, this investigation is very similar to the Life Span Study involving the exposed individuals themselves. The kind of information which is obtained can be collected relatively inexpensively, yet obviously has considerable biologic interest.

The F-1 Mortality Study involves a cohort of 54,000 children. This cohort consists of three equally sized groups of children; 18,000 whose parents, one or both, were less than 2,000 meters from ground zero at the time of the bombing; a group of 18,000 children one or both of whose parents were exposed but at distances beyond 2,500 meters (there was a deliberate effort to omit the zone 2,000 to 2,500 meters), and a final group of 18,000 children, neither of whose parents was present either in Hiroshima or Nagasaki at the times of the bombings. All 54,000 children were born between 1946 and 1958. The

latter data was selected arbitrarily but only after it seemed unlikely that the cohort of children born to exposed individuals would be significantly increased by births subsequent to 1958.

I should probably point out that the 18,000 children in the "heavily" exposed group represent all of the children born to parents with such exposure in the designated years and known to us. The other groups, which are potentially much larger, have been matched by age and sex with the innermost group.

The mechanics of the study are again like those of the Life Span Study in that at periodic intervals an effort is to be made to examine the 54,000 koseki in which these children are inscribed to ascertain whether they are alive or dead and, if dead, to obtain a copy of the birth certificate. Only one koseki check has been made thus far and the results of it have recently been published (Reference 107). This check resulted in the enumeration of all deaths in the cohort prior to or in 1960.

There are a number of problems which these data pose at analysis. These arise principally from the genetically nonspecific nature of the end point which we are measuring, that is, mortality. We know, for example, that among the 18,000 children, neither of whose parents was exposed, the average ages of the parents at the conception of the child are less than in the other two groups. Moreover, the parents had married at younger ages; the number of children which they had had at a specified age was greater, on the average; and the frequency of consanguineous marriages was significantly higher. These three groups also differ in a number of other socioeconomic parameters. There are numerous explanations for these differences which make sense, but of course we can't be absolutely certain of the true one. The parents of these 18,000 children, like all of the other nonexposed individuals, moved into a biologic void, as it were. They came principally from areas surrounding Hiroshima and Nagasaki, but in the early days many of the Japanese repatriated from Manchuria and Southeast Asia settled in these cities.

It is difficult to take into account these factors, many of which may influence mortality. At the least, however, one is obliged to try to obtain some notion of the probable direction of the bias which these factors may introduce. We have attempted, therefore, to secure socioeconomic histories on the parents of a 10 percent sample of the children. On two separate occasions we have inquired, by mail, as

to the educational experiences of their parents, their occupations, and a number of other variables which we thought we could measure with some reliability. In general, we find that the distribution of these socioeconomic variables is such as to increase the likelihood of a child dying who is born to the "heavily" exposed group as contrasted with either of the other two groups.

Let me go back and try to summarize what has been found with respect to the various yardsticks of radiation damage we have enumerated. When one scans the entire array of information presently available and tries to integrate these studies in a meaningful way, one is obliged to conclude, I believe, that in the absence of experimental observations there is insufficient evidence to support a categorical assertion that ionizing radiation is mutagenic in man. However, as I would again like to emphasize, we are not really interested in a proof of radiation's mutagenicity in man because we accept the fact that human genes are mutable—the experimental evidence is, after all, overwhelming. To believe otherwise requires an assumption that man is dissimilar to virtually every other living thing, an assumption that I am certainly not prepared to make. We are left then with the task of trying to get some kind of an estimate as to radiosensitivity. Even then, when we attempt to relate these yardsticks to the probable dose of a given class of individuals and to interpret the data at face value, we frequently find that the observed proportions bobble in every direction. For example, in the F-1 Mortality Study, we found mortality to be highest among the group which received, at most, about 5 r of radiation.

BRILL: Jack, with the availability of dose estimates in the people under 2,000 meters, will it be of value to go over these data once again?

SCHULL: We hope so. When these data were analyzed, we did not have the newer exposure estimates available.

I hope that at this point I have communicated some of our sense of frustration. Extraneous variables seem to confront us in every direction and we haven't found an adequate means of being able to rid ourselves of their possible effects. We are, after all, looking for a very small effect to begin with. For example, if one takes what appears to represent the average exposure of these individuals and assumes that all mutations which were induced will be expressible in some conspicuous form, let's say as an increase in the frequency of

congenital defect, then one would expect the frequency of congenital defect to be augmented by not more than 20 percent of the background rate. So that, as we measure congenital anomalies, that is, as abnormalities, which are life-threatening or seriously handicapping and are recognizable clinically under the circumstances of our examinations, we are talking about the demonstration of a shift from 1 to 1.2 percent in the frequency of congenital defects. If we find something larger than that, we are at odds with all of the experimental evidence. It is for this latter reason that one has to look with considerable askance, in my opinion, on the findings of studies such as the Canadian one (Reference 169), which involves a very small number of children, the offspring of women who were born in the late 1910's and early 1920's who had congenital dislocation of the hip and the dislocated hip was reduced under x ray, where a significant increase in the frequency of congenital defect was found. The dose which the women in this study received—I hesitate to say exactly what it was, but it was not greater than the average in Hiroshima and more than likely much less—seems inadequate to produce a significant effect in the light of the Japanese data and prior theoretical considerations. This tends to be true for almost all of the studies where a significant effect has been found. The effect is invariably in excess of what would logically be expected if all of the experimental evidence is utilized and one assumes that man's genes are no more or no less mutable than those of the mouse, let us say.

Are there any questions before I turn the discussion over to Seymour?

JABLON: Randy asked about dose and I understand you didn't have that. Was it possible to break up that first group by distance?

SCHULL: Yes, the first group actually consists of some nine subsets which were analyzed as such. This analysis merely reinforced the remarks I have already made. In the light of some of the earlier discussion, it may be interesting to note that there was no evidence of an increased frequency of leukemia, as judged by causes of death, among the children born to men or women who had previously been exposed.

LIFTON: Are you going to comment on those early sex ratio findings?

SCHULL: I would like to forget them! But, perhaps a few remarks are in order.

SPEAR: Just one question before you go on to that. How are the distances in these cases established for the populations?

SCHULL: By interview.

SPEAR: Is there anything in the picture that would tend to distort the report?

SCHULL: Although John Auxier and Seymour Jablon may believe otherwise, it seems to me that the genetic data are the most interesting in this respect of any that we have. Remember that each time a pregnant woman presented herself at the ration office to register a new pregnancy another radiation history was obtained. Each history was collected without reference to previous ones. It was possible, since over the period of almost six years, from 1948 through 1953, many women registered two, three, and occasionally four times, to ask, for a very large sample of individuals, how a radiation history obtained at one instance in time contrasted with another taken at a different time but when, mind you, the event was not so far removed that the distortions which come with age and distance from an event could be great.

Several things occurred. As one might expect, histories taken on different occasions did not jibe in all of their particulars. Thus an individual who at one time denied exposure might report himself to be exposed at a different time, or, alternatively, an exposed individual might change his status to nonexposed at a second history. The frequencies with which we encountered changes of these kinds were quite low. Generally if an individual stated that he was exposed at the first history, this position was maintained on all subsequent histories. It was possible, because we also had obtained information as to distance from ground zero and the occurrence of a variety of symptoms associated with radiation sickness, for example, epilation, petechiae, and oropharyngeal lesions, to contrast the occurrence of these variables on different histories. We found with respect to distance that rarely did an individual change his position by more than 300 meters and when a change did occur the number of times individuals moved themselves closer to ground zero as contrasted with the numbers of times they moved themselves further away were about equal. It seems then that errors with regard to distance are reasonably random, and to a statistician this is satisfying.

There are circumstances which may make these data very different from histories that were obtained subsequently. First of all, in those

years the survivors of these two bombings had not as yet been singled out for a great many benefits which they now enjoy, such as free hospitalization, etc. There were very few factors operating then to induce an individual to assert that he was exposed when, in fact, he was not. There were no benefits involved. One could perhaps argue that at that time the survivors were uncertain of the Atomic Bomb Casualty Commission's relationship to the Occupation and therefore gave spurious answers. I do not believe this was so but this is clearly a personal reaction. It seemed to me at that time—and now, for that matter—that the vast majority of persons gave reliable radiation histories, that is, that they were cooperative and candid in describing their radiation experiences.

SPEAR: I was wondering particularly about the possibility of incentives to place one's self more at risk than was the case.

SCHULL: At that time there was no evidence that this was the case.

MILLER: How large an increase in congenital malformations would you have been likely to detect?

SCHULL: We could have detected a twofold increase in congenital defects. That is, if, in fact, the ionizing radiation experience of the parents of these children had increased the frequency of congenital defect by a factor of 2, our sample was sufficiently large that a change of this magnitude or greater would not have gone unrecognized.

I like to view our data as being completely consistent with what one would have expected a priori on the basis of experimental evidence. Thus, where an extremely small difference between exposure groups would have been predicted, "no difference" was found. This does not imply of course that no radiation damage occurred, but merely that the damage was too small to be detected by the means at our disposal.

FRANK: A geneticist in England* whom I talked to about this may have oversimplified the issue by saying that the dose sufficient to produce significant genetic defects in humans would be lethal to the mother anyway. So that the whole problem is an imaginary one. Is that correct?

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* L. S. Penrose, Galton Professor of Eugenics, University College, London, England. Personal communication.

SCHULL: I don't think any one of us would take that point of view.

FRANK: You pointed out that you would expect only a very small increase, so practically it would not matter.

SCHULL: Again it depends upon how one wishes to play the "numbers game." If you take a very small increase and multiply it by a sufficiently large number, the absolute increase in terms of congenitally defective individuals can be any number one wishes. It is important though to bear in mind that even when the risk to a given individual is trivial, in the aggregate, at the population level, that is, a substantial burden of disease and disability can arise.

JABLON: I am sure you didn't intend to say it, but I wonder whether there may not have been some flavor left from your remarks about histories—that perhaps the average error was near 300 yards.

SCHULL: No. The average error was not that great. I didn't intend to say that.

JABLON: No, you didn't. But I just wanted to get in the record that it was the case, was it not, that most people...

SCHULL: Give the same history.

JABLON: Yes.

SCHULL: Within the limits of our ability to measure their responses, of course.

AUXIER: Seymour and I have comments on this because, although the history is accurate and reliable, the histories taken by the shielding group for the dosimetry studies are done in a different way. That is, they didn't just ask the survivors some questions. They had a systematic approach to the problem such that they, figuratively, led the survivor down a path to the house in which they were exposed by means of a large area photo with the area of immediate interest blown up. The survivor had to identify his house first in words and then in terms of a drawing. The probability of uncertainty or of error in the final shielding is much less than I think you would get from just asking the person where he was. Don't you think so?

SCHULL: That is debatable, but I am not sure it is profitable to pursue the matter further now. I certainly agree, however, that the

maps, photos, and the like which are now available are much better than those of 10 or 15 years ago, but the person interviewed has memories, it must be recalled, which have been attenuated by 10 or 15 years. How does one weight the increasing precision of the first kind with the loss in the second?

JABLON: I have a little evidence on that. I don't have the numbers with me, but when the question arose we actually did go out and redo some hundreds of shielding histories years after the original histories had been taken to see how much a variation there would be. While there were a very few rather large moves, for the overwhelming majority of people their movements were not more than of the order of 20 meters or so.

SCHULL: Yes. I am sorry if I left that impression. What I meant to imply was that there were very few moves outside 300 meters in either direction. The vast majority of individuals placed themselves in the same radiation category in each successive history and rarely did a displacement of more than one radiation category in either direction occur.

To go back to the question of the sex ratio which was raised a moment ago. I am sure that most of you know the justification for measuring this variable, but let me run through the argument very quickly for those who may not.

Prior to some of the events about which Michael Court Brown spoke, it was believed that the sex ratio might be a particularly sensitive and specific yardstick of radiation-induced genetic damage. It was thought that changes in the sex ratio would be indicative of the induction of sex-linked lethal mutations, that is, mutations associated with the X chromosomes. And since one derives one's X chromosome in different ways, depending upon whether one is male or female, the sex ratio would be expected to vary in different ways as a function of the sex of the exposed parent. Mind you, this assertion was predicated on the assumption that the only kind of changes which would be reflected in the sex ratio would be changes in the genetic stuff associated with the X chromosome and incompatible with survival. Now, unfortunately, we recognize that there can occur chromosomal alterations which may not even be radiation-induced which can obscure the true sex of an individual. It is thus possible to assign erroneously to the male category an individual who is, in fact, a female and, of course, the opposite may also occur. So the issue has become fuzzier,

and I doubt now that sex ratio data can be interpreted in a quantitative way. Be this as it may, in earlier years and in the absence of the knowledge just cited, the argument used for expecting radiation-induced changes in the sex ratio was largely an extrapolation from Drosophila. It was on this basis that during 1948-53, we were concerned with maternal and paternal exposure and their relationship to the sex ratio.

At that time and on the basis of the theory then current, one would have expected the proportion of males born to women who were exposed to diminish as ionizing radiation increased and just the opposite to occur if the father was the only exposed individual. If both parents were exposed, we assumed, as a first approximation, some linear function of the two changes just cited would result. There was no reason to believe that if both parents were exposed the change in the sex ratio would be nonlinear. So the data were analyzed with respect to several questions: Was the sex ratio diminishing when the mother was the only exposed individual and was it diminishing as a function of degree of exposure? How about the father's exposure? And what happened when both parents were exposed? Unfortunately, no one of the regression coefficients which emerged and by which one could relate the proportion of male births to estimates of maternal and/or paternal exposure could be shown to be significantly different from zero. In the aggregate, however, the data seemed to support the argument which I have just stated, and the best argument that we could make at that time (Reference 167).

We elected to analyze our observations with radiation exposure viewed as a categorical rather than a continuous variable. Each birth was assigned to one and only one cell in a 4 × 4 array in which maternal exposure represented the alternatives along one axis and paternal exposure the alternatives along the other. Originally we recognized five categories of maternal or paternal exposure. But the numbers in the two more heavily exposed groups were so small that we pooled them. The five categories were as follows: Group 1 was unexposed individuals. Groups 2, 3, and 4 were individuals who were exposed at varying distances from ground zero but did not exhibit epilation, oropharyngeal lesions, or petechiae. Group 5 included only those persons who had one or more of the latter three stigmata of radiation sickness.

Obviously, with the appropriate statistical tools, one can partition the 16 cells to which we have referred in such a way as to obtain estimates of the rate of change in the sex ratio as a function of maternal

exposure when the mother alone was exposed; of paternal exposure when the father alone was exposed; of maternal exposure when the mother was exposed but so was the father; and of paternal exposure when the father was exposed but so was the mother. Each of these four estimates represents, in a sense, a test of the general argument previously stated. Thus, we could generate four tests of our hypothesis from every similar body of data, and two bodies of data existed. They arose as follows: When these studies were initiated we did not realize that consanguineous marriages are relatively common in Japan. There are theoretical reasons for entertaining the possibility that there may be differences in the manifestation rate of the same mutation as the individual in whom that mutation occurs becomes genetically more homozygous as a consequence of being the offspring of a consanguineous marriage. Since consanguineous marriages were not randomly distributed among the parental exposure groups, it seemed wise to analyze the data based on consanguineous marriages separately from the data on nonconsanguineous marriages. From these two sets of data collected in 1948 through 1953, eight tests of the argument could be obtained.

We looked at both the magnitude of the change in the sex ratio with parental exposure and its sign. Interestingly enough, in these early data, when both parents were exposed, without exception the sex ratio appeared to be diminishing with increasing maternal exposure and increasing with increasing paternal exposure. When only one parent was exposed, in all of the cases save one, the direction of the change was that which would be predicted on the basis of the genetic theory which prevailed at that time. This was sufficiently exciting that when the clinical program was terminated at the end of 1953, observations were continued on the sex ratio. In 1956, we analyzed the first two years of data collected under the new program which by then was known as the sex ratio program. This gave us a third set of data to contrast with the two which have been mentioned, and 12 different tests of the genetic hypothesis. This opened up the possibility of a significant result emerging from a nonparametric approach to the data. In fact, when the signs of the 12 estimates were considered, an effect significant at about the 3 percent level emerged (Reference 167). This was encouraging. Moreover, when the magnitude of the average of the estimates was examined, it appeared to contrast favorably with the data from Drosophila and the mouse. Thus, where specific locus mutation rates suggested the mouse to be one order of magnitude more radiosensitive than Drosophila, that is, approximately 2×10^{-7} mutations per locus per roentgen versus 1 to 2×10^{-8}, the

sex ratio data could be interpreted as evidence that man too was an order of magnitude more radiosensitive since the rate of increase in sex-linked lethal mutations in Drosophila was 3 percent/1,000 r and appeared to be about 2 percent/100 r in man.

On the basis of the seeming reasonableness of the findings, we elected to continue to collect data. This was our misfortune, for recently when the observations from 1956 through 1962 were analyzed, all that I have said was reversed (Reference 168). At the present time, by any standard that one wishes to play the statistical game, we cannot demonstrate a significant association between either maternal or paternal exposure and the sex ratio.

There is an out, that is, a means to rationalize this apparent reversal of an effect. There are data from the mouse which have been interpreted as indicating that the recoverability of mutational events is directly proportional to the period of time which intervenes between exposure and conception (Reference 75). If a rather long period of time intervenes, presumably because of cell-selective mechanisms, a lower estimate of the rate of mutation will be found than would otherwise be the case. It may be argued, therefore, that our recent findings merely substantiate what is already known in the mouse, for clearly the several sets of data are not in pari materia with respect to the interval between exposure and conception. Whether this is the basis for the disparity in the data is admittedly moot.

ABRAHAMSON: Isn't there one other problem that you are faced with in the human situation? The inactive X hypothesis would suggest that if a female received a sex-linked recessive lethal from her father, she might be killed by it, whereas this would not happen in Drosophila.

SCHULL: All this would do is diminish the recovery rate if inactivation is actually random.

ABRAHAMSON: That's right. Fifty percent of the cells would then be turned off and theoretically any given cell would have that lethal turned off half the time.

COURT BROWN: What is being done about looking at the products of conception by survivors? This is going to be important, certainly from the standpoint of looking for an increased frequency of structural heterozygotes.

SCHULL: Yes.

UPTON: Could you expand that, please, Dr. Court Brown? I am sure that many in the room don't understand the significance. Why should it be important?

COURT BROWN: I think one might expect an increased frequency of individuals with structural rearrangements in the F-1 generation. By now, these will be marrying and in theory one would expect that some of them at any rate will get into difficulty in conceiving children. One would also be interested in looking at the conceptional histories of F-1's who marry.

SCHULL: That's right. Thus far there hasn't been as serious a consideration of the issue as is possible, for most of the F-1's are as yet too young to marry. The average age at first marriage for females in urban areas of Japan is somewhat greater than 24 years, and relatively few marriages occur in the cities before the age of 20. Thus several years have yet to elapse before significant numbers of the F-1 will be marrying.

COURT BROWN: I take it that the authorities are considering the possibility of doing something about it.

SCHULL: Yes.

BUSTAD: Are they recommending against consanguineous marriages in the first group?

SCHULL: To my knowledge, no.

SPEAR: Is it a fact or an old wives' tale that following wars there tends to be a corrective imbalance in the sex ratio?

SCHULL: The sex ratio changes as a function of a great many things. It is known to change with maternal age; the proportion of male births diminishes as women become older. It has a very complex relationship with paternal age, and there are, of course, data of the kind which you have just cited, which, if interpreted at face value, imply that the sex ratio has changed following wars. The published accounts of data of the latter kind do not, however, take into account the fact that the whole age-reproduction structure of a population is apt to be markedly altered both during and after a war. It

is quite probable that what is being ascribed to war is actually a reflection of a change in the age-specific birth rates occasioned by the war.

BRILL: Jack, on the sex ratio question, I am not sure I understand the current thinking. The last time I saw the data I thought your feeling was that, in each of the comparison cells, changes were in the direction that was consistent with a radiation effect hypothesis but that in none was it statistically significant. However, I thought that since all the cells went in the "right" direction you were willing to accept this as sufficient evidence for a radiation effect.

SCHULL: That was true prior to the analysis of the data from 1956 to 1962. But if you take the aggregate now of 140,000 births, the pattern is no longer internally consistent, and still no one of the regression coefficients is significantly different from zero.

There is one final question which I should like to pose rhetorically, as it were, before I turn the discussion over to Seymour. Have we reached a sufficient level of technology that we might, in man, seriously consider mounting a specific locus mutation rate study? This means, of course, that we propose to examine single individuals and assert that a given person's phenotype could only have arisen as a consequence of a mutational event. Let me give you an illustration of the type of situation which is involved.

Suppose that we have a mother of blood group O who has given birth to a child of a phenotype to be specified in a moment. I select mothers on the basis that rarely are there challenges to maternity, particularly in situations such as in Japan, but there are a great many challenges to paternity. If such a mother gave rise to a child who was blood group AB, under usual circumstances a genetic impossibility, and if there was no challenge to maternity, we would be obliged to assume that either the A or the B gene, whichever was derived from the mother, arose as a consequence of mutation. If father-mother-child trios (or less efficiently mother-child pairs) could be tested in large enough numbers and for enough loci, we could conceivably obtain an average rate of induced mutations for the loci under test. The question which one must ask, of course, is can we test a sufficiently large number of persons at a sufficiently large number of loci to make an approach of this kind reasonably likely to yield unambigious results? It seems to me that with the strides which have been made in the automation of a variety of laboratory

tests it is not outside the realm of reason to imagine the processing of tens of thousands of specimens in a relatively short period of time. Some better insight into the answer to the question just posed can be obtained through the "numbers game." Let me try to illustrate what I mean.

Let us begin with the supposition that man's genes are no more mutable than those of the mouse, and accept that the probability of a mutation at a particular locus per roentgen per generation is of the order of 1×10^{-7}. Suppose only one locus could be tested, and let us assume that the population of interest to us consists of all those children one or both of whose parents were within 2,000 meters of ground zero at the time of the bombings. We have already stated that there are approximately [illegible]0 such children. Let us also assume that the average exposure of the parents of these children is 100 r; the true exposure is more than likely less. Granted these assumptions, the number of mutations we would expect would be the dose, 100 r, times the probability of inducing a mutation at the locus in question per roentgen of exposure, 1×10^{-7}, times the number of children studied, 18,000. This product would be the number of mutational events that one might optimistically expect to pick up. Obviously, I am presuming that we can remove all extraneous sources of variation. It doesn't take much of a mathematician to recognize that we would expect only 0.18 children to have a mutant phenotype. Clearly if one is considering a specific locus mutation rate study and has only one locus to test, there is virtually no hope of a meaningful estimate. But an undertaking of this kind ceases to be foolhardy if we can multiply 0.18 by some number n, the number of loci tested, and if n is sufficiently large. That is, if we can scan enough loci, then we are no longer searching for merely 0.18 children but a larger figure. Thus if we had 100 loci, we would be talking about 18 children and if we had 1,000 loci, then 180 children.

To continue, let us assume that we can precisely test each of the 1,000 loci and also assume that we have the technology which would permit us to process the blood, serum, or whatever else may be involved on a scale large enough that the thousands of tests which may be involved on the 18,000 children and their parents plus an adequate sized control are feasible. But have we reached that level of technology and how large, in fact, is the number of loci which could be tested either now or in the very near future? Specimens can, of course, be stored, and at the rate at which new inherited biochemical variants are being discovered, it is conceivable that in 5 or 10 years

we might be able to mark twice as many genetic loci as is now possible. If this was to prove true, it might be worth drawing blood on these 18,000 children and storing it. I am sure that Dr. Court Brown would be pleased to examine their chromosomes, and we would store everything else.

But can enough loci be marked? The most optimistic estimates of the number of loci which we could mark at the present time with the precision which would be demanded here is between 20 and 25. If one is prepared to relax the precision somewhat, the number might reach 50. Finally, if some not too rare pathological characteristics which seem to be simply inherited are included, the number might be 100.

COURT BROWN: Is this including all the biochemical stuff that is coming forward?

SCHULL: Yes.

AUXIER: Also, Jack, aren't a lot of them not applicable to any given mother? For example, they can't all have group O blood to start with.

SCHULL: Yes; that is, some mutations would occur which would not give rise to phenotypes conspicuously at variance with the phenotypes of the mother and father. Suppose we can mark the number of loci which I have mentioned and this number represents a "ball park" estimate at least; can we process large numbers of specimens for the 20 to 25 loci which can be marked at present? That is, do we actually have now the technical capacity or will we have it within a matter of a few years to automate the tests which would be involved? The answer it seems to me is a qualified yes. A number of laboratories have been experimenting with and developing the methodologies to chain together a number of automatic amino acid analyzers and thus permit a continuous flow of tests. Moreover, the results of these tests can be digitalized and the digitalized information punched into cards. It is possible, in fact, to proceed from the specimen of drawn blood to a punched card with little or no human intervention. There are strictures on the types of tests which can be automated; it is not possible at the present time, for example, to automate the detection of a protein where its recognition depends upon its electric charge. Herbert Schienberg at Yeshiva University, who has had considerable experience in these matters, and I have estimated that it would be possible with six automatic amino acid analyzers running full tilt to process about 6,000 bloods a year on the scale here envisaged.

My own reaction to the various questions which I have posed is to believe that an undertaking of this kind is still a bit premature, but we may be closer to the day of its realization than is immediately apparent. To be able to examine the human experience in this context would be marvelous, for we would no longer have to worry about socio-economic concomitants which muddy the water in our data at present. There is no evidence to suggest that any one of the markers which would be used in a specific locus study vary as a function of age of mother, age of father, etc.

ABRAHAMSON: Wouldn't it be cheaper, Jack, to try to do this in tissue culture systems?

SCHULL: But estimates of that kind confound whatever changes may occur in the cells as a result of culturing.

ABRAHAMSON: It is cheaper to try to culture them than it is to try to take all these individuals, I should think.

SCHULL: Are you proposing that we switch the whole business into an in vitro frame of reference?

ABRAHAMSON: At least you could get a whole lot more information a lot quicker and less expensively.

SCHULL: But it depends upon what sort of information one is after. If one is prepared to extrapolate directly from the in vitro to the in vivo experience, what you say is undoubtedly true. But as a population geneticist I find this approach unsatisfying because of its departure from "real life."

BRUES: Dr. Abrahamson is ready to continue the discussion of genetics and to conclude it except for such further discussion as we may have time for.

FREMONT-SMITH: In a highly genetically condensed form!

ABRAHAMSON: I think that Dr. Schull laid the basis for most of the material I want to discuss and has brought you up to the 1945 period in experimental genetics.

I want to mention that much of the work that I will have to review in about 20 minutes may be found in the recent Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (Reference 170).

I have been primarily restricted to a discussion of point mutations in Drosophila and the mouse. However, I do want to say that all of the types of chromosomal aberrations found in somatic cells that were discussed so beautifully yesterday by Dr. Court Brown also occur in the germ cells, the sperm and egg cells, and each of these aberrations has very defined and I think well worked-out genetic consequences in the offspring receiving these irradiated chromosomes.

As you recall, Dr. Schull pointed out that point mutation studies involve either the specific locus experiments or recessive lethal mutation experiments. Point mutation is, in fact, a catch-all term and it includes three different classes of genetic changes. The first, an intragenic change, in which some alteration in the genetic code has occurred, is an alteration that cannot be detected cytologically.

The second kind of genetic change involves deletion of the genetic material. It might be one gene or several genes deleted, but such deletions are not capable of being seen by present-day microscopic techniques.

The third type, called position effect mutations, are known in Drosophila and are believed to occur in other organisms, but the evidence is not as good. This type may occur when there is a structural rearrangement of the genes in the chromosome. Thus a change in location of genetic material can simulate a mutation. And this type of event increases faster than linearly with increasing doses of x-irradiation, while the other two classes of damage are found to increase linearly with increasing x-irradiation dose.

At the very high doses of irradiation, where large numbers of chromosome rearrangements are induced, a large number of class three mutations are included.

There are now known to be a considerable number of factors which will influence mutation rate, and I could probably spend all of my time on this particular subject without exhaustively covering it.

Let us look at the problem this way. First, we apply irradiation to the germ cells of a particular organism and then in some subsequent generation we measure the induced mutation rate in those treated germ cells. What then do we need to know in order to interpret the results? The list should be something like this: We need to know the source of mutagen used—x rays, gamma rays, neutrons, etc. —and

their physical characteristics; the manner in which the irradiation exposure was delivered, for example, acutely or chronically; the dose rate per unit time; whether it was delivered uninterruptedly or if in fractionated doses; what the time intervals between the various fractions were; and, of course, the total dose. With respect to the organism treated we need to know the sex, age, and genotype. For the sample of germ cells treated, we must know if the sample is a homogeneous one, for example, mature sperm, spermatid or spermatocyte, etc.; what stage in a mitotic or meiotic cycle the cells are in; and their metabolic conditions. We need to know the environmental conditions just prior to, during, and after treatment, and if the genetic damage being studied is subject to being repaired. Experimental manipulation of any of these factors has been shown to influence the final yield of mutations. Let me give just a few examples.

During irradiation a change in oxygen concentration from pure oxygen to anoxic conditions can lead to an eightfold reduction in mutation frequency for the same cells treated. Alteration of the oxygen concentration after treatment can also affect the yield of mutations, and, depending on whether female or male germ cells were treated, anoxia can increase or decrease, respectively, the frequency.

With the preceding as a minor introduction, let me present a little of the data available from Drosophila and the mouse on induced mutation frequencies. This will, I think, highlight some of the points I have just been speaking on. I have prepared a table (Table 8) which to some extent summarizes mutation rate studies in different cell stages of Drosophila. The table shows the total recessive lethal mutation frequency per genome per roentgen and also computes the doubling dose for these stages.

FRANK: Would you mind defining recessive in that context?

ABRAHAMSON: By recessive lethal, I mean a gene which will kill an individual when it is present in homozygous conditions; that is, when there are two doses of that mutant gene present, one contributed by each parent; or when it is present in hemizygous, that is, single dose condition, as in the case for males where there is only one X chromosome. In this case a recessive lethal on the X would kill a male receiving it, but would not kill a female receiving it who has also received one normal gene on the other X chromosome. When the recessive lethal is on the X chromosome as I just described, it is called a sex-linked recessive lethal. When it is on a nonsex chromosome or autosome, it is called an autosomal recessive lethal.

Table 8. Stage sensitivities, total recessive lethal mutations in Drosophila for different stages of gametogenesis.[a]

Reference	Germ cell stage	Chromosomes tested	Mutations per genome per r × 10^7	Doubling dose = control - per r frequency	
	Males				
171, Lefevre and Jonsson	Spermatozoan 2500 to 4000 r x rays	3,100	2,500	66 r	
172, Oster	Spermatids 250 to 1000 r x rays	10,800	3,650	45 r	
173, Abrahamson et al.	Spermatogonia 8550 r Cs^{137} (least sensitive cells)	3,400	310	530 r	
174, Oftedal	Spermatogonia 300 r Cs^{137} (most sensitive cells)	5,700	1,800[b]	90 r	
175, Crow and Temin	Control	618,000	164,000 per genome × 10^7		
	Females			♀ Control	♂ Control
176, Parker	Oocytes stage 14		1,200	160 r	140 r
	125 to 750 r x rays	6,700	2,300	80 r	71 r
176, Parker	Oocytes stage 7	18,000	530	360 r	310 r
	250 to 4000 r x rays		1,000	190 r	164 r
177, Himoe	Oocytes stage 7 3000 to 4000 r Cs^{137}	9,000	600	320 r	273 r
172, Oster	Oogonia x ray	12,000	300	630 r	545 r
	Co^{60}	27,000	330	575 r	500 r
178, Muller et al.	Control	35,000	190,000 per genome × 10^7		

NOTES:
[a] Computed whole genome mutation rates from the data of authors listed, as follows.
Total rate = X chromosome frequency times 6.3.
[b] Spermatogonial rate = X chromosome frequency times 12.

Most of the work has been done using sex-linked lethals because the techniques are simpler and take less time; however, a considerable amount of work has involved the autosomes. A number of workers have demonstrated that the rate of induction of recessive lethals is proportional to the length of the chromosome studies. In other words, if you have an autosome that is twice as long as the X chromosome, approximately twice as many lethals will be induced in it. I have used this relationship in order to calculate the whole genome recessive lethal mutation rate. In Drosophila the two major autosomes which are of equal length contribute 5.3 times as many lethals as does the X.

The one exception to this rule exists in spermatogonial cells due to a phenomenon known as germinal selection. It can be shown (Reference 171) that approximately 50 percent of the lethals occurring on the X chromosome (but not the autosomes) will not be received, because, through their metabolic action—or more likely inaction—they cause the death of the spermatogonial cells in which they occur. Therefore to obtain the total recessive lethal rate for spermatogonia, we multiply the X chromosome rate by 12.

I think one example here will enable you to see how the figures in the table were arrived at. The spontaneous, that is, control, frequency of sex-linked lethals is 0.0026; multiplying this by 6.3 gives the total genome rate of 0.01638. The rate per 10^7 is therefore 164,000 newly occurring recessive lethals.

Lefevre and Jonsson (Reference 172) have demonstrated that for fully mature sperm the induced sex-linked recessive lethal frequency is 4 percent/1,000 r or 0.00004/r. Multiplying this times 6.3 gives a total genome frequency of 0.000252, or approximately 2,500 newly induced recessive lethal mutations per roentgen in 10^7 spermatozoan cells.

It would therefore require that 66 r be applied to mature spermatozoan cells to produce as many mutations as occur spontaneously. Then 66 r would be the "doubling dose" for mature sperm. The values presented for the other germ cell stages were calculated in a similar manner.

The table illustrates some interesting facts. The most sensitive stage for the induction of mutations is the spermatid stage. The mature sperm and spermatogonia are less sensitive, respectively. Note, however, that there are two values given for spermatogonia that differ by a factor of 6. The studies by Oftedal (Reference 174),

which give the highest sensitivities, employed the lowest doses (50 to 500 r were used). Oftedal observed that the mutation rate dropped as the dose was increased above 300 r.

In my experiments (Reference 171) a much larger dose, 8,500 r was used and a far lower mutation rate was obtained. Oftedal concluded, and I think correctly so, that the spermatogonial population is a heterogeneous one, and that part of this population consists of cells which are both extremely mutable and are extremely sensitive to cell killing by irradiation. Similarly, in the population are cells of varying resistance to both the killing and mutating actions of the irradiation. If this interpretation is the correct one, then it suggests that the Oftedal experiments (References 174, 179, 180) sampled primarily the very sensitive cells, whereas my experiments (References 173, 181) sampled the much less sensitive cells because the others were destroyed by the high dose employed.

These last mentioned results, in addition to illustrating some of the factors influencing mutation rate, to my mind, bear on what I think may be an even more important problem. That is, the urgency to find out if a similar phenomenon is operating in mammalian systems at low doses. It is important to realize that the mammal may be at least 10 times as sensitive to irradiation as the fruit fly, and therefore if one can generalize the observations from fly to mammal, it could well mean that doses above 30 to 40 r may be wiping out the most sensitive and mutable cell population. One wonders whether it is not this kind of phenomenon which contributes to the discrepancy between observations on human populations when the investigations deal with diagnostic levels of irradiation as compared with considerably higher doses, as was seen for example in our earlier discussions on induced mongolism.

As shown in the table, essentially similar findings are observed in female germ cells with respect to stage sensitivity. Again there appears to be about an eightfold difference in mutability from the apparently least sensitive cell type, oogonia, to the most sensitive cell type, the stage 14 oocyte. Low dose studies on oogonia have yet to be carried out and therefore heterogeneity may well exist.

With respect to point mutation studies in the mammal, the data (Tables 9 and 10)* I will present are those of Dr. William Russell

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* Tables 9 and 10 are modified from Table XII of the U.N. Report Supplement No. 14 (A/6314).

Table 9. Natural and induced mutation rates at seven specific loci in adult mouse spermatogonia (Reference 183).

Source and reference	Exposure (r)	r/min	No. tested spermatogonia	Mutations per locus per gamete per r x 10^8	Doubling dose (r)
X ray (182)	300	80-90	65,548	29	29
X ray (182)	600	80-90	119,326	22	
X ray (182)	1,000	80-90	31,815	10.3	
X ray (182)	600	9	40,326	13.5	62
Cs^{137} (182)	600	0.8	28,059	8.5	
Cs^{137} (182)	300	0.009	58,457	8.0	105
Cs^{137} (182)	516	0.009	26,325	5.2	
Cs^{137} (182)	861	0.009	24,281	8.2	
Cs^{137} (182)	86	0.001	59,810	16.2	52
Cs^{137} (182)	300	0.001	49,569	14.3	
Cs^{137} (182)	600	0.001	31,652	9.8	
$Cobalt^{60}$ (183)	600	24	44,352	17.6	48
Fission neutrons (182)	59	0.79	17,041	171.0	5
Control (182)	—	—	544,897	840 per locus per 10^8 gametes	

and colleagues of Oak Ridge National Laboratory and most of the data have been recently reviewed in the U.N. report I mentioned earlier. (Space and time limitations prevented me from including all the data found in the U.N. report, but it should be stated that the English workers have corroborated Russell's results.) His studies involve the induction of mutations at seven specific gene loci in mice. The technique essentially is to mate a normal but irradiated animal to an animal which carries the recessive mutant genes at these specific loci. If the progeny show any of the seven visible mutant types, then the normal gene has been mutated. This is in a sense a much more restrictive experiment than the lethal experiments employed in Drosophila in which any gene on a chromosome which has mutated to a lethal can be detected.

Table 10. Natural and induced mutation rates at seven specific loci in adult mouse oocytes (Reference 183).

Source and reference	Exposure (r)	r/min	No. tested oocytes	Mutations per locus per gamete per r x 10^8	Doubling dose (r)
X ray (182)	400	90	12,853	45	3[a]
Cs^{137} (182)	400	0.8	36,083	13	11
Cs^{137} (182)	400	0.009	37,049	2	70
X ray (184)	50	81	127,391	22[b]	6
X ray (184)	50	81	54,621	0[c]	
Fission neutrons (185)	63	79	43,000	194[b]	1
Fission neutrons (185)	63	79	40,092	0[c]	
Fission neutrons (185)	63	0.17	46,301	108[b]	1
Fission neutrons (185)	63	0.17	80,391	0[c]	
Control	—	—	98,828	140 per locus per 10^8 gametes	

NOTES:
[a] Doubling dose computed using oocyte control; to compute using sperm control rates, multiply by 6.
[b] Sampled within first seven weeks after irradiation.
[c] Sampled more than seven weeks after irradiation.

I have arranged the data in the table in a way which will permit me to illustrate some of the important observations that have resulted from these studies.

For example, if you examine the first three entries in Table 9, you see that increasing the dose of x rays to spermatogonia in fact results in a significant decrease in the number of mutations induced per roentgen for the 1,000-r treatment. Again, this is interpreted as meaning that spermatogonial selection is taking place at doses above 600 r and the more mutable cells are lost.

The second kind of result to which I would direct your attention—certainly the most important from the standpoint of theory and implications—is that which is known as the dose rate effect. For the same total dose, a high dose rate yields more mutations than a low dose rate. Within the x-ray series, 9 r/minute produces about one-half as many mutations as 90 r/minute for equivalent doses. Since I have already calculated the mutations induced per roentgen, we can ignore the doses used and examine the table with respect to dose rate and mutations. All of the gamma irradiations, Cesium-137, delivered at dose rates below 1 r/minute produce a significantly lower mutation than do x rays delivered at 60 or more r/minute. This phenomenon is even more pronounced when one examines the dose rate effect in oocytes where the two extreme dose rates, 90 r/minute and 0.009 r/minute, result in over a 20-fold difference in mutation produced. Russell's interpretation for this phenomenon, which is the widely accepted one, is that repair of the genetic damage is possible at the intracellular level and that both higher doses and high dose rates interfere with the reparation processes.

If next we examine the question of relative biological effectiveness (RBE) of different types of radiation, by comparing the results of fission neutrons with x- or gamma radiation (and I have chosen only one of a number of experiments to illustrate this), we see that the low dose rate neutrons produce over five times as many mutations (171) as high dose rate x rays (22 to 29). Comparison of neutrons to low dose rate gamma shows about a 20-fold difference in the mutation producing capacity.

Let me try to summarize some of the more recent mutation studies on female germ cells, the oocytes (Table 10), because regardless of the mechanisms involved, the implication to human radiation genetics may be considerable.

First, the longer the interval (in weeks) between irradiation of the female and the time of conception, the lower the mutation rate. This is dramatically so when the irradiation source is neutrons, and preliminary studies with x rays appear to be yielding similar results.

Second, fewer mutations are recovered from irradiated oocytes when a 400-r dose is given in eight equal fractions separated by 75-minute intervals than when an uninterrupted 400-r dose is applied (the dose rate is 90 r/minute in both treatments), 20 mutations per locus per hundred million gametes per roentgen as compared with 50 mutations, etc., respectively (Reference 186).

COURT BROWN: How would you square off this with the classical work on mutation rate that showed nothing?

ABRAHAMSON: First, the classical work was primarily restricted to irradiation of mature sperm, an essentially metabolically inert cell type as compared with spermatogonia and oocytes, and the great majority of evidence is still consistent in that there is no dose rate or dose fractionation effect on this cell type regardless of the species.

With respect to dose rate effects in Drosophila on other germ cell stages, there is as yet no strong evidence for the phenomenon. Muller, et al. (Reference 178) have been unable to demonstrate clearly such an effect in oogonial studies, although they think it probably exists. Purdom (Reference 187) was unable to establish conclusively a dose rate effect for spermatogonia, and Himoe (Reference 177) has found no evidence for a dose rate effect in oocytes. All of these studies involved recessive lethals.

On the other hand, there have also been dose rate effects noted for spermatogonia and oogonia in the silkworm Bombyx mori by Tazima (Reference 188), and this is a complex story. Dose rate effects have been reported by Baldwin (Reference 189) for the wasp Dahlbominus. More work is required though before a clearer picture emerges.

Since you ask me to square it, let me point out some things that have disturbed me about the mouse work. Primarily, I am disturbed by the ascendancy of the repair concept for point mutational damage. With Drosophila and other organisms, we can simulate all the phenomenon found in mice—dose rate effects, dose fractionation effects, and aging effects—when we study the production of chromosome aberrations. So the overriding question with respect to mechanism is, are the mutations in the mouse system truly point mutations or are a substantial proportion of them small deletions, in other words, chromosome aberrations? I know that the dose-frequency relationship is linear, which would argue against a contribution from gross chromosomal aberrations such as translocations or inversions, but deletions may still increase linearly with increasing dose. With respect to the implications to humans, these questions I raise may have little significance but I am not yet sure of that.

Second, let me point out that in Drosophila, we and others have shown that x rays may have an RBE of 1.7 with respect to gamma rays. and I would like to see more studies carried out in mice at

DE BOER: Is there any explanation for the greater effectiveness of the x rays over cesium on energy?

ABRAHAMSON: I think it really relates to the LET phenomenon that was discussed by Dr. Brues.

BRUES: The x rays are a quarter of a million volts and these gamma rays are over a million.

SCHULL: Russell is on record asserting that radiation quality does not affect the yield appreciably in mice (see, for example, Reference 158).

ABRAHAMSON: Yes, I know. I think I would feel more comfortable if there were comparable x- and gamma-ray studies at, say, 90 or 9 r/minute.

BRUES: You mentioned the greater sensitivity of the mouse relative to the fly in terms of recessive lethal mutations.

ABRAHAMSON: In terms of almost any kind of mutation.

BRUES: Is this because there is, so to speak, more material in the genome in the case of the mouse or is it much more complicated than that?

ABRAHAMSON: I would think it is more complicated than that.

LIFTON: Could one ask a general question? Could either of you take the experimental data and the situation that one has observed so far in Hiroshima and in just a few sentences make a statement about the vulnerability of people in Hiroshima and Nagasaki to genetic impairment, an overall impression of how the situation stands?

SCHULL: I am not sure I understand exactly what you mean.

LIFTON: As I understand what you have said about it, you expect to find some sort of impairment. You have found none in control studies. Where does the matter stand now? What is your sense of these people in terms of their genetic situation? What could you say about what might be expected or not expected in the future regarding genetic impairments?

SCHULL: We could estimate the ultimate impact of their irradiation experience on the genetic biology of these two populations, but our estimates probably would not be particularly accurate, for we know so very little about the factors which govern the persistence and spread of mutant genes in a human population. The latter knowledge is essential, however, to any meaningful evaluation of the radiation-induced genetic burdens of the inhabitants of these two cities.

The only statement which one can make with certainty is that the increased mutational burden will ultimately manifest itself in some form. But the rate at which these new mutations will appear is not clear.

BRUES: I think what may be troubling some nongeneticists here—and I am trying to bridge the disciplinary gap—is actually that from what we have seen in the way of numbers, a good many persons in the Japanese cities have received exposures corresponding to a doubling dose so that they carry a double load of mutations and have passed them on, and yet this has been insufficient to produce any effects striking enough to be quantitated by studies of the offspring.

LIFTON: And, of course, is probably troubling the geneticists, too.

ABRAHAMSON: In experimental genetics our breeding procedures permit us to uncover the induced recessive mutations. You don't normally do this in a human population; neither the same testing procedures nor precision of testing is available in the human situation. All of the mutations that are observed in the human situation have to have some degree of dominance or be sex-linked in order to be detected in the next generation after irradiation.

LIFTON: Am I correct in saying that the only expectation, the only basis for expected genetic impairment in Hiroshima and Nagasaki, is experimental evidence?

SCHULL: Yes.

LIFTON: So far. But applying that experimental evidence to subsequent generations, does one put a certain end point at how long this process might manifest itself, or is it limitless in terms of generations?

SCHULL: It is limitless.

LIFTON: One could encounter clinical impairments in these subsequent generations.

SCHULL: Yes.

WHITE: You would have to assume that man is like a mouse or a Drosophila to go ahead with what you want to do, and I don't think this is valid.

SCHULL: There is evidence from Drosophila which is pertinent and which suggests that the largest single increment of mutational damage to be recovered will appear in the first generation following irradiation. But this increment will only be perhaps 6 percent of the total mutations induced; the remaining 94 percent will "leak out" over infinity minus one generations.

LIFTON: I see.

SCHULL: A few moments ago, Seymour presented a number of estimates of the doubling dose for various cell stages in Drosophila and the mouse. These figures lead one to ask what do we know about the doubling dose in man. Unfortunately we do not know very much, and there are sizable differences of opinion with respect to the importance of some of the observations which are available to us. But the consensus seems to be that the doubling dose in man probably resides between 30 r as the lower limit and 100 r as the upper.

ABRAHAMSON: May I just add one other point. Doing a completely different set of calculations, which is the one in the U.N. report, from the one that Jack just quoted, the double dose was calculated at being around 70 r. So it fits again in that range from different sets of data very neatly.

MILLER: What would be your advice to a prospective mother who survived the bomb in Hiroshima with respect to the health of her offspring?

SCHULL: I would say that she accepts without question risks that are far higher than the risks that will accrue to her children as a consequence of her exposure.

MILLER: What risks that are far higher?

SCHULL: The drugs which she is undoubtedly taking during her pregnancy are quite probably mutagenic.

WARREN: Measles.

SCHULL: Measles. The caffeine which she consumes in the tea that she drinks may involve a risk greater than the radiation risk of the infant, etc. I do not believe that the hazard which accrues to the offspring of an individual as a direct consequence of his or her exposure is sufficiently large to be viewed as a legitimate, medical basis for intervening in that pregnancy.

BRUES: I think we are making a good transition, but we shall have to turn quickly to the next topic.

BRILL: From the rates of induction of mutations at the loci you have been talking about and the tremendous populations or exposures that would be needed to determine significant effects, it seems that the only positive human genetic information one can hope to quantitate in the Japanese survivors is in the cytogenetic area. I would not think that there is much likelihood of determining these effects short of a broad-based cytogenetic study. Would you agree with this, Jack, based upon your findings to date? We understand, of course, that these techniques were not available when the study was designed and conducted.

SCHULL: Retrospectively, perhaps the best genetic yardstick is a cytogenetic one, but problems will still exist. For example, it is virtually impossible at present to say what the impact of X chromosome breaks upon a population will be. We know nothing at all about the relationship between such breaks and the reproductive fitness of an individual. In the final analysis, however, it is the frequency with which such changes would be transmitted from one generation to the next which concerns us.

BRUES: We may return to this subject, but, if so, we should do it in the context of a discussion on the psychosocial matters.

SESSION IV

PSYCHOSOCIAL EFFECTS

Chairman: Austin M. Brues
Initiator: Robert J. Lifton

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INTRODUCTION

LIFTON:* In getting to the psychosocial side of the effects of the atomic bomb in Hiroshima, one hopes, so to speak, to examine the men and women behind the blood cells that we have been discussing or maybe we should say the man behind the sperm! [Laughter]. But in any case, I would emphasize the importance of human dimension, which is hard to evaluate with the same precision as other dimensions and can be too readily overlooked. I would say that the true scientific endeavor really has to be a continuing dialog between what might be purely technical information, that is, the generalization of the effects of one aspect of the human experience, and the overall psychological experience or the psychosocial and human experience itself, which is really what I hope to introduce into the Conference today, and subsequently.

I would stress the element of dialog right here, because my sense of the value of this kind of conference is rapid-fire exchange along the lines that Frank Fremont-Smith introduced at the beginning. In addition to the statements from Scott Matsumoto on the city at large from his experience in Hiroshima, and perhaps comparing the two cities (as I would infer from the brochures he passed around), and from Jerry Frank, as he sees fit, on the basis of his general thinking on these issues, I would hope that all of you will intervene very readily. I really don't care if I don't cover too much of this material in a systematic way, because I have no hope whatsoever of covering it all. Therefore, do interrupt and offer your comments, because there are so many of you who have lived in Hiroshima or

*The findings that Dr. Lifton presents here are elaborated in his book Death in Life: Survivors of Hiroshima, Random House, New York, 1968, and in two earlier papers "On Death and Death Symbolism: The Hiroshima Disaster", Psychiatry, vol. 27, 3: 191-210, August, 1964; and "Psychological Effects of the Atomic Bomb in Hiroshima: The Theme of Death", Daedalus, 92: 462-497, 1963.

have been in Hiroshima for considerable periods of time and have had various experiences of one kind or another with these human dimensions, and I think we should hear about them and share them.

Now, this will be a thematic statement. I won't be comparing two populations. It is not a comparative study. Rather it is a statement of the major psychological and psychosocial, or, as we call them these days, psychohistorical themes that one can glean from a study of a population in Hiroshima.

The method of study involves something that I have been working on for over 10, really almost 15, years: an effort to apply an interview technique in studying both depth-psychological and larger historical dimensions of experience—whether various struggles with populations that I have been interviewing in Hong Kong with Chinese and Westerners, or in Japan with Japanese youth. Of course, it involves aspects of death and death symbolism which are not very well dealt with, not very thoroughly considered, really, in the psychiatric and psychological literature. There are reasons for neglect of these which I don't want to go into at any length here, but it certainly has to do as much with resistance of investigators as with any other factor.

SCHULL: Will you give us a definition of what you mean by psychosocial?

LIFTON: It is simply a very general dimension that includes the interaction of the individual with the various groups that he comes into contact or is influenced by. It is a very loose term that really describes a dimension rather than an exact approach.

PSYCHOLOGICAL RESPONSE IN HIROSHIMA

LIFTON: The work that I will be describing was done over a six-month period, mostly from April to September of 1962, the majority of which time I was living in Hiroshima. Actually, I will be very personal about this presentation, because in this sort of work, the personal element is very much a part of one's data, as I will try to indicate from the very beginning.

For the previous two years, I had been doing a study of Japanese youth in Tokyo and Kyoto. I had worked and lived in Japan on and off since 1952. Toward the end of that study, I visited Hiroshima, just looking at the issue of the atomic bomb without any original intention of doing a study. But I found, much to my surprise, that no intensive psychological study had been undertaken in Hiroshima. This to me was extraordinary, because here was one of the great and disturbing events in recent human history, and it hadn't really been approached from a psychological dimension. There are many reasons why it is difficult to approach from this dimension. Those of you who have worked on physical aftereffects are, I think, very well aware of the many barriers and complexities that present themselves. But, nonetheless, it did seem rather surprising and, in a way, astounding to me. I was able to arrange to stay in Japan for an additional six months to undertake the study.

Actually, I think one often finds, as one becomes interested in this psychosocial or psychohistorical dimension, that the larger and perhaps the more profound and the more important the human problem, the more it is neglected. I may be exaggerating, but only slightly. It is always easier for us in psychology and psychiatry, as well as in other disciplines, to focus upon more technically manageable dimensions and to neglect or in some way not confront these larger ones.

FREMONT-SMITH: Is this partly because the larger one involves ourselves?

LIFTON: I think they all involve ourselves but the larger ones are harder to get at intellectually scientifically, as well as involving ourselves.

Concerning the way I went about this study, I will just sketch it briefly and then go into the data, but I want you to have a sense of what I did. I used the modified interview method that I have mentioned. I decided that I wanted to have at least a part of my group selected at random from the list of about 95,000 survivors kept by the City of Hiroshima, and another group who were especially selected because of their intense concern about the bomb. The first group was simply taken from the list, and the other group consisted of people who were in some way articulate about the atomic bomb: physicians who had themselves been survivors at the time, religious leaders, university professors, city officials, writers, poets, artists, and leaders of survivor organizations. Actually, between the two groups, as I got into the work, I found very little difference in their basic psychological responses, but the approach was extremely useful in terms of the level of articulateness and the ways in which they could complement each other

I was interviewing a group of people who were called hibakusha in Hiroshima. Those of you who have been there know that the word simply means "explosion-affected person" (or people). It is a coined word. In a way it takes on importance of a psychological kind, as we will see. The term implies in its tone a little bit more than simply having been in the bomb area and a little bit less than having been physically affected by it. I think that is a fair way to put it. It doesn't actually say that one has in any way been injured by it; it means literally an explosion-affected person. There is a specific set of categories which make one eligible to be considered a hibakusha in a medico-legal sense, and this takes on great importance when one considers the vast array of medico-legal regulations that have been built around them.

Those, of course, who were in the City of Hiroshima at the time of the bomb are hibakusha. That does not take into account any precise set of measurements from the hypocenter, because the city extends roughly in its boundaries, or did then, 4,000 meters from the hypocenter, and in some places up to 5,000 meters. But if one was in the city as it then was defined, one was a hibakusha. Those who came into the city within 14 days and entered an area within 2,000 meters from the hypocenter are considered hibakusha. Those

who handled bomb victims at the time in any way, anywhere, either dead or injured, and those who were in utero and whose mothers fit into any of these other three categories were all technically hibakusha.

The people that I interviewed varied in where they had been, from extremely close to the hypocenter, within 1,000 meters in a few cases, to those who had been 4,000 meters and more away. The psychological responses cannot be coordinated with any precision in terms of distance. We have seen that even with physical after-effects, the correlation is by no means exact, but much less so in the case of psychological effects, because these depend very much on the kind of exposure one had. One could have been at one place at the beginning and then walked and seen a great deal. Also important was the kind of person one was before, personality traits that one had in the past, which I could not go into in too great detail. So what I will be talking about will be general themes that were true in greater or lesser degree and, with much variation, for all of those whom I interviewed.

I tried to see all of these research subjects for at least 4 hours of interviews. That would involve two long sessions. I always had a research assistant with me to interpret. I speak some Japanese and could understand much of what went on in the interviews without the research assistant, but not enough to conduct a free psychiatric inquiry.

ANGEVINE: You made a random selection. I would like to know how the cases were selected. I believe that when a random selection of animals or people is made, there is a proper way of randomizing them.

LIFTON: They weren't randomized in any way; just simply taken off the list. Actually, I simply took every 500th name.

I should say something about the way I approached these people. I felt that it was a very complex situation for an American psychiatrist to come into the city and ask people how they felt about the atom bomb. It is rather Kafkaesque in a way. So I trod lightly and always tried to be intoduced by other people whom I knew in Hiroshima, whom I met or was introduced to in turn through various work I had done in Japan, many of them in academic circles.

I went to the homes of the people in the random group with a social worker from Hiroshima University, and would present my name card along with the card of the social worker with me. I would explain to them very briefly and very simply what I wanted to do, why I wanted to talk to them, what my purposes were in conducting the study. My own sense of purpose, I felt, was important, as conveyed to them, in obtaining their cooperation.

FREMONT-SMITH: What would you say to them?

LIFTON: I would say, "There has been a great deal of discussion—everybody is concerned about the effects of the atomic bomb—but very little had been done in the way of an investigation of psychological or human effects. I would like to make a systematic study, so that these things can be better understood, in the hope that this will contribute to some sort of control of these weapons and in a small way to the prevention of their use."

FRANK: Do you think that this tended to make them exaggerate the bad effects?

LIFTON: Actually, people varied in their responses. Some might have tended to exaggerate; others tried to demonstrate their stoicism and to minimize all effects. Very quickly in the interview situation, I think, one gets across to them the sense that one wants to hear what they really were feeling all the way through and doesn't want any special story, so to speak, any special message from them other than that.

FRANK: That certainly is what you want, but are you sure that they accepted that definition of the situation.

LIFTON: I think so. On the other hand, I also had another way of checking—through earlier, written accounts of various kinds not psychologically evoked. And the descriptions 17 years later of what people went through and what they saw were extraordinarily similar to, say, what Dr. Hachiya wrote in his book (Reference 61) and what others wrote in their early descriptions.

BUSTAD: Did any of them fail to cooperate?

LIFTON: There was just one person who refused to meet me and there were two or three others out of 75 or 76 that I originally approached who didn't show up for a second interview.

KNORR: It is not a question of failure. It was the degree of cooperation that you can observe.

LIFTON: The degree of cooperation, the way they responded, was striking. There was an odd intensity about being approached by an American in this kind of situation, and many of them expressed or obviously experienced various complicated feelings.

KNORR: But could this have falsified in a sense the testimony that they gave?

LIFTON: This is always possible. One has to use one's own clinical judgment in evaluating what kinds of data one gets, and, of course, there could in some cases be inconsistencies in what people said which could be readily seen. One could sense certain exaggerations. But, frankly, there was more of a tendency, when they got down to actual experiences, to minimize or deny than there was to exaggerate. This has to do with various psychic defenses employed.

KNORR: What worries me about this is what might be called the postevent manipulation of the experience, which, of course, could arise from a great many different motivations, sources, etc., and might differ greatly for different groups of people—intellectuals against nonintellectuals, etc.

LIFTON: Let me put it this way. I think that anybody whom you interview after an event is expressing his sense of that event from his standpoint of where he is at the moment of the interview. In other words, he is never exactly back in that situation. Here is a man now, 17 years later, whether a manual laborer, a university professor, or whatever. A lot of things go into the interview and, of course, one tries to understand what they are. But as I led them rather gently back into their experience, the degree to which they did relive it was striking. And this tendency had been described by other people who have worked with survivors of a catastrophic experience undergone many years before. They can project back into that experience and convey a good deal of the emotional aura of that past time. But I would agree that I was really getting a recapitulation of an experience from the standpoint of now, the interview situation.

KNORR: In the sense from a different person really. I mean new sediments of life experience.

LIFTON: Not exactly the same person who was there at the time, yes.

MATSUMOTO: Each year around August, Hiroshima is flooded with all kinds of reporters from various parts of the world who come to write news articles on some aspect of the survivors. Often I have been asked to get together a group of survivors for the reporter to interview. A noticeable factor has been that the tone of the interview seems strongly influenced by the sentiment of the particular reporter, who usually has a preconceived idea of his writeup. The survivors will fit themselves into the interview situation. They could be very somber or gloomy if the mood of the reporter seems to so indicate, or they can be more cheerful—looking-toward-the-future sort of discussions—where they do not go too deeply into their atomic bomb experiences. I was surprised to find often that the same person, according to the type of interviewer, would present a dissimilar picture of his experiences.

LIFTON: I think that is true, and it is consistent with a vast body of psychiatric experience in investigating a large number of phenomena. The setting that you create—the approach, spoken and unspoken, of the investigator—has an extraordinary influence upon the findings of the investigation. My sense of what I was trying to get was a balanced picture of what they had been through. And one of the things that I did to try to evoke this was to talk to them for a considerable period of time before I asked them anything about the atomic bomb experience, per se. That is, about what they were doing now about the rhythm of their lives, their experience over the last few years. And only then would I gradually work my way back to a recreation of the atomic bomb experience. I used a fairly consistent approach with these 75 people, encouraged the widest gamut of responses, and indeed encountered in them highly varied and very divergent qualities of feelings.

What I also did was tape-record almost all of these interviews, especially with the randomly selected group, so that I had a record on tape and, eventually in Romanized Japanese and Engli.. transcription, of the exact interview itself, which could be w.. d from directly.

JOHNSON: While we are speaking about the conditions of the study, would you say something about what you think the limitations in communication were. Did you feel that you were getting through nuances of the language with your own experience or with an interpreter? Was he translating for you into terms which were comprehensible to you? In other words, was his background thrust upon the thoughts?

LIFTON: I worked with two assistants, one of whom is a young woman with whom I had worked for two years in Tokyo and Kyoto, and another was a man who was from the Hiroshima area. Both were completely bilingual. The limitations of using any such arrangement with an interpreter are considerable in communication. But for some time, beginning with work in Hong Kong and then in Japan, I have been trying to evolve ways of transcending some of these limitations through spontaneity within a three-way communication system. This means that the assistant must be trained to feed back information quickly and to stay within the vernacular and the idiom, not to give any kind of overly "proper" foreign-style translation. The whole interview situation had to move fast so that at times I would simply wave if I understood things quite well. Of course, I might miss some nuance, although I felt I did have a fairly sensitive idea of what was going on as we went along. Some of the things that I did miss I could have a second chance of recapturing from the kind of record that I had. And I went over certain portions of the record with various people and parts of the tapes with a Japanese psychiatrist colleague.

FREMONT-SMITH: How did you structure the difference between the first interview and the second with the same individual? Or did you have any system in this respect?

LIFTON: It depended upon what we had covered in the first interview. In other words, the first interview was likely to be sort of a general exploration of their lives, and then a beginning exploration of the atomic bomb experience, in fact, a fairly thoroughgoing one of the immediate experience and the overall response to it. The second interview was likely to be more concerned with their long-range sense of themselves as hibakusha, as part of a very particular community, part of a group identity.

I also questioned them about specific things: about problems of memorialization of August 6th; about an issue that was then a very important one psychologically, a very significant one, the issue of the

Dome, whether the so-called A-bomb Dome should be retained in Hiroshima; about the question of Japanese rearmament; and about the issue of nuclear warfare in general, which would in some ways be some sort of bridge between their individual experience and their later sense of themselves as a special group in the world area.

UPTON: This term hibakusha interests me very much. I haven't heard it before. How did this group of people arise and come to be known this way? How did this word arise, and what various types of individuals does it include? Does it have an official status?

LIFTON: Yes, it does have an official status. It arose through the eventual setting up of medico-legal arrangements for those who were exposed to the bomb, especially medical and economic benefits, which became, of course, very much a part of the whole psychosomatic picture.

MATSUMOTO: I may be entirely wrong, but I think the Japanese use hibakusha much as we would use the word "survivor," without emphasis on the "atomic bomb" but being "a person experiencing a bombing." Ruth Benedict (Reference 190) has taken the Japanese word giri, which means moral obligations, for a critical discussion of the Japanese culture, and Caudill and Doi (Reference 191) have written on the word amaeru, which means the wish to be loved or dependency needs, as a concept in understanding Japanese personality structure.

My question is, can you take one word and put special emphasis on it as the magic key to explain the attitudes and behavior of a group? Certainly there is a special meaning in the sense that, if you are a hibakusha, you are a survivor, but whether there is additional emotional meaning to the word, and to whom, I would question.

LIFTON: I have been interested in that problem. In fact, I asked people to associate freely to the word hibakusha, just to the word, as a way of finding out what it means to them. Their associations were very vivid ones, often associations to the bomb itself, or to the fear of being physically incapacitated or of eventually being discovered to have leukemia. It isn't that hibakusha has any special meaning in itself, but any word can come to epitomize various kinds of experiences, in this case various fears, and a special stigma.

I would have one correction on what you said, Dr. Matsumoto, in reference to studies of the word amaeru. Studies of this and of related Japanese words were not initiated by Americans but rather by Dr. Takeo Doi, a Japanese psychiatrist affiliated with the University of Tokyo. His work follows well-established psycholinguistic principles, and his stress on amaeru was picked up by others. Hibakusha in itself is hardly even a real word. But it still comes to take on significance, not only as designating a certain group in a medico-legal sense, but a kind of superimposed psychological significance in terms of what it feels like to be one.

UPTON: I take it that the word is perhaps analogous in some respects to our word "veteran"? There are veterans to whom this status means a great deal and others who think little or nothing of it.

LIFTON: That's right. I think in some ways one could draw the analogy, and indeed extend it to certain problems of benefits and certain dilemmas which arise.

SCHULL: How can you be sure that the individual interviewed is associating freely? Many of us who have spent considerable time in Japan are impressed with how difficult it is to establish an open relationship with a Japanese family or individual, even when there exists evidence of a reasonably warm and personal relationship between oneself and the family or individual in question.

MILLER: Or even with the doctors that we worked with daily for two years.

LIFTON: Right. When you set up an interview situation like this, you are creating a situation in which you don't have a personal relationship in the sense of either a friend or a colleague. You are creating a relationship whose specific and only purpose is for this sort of exchange of information and feelings. It is a two-way exchange but it comes mostly from one direction. People who can be extremely reticent in social situations, and you can see this in ordinary psychiatric work, can overflow with feelings and express themselves very freely when a situation is particularly structured to encourage doing just that as the expected and appropriate thing to do. This may seem to be extremely difficult across the cultural and language barriers that we have been talking about, but, in fact, the flow was very ready and rather free, and in most cases within a very short period of time, rather spontaneous. This does not

mean that I then had an intimate relationship with these people in the sense that there was an expectation of the various qualities that may go into a relationship with a colleague or a friend. It did mean that a situation was created where they could speak freely.

FREMONT-SMITH: Are you saying that because it was professional situation there was a freedom of expression which they would not have had with you in a social situation?

LIFTON: That's right.

COURT BROWN: You didn't get any sort of transferency?

LIFTON: To a certain extent there was, but I don't think it was present to a very great extent. There were some people who wanted to arrange more interviews and come and see me again and again, and who, without being necessarily aware of what they were seeking from me, sought relief from certain anxieties and conflicts. I sensed a great deal of general relief from ventilation of these things. Others were rather disappointed that I didn't do a physical examination because their fears were about physical aftereffects and they wanted this investigated too.

FREMONT-SMITH: May I just make one point here? You had spent a good deal of time, or was it a good many years, in interview situations with Japanese and Chinese before you came to this particular situation; is that right?

LIFTON: Yes. I had worked on and off in this way for 10 or 15 years, in Japan and in Hong Kong.

FREMONT-SMITH: So that you had had psychiatric experience both as a psychiatrist and as a psychiatrist in the Oriental set-up before you came to this experience, which is, I think quite different if you ...

SCHULL: Those experiences have all involved this race, of necessity, and I find this disturbing. Let me try to illustrate why. Several of us at the University of Michigan are now involved in a study in Detroit which is attempting to determine the effect of stress upon blood pressure. There already exists, of course, a number of observations which suggest that under stressful situations the blood pressure is altered. Our study involves Negroes and Caucasians

living in both economically deprived as well as moderately well-off areas of the city. Stress is measured both at the neighborhood and the household level as a series of scores which incorporate such things as rates of crime, sense of threat to person, etc. At the outset and in the best of statistical designs, we hoped to randomize the race of the interviewer and the one interviewed. This proved impracticable, for it soon became obvious that one's ability to precipitate a stressful situation in the individual interviewed through a series of provocative questions depended upon the race of the interviewer.

LIFTON: Yes.

SCHULL: As an illustration, the word "whitey" carries implications when used in communication between two Negroes which will most certainly elude a Caucasian. How do you make allowance in your interviews for problems of this kind?

LIFTON: In some ways, communication problems between white and black Americans are more difficult than between Americans and Japanese, not linguistically, but in terms of a painful history that they have shared. Of course, my being a white American in this interview situation affected it and was an enormous influence. To the extent that a situation of free expression develops and manifests itself, this is forgotten. And if one listens to an follows the tapes, one can hear and sense an increasing disappearance of this barrier. But another way of checking on the data is, as I said before, the kinds of descriptions that have been written about and reported on that were made to other Japanese, or simply written down earlier. And then one has to use one's sense as a clinician with experience in this area of the world.

I also would talk to them about this issue; that is, their sense of talking to an American, how they felt about it. There was certainly no lack of their capacity to convey this nor was there any absence of psychological defenses.

ANGEVINE: Did they know that you were a psychiatrist or did you present yourself as a physician? If a psychiatrist came into my office to ask me a question, I might answer him entirely differently than I would an internist asking the same question.

LIFTON: For those who knew what a psychiatrist was, I, of course, explained what I was. Those were mostly in the special group. The ones in the random group tended to be people from the lower rungs of the socioeconomic ladder, because that is where most people are in most societies, and they had only the vaguest idea of what a psychiatrist really is. So their sense of me was as a physician or as a scholar, a professor from Yale University, as indicated on my card, and I didn't try to describe to them what a psychiatrist was or try to give them a sense that their minds were being probed for abnormalities, which, in fact, was not the case.

CASARETT: How would you characterize your samples with respect to variability in recorded biomedical effect of the atomic bomb?

LIFTON: You m[illegible]n acute symptoms, symptoms of acute irradiation?

CASARETT: Any recorded effects that were regarded as effects of the bombing.

LIFTON: A small minority had recorded effects, and a small minority had what you might call visible effects such as keloids.

CASARETT: Were cosmetic factors present when you interviewed them?

LIFTON: Just a few, because, as you know, only a small minority of the population now have them. Again, my sense of the data was that in these cases this gave a new dimension to what they experienced but it was a kind of continuum. Keloids, for instance, meant a great deal to people who did not have them as well as to those who did. They are symbols or stigmata of the atomic bomb experience both in the medical sense that we use "stigmata" and, in a way, in a psychological and a theological sense. But they conveyed to almost everybody an image of aftereffects, a vision of danger which they were susceptible to. With those who had keloids, the actual visible manifestations, all this was much more intense. In fact, those who had keloids were more touchy and much more anxious during the interviews.

TAYLOR: Could you correlate anxiety with what might not have happened to them but with someone who was very close to them, such as a wife or a son?

LIFTON: Yes. That is happening to them, if somebody close to them dies, especially if they witness the death; it is something that they experience.

DE BOER: Do the Japanese read a great deal about their own history or, say, do they read the newspaper accounts frequently?

LIFTON: People had different attitudes. Of course, everybody read newspaper accounts. Hiroshima is a city preoccupied with the atomic bomb. As Scott Matsumoto was saying, there is a kind of yearly rhythm in which, as August 6th approaches, you can feel the anxiety waves of the city intensifying, mixed in with all sorts of journalistic interests.

But the newspapers are concerned with atomic bomb issues all year round in Hiroshima, with reports of medical aftereffects or reports of various things that happen to survivors. In fact, this kind of social dimension constitutes a considerable part of my own study. Everybody reads or is aware of these reports, but some people have a psychological style of avoiding them, trying never to read anything about the atomic bomb or never going to a movie that deals with the atomic bomb, etc. Others have the opposite tendency of trying to see and read everything about it.

UPTON: To what extent is this preoccupation confined to the cities directly affected? Does this get national press attention throughout Japan?

LIFTON: There are two dimensions. It does get press attention throughout Japan. Japan is a nation which is, of course, unusually sensitive and responsive to the whole issue of the atomic bomb and of nuclear weapons in general. And certainly the Bikini incident in 1954—there was a Japanese boat, the "Lucky Dragon," with people on it exposed to effects of irradiation from American H-bomb testing fallout—became a national experience with the enormous emotion that was evoked. But there is still a very different dimension, I think, in Hiroshima and Nagasaki, a very specific sort of intracity concern—more so in Hiroshima than in Nagasaki—which is not comparable to the rest of the population. The rest of the population has complicated, sometimes very ambivalent feelings toward people in Nagasaki and Hiroshima. This has to do with a certain kind of "radiation" of the survivor ethos outward. I have been interested in this, too. Well, that's a long story; but, in fact, I think that

probably it is better not to go into it, into hearing all the psychological details. However, there are the two dimensions of feeling: a general one for all of Japan and a special intensity in those two cities.

MILLER: Can I ask you about methods? Did the tape recording have any effect on the interviews?

LIFTON: No. That is, I would always tell them I was doing it. I have learned to ask people but to ask them in such a way that is a fait accompli: "I'm making a tape recording. That is all right with you, isn't it?" They would nod and say, "All right." It wasn't anything they thought about too much. Nobody objected, and it was just turned on.

FRANK: This is a universal experience, I would say, by now. Certainly in psychiatry the only person who is bothered by a tape recording is the psychiatrist. The patient takes it for granted. There must be some kind of implicit question about the confidentiality here, though. Did they know how this material was going to be used or did they set any restrictions on how it might be used?

LIFTON: No.

FRANK: Did they want it to be used, do you think, in wide publicity?

LIFTON: I would tell them that I was using this to make a study, and that I would not use any names nor reveal anyone's identity. That issue wouldn't come up again, and usually wouldn't come up at all with the random group. But I felt that it was wise to say this. It wasn't usually a problem. There were some in the special group who were used to being publicly on record about it and would have welcomed their names being used. Anyhow, I tried to make this clear.

FRANK: The ones who would welcome their names being used would be the ones who would most likely exaggerate, I would think.

LIFTON: Yes, or who had a particular angle, a particular thing they wanted to emphasize. It might not be an exaggeration. They might in fact want to counter another's view. Hiroshima is a city that is full of antagonisms among individual survivors, among

survivor groups, among different political and social groups. And to some extent, these antagonisms themselves can be looked upon as a product of the atomic bomb.

FRANK: We are dealing here with a representative of a victorious country, of high status in that country, talking to people who were of low status in Japan. I think that you would get one impression that would be an angry feeling toward Americans.

LIFTON: I think this was relatively limited, but by no means absent. There were several interviews where people of ostensibly low status in Japan spoke very forthrightly about how they felt about Americans, how they felt about an American coming along 17 years later and asking questions. Where were they, so to speak, when they were needed? What about Americans who dropped the bombs now asking them about it? This came up in a few interviews and there are many other indications of indirect hostilities toward Americans over time. Of course, the whole set of feelings toward Americans in Hiroshima is by no means simply hostile, as we have said before. It is ambivalent, which is saying something very different. There is a lot of positive feeling.

AYRES: Could you sense any ambivalent feelings toward Japanese interpreters and so on? The "Uncle Tom" type of feelings?

LIFTON: I don't think so particularly. The interpreter was looked upon as somebody of a certain intellectual level with social standing in the community. He was cooperating with me, but there aren't those clear battle lines in Hiroshima, to make that "Uncle Tom" analogy, as there are with the white and Negro in this country.

JOHNSON: Would you care, first of all, to make some distinction between what we would, as Westerners, consider the obvious kind of hostility which would be generated, Westerner to Westerner, versus what, because of the cultural differences or a difference in hostility, Easterner or Japanese to American, or do you recognize any difference?

LIFTON: Yes, of course. Every culture has a different style of handling its hostility or potential hostility and there is no reason why we can't go into this now in terms of the question of Japanese hostility toward Americans.

On the one hand, when this issue comes up, there are some people who expect the most vehement sort of naked white heat, of overwhelming hostility on the part of the people who have been atomic bombed. There are otheres who believe that there is no hostility at all because the Japanese had this extraordinary turnabout as noted by so many in their welcoming the Occupation forces at the end of the war. Actually, the truth lies somewhere in between. But I think that the Japanese way of responding was rather impressive, and had to do with a rather rapid capacity to shift their sense of authority to the Occupation with much less hostility than was anticipated. It had to do with Japanese cultural patterns of dampening hostility, so to speak, but it didn't mean that hostility was eliminated.

Part of the American approach, as much as possible, was to avoid sending American troops directly into Hiroshima at the time of the early Occupation. British Commonwealth troops did most of the actual occupying in those early years. But it probably wasn't even necessary to do this, because the response to individual Americans and to American troops was rather warm and the Japanese were struck by the contrast between the images that had been spread by the wartime propaganda, of monsters who would rape and loot at will, and the rather friendly GI's who handed out candy and so on.

Thus Japanese feelings toward Americans are compounded of a series of involvements. They have to do with their response to losing the war; with a shift in authority and a mixture of resentment and developing affection, with perhaps a realization by most Japanese that this was an unusually kindly military occupation; a sense of resentment around the atomic bomb specifically, which over a period of time became as much concerned with the dissemination of information about the bomb and with the handling of later atomic bomb problems as it was with the bombing itself.

Hence, early Occupation policies of limiting dissemination of information about the atomic bomb or publicizing the atomic bomb were often a source of resentment. This was again mixed because many who had been through the bomb didn't want to hear anything about it and had mixed feelings about being confronted with it. Later on, feelings toward Americans became very much involved with the attitudes of individual Americans toward the use of nuclear weapons, particularly statements by Americans concerning their feelings about having used the atomic bomb in Hiroshima. An example is Truman's famous and celebrated statements that he did the right

thing, that he was sure he did the right thing, that he didn't lose any sleep over it, etc. Predictably, these statements were not well received in Hiroshima and they created great hostility. This was mentioned to me again and again.

JOHNSON: Would you add to that array a disillusionment in a military-controlled government which ruled Japan before and after the war?

LIFTON: Much of the Japanese hostility and disillusionment was directed at their own leaders, because they felt, as anyone who has lived in Japan and has talked to the Japanese about this knows, very deceived by these military leaders, especially after they experienced such suffering in relation to a weapon of this kind.

Also, in terms of Americans, there is the whole issue of the American research group in Hiroshima, the ABCC, which had been there a long time before I came and which had become a source of ambivalent feelings with the Japanese in Hiroshima, expressed through imagery that many of you are familiar with, the image of being made into guinea pigs. This is a complicated question in itself, but one could say that some of the feelings about the atomic bomb were displaced, as is always true in catastrophes, toward groups which then go in either to study or to help, and there are also manipulations of some of these emotions by various groups which had their own purposes within the Hiroshima situation.

JOHNSON: And the ambivalence is further demonstrated by an over 80 percent voluntary participation rate in ABCC's clinical study?

FREMONT-SMITH: 80 percent of what?

JOHNSON: Voluntary participation in the clinical examination. I am pointing out that this ambivalence is expressed in the form of what we consider magnificent cooperation.

WARREN: Did any group consider the alternative, that they could have been subjected to the bombing that Osaka had?

LIFTON: There were many who were eager to discuss with me some of the larger philosophical and ethical dimensions of the use of the bomb. Many brought up quite frankly such issues as whether

the Japanese, had they had a weapon like this, would have hesitated to use it. Others brought up the concentration camp experiences and compared survivorhood of that with survivorhood of the atomic bomb in Hiroshima, raising various ethical comparisons. And don't forget that, in terms of feelings toward Americans as such, over a period of 17 years things have leveled off and toned down. We have been living with each other for a long time, Japanese and Americans, and there was a capacity to air these things.

I suppose we should proceed. Various people have advised me to hurry up and get into what I found. Otherwise I may never have a chance to! [Laughter] But I meant what I said when I indicated that I welcomed interruptions.

I was also advised to state all of my conclusions first; otherwise I may not have a chance! [Laughter] But I am not sure I can state them that easily or quickly. In any case, just to give some kind of order and structure to the findings, I think it would be useful to think of four stages of response.

There are really four stages of death encounter, as I have called it, which include: the immediate exposure at the time; the encounter with acute irradiation; later concerns about long-term radiation effects; and finally general social dimension or sense of group identity as survivors, with all that entails.

So if one keeps these stages in mind, the first one is the overwhelming experience immediately following the bomb's fall.

Involved here is a sense of surprise and unpreparedness in connection with the bomb. We could spend a great deal of time on this general issue, but I would say just in terms of what existed in Hiroshima was that on several different levels people were radically surprised and unprepared. In the first place, Hiroshima, as you know, had been relatively untouched by bombs of any kind, except for one or two stray small bombs that had fallen on the city. It had had regular air raid alerts from planes passing over it when heading to other places.

On the morning that the bomb fell, there was an air raid alert sounded about 7:10 a.m. and an all-clear sounded at approximately 7:32. These times, incidentally, are contested. There are different times given from every different source that one goes to.

I followed those given by the city itself. There is an account which indicates that the actual three planes on the mission were spotted and were mentioned on a broadcast on the Hiroshima radio after the all-clear had been sounded—words to the effect that "there are planes which may be approaching Hiroshima but we will keep you informed." This did not reestablish a state of alert, and there was a situation of more or less complete surprise on this immediate level.

On the psychological level, one must consider the sense of invulnerability which all of us maintain, both ordinarily and in situations of danger or threat. A third level of unpreparedness had to do with the dimensions of the weapon, which no one in Hiroshima had in any way anticipated. People in Hiroshima expected bombing of some sort. In fact, there was an eerie feeling about the situation, in their awareness that they were one of the very few large cities in Japan that had not been bombed. There were all kinds of rumors as to why Hiroshima had not been bombed.

Some of these rumors were more or less magical and wishful, such as the rumor that Hiroshima was not being bombed because it was so beautiful an area that Americans were saving it to build villas later on during the Occupation. This rumor, of course, suggests that the Japanese knew how the war was going in general. There was also the rumor that perhaps Hiroshima would not be bombed because so many people had emigrated from that area to the United States. And then there were some even more wishful rumors to the effect that some very important foreigner might be living in Hiroshima. There was even one rumor that some relative of President Truman was living there.

But there was also the feeling that perhaps America was saving Hiroshima for something very special, for some particular kind of devastation, which, of course, turned out to be more or less the case. In any event, on all these levels, there was a state of surprise and unpreparedness.

The first stage of the actual immersion in death begins with the array of dead and near-dead that each survivor found himself in the midst of. Of course, this varied enormously in terms of where one was at the time the bomb fell. But following the early statistics of Oughterson and Warren, if one was, for instance, within 1,000 meters, or six-tenths of a mile from the hypocenter and out of doors

(that is, without shielding), then more than nine-tenths of the people around one were fatalities. If one was unshielded at 2,000 meters, or 1.2 miles, then more than eight of ten people around one were fatalities. But beyond that, reaching farther out into the entire city, there was likely to be some sort of involvement in the experience of the bomb.

Recall of the experience was very vivid, as I suggested, despite the 17-year interval, due to a certain kind of indelible imprint that has been described in various survivor experiences. But for those closest to the hypocenter, first memories of the event were frequently no more than those of a sudden flash, an intense sensation of heat; or the recollection of being knocked down or thrown across the room or finding oneself pinned under debris; or of simply awakening from an indeterminate period of unconsciousness (which could merge with later elements of amnesia, as has been mentioned, although the factor of amnesia didn't seem to be too great). Nonetheless, many stressed—and this is partly with a retrospective reconstruction but still having significance—feelings related to death and dying, which were often among the initial emotions they experienced. And these included (I am going to do a certain amount of quoting from actual accounts* because I think these give a certain immediacy that is important) such emotions as:

> "My first feeling was, I think I will die." "I was dying without seeing my parents."

Or—remember that these are all different people:

> "I felt I was going to suffociate and then die, without exactly knowing what had happened to me."

But beyond this sense of imminent individual death was a feeling expressed to me by many that the whole world was dying. Thus, for instance, a science professor who was covered by falling debris found himself temporarily blinded and his recollection was, and I am quoting:

> "My body seemed all black; everything seemed dark, dark all over. Then I thought, the world is ending."

*Editor's note: The quoted expressions are from Robert Jay Lifton. 1964. On death and death symbolism: The Hiroshima disaster. Psychiatry 27(3): 194.

And a Protestant minister in a different kind of language put it this way:

> "The feeling I had was that everyone was dead. The whole city was destroyed... I thought all of my family must be dead. It doesn't matter if I die... I thought this was the end of Hiroshima, of Japan, of humankind."

And a woman writer later wrote of what she perceived as the collapse of the earth, which, it was said, would take place at the end of the world. These were typical manifestations of the sense that the whole world was dying or was ending.

Rather than the wild panic that people sometimes imagine would take place with a disaster of this kind, most people described it to me as something more in the nature of a ghastly stillness or a kind of deathly silence, and a sense, whether literally true or not, of slow motion. In the silences they described, there were low moans from those who were incapacitated; the rest fleeing, but usually not rapidly, from the destruction outward toward the river (and I should say that branches of the Seto River run throughout Hiroshima), toward where they thought were family members, some sort of medical personnel, or simply toward accumulations of people, or in many cases merely moving along with an accumulating crowd and with no clear destination.

I think that it is unnecessary to say, because it has been said earlier in our meetings, that the bomb fell in the center of the city. The bomb was apparently directly on target and there were military targets right near the center of the city. So it really involved the entire city almost immediately in this disaster.

This feeling of death in life was described to me in many different idioms. But, to give you again some of the flavor of actual recollections, one store clerk put it this way:*

> "The appearance of people was... well, they all had skin blackened by burns... They had no hair because their hair was burned, and at a glance you couldn't tell whether you were looking at them from in front or in back... If there had been only one or two such people... perhaps I would

*See footnote on page 312.

not have had such a strong impression. But wherever I walked I met these people... Many of them died along the road—I can still picture them in my mind—like walking ghosts. They didn't look like people of this world. They had a very special way of walking, very slowly... I myself was one of them."

I should point out at least two psychological elements of this passage. One is again this other-worldly grotesqueness, the sense of a kind of supernatural dimension; images of more dead than alive human figures. And the second is the identification of the survivor, of the man who is still alive, with this supernatural world and with those who died or are dying. This identification with the dead is a central theme of the entire disaster, an important one from the very beginning and continuing indefinitely into the present in various ways.

FRANK: Don't you think that this is peculiar to atomic disaster or would this be true of any disaster where people were killed?

LIFTON: What I have said so far would probably be true of any large disaster, except that the atomic disaster has sudden totality, and is of an extraordinary dimension. So that it may be truer, more vividly true, for an atomic disaster.

There are, in a sense, several dimensions to keep in mind simultaneously. One is that which is true for all disasters. A second dimension is that which is specifically related to atomic disaster, or at least much truer of atomic disaster because of its greater dimensions and because of the factor of irradiation which is a very specific and unique aspect of atomic disaster.

A third dimension is, of course, the cultural element in the response. But as one gets into responses of the catastrophic dimension, I would say—and this is the finding of other people as well, not just my own—that cultural aspects do not tend to dominate responses. There are certain universals that tend to emerge even though they may be, so to speak, stylized or molded into cultural forms.

FREMONT-SMITH: Were there cultural aspects of attitudes toward death which were reflected in this experience?

LIFTON: Yes, there are cultural attitudes toward death which are reflected in the experience right along. I take the position on this—and, of course, it is a large subject—that they tend not to create responses to the disaster but to mold them along certain lines. Even in terms of cultural attitudes toward death they are simply weightings and emphases. For instance, the Japanese place great emphasis upon continuity with the dead, in the little family shrines that they have, and in their various traditional ways of remembering the dead and staying in close touch with the dead, and that would influence the identification with the dead that I have mentioned. But in all cultures, there is a kind of equilibrium between one's need to stay in some sort of touch with the dead—whether specifically prescribed by religious means or whether of a more symbolic kind—and one's need to rid oneself of the dead and keep away from them, one's fear of the dead. And this same ambivalence toward the dead is very much a factor in Japanese culture. So even the Japanese attitude is not unique; it is simply a weighting of universals.

In terms of the further psychological meaning of this earlier immersion in death, we are struck by extreme feelings of helplessness and abandonment, but in a specific context of threatened annihilation. And, of course, the fear and anticipation of annihilation dominate this phase for the survivor, or the hibakusha. One must ask what it is that the survivor fears is about to be annihilated. And here one must say that it is, of course, his overall organism insofar as he is capable of perceiving it, that is, his sense of self. It is also his sense of individual identity, and the field or context in which he exists as well. It isn't only the self that one fears being annihilated but also one's surroundings or "nonhuman environment." One begins to appreciate what the existentialists mean when they speak of "being-in-the-world," an inseparable relationship between self and the world around one, which is also, incidentally, emphasized in Japanese cultural tradition.

This anticipation of annihilation had to do with these overwhelming assaults from without, external threats, as well as with internal psychic responses, all combining to create a fundamental kind of anxiety which has been called basic fear, basic anxiety, or in more lyrical terms, fear of the universe.

In any case, this experience was so overwhelming for many years that I think that they would have been unable to avoid something

approaching psychosis were it not for an extremely widespread and effective defense mechanism, which I call psychic closing-off. This becomes another important theme of atomic disaster, a pattern which, in more chronic or prolonged form, I speak of as psychic numbing. What this really means is that in the face of grotesque evidences of death and near-death, sometimes almost immediately—within seconds or minutes—people simply cease to feel. They have a clear sense of what is happening, they know cognitively what is going on around them, but their emotional responses are unconsciously turned off.

Again to illustrate this, a physicist, observing the process in himself, compared it with an overexposed photographic plate. And a clerk who witnessed others dying around him at a temporary first-aid area reached a point at which, as he described it:

> "I just couldn't have any reaction... You might say I became insensitive to human death."

The woman writer I quoted before spoke of a ...

> "feeling of paralysis that came over my mind."

This was almost universal in survivors who were immersed in a scene of death of this kind.

FREMONT-SMITH: This would be particularly true for those who were surrounded by other dying people and perhaps less so if they were isolated by themselves.

LIFTON: That's right.

FREMONT-SMITH: In other words, it was the overwhelming impact of so many.

LIFTON: This is mainly a response to witnessing death taking place in others.

FREMONT-SMITH: In many others.

LIFTON: In many others, yes. But, of course, it is not limited to this. Again, any psychological pattern one describes has something to do with the human psychological potential which is then evoked under these extreme conditions.

Jerry, do you want to say anything?

FRANK: I can't say anything except simply that a man in battle doesn't know when he is wounded. He is overwhelmed by stimulation.

LIFTON: Exactly. This is a universal kind of response, and it has been noted in the "apathy" observed in survivors of any kind of disaster. It is a pattern that has been constantly observed but I don't think sufficiently explored as a general psychological phenomenon, especially in its relationship to death and dying.

TAYLOR: Is there any indication of whether it is necessary that the intensity be high because of large numbers of people?

FRANK: No, I don't think so.

TAYLOR: Or is it equally effective to have the intensity be high because the death seen is that of a person very close? I ask this because I have not heard that this was a common occurrence when people observed a single death in their own families, for example, under just ordinary circumstances. It doesn't seem to be such a common thing.

BUSTAD: It depends upon the situation. In infantry combat, at which I made a living for a while, you may not be suffering a lot of casualties but you can walk along a road or street and you come to a rest break, maybe on a fast march in pursuit. You lie down and you may lie next to a dead person and not think anything more about it.

FRANK: I think one has the wrong handle here. It isn't the deaths; it is the shock. It is the overwhelming stimulation of some sort. The Pavlovians term this reaction inhibition (Reference 192) or some word like this. It is some kind of apathy that comes when one is overwhelmed by stimuli. I don't think that the fact that there are a lot of corpses is the crucial point.

LIFTON: The crucial point in this kind of response is the relation of stimuli to death, but that could be argued. I think that in the battlefield analogies this is very much the issue. At least the way that I have understood it, although I am sure one could look at it in other ways, is that one is closing oneself off from death itself, with a controlling inner fantasy. "If I see nothing, then death is not

taking place"; or "I know he is dying but I am not related to him; I am not responsible for him." And in a sense one needs this kind of defense to carry on with some sort of functional capacity in the midst of death taking place around one. One would not meet this level of psychic assault under ordinary conditions nor would one, so to speak, turn away from death in the same degree or feel that one had the right to do so.

UPTON: It is true that one needs not to be overwhelmed in order to take proper action to survive. On the other hand, certain degrees of sympathy and cooperation may themselves be necessary to survive.

LIFTON: You have raised what I think is really a basic and paradoxical issue, which applies both to immediate and long-term responses to catastrophe. It also applies to the rest of the world in the contemplation of catastrophe. Under extreme conditions the response of psychic closing-off is life-enhancing—in respect to physical and psychological life. In some very important ways, you need it in order to keep going, to remain alive. But at other times, especially when the response itself is prolonged and no longer appropriate to the threat, it may be exactly the reverse. It can turn into the reverse very quickly, as you suggest. It may well be that some of these people who described precisely this pattern of psychic closing-off as they walked aimlessly through the city could have helped people if they had less of it. Their inability to feel any appropriate emotional response—their emotions being "turned off" in this way—had already begun to be dysfunctional. Like many kinds of repair mechanisms, psychic closing-off or numbing can overstep its usefulness and become a kind of problem in itself.

FREMONT-SMITH: Isn't there a contrast between the kind of accident that can take place, a sudden accident, an automobile accident, when the majority of the people are helpless and then someone will step in and help straighten out the situation? In this situation, with the little reading that I have done, the people were overwhelmed partly by the fact that there was nothing that they could do that was useful because everybody else was dying, everybody else was almost destroyed, and as you said, they walked around partly aimlessly because there was nothing constructive that they could do.

LIFTON: As to the degree to which people could help each other, in general, people were very often able to act effectively in saving

themselves or their immediate family members, whom they tended to think of and to help, particularly if they were in the same place, the same house, or the same area. They did relatively little in helping anyone else. This is again related to Japanese cultural patterns with great stress on the family, with relatively little development of the idea of broader social responsibility for others. But it also had to do with the dimensions that Frank mentioned and with this overall process that I am describing, with the magnitude of the disaster itself. One felt so overwhelmed that the mechanism of psychic closing-off was immediately called forth, and one felt that there was so much of death around that one could do nothing.

There were, however, numerous exceptions, people who did help others, and there was much else that went on. But these are the general patterns.

FREMONT-SMITH: Did the psychic closing last or did it begin to open up and then have its effect later?

LIFTON: It then became a source of conflict later on. For instance there was one man who was a noncommissioned officer at the time. He was outside the city on leave and then came into the city after the bomb fell, in accordance with pre-existing regulations. When he came in, he was one of the relatively few intact men around, especially in the center of the city in the military area to which he returned. Insofar as there existed a chain of military command, he was soon ordered to take charge of burning corpses. He had never in any sense had anything to do with cremation, and was just an ordinary man. I have his quotation, which I won't dig out now, but in essence what he told me was that he was rather surprised at the ease with which he did this. He simply treated them like "goods" as he put it, which you just had to burn. He said, "I had no feeling for them at all." Later on, he was horrified at his lack, or near lack, of feeling for them, at that time. And this was another kind of feeling one had, tied in with guilt, as one comes out of the numbing. Of course, one is never entirely in it; it is never a total mechanism, although it comes closest to being most total, I think, at the moment of impact.

COURT BROWN: What do you mean by psychosis in the context that you use it? As I understand a psychotic is a psychopath whose features approximate a well-recognized syndrome like schizophrenia paranoia, manic depressive psychosis, all of which we would expect to have a biochemical background.

LIFTON: The last phrase may be questioned, but go ahead. [Laughter]

COURT BROWN: Okay. What do you mean when you say he avoided psychosis? He avoided becoming a schizophrenic or did he avoid becoming a psychopath of undetermined origin?

LIFTON: No. I mean putting it very simply, I wasn't talking about actual clinical entities.

COURT BROWN: This is a very important point, because did, in fact, this experience tend to trigger psychosis in individuals who were prone? Is there any follow-up information on this?

LIFTON: Let me answer the questions one at a time.

In terms of the first question, I was talking about psychosis not as a discrete and chronic entity but as a potential response. It has been shown and observed that under certain kinds of conditions people can become temporarily psychotic or psychotic-like in their behavior without being clinically schizophrenics or manic depressives or any form of psychotics of that sort. What I really meant was that psychic closing-off defended against a more cataclysmic psychological reaction. Perhaps it might be better to state it that way.

WARREN: Antisocial?

LIFTON: No. All I meant was that the defense was against some sort of cataclysmic psychological reaction. In terms of the second question, which is an interesting and important one, there are no good statistics. I tried to get them and I talked to a lot of people about them but they really do not exist. And my sense of the matter was that I could not uncover any clear evidence that the psychosis, per se, increased at that time. This is in keeping with others' findings that, under extreme conditions, say wartime conditions, psychoses do not necessarily increase. They can, under some conditions, decrease. It is a very tricky kind of pattern for any clear-cut statistical arrangement.

There was some later imagery of people going mad, acting "crazy," or behaving bizarrely. But when you talked to doctors who were in charge of treating people, or to those who had some sort of authority at that immediate time, they could not really describe any great

number of psychotic reactions. There were also people who became incoherent before they died, on the basis of physical effects.

MATSUMOTO: At Hiroshima University a professor of neuropsychiatry has tried to obtain figures, but as far as he can surmise, he feels that there has been no increase of psychosis because of the atomic bomb experience. For what few cases they have, it seems very difficult whether to relate the psychosis to atomic bomb exposure, per se, or to some other personal experience.

LIFTON: What, of course, does happen in Hiroshima, as a psychiatrist there told me, is that people incorporate their atomic bomb experiences into their psychoses. It becomes part of delusions or hallucinations or other psychotic content, but that is saying something very different from seeing it as a specific cause.

FRANK: Statistics have been presented which show that the rate of psychosis ranged between 2 and 3 percent regardless of peace or war or anything else, but the rate of exhaustion syndrome goes up with increasing length of combat (Reference 193).

LIFTON: That is a good point. The fact that there was not a definite invasion of psychoses does not mean that there weren't severe psychic incapacities—mostly along the lines which I emphasized earlier and which would fit in with an "exhaustion" or numbing syndrome rather than a clear psychosis.

AUXIER: In order to quantitate the thing, you face the same problem that you have on the biological side, which is the fact that you are dealing with survivors. When you read these hundreds and thousands of shielding situations you are impressed with the fact that there were people who walked away from members of their families trapped in debris, and you can't know about the ones who stayed and tried to help but then failed to get out alive. There could be a large bias there.

LIFTON: Of course. In any case, this pattern of psychic closing-off could well be one of the most fundamental human responses to catastrophe. It had, in a psychological sense, a kind of global quality in its throwing out a protective, symbolic screen around the organism which permits it to resist the impact of death. I have said it was incomplete and not entirely effective in an absolute sense. I have also mentioned one of the elements that interfered

with it, that of guilt—guilt over survival, guilt over what one did in the process of surviving—that experience of self-condemnation which is a very early imprint in any kind of survivors for continuing to live in the midst of the deaths of others.

The guilt of the survivors is another enormous topic, but one aspect of this relationship between death and guilt has to do with what I have called the image of the ultimate horror. Most survivors I spoke to would have one particular image which was the most disturbing thing that they could recall, and which they could describe in very vivid and often grotesque detail. That image would seem to epitomize the entire experience. In that image, of course, would be scenes of death and dying; it might include family members who died or it might, as it often did, include women and children dying grotesquely, because women and children evoke and represent what is felt to be the most vulnerable and most pure in any culture. As an epitomization of the entire catastrophe as psychically perceived, this image of ultimate horror became what I have called an indelible imprint.

A special form of ultimate horror included scenes of refusing water to the dying. Almost anybody whom you talked to in Hiroshima would emphasize recollections of the dying asking for water. When I thought about it, I wondered, was it just in relationship to their physical state, a shock-like state which made people seek water? And insofar as there was any kind of instruction given by the authorities to the people, they were told not to give water to the injured because it might harm them. Most were, in any case, unable to do so. But it turned out that the offering of water and the request for water have a certain symbolic significance in Japanese culture; it is related to mythological imagery about water preventing the soul from leaving the body, which the soul does at death, or calling back the soul if it has just left. Water is thus perceived as life-sustaining and the survivors' refusal of water to the dying for whatever reasons had the symbolic significance of refusing life to others while clinging to life oneself.

BUSTAD: But it was also very hot.

LIFTON: Yes. It was extremely hot.

Proceeding to the second state of the general psychological sequence I mentioned before, it is dominated by the theme of "invisible

contamination." This, of course, has to do with problems of acute irradiation. I don't think there is any need with this group for me to go into details about the symptoms of acute irradiation. They have been mentioned several times.

UPTON: I am not sure I understand. Were they aware that this was a radiation device?

LIFTON: No, not at all. In terms of knowledge about the bomb, things were very confused. Don't forget, the beginning symptoms of acute irradiation manifested themselves very quickly. Sometimes within a few days or even within 24 hours there were beginning gastrointestinal symptoms. And then during the next few weeks they began to manifest themselves more fully. But at that time and for the first few days practically nobody really knew what had happened.

People had all sorts of images about what the bomb was. There were various kinds of mythology about the bomb and different kinds of corpse mythology. In terms of what the bomb was thought to be, some thought it was an ordinary bomb which had fallen right on them. This is called, by some disaster workers, the "illusion of centrality" —the illusion that the disaster is happening immediately around one. But in this case it is a bit more complicated because in a way the disaster immediately did involve such a large area of the city. Some described the bomb in terms of a large short circuit or a vast electronic failure, because of its general manifestations.

Of course, the Japanese leaders in Tokyo knew immediately it was an atomic bomb. This was announced by President Truman and they picked up the shortwave broadcasts. They sent in a team of scientists and physicians who arrived in Hiroshima on the 8th of August. And there was beginning knowledge quickly among surviving leaders in Hiroshima, some university people, and some physicians that this was an atomic bomb. But as to when exactly who knew what, the situation was extremely confused; people half knew it, without knowing what it meant to know it, and half didn't know it. The average person knew nothing about the weapon. What they came to suspect as they began to experience or observe symptoms was that the weapon emitted some kind of poison. The Japanese made an official announcement that a "special weapon" had been used. They didn't announce it as an atomic bomb until some time afterward.

BRUES: As far as I could tell, Dr. Hachiya, in his very detailed diary (Reference 67), never tells the reader when or how he became aware of what it was.

LIFTON: He does have a very fascinating section and, I think, a very interesting and informative one, in which he describes his own struggles with exactly what the symptoms in his patients meant. At first he thought it was some sort of a cholera-like epidemic, because gastrointestinal symptoms were especially prominent in the early stages. Then he had some idea that the manifestations he was seeing had something to do with atmospheric pressure but he didn't know quite how or why. Then he began to hear various rumors about an atomic bomb, but only gradually, with a lot of fits and starts, did he come to grasp this. And he didn't make a clear-cut evaluation of his findings with any confidence until he heard the famous lecture given by Tsuzuki a month later in Hiroshima.

Tsuzuki was a leading Japanese authority on radiation. He was actually a surgeon who had had a background in radiation work and had, I think, spent a period of time in America. Tsuzuki was the major consultant to the Japanese military, and he came to Hiroshima to make some studies and, of course, cooperated with the American team, which came in later.

WARREN: Dr. Tsuzuki was my guide and protector at this time, and much of the information that was the basis for his lecture in mid-September came from our party at the time.

Could I explain some data? It was about two weeks or mid-August after a lot of autopsies and bacterial investigations of the intestinal tract before the Japanese doctors were able to eliminate cholera. Histological preparations were made at the local Omura Tuberculosis Sanatarium to identify extensive disintegration of the intestinal epithelium, especially in the colon. They also found some bone marrow and lymph node damage which led them to suspect this as a contributing lesion. But, of course, the bone marrow didn't really fall apart except with excessive doses in the survivors who lasted a week. The bone marrow really didn't begin to fall apart until about the fifth and sixth weeks when large numbers died after a day or so of weakness, pallor, and purpura.

Tsuzuki's lecture came at about this time. Just after the lecture, while we were grounded in Hiroshima by the early typhoon and rain, our planes couldn't come in and get us for several days. A

newspaperman who had had a journalistic training in Los Angeles made his way into our bivouac and had dinner with us and said, "Do you know that the word is going around in Tokyo that the use of the atomic bomb was not a good thing; in fact, it's your newspapermen who have raised the ethical and moral question?" This was quite a surprise to the Japanese who had not questioned at any time the use of any weapon that might be of an advantage. We discussed this quite extensively almost all that night.

LIFTON: Of course, at that moment they weren't in a position to question anything.

WARREN: No. But still they could have cried all over. They weren't bringing this question up at all and it wasn't until later, when it became a popular subject among our newspapermen and others, that the Japanese newspapers began to join in. Tsuzuki and Motohashi, who were high in the government, did not. Apparently, Tsuzuki had been in on the meetings of the Cabinet where the question was asked, after Hiroshima was bombed: "Is this an atomic bomb, can the Japanese make one, and is there any defense?" And the answer from Nishina and others present was that they had just come back from Hiroshima and they said, "Yes, it's an atomic bomb. There's no defense, and we can't make one." This was probably the straw that helped break the back of the military control and they stepped down and relinquished control to the Emperor, who the next day asked the same three questions and then decided that he would surrender.

Tsuzuki, with no hesitation, said that the fact that this was beyond the capacity of the Japanese to deal with enabled the Emperor to surrender without losing face and without committing harakiri. This relieved also the other people in command and in responsible positions from doing the same thing. We discussed this matter for days before returning to Tokyo. If you recall, prior to this time, whenever we had overrun or the Japanese had bypassed and abandoned troops and civilians on some of the Pacific Islands, that some of the officers who were left had committed harakiri and some of the civilian components had actually walked into the ocean and committed suicide.

Tsuzuki was quite vocal about the fact that a large part of the people in the Empire knew they were going to lose but they had to save face so that when they lost, a great many planned to commit

harakiri. When I got ready to leave, the prefectural governor of Nagasaki gave me his personal harakiri knife since "he wasn't going to need it anymore." The kamikazis were all set with the last gasoline they had. We saw some of these installations around Nagasaki particularly. They were going to go all out in a bansai suicidal effort to end it all when it happened. They, too, were greatly relieved by not having to do this.

All this accounted in great part for our easy and cordial reception. Remember that my party was down in an unsecured area. MacArthur had not come in yet. We went with his grudging permission and with the protection of Tsuzuki delegated to him by the Emperor and with the beneficience of Dr. Juneau from the Swiss Red Cross. We did not expect to get back. We all were quite surprised at our reception and after the first day we left our weapons in the hotel; we didn't need them.

I can verify what Dr. Lifton has said about the suspicion and investigation by the Japanese police, of people who might have influenced the Americans so that they did not bomb Hiroshima. The newspaperman who brought this news about the criticism of the use of the atom bomb also referred to this fact since he had spent some time previously in Hiroshima. There were quite a large number of Christians in Hiroshima and the people had wealthy relatives in California. The military authorities had actually arrested some Christian men whom they suspected of being involved in an arrangement not to bomb Hiroshima. Of course, several other cities were not bombed and they suspected, which was true, that they were being reserved for some special treatment.

During most of September and early October, we talked with the teachers in Nagasaki, a few of the managers of the Mitsubishi Works who came back to see what they had left, and a few of their secretariat and the prefectural governors, especially the one in Nagasaki. He was around the hill in the center of the city and he was not involved directly, although he felt the blast and saw the light. You got the impression that there was widespread initial shock, and individuals would go around in a numb or amnestic state and they might or might not respond to questions. Then they suddenly recovered and might find themselves, like the doctor I talked about the other day, say 10, 15, or 20 miles out in the country and not know how they got there.

Apparently, also, the people were more interested in their own survival than they were in helping others, except for family members and doctors and nurses. There was quite a bit of distress in the survivors from the lack of water at the first-aid stations. These often were not much more than a couple of pieces of corrugated iron set up on some posts to protect patients from the rain. It was difficult to get water anywhere, for the pipes were all broken. The prefectural governors, particularly the one in Nagasaki, delivered two bowls of rice and a pinch of tea per casualty per day. This was why these aid stations collected the injured.

By the time I got there many badly injured patients had been distributed to the local farmers, particularly the burn cases. There were almost no burn cases in the aid stations. The others that were ambulatory or could be carried in came because of the food since the local farmers didn't want to use the scarce food supply which they had if there was some available from the prefectural government. This was why we were able to see the purpura casualties in such large numbers. After the purpura cases died off, the burn cases were brought in for the food. Thus I saw mostly purpura cases and Shields Warren and later groups saw mostly burn cases.

The peculiar thing, as I said before, was that the ones with purpura and the low white counts and a beginning drop in the red count, were collected here in large numbers, in distinction to the burn cases, probably because they could still walk in, their main symptom at this time being weakness.

We frequently had as assistants interpreters and aids from the Japanese population who had a lemon color, a little epilation, and a beginning purpura, who would work one day and be dead the next. It was that acute a collapse. At first, we did blood counts and we had brought some penicillin and some plasma in and we were going to try the effects of these things. But the minute we punctured the skin, we were unable to stop the bleeding. The oozing continued and since the patient died that night or the next day, venipuncture or blood counts became very unpopular. So we had to stop even the blood counts. Even the Japanese couldn't do it anymore. Some white blood counts were as low as 50 cells and there were no platelets in the last days of life.

I think that there was chaos, also, in Osaka and Yokohama but not to the extent there was here. Hiroshima particularly had a big

military group and a military hospital and the city was practically run by the military government. Most of the military were killed. We had a meeting with the acting mayor probably on the fifth week and he said he didn't know much about the situation because he had not functioned before as the mayor, who had died. The military governor had died a week before and there was a little major from Kure representing the military government. He knew nothing about what went on in the city or the number of troops there. This gives you an idea of the collapse of the administration in Hiroshima.

On the other hand, in Nagasaki, the prefectural government survived and was on the job immediately. The military was distributed mainly along the Bay usually working in or supervising the work of these big factories. Therefore, they did not dominate the picture afterward except that they sent in parties to remove the ill and aged to the aid centers and to burn the bodies.

It was particularly characteristic in Nagasaki, where the destroyed area was almost undisturbed, to find large piles of human ashes, particularly outside the medical school. But then about the time we got there, they ran out of lumber and combustible materials and were importing oil, if they could get it, to burn the bodies. The fires were still going all night through the seventh and eighth weeks from the large number of those dying from bone marrow collapse at that time. This had quite an impact on the uninjured ambulatory people who tended to stay out of the place for fear of poisoning of some sort.

I am no psychiatrist, but we wanted to know what happened, how did this affect the people? I got the impression that it was the overwhelming, almost instantaneous, catastrophe that was most important, particularly in this early period or through September, except for the officials that I mentioned and the doctors. Even they were convinced that "one plane, one bomb, and one city" was overwhelming. The radiation part of the big weapon wasn't generally known by the population until early October or about when Shields Warren took over the area.

LIFTON: Extremely interesting.

In connection with some of the things that Dr. Warren said, there was a lot of almost compensatory bodily mythology that was expressed to me about what kind of injury one had and whether it was good or bad,

whether one would live or not. For instance, a distinction was made between burns and the "poison," which was, of course, the irradiation. It was felt by many burned people that they were fortunate because those who got burns didn't die from the poison, or even that severe burns protected one better than lesser burns. I think this is a kind of psychic compensation, and also the need to have some sort of rule and order, some sort of sense of causation, a need which is very profound. There was also times the idea that a vertical wound was better (less fatal) than a horizontal wound. It went to that extent.

In terms of the administrative disorder in Hiroshima, I agree with everything you said. I think that the period you were describing was that in which there was an elderly man who had become mayor when the former one was killed. He was rather helpless. He was described politely by people as being rather ineffectual. Ther a younger man stepped in, who subsequently became mayor of the city and served almost continuously with few interruptions over the whole post-war period. He became something of an early hero because he distributed clothes and goods rather energetically. But all that was some time after the period you have described.

WARREN: Yes.

LIFTON: Certainly the administrative chaos was important. Of course, the difference between Hiroshima and Nagasaki early in these matters is very important. Hiroshima was pretty much destroyed as a city, whereas more of Nagasaki was intact than was destroyed.

WARREN: More than half of it was around the corner from the hill and almost intact.

UPTON: This dawning awareness of intoxication of some sort, I think, must be extremely important here, because it does distinguish between simply a large conflagration that one could have had with it and the long-term effects that one has with this kind of a weapon.

LIFTON: Absolutely. In a sense, at the moment when the symptoms began to appear, one could say this was the turning point between a nuclear disaster and what you might call a non-nuclear disaster of an extraordinary dimension. Maybe I can't even make that statement. But for the dimension, it would be a turning point.

WARREN: Wasn't this, though, first known among the doctors? Of course, I was an enemy, but, nevertheless, the doctors were able to talk to us on a professional basis. Their point of view was, well, this is very interesting but what can we do about it? And there was no answer to this.

I can remember that I tried to avoid actually seeing patients as a consultant. But in Nagasaki I could not avoid this on the plea of the prefectural governor. So I went out to a farm with him and with Tsuzuki and one of our men and saw a woman who had been brought in who obviously was badly damaged by the radiation. Her blood count must have been down. They had no count, but she was beginning to be yellow; she had lost her hair. She had a little diarrhea earlier but had gotten over that and was obviously going to die. What could I suggest? I thought and thought and I couldn't think of a thing to do. We had no blood at this time at this place and no plasma and no penicillin. We had left this all in Hiroshima. And I said, "Well, can you get any liver? This might help some," thinking that if they had something to do, this would help the bystanders but maybe not the patients. Of course, they had no liver.

LIFTON: They had nothing.

WARREN: They had nothing. They had a little raw fish but there wasn't enough fish liver to do anything. So this caused a great deal of gloom in the people who knew about this locally and it was one of the psychological hazards that I wanted to avoid because I just added to the gloom. There was nothing you could do.

LIFTON: That brings up an important point. Survivors were faced with the bizarreness of the situation and the strangeness of the symptoms. Yet, beginning knowledge of what caused them did not necessarily help them, or overcome their fears.

Through word-of-mouth information and misinformation about the atomic bomb, the word got around among ordinary people that some mysterious poison emanating from the weapon was causing all this, and, whatever their sense of the cause, they were quite impressed not only by others dying around them but by the way in which they died. This constantly came up in their later accounts. They described what amounted to a gruesome form of rapid bodily deterioration which seemed unrelated to the more usual and "decent" forms

of death. This was again and again conveyed to me. I will cite perhaps one account to illustrate that sort of feeling. This was a middle-aged writer and businessman who described his daughter's sudden illness and death, as he recalled it. He said:*

> "My daughter was working with her classmate at a place 1,000 meters from the hypocenter... I was able to meet her the next day at a friend's house. She had no burns and only minor external wounds."

(Incidentally, many children were working near the center of the city, mostly on clearing fire lines and other work of that sort, and they were, of course, caught there.)

WARREN: Adolescents, yes. Smaller children had been moved to the countryside.

LIFTON: Adolescents and some younger children, school children in any case.

> "She was quite all right for awhile but on the 4th of September she suddenly became sick... The symptoms of her disease were different from those of a normal disease... She had spots all over her body... Her hair began to fall out. She vomited small clumps of blood many times. Finally, she began to bleed all over her mouth. And at times her fever was very high. I felt this was a very strange and horrible disease... We didn't know what it was. I though it was a kind of epidemic, something like cholera. So I told the rest of my family not to touch her and to disinfect all utensils and everything else she used. We were all afraid of it and even the doctor didn't know what it was. After ten days of agony and torture she died on September 14th. I thought it was a very cruel thing that my daughter, who had nothing to do with the war, had to be killed in this way."

There are many theses in this statement. The first and perhaps the most important is the bizarreness and the strangeness and the

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*The quotation is from Robert Jay Lifton. 1963. Psychological effects of the atomic bomb in Hiroshima—The theme of death. Daedalus. 92(3): 473.

sense of the grotesque which they felt with these symptoms. The second was their feeling toward their family members who were dying, of not only intense concern and anxiety, but also, in a sense, a desire to push them away, to protect themselves from this "epidemic" that they were spreading—to protect themselves from the dead and the dying, —which is always the feeling of any population under such conditions.

The grotesque way of dying becomes extremely important. The whole subject of the way in which one dies is very important in Japanese thought and in all thought, and one of man's most fundamental fears is the fear of premature death, of unfulfilled life, of some sort of humiliating death rather than a calm death at an appropriate time after a fulfilled life.

ABRAHAMSON: Isn't there one other point in that her father says that it was a cruel thing that she had to die. Doesn't this imply that they were and still are upset by the fact that we didn't use this weapon in a nonweapon sense, that is, as a demonstration outside a populated area?

LIFTON: Of course, there is a lot of that feeling in Hiroshima, and it was often brought up to me. But I am not sure that it was included in the statement he actually made. It is a more general kind of statement. It was cruel fate and it was cruel of the forces that caused her death, which included the Americans. It was not a direct expression of hostility but, on the other hand, people who lost children like this often tended to retain a certain dimension of bitterness of which they could not rid themselves. This particular man, whom I remember very well, told me in no uncertain terms that he could never forgive Truman for using the bomb, that he considered him "a cold-blooded animal," and that he or his family would experience some kind of retribution. And all this was, of course, related to his daughter's death.

WARREN: Wasn't the problem here that they thought that she had gotten by with it?

LIFTON: Yes.

WARREN: She had a few superficial lacerations but nothing else. Then suddenly this hidden thing cropped up with a prolonged period of illness, and she died with everybody there to witness all the agony

and the difficulty and with the uncertainty whether it would spread to them. This is the difference between an ordinary kind of a weapon and this one.

LIFTON: Exactly. Very quickly people began to form a hazy but still emotionally powerful image of the weapon that had hit them, and the image, as you see from the sequence I have described, was of a weapon, a single weapon which not only could destroy a whole city but which could leave in its wake a poison which could manifest itself at any time subsequently—even after a period of latency in which nothing happened and in a way that was likely to be fatal. This kind of image about something you have gone through has tremendous emotional force.

WARREN: Yes. There is a lot of fear and uncertainty.

FRANK: In the Hiroshima Diary (Reference 61) there is a rumor that the city itself would be poisonous for about 75 years.

LIFTON: Yes. The issue of the rumors at that time in Hiroshima was a very important one in relationship to death imagery. There were three main rumors, although there were others, that people told me about consistently. These three swept the city during the period immediately after the bomb. The first rumor simply held that all those who had been exposed to the bomb within the city would be dead within three years—everybody. The psychological message here is very simple: none can escape the poison, the epidemic is total, all shall die. It is a naked death imagery.

But there was a second rumor which was told to me even more frequently, and I thought with greater emotion. That was that trees and grass and flowers would never again grow in Hiroshima; that from that day the city would be unable to sustain vegetation of any kind. Here, I think, the message was that nature was drying up altogether, life was being extinguished at its source, suggesting an ultimate form of desolation which not only encompassed human death but went beyond it. I think that was the kind of message of that rumor in a culture which had always placed great emphasis upon nature symbolism.

The third rumor, which Jerry mentioned, was that for a period of 70 to 75 years Hiroshima would be uninhabitable; nobody would be able to live there. It is, of course, related to the other two,

but conveys the sense that the city would be deurbanized and devitalized by this one event.

BRUES: That figure of 70, curiously enough, arose from a press release made by a scientist not closely associated with the thing a couple of days after the news came from New York.

LIFTON: I would like to know about that. I have been searching for the source of that rumor for a long time. The Japanese thought the rumor had come from a foreign source.

BRUES: This was a physician or scientist whose name was Jacobson.*

LIFTON: You mean he put it out in association with the military?

BRUES: No. This man, as far as I know, was unconnected and simply felt the impulse to make a press release on the subject.

LIFTON: This rumor appeared in the Japanese papers—and in the Hiroshima papers as soon as it was reestablished—and it was prominently mentioned as having a foreign source.

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*A study of newspapers indicates that Dr. Harold F. Jacobson, a chemist who had worked with the Army Engineers until 1943, and in 1945 had become a popular technical writer, let it be known to the press that the bombed area in Hiroshima might be uninhabitable for 70 years. This release was given out within 48 hours of the Hiroshima bombing, and as far as I can tell, was the earliest statement suggesting that residual radioactivity might be important. At any rate, this news seems to have reached Japan before anything else except President Truman's announcement. On the afternoon of August 8 he was interviewed by some intelligence officers and made a statement watering down the report, and at that time the War Department categorically denied his first statement. It appears that for some time thereafter there was a good deal of confusion in the press between residual radioactivity and direct radiation from the weapon; in fact the latter was generally appreciated only some days later.

UPTON: Can you say anything about the impact of these rumors? How long did they go uncorrected? Did they drive many people out of the city?

LIFTON: That brings up the whole psychology of belief. Within about one month, people began to return to the city. Almost everybody was outside at least the central area of the city right after the bomb because it had been almost totally destroyed. But there was immediately some scavenging in the ruins, and there were people living on the fringes all the time. Between one and three months a great number of shacks went up, showing that by then there was considerable doubt about the rumors. But even as the rumor was being disproven—one could see lots of grass in various places and there were certain kinds of vegetation that grew very rapidly—people still half believed the rumors. They would see evidence in front of them that the rumors weren't true and they would still half believe them, because the rumors were significant not only in literal content but in what they symbolized, as I mentioned before.

So there was enormous variation in degree and duration of belief in them. But gradually over a period of several months they began to lose their hold. One can gauge this from the real estate prices in Hiroshima, as a matter of fact. Of course, they went very low indeed right after the bomb. But later on, after several months, and especially during the period between six months and a year, they began to rise again, as people began to act upon the assumption that the city was going to reappear and again be a city.

WARREN: Weren't there lots of problems with property rights, etc., because the squatters moved in and there were no good records and no survivors?

LIFTON: Very much so. One of the incentives for coming back to the city was to reclaim one's area, one's land, from squatters. But even apart from this there was strong general tendency for people to want to come back—a powerful urge people have to move back to their homes under the most extreme conditions. It has been described almost everywhere.

Another factor was the fascination for the center of the disaster area, which appeared very early and which also appears in all catastrophes. This is related to a general fascination we all have for catastrophe and for accidents. It is partly related to a reassurance

that one is really alive. The more one, so to speak, "receives" the catastrophe and the fact that all of these others died, the more one is reassuring oneself of one's own survival.

BUSTAD: I was interested in another rumor; perhaps it isn't important here. That is the one we talked about earlier, that they did in fact have an atomic bomb and were going to drop it, as announced in your recalling the incident in the hospital.

LIFTON: There were rumors to that effect. There is an extraordinary incident which Dr. Hachiya described in his Diary (Reference 61) which is well worth reading and pondering on. At the low point, just a few days after the bomb, when people were just dying on the floor of the hospital and he and other doctors were still alive, but helpless to do anything about the situation, somebody, who had been in touch with the military, came running in, shouting that Japan had the weapon, that Japan had atomic bombs, and that they had been dropping them on the West Coast—one of them on San Francisco. It looked as though the course of the war was changing. Dr. Hachiya describes the near dead rising in joy, singing the National Anthem—a rather dramatic scene in the hospital in which the dead seemed to come to life.

FREMONT-SMITH: How soon was this after the bomb?

LIFTON: I think it was a few days later; I don't know the exact day.

This suggests many things, one of them that the psychological use of the rumor was to reassert power through identifying with the enemy's weapon and having one oneself. Indeed, it did reassert power, even if momentarily, including almost the power to live.

There were other rumors that America only got the bomb through having stolen it from German scientists, or that the Japanese were in some way involved in the making of the bomb. Again, these were efforts to reassert some sense of pride or power in connection with this overwhelming weapon.

JOHNSON: There is a letter to the Editor of the Bulletin of the Atomic Scientists (Reference 194) that came from Yale and described a large, unused portion of funds allocated to Project Aeropower. The letter stated, in brief, that the Japanese scientists

planned to set up what seemed to have been a pilot operation to concentrate uranium. This letter gave the information that in 1943 the Atomic Nucleus group in Tokyo was apparently ordered to attempt the making of an atomic bomb. The laboratory was burnt during an air raid over Tokyo early in 1945, and the allocation remained largely unspent.

WARREN: It was Nishina, wasn't it?

JOHNSON: I don't remember.

WARREN: He had a cyclotron, but this was engaged in physics experiments and not in this other business.

LIFTON: There was talk. I quoted a physicist before, and he turned out to be the ranking surviving physicist in Hiroshima, because his senior colleague was killed by the bomb. I discussed with him any prior sense among Japanese physicists of an atomic bomb or of anybody having one. His feeling was that, like most physicists throughout the world, they knew it was theoretically feasible, but they thought that in a practical sense it was absolutely impossible. Occasionally, they did talk about it among their colleagues.

He had two sources of disturbance about his role right after the bomb fell in Hiroshima. One was that his senior colleague died—and another colleague too—and he couldn't help enough. He had great guilt over what he did or didn't do in connection with them. But, second, he was consulted almost immediately after the bomb fell by the local authorities in Hiroshima, including military authorities, before the thing had been fully coordinated with the Tokyo authorities, and he was asked whether this was an atomic bomb. And he said, no. They must have had some information from somewhere, maybe from the shortwave radio broadcast, that an atomic bomb was used, and he later felt humiliated over the mistake that he made.

WARREN: They didn t have electroscopes handy so that Nishina had to bring his own electroscope, as I understand.

LIFTON: In any case, this does illustrate the extraordinary confusion even among people who were knowledgeable. The capacity to act upon even accurate knowledge was impaired among scientists

in Hiroshima. First of all, there wasn't enough understood about the symptoms and about the conditions for even the best informed to know exactly what to do. Nor are the best informed even now immune to various emotions concerning fear of aftereffects that would not seem to be in a literal scientific sense "justified." So these emotional reactions, frequently from the beginning and indefinitely, transcend logical knowledge. The logical knowledge can be helpful in its effect on a population, but it doesn't eliminate these emotional reactions.

There were other rumors, which we need not go into, about what would happen after the bomb. There was the fear that some other new kind of weapon would be dropped, that new poison gases or various kinds of burning oil would be used. Or else that now that America had dropped—this was a sort of a folklore at the time—a terrible "hot bomb," they would next drop a "cold bomb" or an "ice bomb" that would freeze everything so that everyone would die. These rumors reflected the idea that the atomosphere had been so totally disrupted, and the Americans had such unlimited disruptive power, that anything was possible from that point.

At this second stage, then—the second encounter with death—the dominant psychological theme is that of a fear of epidemic contamination to the point of bodily deterioration (of the kind that I have described), and a sense of individual powerlessness in the face of an invisible, all-enveloping, and highly mysterious poison. There was also the sense, often largely unconscious or else indirect communication, that this total contamination—contamination which seemed to be limitless in time and space—must have a supernatural or at least a more than natural origin. It must be something in the nature of a curse with which one's group is afflicted as a punishment for some form of wrong-doing that has offended the supernatural forces which control life and death.

When I say this, of course, I would emphasize that the Japanese were quite aware that this was a bomb that was used in warfare, that it was a weapon based upon physical laws. But at the same time their emotional responses included these images of supernatural dimensions. This sense of retribution for some earlier form of evil is, incidentally, the kind of reaction that any group may have in response to extreme catastrophe. In fact, it still occurs in ordinary people anywhere in response to illness. One was in some way bad or evil and therefore one was struck down. It is an almost

innate idea of cause and effect in response to bodily attack or insult. But among the Hiroshima people it was sometimes expressed in Buddhist imagery, that one had done something in a previous incarnation and now one was being punished or one's family was being punished.

WHITE: Did any of this ever become connected with Pearl Harbor at all, with bombing Pearl Harbor?

LIFTON: You mean in terms of the retribution?

WHITE: Yes.

LIFTON: Yes, I would say it was and sometimes almost directly Many brought up problems of Japanese responsibility in initiating the war, including Pearl Harbor. And their feelings about their military leaders, which we referred to earlier, could to some extent be connected with this sense of retribution, as would their association with Japanese militarism, with Hiroshima's history as a very military-oriented city, not only at this time but decades before, going back to the end of the previous century.

The third enounter with death was associated with later radiation effects, not months but years after the atomic bomb itself, and can be summed up in the scientifically inaccurate but emotionally charged term "A-bomb disease."

I suppose it really is worth a word about the history of the term A-bomb disease. Nobody can really give the history with full accuracy, but it is a kind of abbreviation of the early terminology that was used for "atomic bomb disease" or "atomic bomb injury" during the first week after the bomb fell.

There is disagreement about this, about where the term came from. It was certainly used by Japanese military physicians rather early. Some thought it came from Tsuzuki's lecture, but some said it came earlier than that, from other physicians. There is even a question as to what Tsuzuki meant, because the word shō, which is part of genbakushō (which is part of the Japanese term for A-bomb disease) can mean either "injury" in a specific surgical sense or "illness" or disease in a more general sense, depending on the way it is written. Some doctors thought that Tsuzuki meant it in the first way because he was originally a surgeon, but others felt he

meant it in the second way. In any case, it later took on the second meaning, as disease in general, which is the way in which it has evolved.

It didn't become a popular term among ordinary people until much later on. This term wasn't used very widely among the ordinary population until the Bikini incident in 1954. It reestablished intense focus upon the whole Hiroshima situation. It created a lot of complicated issues of one kind or another. But in any case, the term A-bomb disease eventually became very widespread in Hiroshima, and the difficulty about this in a psychological sense is that the model for A-bomb disease eventually came to be leukemia, based upon the kinds of problems surrounding the incidence of leukemia that have been discussed here before.

The symptoms of leukemia also are sufficiently similar to those of acute irradiation—since both, of course, affect the blood-forming organs—that the association could be readily made in people's minds. In terms of psychic imagery, people came to look upon harmful effects from the weapon as a continuous experience in which the symptoms of acute irradiation—and one's sense of vulnerability at that time—have never ceased. One might, it is feared, develop A-bomb disease—often meaning leukemia, not in a specific way but in a vague, fearful way—at any time subsequently. There is a sense of endless exposure to those initial deadly effects—symptoms of purple spots, falling hair, gastrointestinal effects—in the way this is perceived by the population

BRUES: At the time I first knew Tsuzuki, 18 months later, he used the term for the acute radiation syndrome (Reference 195). Whether he included leukemia, which I think had not then been identified as a sequela of bomb irradiation but was known as an effect of radiation, I am not sure.*

WARREN: I don't think so. I think he must have used it because when we talked about it, it was a radiation syndrome, an injury, and not a disease.

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*Dr. Brues added subsequently: A man exposed at Nagasaki developed a marked leukopenia and recovered within a month. Two months later he died of acute monocytic leukemia. Dr. Tsuzuki wrote, late in 1946, "Since then we have not heard of any similar cases, but we shall not assume that this case might be an accidental one." (Reference 195)

LIFTON: I mentioned earlier the idea of an ultimate horror, that is, the sort of image that epitomized all the anxiety and pity and guilt tied in with the experience. The occurrence of leukemia in children became a continuation of the ultimate horror on a kind of chronic basis. It symbolized once more the bomb's desecration of those held to be most pure and vulnerable, the desecration of childhood itself.

ANGEVINE: I would like to ask Dr. Matsumoto to comment on the A-bomb disease. This is the first time I have heard that it was connected with just leukemia.

LIFTON: No. Don't misunderstand me. It isn't connected with just leukemia. What I am saying is that the fear that is contained in the term often takes leukemia as its model. It is used for everything. I am coming to that. It is used for every possible kind of condition and feared condition—from a common cold to acute fulminating leukemia, and everything else between. So it has no precision at all, but the feared model is that of leukemia, which is what gives the anxiety such intensity. That is what I am suggesting. Perhaps I didn't make myself sufficiently clear.

BUSTAD: I suppose there are all sorts of hypochondriacs relative to this disease.

LIFTON: Yes.

MATSUMOTO: It is still a highly emotionally charged term. There was a recent article in the Nagasaki Journal by a doctor at the Nagasaki University School of Medicine, Professor M. Tomonaga, whom some of you know. It points out that the use of the term is harmful in the sense that it adds to anxiety (Reference 196). In the early days it was used to define leukemia. It is now used to include cancer, liver diseases, and a wide range of disease.

UPTON: Genetic effects too?

MATSUMOTO: It has no clear medical definition, but the newspapers keep utilizing the term. Every death of a survivor is said to be caused by the atomic bomb disease, whatever it may be, although often the deceased may be 80 or 90 years old.

UPTON: Does this extend to effects on an offspring?

MATSUMOTO: The term has not been used widely for offspring.

WHITE: Not yet.

MATSUMOTO: Not yet.

LIFTON: The fear of impairment to offspring is very great. The term A-bomb disease, as Dr. Matsumoto says, seems to be applied more to threats to one's own body, to the survivors themselves, but it is used for every possible condition; and, of course, standards vary among Japanese physicians and among journalists concerning the precision or imprecision with which these things are described.

JABLON: Isn't it true, Scott, if the child of a survivor were to die of leukemia or another form of cancer, that there would be an excellent chance that the newspapers might refer to this?

MATSUMOTO: Yes.

JABLON: As A-bomb disease?

LIFTON: Yes.

WARREN: Does the Buddhist have the same backgound as the Christian ethic has, that the sins of the fathers are passed down to the sons and the subsequent generations, so that this would be a factor? If they were sinning and therefore the atom bomb was...

LIFTON: There is an enormous sense in East Asian culture, in general, of generational continuity and of one's responsibility to past and future generations. The Confucian ethic strongly emphasizes one's posterity. Whatever one does that is good or bad is going to be passed on to one's posterity. This would not be exactly the same as the sins of one's father, but it might be equivalent.

UPTON: I think that the question that I was trying to ask is, does A-bomb disease imply a subtle form of impairment which is not necessarily overt in obvious sickness?

LIFTON: It implies that and everything else as well. In other words, it means that anybody who has been through the bomb is susceptible to exactly what you mention.

UPTON: But by susceptibility one again implies the susceptibility to the occurrence of some overt sickness rather than the existence of a latent impairment.

MATSUMOTO: I recall conversing with a local Japanese reporter, back in 1959, who said that if a survivor was killed in an airplane crash he would still have died from atomic disease in the newspaper report.

LIFTON: That may be so, but it gives you a sense of the situation created by the bomb. I talked with a lot of physicians in Hiroshima because, as part of the whole problem of so-called A-bomb disease and the psychosomatic issue of bodily fears, I was interested in the way in which the doctors themselves responded. It was a rather revealing problem to look into, although a complicated one. Doctors in Hiroshima varied enormously in their use of the term A-bomb disease. It was used by doctors, although the closer a doctor was to scientific terminology the less he tended to use it. I felt that doctors fell into four general categories.

The first was what I called the all-embracing concept of A-bomb disease. In this category were doctors who felt that everything might be A-bomb disease and in fact everything that a survivor had should be looked upon as related to the A-bomb. They could justify their position by saying that, after all, these people went through a catastrophic experience, and if they got heart disease, the experience might have affected them in some way, and so on. They include not only leukemia but other conditions in controversial areas which some doctors in Hiroshima associate with A-bomb aftereffects, such as various anemias, accelerated aging, impaired growth and development, etc. All these they would accept without question as connected with A-bomb disease, as they would other conditions usually considered to have nothing to do with the atomic bomb.

ANGEVINE: As you talk about A-bomb survivors, do you use A-bomb disease as a disease yourself?

LIFTON: No.

A second group had what I called a moderately inclusive concept. They could include leukemia and cancer, and usually the controversial conditions of anemia, impaired growth and development, and so on.

There was a third group whose approach was that of skepticism. These doctors felt that the term A-bomb disease was misleading, would accept only leukemia and possibly cancer as A-bomb related, and had great doubt about everything else.

Finally, there was a fourth category, outright rejection. Doctors in this category would want to dismiss the entire concept and the term A-bomb disease, would insist upon very clear-cut statistical evidence before saying anything was related to the bomb, and would raise certain theoretical issues about its importance for leukemia as well.

Now, I am speaking about a tone of discussion as well as about the content of these general positions.

In the first group were likely to be older Japanese physicians with less attachment to the scientific tradition; they might or might not be survivors themselves. When I talk about various background elements of physicians I don't mean to suggest that any one of them in itself determines a point of view, but rather that many such elements go into a point of view. It is not a simple cause-and-effect relationship but an overall formulation. The attitude of an individual doctor would be partly affected by a sense of guilt toward survivors whom he couldn't help enough; or if not a survivor but dealing with a community of survivors by a Japanese psychological tendency to "merge" with the surrounding community—so that to understate what survivors were experiencing or deny them economic and medical benefits, or fail to recognize their suffering, might be perceived as a way of betraying them symbolically. All of these went into this first position.

If we consider the other categories, as you move toward three and four, you are likely to get younger Japanese physicians more exposed to the scientific ethos, or American physicians were likely to be in three and four. So that you have a generational element, a Japanese-American element, and the element of one's relationship to the scientific tradition. Of course, as you move all the way into four there are such elements as anxiety and guilt and the need to push away the patient and minimize either his suffering or the effects of the bomb in general. All physicians approaching these problems, or investigators of any kind, are to some extent susceptible, within their formulations, to an array of emotions that the whole subject evokes.

FRANK: There is one dimension that you haven't touched on yet, which was called to my attention when I met one of the Hiroshima maidens. She said that of course she could never marry. When you talk of posterity, is this a general notion, that you are contaminated as to having children?

LIFTON: Survivors, especially in the immediate postbomb years, underwent considerable discrimination in jobs and marriage. There was always a kind of rationale given for the discrimination. The rationale in employment opportunities was, because they are survivors they are susceptible to various aftereffects.

There is one symptom that I didn't mention, simply that of fatigue, which is the most common symptom expressed by survivors, and there is great difficulty in tying it in with any kind of definite syndrome.

FRANK: This is apparently a psychic symptom. I have seen it in soldiers suffering from schistosomiasis who didn't seem to recover and who did not have any evidence of infection. Their universal symptom was chronic fatigue (Reference 197).

LIFTON: It looks to be primarily, and possibly entirely, an emotional symptom. But it is persistent and constantly referred to, and is associated by the individual who experiences it and by many if not most doctors in Hiroshima, as somehow connected with the A-bomb.

FRANK: I supposed you might be fatigued for awhile, from the experience you have been through, and stick with it, you know.

LIFTON: Yes. It really can embody, I think, all the psychological elements we have been talking about, and in any kind of combination.

UPTON: I was going to ask about the other side of the coin and perhaps Dr. Matsumoto will speak on this question later. The individual regards himself in a certain way, but how is he looked upon by society?

LIFTON: That is what I was going to comment on in terms of Jerry Frank's question about marriage and so on. I think the problem has greatly lessened over the years but there has been a

problem. In terms of the justification for discrimination in employment it has been that they are weaker; they have symptoms and they may not be able to work as well. They need more time off.

For marriage the rationale is obvious. Marriages are usually arranged with the help of a go-between, a nakōdo, who negotiates with both families. In the Hiroshima area the fact that someone is a survivor must be taken into account by the go-between. It can be considered a point of considerable magnitude against one, because the main purpose of marriage in East Asian culture has traditionally been the begetting of children and carrying on the family line. If this is thrown in doubt, as it is by psychic perceptions of genetic dangers, then one is not a good marital candidate. But underneath these strongly felt rationales for discrimination, I would suggest, is the profound feeling of a death-tainted population. So you find that in a variety of situations martyrs are honored but survivors are resented. I think this has to do with the feeling among the rest of the population that the survivors are somehow threatening. They are tainted with death and this, of course, is accentuated in a nuclear experience where the whole issue of aftereffects is very much tied in. So their position vis-a-vis the rest of the population is a very important element in the whole thing.

SPEAR: Have you had an opportunity to conduct similiar interviews with non-Japanese survivors? There were some German missionaries involved, I understand.

LIFTON: Yes. One of my research subjects was a German missionary who was still in Hiroshima and with whom I did conduct an interview. Actually it was very useful to be able to do that. But do you have any particular question about it?

SPEAR: Of course, with only one person, this is perhaps a silly question, but I am interested in whether there are different types of responses growing out of different cultures?

LIFTON: Yes. Well, as a general principle, I would say that the kinds of responses I have described occur in any culture and this tended to be corroborated by at least my limited experience with one non-Japanese research subject. His experiences were not fundamentally different; at least nothing that I have said so far was alien to him or would be alien to a Western survivor, or hibakusha. In fact, sometimes he seemed more Japanese than the Japanese themselves.

If we talk about some of the more general philosophical ways of coping with this experience—with attitudes of resignation or "it can't be helped" or it is part of a larger destiny—these were as much or more present in him as in Japanese survivors, except that in his case they were expressed through a Catholic idiom. He expressed other things through his sense of himself as a missionary and through his idea of God. Here we are dealing with what I call formulation of the experience—and I use formulation in a broad sense to include all aspects of psychic form. Perhaps we will get into this later on, but his way of giving form to the experience, in a fundamental sense, was not different from that of the Japanese, nor were his general responses, initially and in terms of later anxiety—the various stages that I have described. He in fact presented a rather complicated problem, having had what appeared to be physical after-effects at the time of the bomb and then later a constellation of symptoms in which the physical and psychological became hopelessly intertwined. So all these things, I think, apply in a universal way.

Let me just note that we can and will say a lot more about these things, especially about the psychosomatic features. But I want to ask Scott Matsumoto to talk about the general social or psychosocial patterns in Hiroshima, and possibly Nagasaki too. Then we can continue with what he suggests to us and perhaps come back to anything else as we see fit.

FREMONT-SMITH: I would like to say a word at this point rather than wait until the very end.

It is obvious that in this Conference we could not possibly do justice to any one of the topics which have been assigned. I might remind the group as a whole that there will be four more conferences, that about half the group has been invited from the beginning to attend all five conferences and the remaining half has been invited as guests for this Conference. There will be another group of guests invited to each of the subsequent conferences, some of whom may be some of the same guests that are here now. You can't guarantee that you won't be included again. We hope you will be. Our two cochairmen will have to make this decision.

Obviously we haven't been able to fulfill the mission of the Conference. We may very well fail to fulfill it by the time we have held all five conferences. But we welcome and need your comments, suggestions, and critiques of those things which you feel could be

handled better or more deeply or need to be given greater emphasis in the remaining conferences.

I think it is only in this way that we will hope to come as close as possible to doing some justice to the really tremendously difficult and important topic which has been assigned to us. So with that, I think we should go right on with the Conference.

SOCIAL IMPACT ON HIROSHIMA AND NAGASAKI

MATSUMOTO: What has been the social impact on the communities exposed to atomic bombing? In Hiroshima and Nagasaki there is still a certain level of anxiety, but the evaluation of social trends must primarily gauge the effect and influence of mass media on both these cities continuously for the past 20 years rather than emphasis on individual psychology. But how does one measure the extent of such social effects?

What I propose to do here, because of time limitation, is to briefly review the major events which have occurred in the two cities since 1945. It is a practical rather than an analytical method. This description should help us to appreciate the cumulative effects of such activities which are continuously played up in the mass media of communication. This in turn perpetuates and intensifies the air of anxiety among the survivors.

As discussed this morning, in 1945 the rumor became widespread that nothing would grow in Hiroshima for 70 years. In September, 1945, Dr. Tsuzuki from Tokyo University began examination of patients. The Joint Army-Navy Commission, with Colonel A. W. Oughterson as Chairman, was organized in 1945.

In 1946 the five-man commission, which included Paul Henshaw, Austin Brues, Melvin Block, James Neel, and Frederick Ullrich, visited Hiroshima and Nagasaki.

In March, 1947 Dr. Jim Neel opened an office at the Hiroshima Red Cross Hospital, which was the actual beginning of the ABCC. In December the Emperor himself visited Hiroshima to extend his condolences to its citizens.

No major events in 1948.

In 1949, the Crown Prince visited Hiroshima. The ABCC began its medical research work at Ujina, while construction for a more

to note that the establishment of the ABCC itself added to the anxiety of the survivors with the question, "Just what is it that ABCC is looking for?" Norman Cousins started the "morally-adopted children" movement in September of 1949.

1950 was the year that the Japanese National Census, at the request of the ABCC, conducted a nationwide supplementary survey on the survivors of the atomic bombs in Hiroshima and Nagasaki. This is the basis for our research samples in both cities.

UPTON: How active did Norman Cousins' movement ultimately become?

MATSUMOTO: He came back at least four or five times to the city and has encouraged people in the United States to "morally-adopt" orphans. In other words, to extend financial support for a specific child in the various orphanages in Hiroshima.

UPTON: Have many children been affected in this way?

MATSUMOTO: Yes. I would say perhaps about 100 or so. A few years back the city presented Mr. Cousins with a certificate of appreciation.

Until 1951, because of the GHQ Press Code, the Japanese press had not been allowed to publish freely on the atomic bombing or its aftermath. After the signing of the Japanese-American Peace Treaty in San Francisco in September, 1951, items such as A-bomb disease, as discussed earlier, began to appear increasingly in the newspapers. In December the Hiroshima Medical Association sponsored a conference on the effects of the atomic bomb, emphasizing leukemia. Dr. Tsuzuki was, of course, an important organizer of this meeting. Also in 1951 Dr. Herman Mueller, the Nobel Prize Winner in medicine, visited Hiroshima. Earl Warren, then the Governor of California, also visited Hiroshima. Some of the important visitors to the city will be mentioned because such visits impressed the people of Hiroshima of worldwide interest and concern.

In January, 1952, Hiroshima City conducted a survey on deaths due to the atomic bomb. In August, the Cenotaph was unveiled, which is a memorial to the deceased and contains the names of over 57,000 persons reported killed in the atomic bombing. This is the year after the signing of the Peace Treaty and Asahi Graph, a pictorial weekly magazine, ran a special issue on August 6th depicting the horror of the survivors at the time of the bombing. Helen Keller visited Hiroshima

in September. A movie called "The Children of the A-Bomb" proved popular nationally.

Then in 1953, eight years after the bombing, in January the Gentaikyō, Hiroshima City Atomic Bomb Casualty Council, was established. This was an organization composed of civic leaders and local physicians who recognized the need for special research and treatment. Twenty-one local doctors began to give special attention and treatment to the survivors. The president of this organization was Mayor Hamai.

FRANK: You mean that up to this time the survivors hadn't been getting any special treatment?

MATSUMOTO: This is the first time the survivors received special group attention, as I say, eight years after the bombing. There was another movie entitled "Hiroshima," and Eleanor Roosevelt came in June of that year.

The year 1954, as noted earlier, was the year of the exposure of the No. 5 Fukuryū-Maru (Lucky Dragon) to the hydrogen bomb at Bikini in March. In September, one of the fishermen, Mr. Kuboyama, died, adding to the furor created by this incident. I believe that Dr. Morton, then the Director of the ABCC, went to Tokyo to examine the patients, and a lot of publicity, much of it unfavorable to the Commission, was published at that time.

UPTON: By the "Commission" do you mean the Atomic Bomb Casualty Commission?

MATSUMOTO: Yes. At that time, because of this incident, there began...

UPTON: May I interrupt, please?

MATSUMOTO: Surely.

UPTON: I am not sure I understand why the ABCC was singled out for unfavorable publicity. Were they identified with it? To what extent is Dr. Morton responsible for the detonation that affected the fishermen?

MATSUMOTO: I believe that the Japanese felt, since the ABCC was in Japan to study atomic illness, that they would like an American radiation specialist to examine the fishermen who were exposed.

LIFTON: To answer that question you would really have to consider a long-standing social problem; that is, the currents surrounding the ABCC in Hiroshima and the qualities of ambivalence we talked about before. I am not sure this is the time to go into them. Perhaps Scott should finish his general sequence first. But the thing doesn't admit of a simple answer, I think.

BRILL: I think that there were some military people also attached to the ABCC at the time who put on their uniforms and went to the Tokyo area to see the survivors. I gather that officers of higher rank or experience might have been better accepted, even though the persons involved were very competent. Some superficial protocol mistake of this kind was of some importance.

FREMONT-SMITH: Wasn't there some difference in judgment as to the nature of the illness suffered by the men on this ship?

BRILL: Yes.

FREMONT-SMITH: That reacted adversely?

BRILL: Yes.

FREMONT-SMITH: Perhaps we all bring up too much.

LIFTON: It just depends upon whether one wants to discuss it now. Apparently there was one point of view, and I am not sure that the situation will ever be entirely clarified, that the condition might have come from transfusions which were given at the time as part of the treatment. There was a national furor and outcry about this.

Of course, it also involves the complicated question about a research organization in Hiroshima which gave no treatment, and which helped to create the Japanese imagery of being used as guinea pigs, although that original decision had involved lots of factors, including a reluctance on the part of Hiroshima physicians to have American physicians doing treatment in Hiroshima. So I think that any such incident brings up a whole realm of feelings which include those surrounding the bomb itself, which then became reactivated, and any "American presence" in Hiroshima is likely to become the recipient of such feelings.

FREMONT-SMITH: Reactivated and projected.

LIFTON: That's right.

MILLER: Wasn't it also true that the American delegation thought that the exposed sailors were not seriously affected by their exposure and the Japanese thought they were?

FREMONT-SMITH: Yes.

MATSUMOTO: This kind of statement in the newspapers created comments about the Commission itself.

This incident started a signature-collecting campaign against the atomic and hydrogen bombs by the various women's groups in both Tokyo and Hiroshima during July and August of 1954. This was the commencement of the "Ban-the-Bomb" Movement in Japan, and it is becoming accelerated as the years go by. In 1954 the Gentaikyō, the local medical and civic group, established an examination clinic in the Citizens Hospital. Before that, they had an office in the City Hall, but they actually went into a hospital setting so that more adequate treatment can be given.

FRANK: There was something called the Japanese League Against the A- and H-Bombs. Is that the one that has become communist-dominated? This is where it started, though?

MATSUMOTO: Yes; this is where it started.

There was another motion picture called "The Record of the Living Beings," a poor translation of the Japanese title, which is about Hiroshima and the survivors. Also, Joe DiMaggio and Marilyn Monroe visited Hiroshima.

1955 was the year that 15 "A-bomb maidens" went to Mount Sinai Hospital for surgery. Much publicity was given to the girls prior to their departure. This was the year that the First World Congress Against the Atomic and Hydrogen Bombs was held in August. The official Japanese name of the group is long so it has been abbreviated to Gensuikyō, "Japan Council Against Atomic and Hydrogen Bombs." The organization is headed by Professor Yasui of Hosei University in Tokyo. The conference was first held in Tokyo and then by the A-bomb anniversary date the group moved down to Hiroshima and later to Nagasaki. This year was the opening of the Hiroshima Peace Memorial Museum and Nagasaki's Cultural Building which contains the A-Bomb Memorial Museum with the Peace Statue installed in Peace Park. Dr. Hachiya published his Hiroshima Dairy.

In May, 1956, the Hidankyō was organized by the survivors themselves. The survivors felt the need to push more intensively for their own benefits, and proceeded to create an organization whose membership was limited to survivors. Its official name is the Hiroshima Prefectural Council of A-Bomb Survivors Organizations. Each year after a conference, a delegation heads for Tokyo to persuade Diet members to pass bills designed to benefit survivors.

In September of 1956, the Atomic Bomb Hospital was established in Hiroshima, and the Gentaikyō, the medical-civic group, established its new offices within the Atomic Bomb Hospital. The Atomic Bomb Hospital was built through donations made by the Japanese public by purchasing New Year postcards. The regular seven yen per card was paid plus one yen extra which went toward the construction of this hospital. The donations amounted to approximately $200,000 for that year. In August the Second World Congress Against the Atomic and Hydrogen Bombs was sponsored by Gensuikyō. Another movie, roughly translated as "Glad To Be Alive," was released.

1957 was the year of the enactment of the Atomic Bomb Survivors Medical Treatment Law by the Japanese Diet which assured free medical examination and treatment to the survivors. This was the result of the efforts of Gentaikyō and Hidankyō, both civic organizations, and the survivors working together on the Japanese Diet. Under the law the Atomic Bomb Casualty Section was established in the City Hall in April for administrative purposes. In June, the Atomic Bomb Hospital was also built in Nagasaki. In Hiroshima, Mr. and Mrs. Ira Morris of France, members of the International Pen Club, visited Hiroshima to establish the Ikoi-no-ie, a rest home for the older survivors. Prime Minister Nehru and his daughter, Mrs. Indira Gandhi, visited Hiroshima in the latter part of the year.

By 1957, at the Third World Congress held by Gensuikyō, quite a squabble occurred among the delegates because the large Soviet Communist donations had not been spent for the direct welfare of the survivors but for the movement itself. The survivors charged that the money had not been spent for their medical treatment. A new movie, entitled "The World Is in Fear," showed two-headed goldfish swimming across the screen, adding to the anxiety of survivors.

In 1958 a statue was dedicated in Hiroshima to the children exposed to the atomic bomb. A young girl, Sasaki Sadako, believed that if

she folded 1,000 paper cranes, she would recover from her ailment. In Japan the crane is a symbol of good luck and long life. She died in 1955 before she was able to finish folding 1,000 paper cranes. This led to the organization of various orizuru, young peoples' groups. They helped to keep Peace Park clean, folded paper cranes to present to patients in the Atomic Bomb Hospital, and carried on other such activities.

A movie entitled "Thousand Folded Paper Cranes" was produced. This was the year when, in June, Dr. Earle Reynolds, who formerly worked at ABCC, entered the Bikini area in his yacht. This was given much publicity in Hiroshima. In August the Fourth Gensuikyō World Congress was held and in December United States Ambassador MacArthur came to Hiroshima.

Up to 1959, each death at the Atomic Bomb Hospital was numbered. The newspapers would start from January first and report "Mr. So-and-so, No. 1 death at the Atomic Bomb Hospital," then No. 2, No. 3, and so on throughout the year. This, of course, became a source of great anxiety among the survivors. Finally, the Board of Directors, headed by Mayor Hamai, of the Atomic Bomb Hospital, decided to firmly request the press to stop numbering deaths that occurred at the hospital. In 1959 also the Hiroshima Prefecture, Hiroshima City, and Gentaikyō held its First Medical Meeting on the Late Atomic Bomb Effects. This became an annual event. The Fifth World Congress Against Atomic and Hydrogen Bombs showed intense political involvements. Much activity was exhibited by the right-wing groups against Gensuikyō. The right-wing people came into the conference and threw water and paste at delegates. In the commotion, the leftist British and West German delegates finally withdrew from this conference. The Liberal-Democrats in Hiroshima refused to participate in the meeting. The Nagasaki Atomic Bomb Welfare Center was established that year. Also the movie "The No. 5 Lucky Dragon" was released as was "Amour Hiroshima," a film that some of you, I am sure, have seen.

In January, 1960, the Gentaikyō of both Hiroshima and Nagasaki held a combined meeting in Hiroshima to exchange views on their mutual problems and policies. This was the year of strong movements against the revision of the United States-Japan Security Pact, and the Kishi Government fell. The Sixth World Congress was held again in August with more leftist activities and with the American delegates finally withdrawing from the meeting. The Crown Prince of Japan attended the A-Bomb Memorial Services in Hiroshima.

Much appeared in the papers about the research laboratory on utilization of the hot springs bath treatment at Beppu City for survivors. The Japanese felt that the spas offered physical relaxation to the survivors, and this is understood as a form of treatment. Discussions began to appear in the papers of the social problem of the "A-Bomb Solitary Aged," the oldsters who had lost their families and had no place to go. At one time, the Atomic Bomb Hospital became filled with many elderly people who actually were not ill but had no place to go. There was partial revision of the Atomic Bomb Survivors Medical Treatment Law which extended medical treatment and established an allowance system.

In 1961, the Gentaikyō in Hiroshima constructed the Atomic Bomb Survivors Welfare Center. The Japanese Government established the Research Institute for Nuclear Medicine and Biology at Hiroshima University with eight departments and a personnel of 120. In June, Professor H. Yugawa of Kyoto University, and the President of Tokyo University got together with others to form a seven-man Committee for Appeal for World Peace. United States Ambassador Reischauer visited Hiroshima in October. The Seventh World Congress Against Atomic and Hydrogen Bombs was held in Tokyo and then later in Hiroshima and Nagasaki. The feeling increased that Gensuikyō harped only on the Americans and the Europeans without any reference or statements on the Russian or the Chinese tests. So there was a splitting off of a No. 2 Gensuikyō which was called the Kakkin, the National Council for Peace and Against Nuclear Weapons. This group, headed by President Matsushita of Rikkyo University, opposed the left-wing bias of Gensuikyō and was clearly anti-Communist.

In February, 1962, the Hiroshima-Auschwitz Peace March was organized by a Buddhist priest and three university students who planned an eight-month "crusade" to Poland from Hiroshima. There was also a "Peace Pilgrimage" in March headed by Mrs. Earle Reynolds and two survivors who would go on to the United Nations and later to the Geneva Disarmament Conference. The counterpart of the Research Institute of Hiroshima University was established within the Nagasaki University School of Medicine as the Atomic Disease Institute. Further revision of the Treatment Law extended coverage to the distance of 3 km from the hypocenter. The Eighth World Congress and now the Second Kakkin Conference were held. Another movie, "Cannot Forget that Night," related the sorrowful tale of a beautiful bar hostess in Hiroshima with an ugly scar on her back who feels that she cannot marry a handsome journalist who had come to Hiroshima looking

for a story. Time magazine printed "The Tale of Two Cities," comparing the reactions of Nagasaki and Hiroshima. Hiroshima was presented as a self-advertising city and as a stark symbol of A-bomb's inhumanity, whereas Nagasaki was a spirit of forgiveness. This was the May 18th issue. I have passed around the travel brochures, and you can see for yourself how each city presents the atomic bomb experience in its official publications.

In 1963 the Ninth World Congress was held, but there was further splitting of the delegates within the conference. Now the Socialists and the Sōhyō trade union delegates left the conference. In December the Tokyo District Court handed down the decision that atomic bombing was a violation of international law, but the five plaintiffs from Hiroshima and Nagasaki could not demand payment of compensation from the Japanese Government. There was also much in the press about the problem of having the United States nuclear submarine coming into Japanese harbors.

In early 1964 Dr. Angevine got into the local papers with the "No Atomic Bomb Disease" statement that he supposedly had made in the United States. This was in February. The second "Peace Pilgrimage" was organized by Mrs. Reynolds in April. For this year there were now three different congresses aimed at the banning of atomic and hydrogen bombs under the sponsorship of three separate political organizations. The Tenth World Congress by Gensuikyō was now strictly under the sponsorship of the Japanese Communist Party and supported the Communist Chinese policies. And the first Gensuikin (this is the No. 2 Gensuikyō) meeting was composed of members of the Japanese Socialist Party and Sōhyō (General Council of Japanese Trade Unions) which supported the policies of the USSR. At one time both groups were waiting at the railroad station as the foreign delegates came in, and each tried to coax attendance to their own meeting. The third conference was the Fourth Kakkin meeting which was composed of the Democratic-Socialists and the Liberal-Democrats with anti-communistic emphasis. Little enthusiasm was now shown by the citizens of either Hiroshima or Nagasaki in these meetings. Thousands of persons arrived in August and they all left soon after. A federation was formed to aid the A-bomb survivors in Okinawa. There was also a movement for the preparation of an "A-Bomb White Paper" in October.

1965 was a special year, of course, being the 20th anniversary of the bombing. There was much commotion about the American nuclear submarine docking in Sasebo in February. In March a Japanese-United

States agreement was reached on the medical treatment of atomic bomb survivors residing in Okinawa, and 13 arrived in Hiroshima for treatment. This year there was much publicity in the newspapers concerning the Kinoko-kai, the "Mushroom Society" which was composed of the parents of the 16 microcephalic children who were exposed in utero. In August again were held the Eleventh World Congress of Gensuikyō, the Second Gensuikin meeting, and the Fifth Kakkin meeting. The Kakkin delegates installed the "Eternal Light" in Peace Park. There were revisions of the Atomic Bomb Survivors Medical Treatment Law in April and again in October which further extended coverage to those persons who entered the city within three days after the A-bombing. In Nagasaki the Obama Hot Spring Rest Center was opened for atomic bomb survivors. With pressures on the government, the Ministry of Health and Welfare stated that it would conduct a national survey on the actual status of atomic bomb survivors in November. There was the formation of the Promotion Committee for Atomic Bomb Casualty White Paper in December.

In 1966, much discussion centered on the question of whether to preserve the Hiroshima Atomic Bomb Dome. The damaged building was badly in need of repairs, but no public funds were available for the preservation of this building. Several years before, Mayor Hamai philosophically thought that rather than to fix it, time would solve the problems. But as the building started to crumble, public sentiment became strong that the dome should be preserved as a memorial. There were again in August the Twelfth World Congress, the Third Gensuikin Congress, and the Sixth Kakkin Congress. Three separate meetings with over 10,000 people in attendance came to Hiroshima and later to Nagasaki. There was by now absolutely no city participation in these meetings.

WHITE: Did they all come at the same time or at different times?

MATSUMOTO: Practically the same time.

LIFTON: They are always held around August 6th.

MATSUMOTO: Yes. In both the Gensuikyō and the Gensuikin Congresses this year the emphasis was on opposition to the war in Vietnam. In one meeting, I understand a professor gave a vigorous speech but did not refer once to the banning of the bomb. His speech was entirely on opposition to the war in Vietnam. By this year there were a series of meetings being held by survivors in other areas of

Japan: Tokyo, Yokohama, and Osaka. In October the Seventh Meeting on the Late A-Bomb Effects, which I have referred to earlier, was held, but it was decided that this would be the final meeting. The reports were getting repetitious and so the decision was made to disband. Announcement was made of the establishment of the Arifuku Hot Spring Rest Center for atomic bomb survivors. Also in 1966 a Home for the Aged of the atomic bomb survivors was established by the City in Ninoshima. There was a new movie "Hiroshima, 1966" and still another movie "Record of Love and Death," which received a cinema award. Much publicity was being given to another new movie released in March, 1967, called "The Song of Chikuma River." The 1966 movie concerns a girl working in a record shop and her love for a boy working in a printing shop who comes down with atomic bomb illness and dies. The newest movie presents practically the same story, but the girl is now a nurse and the boy, a truck driver. One of my Japanese workers brought me a summary of this latest movie from a magazine before I left Hiroshima. According to this movie review, at the end, as the youth expires from leukemia, he requests that the girl disrobe so that "he can have the image of her nude figure burned into his eyes." Perhaps the producer found that, to draw the customers, just the atomic bomb now is not enough.

That is the end of the listing of major events through the years, but I thought perhaps this would give you some idea of the constant pressures on the survivors, and the headlines in the newspapers. Beginning in 1945, the atomic bomb disease was a recurrent topic in the press. Later on, there was a great deal of space devoted to what was termed the "atomic bomb neurosis." The possibility of abnormal births or genetic effects was another constant theme. As the years went by and the situation didn't turn out actually that way, the press turned to other topics, and by 1958-59, there were many comments that many single female survivors were not getting married or that the survivors were unable to obtain jobs. Newspapers also stressed suicides—"Atomic Bomb Survivor Commits Suicide." There were also the numbering of deaths, as I said before, at the Atomic Bomb Hospital.

Into the 1960s the older survivors became treated as a social problem. There were no more A-bomb orphans by then. They had grown to adolescence and had left the orphanages. The focus turned toward the solitary aged. In the mid-1960s the press began to give much space to the microcephalic children. The parents of the 16 microcephalic

children gathered for press interviews because they were very concerned about the future welfare of the children after their own deaths—a problem for parents of all mentally retarded children.

UPTON: May I interject a question here, please? Do the children of the survivors experience discrimination? The survivors themselves, we have heard, labor under a handicap when they apply for jobs, and so on. Do their children similarly encounter these difficulties?

MATSUMOTO: These are all reported as social problems, but the problem, of course, is to evaluate to what extent there is discrimination. Maybe I can get back to this later.

FRANK: There must be a figure on the marriage rate of survivors, for example, for the women.

MATSUMOTO: Surprisingly, I could not find it except for one item found in one of our technical reports issued by the ABCC.

FRANK: Is there a lower marriage rate?

MATSUMOTO: Maybe I can start on that now. The present theme most often seen in the local newspapers is the supposed association of the slum areas in Hiroshima to survivors of the atomic bomb. The poverty-stricken people of the community are connected to survivorship. The number of day laborers who are atomic bomb survivors is reported as large. The impressions on social problems of marriage, occupation, and suicides are noteworthy, but it is very difficult to know the actual facts.

The material I present here are those which I could gather hastily before I left Hiroshima. Table 11 shows the composition of the ABCC Life Span Study and Table 12 the Adult Health Study, because some of the material will refer to these various exposure groups.

Table 13 is on marital status. This is from a report (Reference 198) by Dr. Hollingsworth and Dr. Anderson which looked into the marital status of the Adult Health Study sample in 1958–59, which is almost ten years after the sample itself was established. The female exposure group 1 is within 2,000 meters from the hypocenter with symptoms. No. 2 is the group within 2,000 meters from the hypocenter without symptoms. No. 3 is the group 3,000 to 3,500 meters

Table 11. Composition of ABCC Life Span Study and the pathology study.

Category	Hiroshima	Nagasaki	Total
1. <2,000 meters from hypocenter	21,200	6,600	27,800
2. 2,000–2,499 meters from hypocenter	11,500	5,100	16,600
3. 2,500–9,999 meters from hypocenter	21,200	6,600	27,800
4. Not-in-city	21,200	6,600	27,800
Total	75,100	24,900	100,000

Table 12. Composition of ABCC Adult Health Study.

Category	Hiroshima	Nagasaki	Total
1. <2,000 meters from hypocenter With symptoms	3,428	1,565	4,993
2. Without symptoms	3,428	1,560	4,988
3. 3,000–3,499 meters from hypocenter[a]	3,433	1,559	4,992
4. Not-in-city	3,433	1,559	4,992
Total	13,722	6,243	19,965

NOTE:
[a] For Nagasaki 3,000–3,999 meters.

Table 13. Adult Health Study, Hiroshima 1958–1959. Marital status, percentage distribution by sex and exposure.

Category	Total	Exposure Group			
		1	2	3	4
Male					
Never Married	23.12	17.66	26.14	29.47	20.44
Ever Married: Married	70.46	73.13	66.85	64.48	75.55
Ever Married: Divorced or Separated	1.27	2.24	1.08	1.05	0.80
Ever Married: Widowed	5.15	6.97	5.93	5.00	3.21
Female					
Never Married	15.29	11.40	14.53	18.29	17.59
Ever Married: Married	58.78	57.31	60.43	57.83	59.84
Ever Married: Divorced or Separated	3.60	5.26	4.17	2.17	2.49
Ever Married: Widowed	22.33	26.03	20.87	21.71	20.08

from the hypocenter. Group 4 is "not-in-the city." You will notice that, at least at this period, the percentage of females in group 1 who had not married tended to be proportionately smaller than the other three groups.

LIFTON: Actually, at a conference at Yale, Dr. Hollingsworth stated that he thought that the survivors overmarried. He used that phrase. What he meant by that was that, if anything, the rates of marriage of survivors might be higher than those of the control population. But one must be cautious about conclusions here. In a sense there are several levels of statistics and of experience. There definitely was consideration given to the fact of one's hibakusha status, his or her exposure to the bomb, in connection with marital arrangements. It came up repeatedly during my interviews—the matter being examined by the go-between and looked upon as an important issue. So there were problems of discrimination in marriage. Yet it is quite possible, nonetheless, that an equivalent proportion of the survivor population could marry and did eventually marry.

Another phenomenon described in concentration camp survivors and various survivors of mass catastrophes is a very strong urge to marry and have children very quickly, to reassert life in various ways immediately after the ordeal. I think this could very well be reflected here, too.

UPTON: I would infer then, at the moment, that we don't know whether there is in fact a difference in marriageability among survivors and their children, as opposed to nonsurvivors.

LIFTON: I think that is a fair statement in a certain sense.

JABLON: What do you mean by marriageability?

UPTON: Opportunity in the marriage market.

FREMONT-SMITH: As evidenced by marriage.

LIFTON: You see, another issue is—and this is very hard to gauge in pure statistics of this kind—whom one marries. In other words, is one's marriageability at high levels, relatively high socio-economic levels, impaired? Is one forced to marry at a slightly lower level in terms of the arrangements because this is a kind of demerit, so to speak, in the various evaluations that the nakōdo makes?

FREMONT-SMITH: If there are financial benefits to being a survivor, there might be an advantage to them getting married! [Laughter]

LIFTON: Unfortunately the benefits are rather slim.

FRANK: Do the benefits go to spouses? The figure for widowed, 22 percent, seems very high. Do the women marry older men or does this figure depend on something else?

LIFTON: I just don't know.

UPTON: One can assert that differences do not exist, but the data, such as they are, do not conclusively demonstrate it.

LIFTON: The first question is, can you find objective statistical evidence on marriage rates on this issue? But here is another question. Is there special imagery about the survivor? I would not dismiss this question, because such imagery is very much an expression of survivorhood. They are said to be relatively unmarriageable as compared with the total population. If the marriage rates are not altered, as they don't seem to be, then the thing would be the same kind of anxious response that you get, say, to fear of physical aspects.

I would like to emphasize that all of these are expressions of anxiety that relate to the bomb itself. In any sort of catastrophic event you will get a mixture of responsibility and something less than that on the part of mass media, and the preoccupations of mass media will in turn feed back upon the anxieties of the survivors, which is very much, as Scott has just made clear, the situation in Hiroshima. But one has to see the whole thing as a vicious circle, in which the atomic bomb is a very important part.

UPTON: I guess the question is whether the survivors are to any extent a class of untouchables, as it were; and the marriage rate data give no credence to this hypothesis.

LIFTON: On the other hand, there have been serious conferences and discussions held on exactly that question, because there is a group of outcasts in Japan, the so-called eta or burakumin, and the survivors have been compared with them. So there is a widespread image of this kind surrounding survivors, however difficult to document. And there is some evidence that survivors are at somewhat lower socio-economic levels than other groups of the population. One must also

remember that survivors are only roughly 20 percent of the population in Hiroshima now. So again the image of survivors as an equivalent of an outcast group has been extensively discussed and in this way fed into the general mill of social and group anxiety, which in turn acts back upon the individual psychology of survivors.

WHITE: What do you think started that in the first place?

LIFTON: There is one point in this regard that was not mentioned, in what was otherwise a very complete summary—one datum which has very great importance. In 1949 Hiroshima officially became, by a national Japanese Government designation, the International City of Peace. This designation was by a decision taken in the Diet, a political decision which Hiroshima had sought. It established what I could call a symbolic designation the city has held ever since in its international and national status. It also provided a sizable financial grant from the government which included the money to build various monuments, the Museum and the Cenotaph, and other peace monuments. I would stress that these impulses came strongly from within Hiroshima and subsequently became intertwined with individual, social, national, and international anxieties and concerns that have all become part of this web that we now find. People do indeed make pilgrimages to Hiroshima every year—from England, Denmark, United States, etc.—as expressions of their anxieties about nuclear weapons. In this sense, the Hiroshima experience remains a constant focus for universal fears surrounding the universal danger of nuclear weapons.

WARREN: Didn't they symbolize it, too, by adopting an official peace rose which is planted all over the city, and samples sent around the world?

LIFTON: There have been all sorts of things.

UPTON: The earlier remark about the difference in the response of the two cities comes to mind on looking at the next table on suicides. I am not sure I know how to interpret the data. Probably Dr. Matsumoto intended to discuss them.

MATSUMOTO: The newspapers often give the impression that there are more suicides among the survivor group. Table 14 presents the deaths from suicide from 1950 to 1965 in Life Span Study sample, and these are preliminary overall percentages. Miss K. Fukushima, in the Department of Statistics, ABCC, was kind enough

Table 14. Deaths from suicide in the Life Span Study sample by exposure group, sex, and city, 1950–65.

Exposure Category	Male				Female			
	Population Total	No. of Suicides			Population Total	No. of Suicides		
		Total	Per 1,000	Age-Adjusted		Total	Per 1,000	Age-Adjusted
HIROSHIMA								
<2,000 meters from hypocenter	8,835	40	4.5	4.9	12,503	32	2.6	2.6
2,000–2,499 meters from hypocenter	4,773	24	5.0	5.3	6,748	16	2.4	2.5
2,500–9,999 meters from hypocenter	8,795	56	6.4	6.3	12,473	31	2.5	2.6
Not-in-city	8,295	27	3.3	3.4	11,926	22	1.8	1.8
Total	30,698	147	4.8	4.9	43,650	101	2.3	2.4
NAGASAKI								
<2,000 meters from hypocenter	3,058	4	1.3	1.7	3,744	7	1.9	2.1
2,000–2,499 meters from hypocenter	2,052	8	3.9	4.2	3,090	9	2.9	3.0
2,500–9,999 meters from hypocenter	3,024	20	6.6	6.8	3,718	15	4.0	3.1
Not-in-city	2,870	15	5.2	4.8	3,480	7	2.0	2.0
Total	11,004	47	4.3	4.5	14,032	38	2.7	2.7

to figure the age-adjusted ratios, but they do not seem to differ much. Also, she analyzed the age differences—high at the younger age, down in the middle ages, and then higher at the oldest ages. She compared these with the suicide figures from the National Vital Statistics, and actually the figures for Hiroshima and Nagasaki were slightly lower than the national figures. In Nagasaki the high male rate for the 2,500 to 9,999 meters is difficult to explain. But at least the figures show that there is not an excessively high rate of suicides among those closely exposed to the bomb.

UPTON: The rates appear higher at that distance in all categories, for some reason.

MATSUMOTO: Yes.

UPTON: Do you think the differences are significant here?

MATSUMOTO: Miss Fukushima has done some significance tests. Mr. Jablon, could you please explain?

JABLON: What she said was that in Hiroshima the not-in-city group is significantly lower than the combined exposed groups. But she did not remark that there were any significant differences among the various distance groups. So I suppose there are not. But, as Dr. Matsumoto said, the point that she did make is that all of these rates are a little lower than the Japanese national rates.

LIFTON: I was also interested in the question of suicide. I had a very similar impression. I am interested to see the statistics now, because in looking into and in talking also to psychiatrists and physicians in general about their impressions of suicide, I never found any evidence that suicide was significantly higher in survivors than in other populations. Yet there is again constant imagery in Hiroshima of the survivor who kills himself, often because of fear of A-bomb disease, or after being told that he has A-bomb disease—perceived as a kind of death sentence. So again it is a form of imagery that one should kill oneself under these extreme conditions of fear.

TAYLOR: Do you think there is more imagery among the survivors than among people who are not survivors but, say, are Japanese?

LIFTON: Japanese suicide rates are rather high, as you know, on the international scale. What I felt in survivors was that there was a paradoxical psychological pattern going on which was specifically

related to their experience, therefore, more than in ordinary Japanese. Their preoccupation with death and death imagery was in a sense a way of being able to stay alive. It operated on various psychic levels as a substitute for actually killing oneself. I have not had the time today to convey the full force of survivors' identification with the dead, to the extent of forming a sense of themselves as if dead, or what I call identity of the doomed or identity of the dead. This constant preoccupation with the dead is a way of making their obeisance, of dealing in some measure with their guilt over surviving, and thereby giving themselves a kind of permission to go on living. So, in short, some of the preoccupation with death imagery becomes a psychically useful mode of maintaing one's own life. But I never had the impression of a higher suicide rate, and I found a strong inclination among most survivors to cling to life.

MILLER: Are there any statistics previous to 1950 on suicides?

MATSUMOTO: Not by exposure groups, as far as I know.

LIFTON: I would like to say another word on the Hiroshima and Nagasaki comparison. I think this is a very important subject and one that constantly comes up.

As Scott indicated, the Time magazine article, of 1962, had a somewhat flip approach in the sense that its tone was pretty much that Hiroshima equals bad guys and Nagasaki good guys. "Hiroshima makes an industry of its fate"—that was the phrase they used—"whereas Nagasaki is a monument to forgiveness."

Well, I think it is true that Hiroshima more strongly emphasizes its anxieties and its sense of itself as an A-bomb city; you get many impressions and evidences very much in this direction, including those of the brochures. But one has to think of the differences in the histories and the general identities of two cities to account for this distinction. Nor is the distinction an absolute one; it is only a relative one.

So I would say that in comparing the two there are four psychosocial factors that must be considered. First Hiroshima was the first city to be bombed, which is enormously important. What happened first is, of course, most vivid in terms of what is symbolically expressed later on. Second, equally important and perhaps even more so, Hiroshima was almost entirely destroyed because the bomb fell in the

center of a flat city with mostly wooden structures, while in Nagasaki, as we said before, the bomb fell more toward the outskirts and destroyed perhaps a third or a quarter of the city and the remainder stayed intact. Third, Hiroshima is closer to Tokyo and therefore closer to various movements, either formed in Tokyo or taking much of their impetus and intensity from Tokyo, whether they are pace movements or ideological ones of any kind. And fourth, another important dimension, that of the cities' identities in the past, Hiroshima was merely a provincial capital of no special international distinction or importance, whereas Nagasaki was an extremely important center for interchange between Japan and the West, especially during the 200 years of the Takugama era when the rest of Japan was closed. Nagasaki has had various kinds of imagery about its contact with the West and was even the home of Madame Butterfly.

Thus while postwar Hiroshima has had an identity of little but that of an atomic bomb city, Nagasaki has had a great deal else to call upon. Moreover, Hiroshima was officially made the symbolic A-bomb city by being designated in 1949 the International City of Peace. It amounted to the same thing. Nagasaki at the time was made the International City of Culture, a much more nonspecific and vague designation, which clearly made it not the symbolic A-bomb city. But once all of this has been said, I think, it is a matter of relative intensity, and I think that if I had made the study in Nagasaki rather than in Hiroshima, some of the anxieties and even perhaps some of the psychosomatic concerns might have been relatively less, but only relatively. I don't think these things are absent in Nagasaki.

So in this sense, a fairly absolute distinction like that of Time magazine can be quite misleading.

SPEAR: I was struck with this particular pamphlet on Nagasaki which totally ignores the atomic attack. This surely is deliberate. This would almost be like a chamber of commerce brochure on Manassas never mentioning the Civil War as the outstanding dramatic thing that had happened. Has this led to a kind of hostility between the two cities?

LIFTON: The human condition is such that there has inevitably been rivalry between the two cities. Once the designation of International City of Peace had been given to Hiroshima, there was evidence of a certain amount of jealousy on the part of Nagasaki, which may be why it was given an alternative designation of International City

of Culture. And some people in Nagasaki have a certain sense of humor about this.

When I talked with people in Nagasaki about some of these issues, one of the government officials, who was himself a survivor and had had a particularly intense A-bomb exposure, said to me, "Well, you know how it is. When you have a country fair, first prize is the gold medal and the second prize is the silver medal. We got the silver medal." There is also the element of denial, the other side of the coin, which I think you are suggesting.

My impressions in Nagasaki are very anecdotal. I only spent about a week there and really didn't do any intensive work, but I did do a certain amount of interviewing. There was one physician whom I knew and with whom I spent a great deal of time. He himself was a survivor and he said to me, "Well, we in Nagasaki don't bother with these things. There is too much fuss made about the bomb in Hiroshima. I think we have a more mature attitude here and actually I don't think there is any such thing as aftereffects, perhaps leukemia, but nothing more." He sought to dismiss the subject. But as I got to know him, he gradually revealed that he himself had cut down on his activities because he noticed that he began to fatigue easily. And he had a series of anxieties which he himself rather clearly related to his atomic bomb exposure, as well as recurrent dreams about the atomic bomb.

So what I am getting at is that part of the de-emphasis can be an element of denial of actual fears which are present. One doesn't know whether certain kinds of psychosomatic expressions may be as great in a hidden way in Nagasaki, or even greater, because they are not given individual public expression. None of this is really known because it hasn't been studied on any careful comparative basis.

Another thing is the whole issue—a psychosocial and an ethical issue—of how is an A-bomb city to behave? What are the standards of "proper" behavior for an A-bomb city? I have often raised this question.

FREMONT-SMITH: Or for the rest of the world looking at an A-bomb city.

LIFTON: Yes. The implication about physic health in the Time magazine article is that the healthy thing is to forget it. It has

happened in the past, it is over, so one need not worry about it anymore, whereas the unhealthy thing is to keep on harping on it. That is a judgment of psychic health that might not universally be shared. Obviously, where a continuing preoccupation with one's atomic bomb condition keeps one from an active life and interferes with one's function, then there may be much to be said for such a judgment. But there are certain questionable ethical assumptions being made in all this. And on the other hand, the notion that health lies in total or near-total dismissal (which in fact doesn't exist in Nagasaki either, as I have suggested) is also a very questionable assumption.

SPEAR: So at the very least you get a kind of municipal self-consciousness.

LIFTON: Yes. You get a kind of municipal self-consciousness, which in Hiroshima is much more vivid because almost from the beginning it became, at a local as well as an international level, the symbolic A-bomb city. This sense of itself both emerges from within and is spread from without by anxieties and by various movements that come from the outside world to Hiroshima.

FREMONT-SMITH: Do we have a parallel in the men who were gassed or thought perhaps they had been gassed in World War I and who know that their lungs are rotting away inch by inch and are paid a monthly stipend by the Veterans' Administration to maintain this position? I have seen a good many of them and I am sure many of you have too, still alive, some of whom didn't get even a whiff of gas. But we have, it seems to me, a parallel situation, keeping alive an anxiety—and payment—which prevents them from resuming their place in normal society.

LIFTON: This is very true and there is a whole medico-legal structure in Hiroshima which is extraordinarily complex—a series of arrangements which are economic and medical. They depend upon one's designation either as a general survivor or a special survivor, the latter group including those closer to the hypocenter or those who had more severe experiences. The whole problem inevitably becomes a political issue, which comes up constantly, as Scott indicated, in terms of widening the definitions, of when benefits are available, and for what kinds of conditions for broadening the designation of the special survivor.

UPTON: I was wondering about this last table. Does this bear on that subject, Bob: Do these data tell us something about the kinds of benefits and the exposure categories?

LIFTON: Let me just make a last point and then return to your question. There is a category where one is certified to have an A-bomb-linked condition. Conditions under which one can be so certified include leukemia, cancer, and certain anemias. This means that some survivors, as several Japanese physicians have explained to me, can in a sense be certified for life as suffering from an A-bomb-linked condition. You can imagine the psychosomatic impact and "feedback" of that sort of situation. But this is not simply a matter of greedy people trying to gain more through a form of irresponsibility. It is a part of a mass city response, an anxious response to catastrophe in which recollections of the harmful effects become part of this thing. The city is constantly caught in a psychological and moral dilemma. On the one hand there is the urge to emphasize the effects of atomic bombing for the sake of letting the world know about it, particularly among certain journalistic groups. On the other hand there is awareness of the harm done by this emphasis and by possible exaggerations to the survivors in terms of stimulating anxieties, as doctors as a group tend to point out. So all this is part of the picture to be considered.

Let us go back to that other table and the point you raised.

MATSUMOTO: In the Adult Health Study, medical social case workers are always available in the lobby of the clinic to help any person who has a problem. We did some tabulations to see which exposure group sought the most assistance from the social workers (Table 15). A social welfare fund exists at ABCC from the sum ABCC receives under the Treatment Law. Every time a survivor recognized as such by the law is examined, the Japanese Government reimburses not only ABCC but any hospital for the examination performed. The ABCC director has set this fund aside for social welfare work among the survivors. But according to the table, in both Hiroshima and Nagasaki there is no overwhelming social welfare work being performed for the closely exposed as compared with the nonexposed. In Nagasaki the percentage is higher, probably because the ABCC establishment is in the city close to streetcar and bus transportation, whereas in Hiroshima the facilities are located on a hill involving a long walk for the survivors. Another reason may be that the Nagasaki program is smaller so that proportionately more social case work can be handled.

Table 15. Medical social case work in the Adult Health Study by exposure group and by city, third cycle, 1962–1964.

Exposure Category	Total 3rd Cycle Exams		Medical Social Case Work					
			Medical Care		Social Welfare		Total	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
	HIROSHIMA							
1	2,430	100.0	52	2.1	57	2.3	109	4.5
2	2,338	100.0	41	1.8	70	3.0	111	4.7
3	2,401	100.0	49	2.0	59	2.5	108	4.5
4	2,284	100.0	41	1.8	46	2.0	87	3.8
Total	9,453	100.0	183	1.9	232	2.5	415	4.4
	NAGASAKI							
1	1,118	100.0	61	5.5	19	1.7	80	7.2
2	1,116	100.0	63	5.6	20	1.8	83	7.4
3	1,065	100.0	50	4.7	11	1.0	61	5.7
4	982	100.0	46	4.7	12	1.2	58	5.9
Total	4,281	100.0	220	5.1	62	1.4	282	6.6

FRANK: The social welfare work seems to be less in Nagasaki. It is the medical care that is higher. What does that mean? Actual medical care or medical examinations?

MATSUMOTO: Medical care refers to need for hospitalization, treatment, or some need which would be considered medical rather than a welfare problem. Of course, often both are involved. But each case was categorized into either medical care or social welfare.

FRANK: It suggests that the location of the clinic makes a difference with respect to medical care but not social welfare. You would think just the opposite, really.

JOHNSON: Another factor is that the socioeconomic score is much lower in Nagasaki than it is in Hiroshima.

FRANK: But it shows up in the social welfare figures.

WHITE: This might be a very real difference, though. As I remember the old figures, the mortality rate after the first day following the bombing was 40 percent in Nagasaki and double that in Hiroshima. For Nagasaki the yield was larger, the pressures were higher (References 4 and 17).

FRANK: You mean there was more health damage immediately after the bombing in Nagasaki in spite of more concrete buildings and all the other things?

WHITE: Subsequently the mortality rate among the injured was double.

FRANK: Was it because the bomb was different?

WHITE: I don't think it had anything to do with the difference in design. I think it just had to do with the fact that the yield was larger. There were more people hurt by translational effects and primary blast effects probably. But that figure is in the literature and I don't know whether this is a reflection of it. I haven't any idea. We don't know much about sequelae, Dr. Frank.

MATSUMOTO: Probably the difference lies in the fact that the work emphasis placed by the case workers was different in each of the two cities, but within each city I think it is fair enough to compare the percentage among the four exposure groups.

The other tables are rates concerning two major programs at the ABCC: the autopsy program and the medical program. Just how one interprets these figures in terms of the survivors' attitudes is difficult to state, but for autopsy procurement from 1961 to 1965 (Table 16), a slightly higher percentage of cooperation is indicated by the most closely exposed group. Whether this means the families of such deceased survivors are more sensitive to cause of death, we do not know. The other three charts concern the Adult Health Study. The proportion migrating from the original sample (Table 17) was highest for the not-in-the-city group and higher for Nagasaki than Hiroshima. Also, the not-in-the-city group has the lowest death percentage, and the proportion of deaths is higher in Hiroshima than in Nagasaki (Table 18). Table 19 shows the participation rates among the various exposure groups. In the tabulation, the migrants and the deceased were subtracted from the original sample, as they, of course, could not be approached to solicit cooperation in the medical program. It seems clear that those survivors closely exposed

Table 16. Autopsy procurement in the Life Span Study sample by exposure, category and city.

Exposure Category	HIROSHIMA			NAGASAKI		
	No. of ST-100 Deaths	Procured		No. of ST-100 Deaths	Procured	
		No.	Percent		No.	Percent
1	1,111	628	56.5	278	129	46.4
2	538	275	51.1	231	91	39.4
3	975	391	40.1	256	117	45.7
4	751	341	45.4	244	102	41.8
Total	3,375	1,635	48.4	1,009	439	43.5

tended to have a slightly higher participation rate, especially those with symptoms. Perhaps this can be interpreted as an indication of greater anxiety about their health status.

LIFTON: There has been a lot of evidence, as would be expected from everything we have said, that survivors are preoccupied with their health situation. Various questionnaires have been given them—some Japanese physicians have given the Cornell Medical Index or related indexes to comparative populations—and, of course, the survivors always come out more hypochondriacal and more concerned with their health, and having an endless series of bodily complaints which they tend to associate with A-bomb-linked diseases.

Table 17. ABCC Adult Health Study for three cycles (1958–60, 1960–62, 1962–64) of migrants by exposure group and cycle, sex, and city.

Exposure Category	HIROSHIMA				NAGASAKI			
	Total No. In Sample	No. Migrated (%)			Total No. In Sample	No. Migrated (%)		
		1st	2nd	3rd		1st	2nd	3rd
MALE								
1. <1,999 meters with symptoms	1,316	7.1	7.4	7.8	683	12.9	14.2	16.5
2. <1,999 meters without symptoms	1,310	7.9	7.6	8.1	675	12.1	14.2	17.0
3. Distal	1,309	5.7	6.6	7.0	673	13.8	15.8	15.8
4. Not-in-city	1,316	9.8	10.0	11.0	675	19.6	23.0	24.7
Total	5,251	7.6	7.9	8.5	2,706	14.6	16.8	18.5
FEMALE								
1. <1,999 meters with symptoms	2,116	5.8	6.3	6.9	886	10.3	12.5	13.9
2. <1,999 meters without symptoms	2,114	6.6	7.2	7.7	881	10.4	10.6	11.9
3. Distal	2,121	5.4	6.6	7.1	886	12.5	14.0	15.8
4. Not-in-city	2,117	8.2	9.1	10.0	884	16.7	17.9	19.6
Total	8,468	6.5	7.3	7.9	3,537	12.5	13.7	15.3
TOTAL								
1. <1,999 meters with symptoms	3,432	6.3	6.7	7.2	1,569	11.4	13.3	15.0
2. <1,999 meters without symptoms	3,424	7.1	7.4	7.9	1,556	11.2	12.1	14.1
3. Distal	3,430	5.5	6.6	7.1	1,559	13.1	14.8	15.8
4. Not-in-city	3,433	8.8	9.5	10.4	1,559	18.0	20.1	21.8
Total	13,719	6.9	7.6	8.1	6,243	13.4	15.1	16.7

Table 18. ABCC Adult Health Study for three cycles (1958–60, 1960–62, 1962–64) of deceased by exposure group and cycle, sex, and city.

Exposure Category	HIROSHIMA				NAGASAKI			
	Total No. In Sample	No. Deceased (%)			Total No. In Sample	No. Deceased (%)		
		1st	2nd	3rd		1st	2nd	3rd
MALE								
1. <1,999 meters with symptoms	1,316	10.6	13.1	16.6	683	7.2	8.9	11.1
2. <1,999 meters without symptoms	1,310	11.3	14.0	16.6	675	8.4	9.8	11.3
3. Distal	1,309	10.6	13.5	15.9	673	7.6	9.7	10.0
4. Not-in-city	1,316	8.8	12.2	15.1	675	6.1	7.7	9.0
Total	5,251	10.3	13.2	16.1	2,706	7.3	9.0	10.3
FEMALE								
1. <1,999 meters with symptoms	2,116	5.5	7.2	9.4	886	5.5	6.2	7.0
2. <1,999 meters without symptoms	2,114	6.6	7.8	9.2	881	5.2	5.8	7.2
3. Distal	2,121	6.5	8.3	9.7	886	4.9	6.0	6.5
4. Not-in-city	2,117	4.0	5.5	7.0	884	4.4	5.4	6.3
Total	8,468	5.6	7.2	8.8	3,537	5.0	5.9	6.8
TOTAL								
1. <1,999 meters with symptoms	3,432	7.4	9.5	12.2	1,569	6.2	7.4	8.8
2. <1,999 meters without symptoms	3,424	8.4	10.1	12.0	1,556	6.6	7.5	8.9
3. Distal	3,430	8.1	10.3	12.1	1,559	6.0	7.6	8.0
4. Not-in-city	3,433	5.8	8.1	10.1	1,559	5.1	6.4	7.5
Total	13,719	7.4	9.5	11.6	6,243	6.0	7.2	8.3

Table 19. ABCC Adult Health Study for three cycles (1958–60, 1960–62, 1962–64) of participants in the Adult Health Study by exposure category and cycle, sex, and city.

Exposure Category	HIROSHIMA						NAGASAKI					
	1st Cycle		2nd Cycle		3rd Cycle		1st Cycle		2nd Cycle		3rd Cycle	
	Contacted Total	Percent Exam.	Contacted Total	Percent Exam.	Contacted Total	Percent Exam.	Contacted Total	Percent Exam.	Contacted Total	Percent Exam.	Contacted Total	Percent Exam.
MALE												
1	1,083	90.1	1,045	87.4	995	86.4	546	91.2	525	91.8	494	92.7
2	1,059	89.0	1,028	85.9	987	84.7	536	91.4	513	92.4	484	92.1
3	1,096	86.7	1,045	86.1	1,009	85.7	529	90.4	502	91.2	500	89.8
4	1,071	88.2	1,023	87.9	972	83.8	502	88.4	468	90.2	447	90.8
Total	4,309	88.5	4,141	86.8	3,963	85.2	2,113	90.4	2,008	91.4	1,925	91.4
FEMALE												
1	1,878	90.6	1,830	89.8	1,771	88.7	746	92.2	720	93.6	701	94.2
2	1,834	88.8	1,797	89.6	1,756	85.5	743	91.4	737	96.2	713	94.0
3	1,868	87.8	1,804	89.7	1,764	87.1	732	88.4	709	88.9	688	89.5
4	1,859	86.6	1,807	86.9	1,757	83.6	697	87.2	678	88.2	655	87.9
Total	7,439	88.4	7,238	89.0	7,048	86.2	2,918	89.9	2,844	91.8	2,757	91.5
TOTAL												
1	2,961	90.4	2,875	88.9	2,766	87.9	1,292	91.8	1,245	92.9	1,195	93.6
2	2,893	88.8	2,825	88.3	2,743	85.2	1,279	91.4	1,250	94.6	1,197	93.2
3	2,964	87.4	2,849	88.4	2,773	86.6	1,261	89.2	1,211	89.8	1,188	89.6
4	2,930	87.2	2,830	87.2	2,729	83.7	1,199	87.7	1,146	89.0	1,102	89.1
Total	11,748	88.5	11,379	88.2	11,011	85.9	5,031	90.1	4,852	91.7	4,682	91.4

ATTITUDINAL IMPLICATIONS FOR OTHER POPULATIONS

AYRES: I have a thought which has to do with the implications of this for nuclear war, namely, that although the major weapons effects tend to occur right away and then are over with, the problems that people are going to worry about afterward, which they have the rest of their lives to worry about, may be very small ones in comparison with the whole. In the case of Hiroshima, there is perhaps nothing positive anybody can do in finding a cure for leukemia which hasn't been done yet. But after a nuclear war with fallout, there is, of course the possibility of decontaminating or devising some diets, and so on, to minimize the intake of radioisotopes. It has been pointed out repeatedly and correctly that internal hazards are a trivial problem by comparison to other things. The internal dose that you might pick up even with a lot of megatons at groundburst is not likely to be a major medical problem by wartime standards except for the fact that people will be anxious about it, and it happens to be something where some options remain. I wonder what you think about this notion? How do we get from there to the notion that it is not necessary to decontaminate? I have the feeling that we might spend a disproportionate amount of surviving resources on decontamination, trying to provide special diets, etc.

LIFTON: In other words, on the basis of what we have observed in Hiroshima, whether there might be a kind of preoccupation with decontaminating beyond the medical relevance?

FOUNTAIN: Economic and medical. You have to consider these factors, and the resources may not be readily available for complete decontamination.

LIFTON: Nobody is sure, but I would suspect that is true. I am sure that one could find evidence for that kind of preoccupation even in places other than Hiroshima. Certainly what we discussed in Hiroshima applies, as does the Bikini incident, as evidence in that direction. But even if you think of what happened in Spain recently,

where a hydrogen bomb was lost, the anxieties of the local population seemed to be considerable; then all kinds of other things can get mixed in, political, social, what have you. Even if you come closer to home and consider some of the reactions in urban centers where there has been talk of building some sort of nuclear plant, and the anxieties that are aroused in the population, it would seem that this aspect of radiation, the invisible contaminant, so to speak, has a tremendous capacity to produce anxiety.

AYRES: You might get a kind of pathological preoccupation with removing the irremovable, as with Lady Macbeth.

LIFTON: That is not quite it. I don't know.

JABLON: Have I lost something here? It seems to me that maybe if I interpreted it correctly, we were leading to a conclusion that perhaps there was an undue amount of anxiety in Hiroshima that was not justified by any objective evidence. How do we get from there to the notion that it is not necessary to decontaminate? I have lost the bridge.

AYRES: I brought it up because a common criticism of studies which worry about it is that, in comparison with other problems, it is a trivial one and one should after all, allocate resources as nearly as possible in accordance with the disutility involved. What I am doing is suggesting a prediction that in fact people would, after the event, tend to allocate resources to this problem even though the disutility was perhaps much exaggerated in comparison with, say, automobile accidents, or other things.

JABLON: I see your point. It is plain that this disutility is ex cathedra as far as we are concerned at this time.

LIFTON: I would put the matter in a different way. Let's look at it in still larger dimensions. My own feeling about the ultimate usefulness of this kind of material is less what it would teach one about how to behave if there were another war than in hopefully illuminating some anxieties that are related to all of these weapons and, therefore, hopefully again—and one always has to say that—contributing to some capacity to cope with these weapons.

In Japan there is a tremendous preoccupation with the entire issue of fallout, as you know. So that whenever any country tests—the latest example is China's testing, although I wasn't there to get a sense

of how great the anxieties were—only in Japan, among all countries in the world, are there people who make a point of using umbrellas, of avoiding going out in the rain during times of announced fallout, and who become really concerned about physical harm from fallout with this nuclear testing.

Of course, this has to do with Japan's historical experience as the only country that has been atomic bombed. But I would also suggest, as you are suggesting in another context, that preoccupation with test fallout as such, beyond its relative importance within the overall nuclear weapons problem, is again a kind of displacement of overall anxieties about nuclear weapons. If one is afraid of being annihilated by nuclear weapons, or has all kinds of related fears, one may pick on some aspect of the thing that seems, if not exactly concrete, at least an issue that one can attach this anxiety to.

COURT BROWN: Perhaps there may not be too much anxiety in Hiroshima and the press whips up a good deal of this anxiety. Have you got any feeling for translating this anxiety in terms of morbidity, as presumably there is some sort of equation?

LIFTON: You mean of incidence of disease as an expression of these anxieties?

COURT BROWN: Yes. It is an expression-producing situation presumably which is reflected in morbidity and this in some ways must be regarded as a result of the nuclear explosion.

LIFTON: It has been done to a very limited extent, as in the indexes that I mentioned, where the morbidity becomes mainly a more or less hypochondriacal sort. Whether it will spill over into an actual clinical syndrome is questionable. Apparently, from what data there are, this isn't suggested, because I don't think there is any particular increase in such conditions as, for instance, gastric ulcers or ulcerative colitis, in which one knows that there are important psychic components. It may be that those conditions require the anxiety to have been stimulated much earlier in life to create them.

FREMONT-SMITH: May I comment on what Dr. Ayres said, because he is suggesting, and probably quite rightly, that were we to have a nuclear war, there might not be a wholly rational arrangement of effort.

AYRES: Right.

FREMONT-SMITH: Therefore I go back to a time when I met with a small group that was concerned with economic factors, and made a statement which I liked very much and which I occasionally repeat for my own satisfaction. I will now repeat it: that the laws of emotion were to the laws of intellect as the laws of politics were to the laws of economics; that the laws of the intellect worked extremely well and accurately except when emotional factors entered in, which they always did; and that the laws of economics worked with great accuracy except when the political situation entered, which it always did. I suspect that this would be my answer to you, that the political situation is bound to enter any worldwide situation, or even in a smaller context, and that therefore you must expect irrational behavior in the presence of calamity, and that this is what we have seen on every side, whether it is war gas exposure or semiexposure or fatigue after potential exposure.

So I think that really it is as important to concern ourselves with how we understand and deal with the common phenomena of irrational behavior as to decide what would be rational behavior, which we are not going to get. I think this is the one thing that we know about human behavior—that it is not likely to be very rational.

LIFTON: Especially under extreme conditions.

AYRES: In effect, of course, I don't disagree with you. But we also have the option to plan or to foresee irrational behavior and behave in such a way as to minimize its bad effects.

FREMONT-SMITH: Right. This is just my point.

AYRES: The fact that irrational behavior is bound to come doesn't make it not worthwhile to think about what that behavior may be.

FREMONT-SMITH: No. It is just the reason why we need to focus attention on it, but I don't think we focus attention on it by deciding what would be rational. I think we focus attention on it by seeing what is the form of irrational behavior that we are likely to expect.

TAYLOR: I wonder. There is one form, it seems to me, that did not exhibit itself at either Hiroshima or Nagasaki which would exhibit itself in another bombing, because, as you said several times, right after the explosion no one that was exposed knew about radiation.

FREMONT-SMITH: Right.

TAYLOR: In a new event everyone would, of course. There is a sort of process that took place in Hiroshima that would not in the new case, namely, people who were not killed or wounded who began to show the symptoms of radiation sickness gave the rest of the population some basis for being concerned, but until that happened there was no concern, presumably. Now the situation would be very different Immediately everybody would be concerned about how much radiation they got.

FREMONT-SMITH: And were going to get.

TAYLOR: And were going to get, right. And it seems to me that that puts the immediate stress on the population right after the explosion in a very different form from what it was at Hiroshima.

LIFTON: But it doesn't necessarily help the situation.

TAYLOR: No, no. It may make it much worse.

LIFTON: There is a paradigm in Hiroshima but it is very incomplete at best as a model.

TAYLOR: Is it true that there are really no data from Hiroshima and Nagasaki on this? Is there any detectable change in the psychosocial effect of the radiation once people became aware of the radiation sickness?

LIFTON: That is what I was about to get to. It is difficult to measure in any precise way. I think there are conflicting forces, operating here; that is, on the anxiety-relieving side, in finding out what it is, the universal human need to find a cause and to have some sort of rationale for what is happening to one. On the negative or anxiety-stimulating side is the knowledge that there is indeed an invisible contaminant which has previously unforeseen harmful effects. So that many people I have talked to about just this kind of problem suggested to me that from the latter standpoint, finding out what the thing was didn't in any sense relieve their anxiety. What it seems to have done is to build up a whole new dimension of half-truths—the truth being that radiation is indeed harmful and that there are indeed aftereffects; the half-truths being the enormous superimposition of distortions and exaggerations. I think we could probably anticipate some such pattern if a war occurred now, where there is knowledge

of such things as fallout and radiation. After all, it wouldn't necessarily be a reassuring knowledge nor would the knowledge cover the full implications of the dangers. In other words, it could never be that precise.

TAYLOR: It is a kind of a big leap, but one is tempted to say that this uncertainty about the dose that somebody got might lead to a qualitatively larger amount of irrational behavior right after the explosion and, for example, have an important effect on civil defense measures.

FREMONT-SMITH: Take the Wind Scale accident. Most of you remember the Government's announcement with great assurance that there was no danger to anybody. Now, when the Government does this and then has to back away a few weeks later and kill a few cows or pour the milk in the river, then public confidence in the Government's statement about the danger becomes very much jeopardized and then we get closely into the combination between the political and the truth.

AYRES: I make the prediction that, given the usual amount of bungling and confusion, about two weeks after any attack nobody would have any confidence in the Government.

FREMONT-SMITH: I think that to date, including our own AEC—and I will put this in the record—it is highly likely that this would be the case, unfortunately.

LIFTON: What happens, apparently—again drawing upon the Hiroshima experience for another relevant pattern—is that a pseudoscientific instrument is quickly embraced. For instance, there has been a preoccupation with blood counts in Hiroshima, so that people are constantly having their blood counts taken. They are constantly evaluating their health in terms of a precise figure as though that figure were an exact indication of how healthy they are or are not. Of course, those doctors in Nagasaki and Hiroshima with a real knowledge of hematology are extremely critical of this practice and try to discourage local physicians from in any way taking part in it. But it goes on because the survivors feel they must have some seemingly concrete, semiscientific measurement.

ABRAHAMSON: What would be the effect on the people of a nation who exploded such a bomb? In other words, putting this into "a for

instance," what if America should drop another bomb? What would be the effect on the American population? Suppose that we did it in Vietnam.

LIFTON: I think anybody is guessing when he answers that.

FRANK: We had an experience in the last war. I think that people justify whatever they do. Americans were morally outraged by the bombing of Rotterdam, but before the war was over we carried out much more destructive bombings, including fire bomb raids, and finally atomic bombing, without compunction.

LIFTON: This raises the problem of applicability of the principle of psychic numbing to the larger population. In a second study, I have been interested in the attitudes and feelings of nuclear scientists at the time that they were working at the Manhattan Project and creating the bomb; of the military, especially the pilots, in dropping the bomb; and then of the response in the country in general to the use of the bomb. Without going into it in great detail, there is an elaborate structure of reason for justifying these actions during wartime because wartime itself creates a sort of mass psychic numbing which permits you to do things you would ordinarily not permit yourself to take part in. Psychic numbing becomes an extraordinarily widespread pattern which allows people, through organized group behavior, to do things that they would be strongly opposed to under other conditions.

So one could, on the one hand, presume that, as Jerry is suggesting, a population can increasingly accept the use of extreme measures by its own people, as happened increasingly not just with the atomic bomb, but prior to it. The atomic bomb was a culmination of a wartime pattern moving back and forth between Germany and the Allies which Germany initiated with mass bombing of unprotected populations, and which we adopted, doing the same to the populations in Germany, eventually ending up with the use of the atomic bomb as a means of ending the war.

AYRES: Incidentally, I believe the British initiated all of the escalations in mass bombing. The Germans retaliated but more effectively.

FREMONT-SMITH: Rotterdam wasn't British.

FRANK: I think the British stated the policy of mass bombing first but were not able to do it as well. They made the first attempt at mass bombing. However, that is irrelevant.

LIFTON: I might say that in this kind of study of the atomic bomb, or of nuclear weapons problems, what we really have to get hold of are the phenomena occurring—the psychological or human patterns that take place—and name-calling or designating villains or heroes doesn't really serve our purpose. What does seem clear is that nations are capable of extraordinary degrees of this pattern of psychic numbing, and on a very large scale. But I would also say that it takes a certain amount of psychological work to achieve this psychic numbing, and there are always elements of individuals or groups rebelling against it or questioning it, in complicated admixtures.

COURT BROWN: Everyone seems to have forgotten the town of Groningen, which I suppose was the first one.

FRANK: We would start out with small nuclear weapons after a lot of preparation of the public. I think psychic numbing is too simple a principle here. There are several principles operating. Adaptation is one.

LIFTON: But you are adapting yourself to something that would not be permissible ordinarily, you see.

FRANK: It is a moral adaptation, you might say, but it is the same principle. You just get used to it after a while, you know.

LIFTON: Yes.

FREMONT-SMITH: It is analogous to numbing.

FRANK: But I think there is a more active principle involved which is termed—I can't think of another good word for it—the reduction of cognitive dissonance, that horrible phrase which simply means you have to justify yourself. If you are forced into a line of action by any means, in order to live with yourself you have got to decide it was the right thing to do or the right thing to have done, and you build up a justification for it afterward. It is more than just neutrality; you have to defend yourself. You have got to say, this was right and proper. It is a universal phenomenon.

FREMONT-SMITH: To come back to the question about Vietnam, I think the answer might be dependent upon whether we had formally declared war, because if we had done so, there would be a different attitude toward using certain weapons for a good many of the community than if the effort were made currently.

LIFTON: It isn't only a question of a formal declaration of war. I think the important issue here is that Vietnam is a very controversial war as wars go. It is considered an enormous moral dilemma for our country. It has gotten more opposition than any war in modern history that America has been involved in. World War II was a war that was, by and large, supported enthusiastically by the great majority of the population, a war we considered thrust upon us. Toward the end of World War II, any measure that would defeat the enemy became acceptable, so that there could be more psychic numbing, if I can still use the term in a broad way. Now the numbing would be less effective, so to speak, and many issues would arise.

FRANK: I guess the point I am trying to make is that I don't think the fact that they are nuclear weapons makes a particle of difference. We have gotten used to napalm now which is just as horrible as nuclear weapons in another way. I don't think the fact that it is nuclear makes any difference.

AYRES: I disagree with you. I think that we have indoctrinated ourselves to think that it does make a difference, and, therefore, it does make a difference. We used napalm in World War II and have essentially never stopped using it, but we have created barriers to the use of nuclear weapons.

FRANK: I hope we have.

LIFTON: I partly agree with Jerry, but partly disagree with both positions. It isn't that we have indoctrinated ourselves, but that the nature of the weapon itself creates imagery in our minds that is different from that of so-called conventional weapons. Of course, it is no better to die from one than from another, but there is an element of totality and of world destruction in our way of envisaging nuclear weapons, which is based upon a good deal of actuality in terms of what these weapons do. Without even arguing the point as to whether they would totally destroy civilization, I would say that their dimensions are so vast that they take us psychically into a perception of infinity. In this sense our imagery about them is different.

AYRES: Yes, but there are nuclear weapons whose yield is bigger than a ton of TNT and there is no distinction in terms of magnitude now. The distinction is between nuclear and non-nuclear and in fact the very title of this Conference makes that distinction.

FREMONT-SMITH: Don't you think that in the public mind the distinction is between overwhelming and devastating as opposed to the napalm, which is for a small group?

AYRES: In part that is true, but there is a distinction also in the minds of the leadership, that is, the policy-makers, which probably is more important.

TAYLOR: Nuclear or non-nuclear what? If it is a nuclear strategic weapon, that's different. One must define "nuclear." They are very big, great devastating things. A tactical weapon may be very small.

LIFTON: It is a different weapon you can say in terms of mechanism, in terms of its physics. It is of a different order from what we call conventional weapons. Starting with that point, imagery forms around it. The imagery is related, I would say, to two particular elements that make it different in the way it is received psychically from conventional weapons. One is its potential dimension. It is true, of course, that you can make "little" nuclear weapons, but you can also make big ones capable of destruction that no conventional weapons can approach. Second, the issue of radiation. Because of these two factors the imagery is bound to be different.

AYRES: Even if you used one small tactical weapon, then you don't know how to make another barrier, because there are no more barriers in size.

UPTON: Don't we create a barrier with gas? I think we have the same problem with gas. We condone the use of tear gas by the police on rioters in a city, but we don't condone the same use of gas by military personnel on ground that this use of gas makes...

TAYLOR: We tolerate it, though. We have been tolerating what has been happening.

FREMONT-SMITH: There is an awful lot of gas in a napalm bomb and a phosphorous bomb is mostly gas; we don't call it by that and therefore it doesn't count. I have seen them explode and they are gas warfare in no uncertain terms, as was the Hamburg fire storm.

AYRES: Lots of people die of carbon monoxide.

FREMONT-SMITH: So that the name isn't in any way a sharp distinction.

UPTON: The word "gas" is a bad word.

FREMONT-SMITH: Exactly. But you can call it napalm or phosphorus and it doesn't matter because it is not called bomb.

TAYLOR: To get back to the question, it seems to me that I have a very simple and what I think is an obvious answer to the question, how would we feel about dropping bombs now in North Vietnam? I think that the primary rationale would be fear of retaliation and not the moral question about what we have done—just plain straight fear of retaliation.

FREMONT-SMITH: Fear of Chinese retaliation.

TAYLOR: Yes.

LIFTON: A good deal of our thought and behavior around nuclear weapons has to with that anxiety. It is another reason for much more serious efforts at thinking about psychological aspects of death and death symbolism that those we have made so far in psychiatry. It is an interesting phenomenon that during the last decade or so psychological studies of death totally unrelated to nuclear weapons, of the dying patient, for instance, have enormously increased. One could postulate that there is a kind of feedback from a general concern with death associated with nuclear weapons which affects the choice of topics for research in seemingly unrelated medical endeavors. But in any case, the element of death anxiety becomes extremely important in all of our thinking about nuclear weapons, and when I say "death anxiety" I don't mean simply the fear of being killed, but what we spoke of briefly before, the way in which one dies, the circumstances, the phase of one's life, the degree of fulfillment, or interruption, etc.

WHITE: How do you line up the fear of the unknown in this regard?

LIFTON: Very much part of it.

FRANK: One line of thought Dr. Ayres started in my mind is that it may be foolish to think about the long-range effect of nuclear war, because at any given level of nuclear war the immediate effects are so vastly greater than the long-term effects that the latter do not really matter.

TAYLOR: Is that fear really?

AYRES: It is a rather standard criticism of effort to look at long-range problems.

FRANK: Is it true? I am raising the question. That is what struck me from what you were saying.

AYRES: It is true until the immediate effects are over, but when they are over it is the long-range effects which are still ahead and where some options may remain in terms of taking various kinds of actions.

FREMONT-SMITH: They are not over; they are psychologically by no means over any more than they are over in Hiroshima yet.

UPTON: I think one must also bear in mind the scale, because the scale of a 30-kiloton weapon and its long-term effects differs quite drastically from the scale of the long-term effects of a 30-megaton weapon.

FRANK: But the initial damage done by a 30-megaton weapon is also very much greater than that done by a 30-kiloton weapon. If our society gets destroyed, it will be destroyed by the initial attack and not by the long-term effects, as I understand this point.

TAYLOR: But what do you mean by destruction? Lots of people will argue that what will destroy a civilization will be the inability for the remaining nine-tenths or three-quarters or three-tenths, whatever the fraction of the remaining population, to survive for reasons that are very complicated and long-range and not as a direct result of being killed, because they were not killed.

FREMONT-SMITH: It would depend upon shelter and a great variety of other factors that would enter into it. So it is entirely conceivable that there would be a large survival of 40 percent of the population, immediate survival. Then the question that you bring up would enter into it as to whether an organized society could be reconstituted.

FRANK: But if the line of reasoning that Dr. Ayres opened is right, the way to really make sure you win is to destroy 90 percent of the population right off the bat and not worry about the long-term effects. Isn't that true with nuclear weapons?

TAYLOR: Yes. Neither the Soviet Union nor the United States can do it right now with either of their stockpiles, not 90 percent.

COURT BROWN: What population? You are not talking about the world population, you are talking about the American population.

FRANK: Yes.

COURT BROWN: There are other populations in the world that are going to be at risk. We think that the risk will be appreciable.

FREMONT-SMITH: And long lasting.

TAYLOR: Won't it be from zero to very large and sort of everything in between?

WARREN: Yes. But it won't be total.

FREMONT-SMITH: May I throw in one other element that you are speaking about, Bob—new kinds of research. I have forgotten the name of the club, the big fire in Boston, the Coconut Grove. This initiated studies on mourning. Perhaps there had been some before, but some very important studies were done on mourning resulting from the Coconut Grove experience, and this was partly due to the mass effect and the sudden loss and the fact that a lot of young people lost their lives.

It seems to me—and this may be something that Bob would bring up next time—that there was a great deal of emphasis on mourning in Hiroshima and Nagasaki, or am I wrong on this?

LIFTON: This is a long story and you are entirely right about it. I have begun to think about the whole survivor ethos, and in the latter part of the study that I recently completed I compared patterns in Hiroshima survivors with concentration camp survivors, and also to a certain extent (from old descriptions) with survivors of the plagues of the Middle Ages, as well as with ordinary survivors of family members in contemporary life. There are certain consistent

patterns one can delineate, and what we usually call mourning is a central part of the survivor ethos. I mentioned once or twice before the need for the survivor to formulate, that is, to bring form to, to bring psychic order to his survival. When Freud originally described the mourning process, he emphasized loss, as well as ambivalence and guilt toward the dead person on the part of the survivor. But he also said that one had to gradually learn to imagine a world in which that dead person no longer was present.

This is the beginning of an idea of psychic formulation—or reformulation—of the world by the survivor. It involves a constant mourning process, and I came to the recognition that in Hiroshima various impediments existed to the mourning process; that is, to resolving the mourning process by means of formulation. These impediments include simply the massiveness of the experience. In a way it is not too strong a statement to say that many Hiroshima survivors, psychically speaking, have never gotten over that one moment. The losses then that they have sustained of specific family members, as well as their unresolved relationship to the anonymous dead, are internalized; then to achieve formulation one needs some sense of relevant activity or involvement that gives significance to the catastrophe one has been a part of.

Therefore, the involvement of survivors themselves in aspects of peace symbolism and peace movements has a certain kind of psychological significance that it didn't have for outsiders. But they became profoundly disillusioned with these elements of expressions, and would no longer see them as purposeful results of their experience as indeed happened in relationship to the political manipulations of the peace movement. This was another impediment to this formulative process and to the process of mourning. There were other impediments, too, related to the social change and general historical confusion which they were also in the midst of.

FREMONT-SMITH: Wasn't it a fact, particularly in the midst of the Japanese culture—and here I am showing my ignorance—that the bodies of the dead were not available. They were just lost, burned or disappeared; and individuals could not find any remnants of their lost wives or children around which some kind of formalized mourning could be organized. Is that a factor?

LIFTON: Very much so. Again, I have had much contact with psychiatrists working with concentration camp survivors and they

speak of the phenomenon of the "missing grave" as an impairment to the resolution of the mourning process in concentration camp survivors. In a sense it is the same phenomenon, the "missing grave" or the "missing dead."

Many Japanese survivors actually tried to fetch the remains of family members, but, of course, it was usually impossible because of the bomb effects and the mass cremations. There were some efforts to keep bodies labeled and to retain some bodily remnants, as is ordinarily done with cremation in Japanese culture. But it was impossible to do with any accuracy. This is one very important impediment to the mourning process.

DE BOER: How many of these people who have been subjected to this are now manning the peace marches, for example? In my own family, we lost several members during the war but none of us ever participated in a peace march. On the other hand, I know several people who never lost any member of their family, but now they are in the forefront of the peace marches.

LIFTON: Right. It is a kind of a vicious circle in a more or less impossible problem for them to solve. It was generally true that if survivors could feel that their experience could have meaning for the world, this contributed to its formulations. It doesn't mean that they would all then become peace movement activists. Some would prefer the process to take place quietly. And some of the quietude or "deadness" from the disaster itself in a sense stayed with people, and aspects of the psychic numbing were almost life-long with some survivors, a kind of constriction of the personality in ways which I don't have a chance to talk about now.

In terms of activist movements, I think the survivor participation is rather small. Many were at first either involved or at least sympathetic but they became quickly and bitterly disillusioned. Some tried to ignore the political infighting, but they could not usually do so. They became bitterly upset by it because they felt that it was "an insult to the dead." Much of their struggle with guilt, which I have not sufficiently stressed as a life-long struggle, involves their sense of how they behave toward the dead and toward what they perceived to be the moral commands of the dead. This is another way of saying that the dead are very much present in survivors' efforts to formulate the experience. Something perceived as an insult to the dead made them extremely anxious.

DE BOER: Did I understand you correctly when you said that the Japanese had no feelings of anxiety initially, did not see anything wrong about using nuclear weapons and would have used the same had they had them? This is important, because for that matter neither did or do the Germans, and both are considered our allies today.

It is now 20 years later, and I am wondering what some of the younger Japanese are thinking about developing these weapons for their own defense. I know in Germany there is a definite resurgence of militarism, anti-Semitism, and nationalism. In fact, had we not separated Germany, they might have been a nuclear power today.

I think one of the most important things that confront us today is to relate to the coming generation our experiences from the past in terms of the truth and not of conjecture. The facts are that we know very little about the long- or short-range effects of nuclear war.

MC CULLOCH: I happened to have been present at some of the gab fests with the youngsters around MIT. None of them are particularly interested in the momentary effect. All of them are interested in fallout. This is all you hear in the coming generation.

UPTON: I would like to raise a question here. It may be too late now, but it seems to me that one of the problems that we haven't considered sufficiently is the ability of a city to rally, to adapt, to repair itself. All the time we have disasters in this country and circumscribed areas become disaster areas which call on the resources of the nation to recover from some calamity. On the other hand, we know that in nuclear accidents there is an aura of danger that surrounds the accident which frequently hinders rational efforts to help. I am wondering whether in the Japanese disasters, once knowledge of the nuclear aspects was had, this aspect got in the way of the aid to the areas affected?

LIFTON: That is difficult to say. In a general sense my impression—and, of course, I tried to look into the whole issue of the recovery of the city, too—was that most of the hibakusha whom I interviewed felt that recovery would have been impossible without outside energies of some sort; that without these the city would have been unable to reconstitute itself. Yet, on the other hand, they like to emphasize, for various psychological reasons, their own efforts in reconstructing their city. The help from the outside was in many ways slow in coming, partly because Japan was rather thoroughly

devastated in other areas too, not just Hiroshima and Nagasaki. There were physicians who came from Kyoto, Okoyama, and other places, some fairly quickly, but in trickles rather than on a large scale.

UPTON: Did fear of self-contamination enter into their attitude toward the cities?

LIFTON: I don't know how much.

TAYLOR: They didn't know it was contaminated with radioactivity but they did know that there was some peculiar disease, which might have been more inhibitory.

FREMONT-SMITH: Like an epidemic of cholera or something like that.

TAYLOR: Yes.

LIFTON: I don't know too much from actual evidence, but I would guess that it did.

UPTON: I think this fear of fallout, which has been magnified and exaggerated, could indeed be a problem in dealing with any future incidents of this type, and I gather from what you say that this didn't seem to be a problem at the time.

BRILL: What you are usually talking about when you mention fallout is global fallout. What we are talking about now is near-in fallout. We are talking about an acute weapons conflagration involving multiple megaton weapons, and this cannot be disregarded quite so summarily.

LIFTON: Another very basic point about this thing is terribly important to make. When we say that fears are exaggerated, this can be a misleading idea. It may suggest that if people weren't so irrationally afraid, everybody would be better off. But these fears are not so much exaggerated as displaced. In other words, the fear has to do with death anxiety, based on the nature of the weapon and the exposure itself. It is a fear that we all in some measure share regarding nuclear weapons. So that when it is expressed about fallout it may be exaggerated in terms of the harm from the fallout, per se, but it may not be inappropriate for the nuclear weapons danger in general—although when the fear itself becomes very great, it can be immobilizing.

FOUNTAIN: Survival comes from increments. If you don't survive the first 24 hours, then you, as an individual, don't have a fallout problem.

WHITE: You have to survive seconds first, then minutes, then hours, etc.

FOUNTAIN: Then you become involved with fallout. You can't disregard it. But in any conflagration the major casualties are going to be caused by the initial effect of the nuclear weapon.

AYRES: The chance of ultimate survival may depend on taking limited resources, surviving after a couple of months and doing your best with them. What we have been talking about are possible impediments to that, things which may cause you to direct your efforts to irrational measures.

TAYLOR: Are you saying that fallout will not be a major contributor to casualties? Because I don't believe that at all.

BRILL: I am very much conerned about this turn in the discussion. We have only scratched the surface on what is known about the somatic effects of external irradiation and talked even less about the genetic effects. The weapons we discussed were in the 10- to 20-kiloton range, two in number, separated in time and distance, both air shots. To generalize from this cursory review of war gamesmanship is foolhardy, especially since effects of internal emitters, and genetic hazards themselves, have only barely been touched upon in the discussion.

I must confess to a feeling of grave concern over the attitudes of some of the participants in this symposium expressed in informal evening discussions and in the proceedings of the meeting as well. These suggest to me that we are beginning to accept small nuclear weapons, so-called tactical weapons, as no different or less acceptable than other instruments of war. The critical concern seems to involve an assessment of retaliatory capability. I am unable to accept this line of reasoning and feel that morality cannot be disregarded and operational planning conducted in vacuo. Concern regarding short-term survival is too narrow a perspective.

UPTON: Who is not concerned about fallout?

BRILL: I had the impression that you said fallout was not a major problem in nuclear war. After two months of living in such a field as this you wouldn't have any people to worry about.

COURT BROWN: Are we looking at survival in too limited a way? Many of us are concerned not so much about the immediate disintegration of the American society or the Russian society, or whatever. We are concerned about the very long-term effects thereafter in terms of the rest of the world, and we are concerned, in thinking about survival, in terms of the survival of cultures, in the survival of a way of life which depends upon the integrity of a genotype. No one seems to be considering this at all. Everyone is considering the immediate effects, the immediate killing effects. No one seems to be worrying about the long-term effects. Surely they must be taken into account.

FREMONT-SMITH: I entirely agree, and may I say that since we have four more conferences, fortunately we are going to be able to take them into account.

LIFTON: In terms of this particular issue about the survival of culture and of the genotype and so on, I would emphasize something I didn't have a chance to present in an orderly way. The psychic sense of Hiroshima survivors—from fears of A-bomb disease and of genetic effects—was of being involved in an endless chain of potentially lethal impairment, which, if it did not manifest itself in one year or in one generation, might well do so in the next. This kind of anxiety, I believe, is a beginning point for the sort of concerns that you are describing.

FREMONT-SMITH: Gentlemen, we must draw this to a close although I hate to do so.

We were talking about death. Gertrude Stein on her deathbed made a statement of great scientific significance which bears on the topic of our discussion. She was very restless and very unhappy, as people are sometimes on their deathbeds, and Alice Toklas, her constant companion, was trying to sooth her.

Gertrude kept saying, "What is the answer? What is the answer?" and Alice didn't quite know how to deal with this. She tried to soothe and calm her, but Gertrude wouldn't be calmed. "What is the answer? Alice, what is the answer?"

Finally Alice Toklas felt all she could do was to give Gertrude the truth, as she always had, and she said, "Gertrude, there is no answer."

"If no answer," said Gertrude, "then what is the question?"

Ladies and gentlemen, what is the question? We will come to that at the next conference.

Thank you all very much.

REFERENCES

REFERENCES

1. Warren, S.L. and G.H. Whipple. 1922. Roentgen ray intoxication. I. Epithelium of small intestine sensitivity to x-rays. II. Sequence of clinical, anatomical and histological changes following a unit dose of x-rays. III. Speed of autolysis of tissues. J. Exptl. Med. 35(2): 187-225.

2. Glasstone, S. 1962. Effects of Nuclear Weapons. 730 pp. U.S. Govt. Printing Office. Washington, D.C.

3. White, C.S. 1966. The scope of blast and shock biology and problem areas in relating physical and biological parameters. Proc. Conf. Prevention and Protection Against Accidental Explosion of Munitions, Fuels and Other Hazardous Mixtures. N.Y. Acad. Sci. Oct. 10-13. (in press).

4. White, C.S., I.G. Bowen and D.R. Richmond. 1964. A comparative analysis of some of the immediate environmental effects at Hiroshima and Nagasaki. Health Phys. 10: 89-150.

5. Damon, E.G., D.R. Richmond and C.S. White. 1964. The effects of ambient pressure on the tolerance of mice to air blast. Aerospace Med. 37: 341-347.

6. Richmond, D.R., E.G. Damon, E.R. Fletcher, I.G. Bowen and C.S. White. 1966. Proc. Conf. Prevention of and Protection Against Accidental Explosion of Munitions, Fuels and Other Hazardous Mixtures. N.Y. Acad. Sci. Oct. 10-13. (in press).

7. Bowen, I.G., E.R. Fletcher, D.R. Richmond, F.G. Hirsch and C.S. White. 1966. Biophysical mechanisms and scaling procedures applicable to assessing responses of the thorax energized by air-blast overpressures and nonpenetrating missiles. Proc. Conf. Prevention of and Protection Against Accidental Explosion of Munitions, Fuels and Other Hazardous Mixtures. N.Y. Acad. Sci. Oct. 10-13. (in press).

8. Richmond, D.R., E.G. Damon, I.G. Bowen, E.R. Fletcher and C.S. White. Air blast studies with eight species of mammals. Tech. Progr. Rept. DASA-1854. Defense Atomic Support Agency. Dept. Defense. Washington, D.C. (in press).

9. White, C.S. and D.R. Richmond. 1960. Blast biology. In Clinical Cardiopulmonary Physiology. R.C. Kory and B.L. Gordon, Eds. Chap. 63, pp. 974-992. Grune and Stratton. New York.

10. Benzinger, T. 1950. Physiological effects of blast in air and water. In German Aviation Medicine, World War II. Chap. 14-B, Vol. 2, pp. 1225-1259. U.S. Govt. Printing Office. Washington, D.C.

11. Desaga, H. 1950. Blast injuries. In German Aviation Medicine, World War II. Chap. 14-D, Vol 2, pp. 1274-1293. U.S. Govt. Printing Office. Washington, D.C.

12. Rössle, R. 1950. Pathology of blast effects. In German Aviation Medicine, World War II. Chap. 14-C, Vol. 2, pp. 1260-1273. U.S. Govt. Printing Office. Washington D.C.

13. Leavell, B.S. 1945. Acute heart failure following "blast injury." War Med. 7: 162-167.

14. Chiffele, T.L. 1966. Pathology of direct air-blast injury. Tech. Progr. Rept. DASA-1778. Defense Atomic Support Agency. Dept. Defense. Washington, D.C.

15. White, C.S., T.L. Chiffelle, D.R. Richmond, W.H. Lockyear, I.G. Bowen, V.C. Goldizen, H.W. Merideth, D.E. Kilgore, B.B. Longwell, J.T. Parker, F. Sherping and M.E. Cribb. 1957. The biological effects of pressure phenomena occurring inside protective shelters following nuclear detonation. USAEC Civil Effects Test Group Rept. WT-1179. Office Tech. Serv. Dept. Commerce. Washington, D.C.

16. White, C.S., I.G. Bowen and D.R. Richmond. 1965. Biological tolerance to air blast and related biomedical criteria. USAEC Civil Effects Test Operations Rept. CEX-65.4. Office Tech. Serv. Dept. Commerce. Washington, D.C.

17. Bowen, I.G., A.F. Strehler and M. Wetherbe. 1956. Distribution and density of missiles from nuclear explosions. USAEC Civil Effects Test Group Rept. WT-1168. Office Tech. Serv. Dept. Commerce. Washington, D.C.

18. Oughterson, A.W. and S. Warren. 1956. Medical Effects of Atomic Bomb in Japan. McGraw-Hill. New York.

19. Fletcher, E.R., R.W. Albright, R.F.D. Perret, M.E. Franklin, I.G. Bowen and C.S. White. 1963. Nuclear bomb effects computer (including slide rule design and curve fits for weapons effects). USAEC Civil Effects Test Operations Rept. CEX-62.2. Office Tech. Serv. Dept. Commerce. Washington, D.C.

20. Richmond, D.R., V.C. Goldizen, V.R. Clare, D.E. Pratt, F. Sherping, R.T. Sanchez, C.C. Fischer and C.S. White. 1962. The biologic response to overpressure. III. Mortality in small animals exposed in a shock tube to sharp-rising overpressures of 3 to 4 msec duration. Aerospace Med. 33: 1-27.

21. Richmond, D.R., R.V. Taborelli, F. Sherping, M.B. Wetherbe, R.T. Sanchez, V.C. Goldizen and C.S. White. 1959. Shock tube studies of the effects of sharp-rising long-duration overpressures on biological systems. USAEC Tech. Progr. Rept. TID-6056. Office Tech. Serv. Dept. Commerce. Washington, D.C.

22. Richmond, D.R., V.R. Clare, V.C. Goldizen, D.E. Pratt, R.T. Sanchez, and C.S. White. 1961. Biological effects of overpressure. II. A shock tube utilized to produce sharp-rising overpressures of 400 milliseconds duration and its employment in biomedical experimentation. Aerospace Med. 32: 997-1008.

23. Fisher, R.B., P.L. Krohn and S. Zuckerman. 1941. The relationship between body size and the lethal effects of blast. Rept. R.C. 284. Ministry Home Security. Oxford Univ. Oxford, England.

24. Schardin, H. 1950. The physical principles of the effects of a detonation. In German Aviation Medicine, World War II. Chap. 14-A, Vol. 2, pp. 1207-1224. U.S. Govt. Printing Office. Washington, D.C.

25. Bowen, I.G., A. Holladay, E.R. Fletcher, D.R. Richmond and C.S. White. 1965. A fluid-mechanical model of the thoraco-abdominal system with application to blast biology. Tech. Progr. Rept. DASA-1675. Defense Atomic Support Agency. Dept. Defense. Washington, D.C.

26. Richmond, D.R. and C.S. White. 1962. A tentative estimation of man's tolerance to overpressures from air blast. Tech. Progr. Rept. DASA-1335. Defense Atomic Support Agency. Dept. Defense. Washington, D.C.

27. Damon, E.G., J.H. Sherrod, T.L. Chiffelle and D. R. Richmond. 1965. The effects of a 500-ton explosion in cattle exposed in the higher pressure regions. Biomedical Program—500-Ton Explosion—Final Report. Tab "A." Tech. Progr. Rept. DASA-1656. pp. 2-1 to 2-13. Defense Atomic Support Agency. Dept. Defense. Washington, D.C.

28. Damon, E.G., C.S. Gaylord, J.T. Yelverton and D.R. Richmond. The tolerance of cattle to long-duration reflected pressures in a shock tube. Tech. Progr. Rept. DASA 1855. Defense Atomic Support Agency. Dept. Defense. Washington, D.C. (in press).

29. Brody, S. 1964. Bioenergetics and Growth. Hofner. New York.

30. Blocker, V. and T.G. Blocker. 1949. The Texas City disaster—A survey of 3000 casualties. Am. J. Surg. 78: 756-771.

31. Ruskin, A., O.W. Beard and R.L. Schaeffer. 1948. Blast hypertension—Elevated arterial pressures in the victims of the Texas City disaster. Am. J. Med. 4: 228-236.

32. Ruskin, A. and O.W. Beard. 1948. The Texas City disaster—Cardiovascular studies, with follow-up results. Tex. Rept. Biol. Med. 6: 243-259.

33. Chase, W.H. 1934. Anatomical and experimental observations in air embolism. Surg. Gyn. Obstet. 59: 569-577.

34. Richmond, D.R., I.G. Bowen and C.S. White. 1961. Tertiary blast effects: Effects of impact on mice, rats, guinea pigs and rabbits. Aerospace Med. 32: 789-805.

35. Auxier, J.A., J.S. Cheka, F.F. Haywood, T.D. Jones and J.H. Torngate. 1966. Free-field radiation dose distributions from the Hiroshima and Nagasaki bombings. Health Phys. 12: 425-429.

36. Miller, R.W. 1956. Delayed effects occurring within the first decade after exposure of young individuals to the Hiroshima atomic bomb. Pediatrics 18: 1-18.

37. Richmond, D.R., M.B. Wetherbe, R.F. Taborelli and T.L. Chiffelle and C.S. White. 1957. The biologic response to

overpressure. I. Effects on dogs of five to ten-second duration overpressures having various times of pressure rise. J. Aviation Med. 28: 447-460.

38. Cassen, B., P. Kalian and H. Gaas. 1951. High speed photography of the motion of mice subjected to laboratory-produced air blast. UCLA-AEP Contract Gen-12 Rept. 138. pp. 4-15.

39. Cassen, B. and K. Kistler. 1954. Development of acute pulmonary edema in mice and rats and an interpretation. Am. J. Physiol. 178: 49-53.

40. Wycoff, C.C. and J.E. Cann. 1966. Experimental pulmonary air embolism in dogs. Calif. Med. 105: 361-367.

41. Dunne, B.B. and B. Cassen. 1956. Behavior of shock waves entering model bomb shelters. Paper 919. Proc. Am. Soc. Civil Engrs. Structural Div. pp. 1-2.

42. Johnson, M.-L.T., P.B.Gregory and R.C. Milton. 1966. Dermatologic observations in atomic bomb survirors. Draft, Atomic Bomb Casualty Comm. Tech. Rept. Dec. (in press).

43. Treves, N. and G.T. Pack. 1930. Development of cancer in burn scars: Analysis and report of 34 cases. Surg. Gyn. Obstet. 51: 749-782.

44. Bloom, A.D., S. Neriishi, K. Kamada, T. Iseki and R.J. Keehn, 1966. Cytogenetic investigation of survivors of the atomic bombings of Hiroshima and Nagasaki. Lancet 2: 672-674.

45. Miller, R.J., T. Fujino and M.D. Nefzger. 1966. Lens findings in atomic bomb survivors: A review of major opthalmic surveys at the Atomic Bomb Casualty Commission, 1949-1962. Atomic Bomb Casualty Comm. Tech. Rept. 26-66.

46. Sinskey, R.M. 1955. The status of lenticular opacities caused by atomic radiation: Hiroshima and Nagasaki, Japan, 1951-1953. Am. J. Ophthalmol. 39: 285-293.

47. Woods, A.C. 1959. Cyclotron cataracts. II. Am. J. Ophthalmol. 47: 20-38.

48. Merriam, George R. and Elizabeth F. Focht. 1962. A clinical and experimental study of the effect of single and divided doses of radiation on cataract production. Trans. Am. Ophthalmol Soc. 60: 35-52.

49. Report of the RBE Committee to the International Commissions on Radiological Protection and on Radiological Units and Measurements. 1963. Health Phys. 9: 357-386.

50. Miller, R. W. 1963. Distant visual acuity loss among Japanese grammar school children. The roles of heredity and the environment. J. Chronic Diseases 16: 31-54.

51. Brown, W. L. and A. A. McDowell. 1960. Visual acuity performance of normal and chronic irradiated monkeys. J. Genet. Psychol. 96: 133-137.

52. McDowell, A. A. and W. L. Brown. 1960. Visual acuity performance of normal and chronic focal-head irradiated monkeys. J. Genet. Psychol. 96: 139-143.

53. Gregory, P. B., R. C. Milton, M.-L. T. Johnson and T. Taura. 1966. Spleen shielding in survivors of the atomic bomb. Atomic Bomb Casualty Comm. Tech. Rept. 17-66.

54. Caidin, Martin. 1960. The Night Hamburg Died. Ballantine. New York.

55. Zeldis, L. J., S. Jablon and M. Ishida. 1964. Current status of ABCC-NIH studies of carcinogenesis in Hiroshima and Nagasaki. Ann. N. Y. Acad. Sci. 114: 225-240.

56. Leutz, H. 1966. Fireproof shelters with secured ventilating systems. Proc. Symp. Protective Construction. Apr. 19-23. pp. 134-138. Natl. Acad. Sci.-Natl. Res. Council. Washington, D. C.

57. McDonnel, G. 1961. Effects of nuclear detonation on a large biological specimen (swine). WT 1428. Operation Plumbbob. Aug. 11. (For Official Use Only).

58. Shouse, S. S., G. H. Whipple and S. L. Warren. 1931. II. Aplasia of bone marrow and fatal intoxication in dogs produced by roentgen radiation of all bones. J. Exper. Med. 53(3): 421-435.

59. Oughterson, A. W., G. V. Leroy, A. A. Liebow, E. C. Hammond, H. L. Barnett, J. D. Rosenbaum and B. A. Schneider. 1951. Medical effects of atomic bombs. Rept. Joint Comm. Investigation of the Effects of the Atomic Bomb in Japan. AEC Document NP-3041. Vol. VI. U. S. Atomic Energy Comm. Tech. Inform. Serv.

60. Davis, L. W., W. L. Baker and D. L. Summers. Analyses on Japanese nuclear casualty data. Tech. Rept. DO-FR1045

and 1054. Atomic Energy Comm. Office Civil Defense Projects. Dikewood Corp. Alburqerque, N. Mex. (in preparation).

61. Hachiya, M. 1955. Hiroshima Diary. Warner Wells, Ed. 233 pp. Univ. North Carolina Press. Chapel Hill, N.C.

62. Glasstone, S. 1950. The Effects of Atomic Weapons. U.S. Govt. Printing Office. Washington, D.C.

63. White, C.S. and D.R. Richmond. 1959. Blast biology. Tech. Progr. Rept. USAEC Rept. TID-5764. Office Tech. Serv. Dept. Commerce. Washington, D.C.

64. U.S. Bureau of the Census. 1958. Statistical Abstract of the U.S. 79th edit. Washington, D.C.

65. U.S. Bureau of the Census. 1963. Statistical Abstract of the U.S. 84th edit. Washington, D.C.

66. Auxier, J.A., J.S. Cheka and F.W. Sanders. 1961. Projects 39.1 and 39.2. Operation Hardtack (Phase II) Rept. WT-1725 (Classified).

67. Thorngate, J.H., D.R. Johnson and P.T. Perdue. 1966. Neutron and gamma-ray leakage from the Ichiban critical assembly. USAEC Rept. CEX-64.7.

68. Storm, E. 1952. Gamma-radiation exposure as a function of distance. Operation Buster-Jangle. WT-408 (Classified).

69. Storm, E. 1956. Gamma radiation as a function of distance. Operation Upshot-Knothole. WT-827 (Classified).

70. Cheka, J.S., F.W. Sanders, T.D. Jones and W.H. Sinpaugh. 1965. Distribution of weapons radiation in Japanese residential structures. USAEC Rept. CEX-62.11.

71. Sanders, F.W., F.F. Haywood, M.L. Lundin, L.W. Gilley, J.S. Cheka and D.R. Ward. 1962. Operation plan and hazards report. Operation BREN. USAEC Rept. CEX-62.02.

72. National Bureau of Standards Handbook 88. 1963. Radiobiological dosimetry.

73. National Bureau of Standards Handbook 84. 1962. Radiation quantities and units.

74. National Bureau of Standards Handbook 59. 1954. Permissible dose from external sources of ionizing radiation.

75. Russell, W.L. 1965. Effect of the interval between irradiation and conception on mutation frequency in female mice. Proc. Natl. Acad. Sci. (US) 54: 1552-1557.

76. Bender, M.A. and P.C. Gooch. 1966. Cytogenetic methods: Somatic chromosome aberrations in occupationally irradiated humans. Symp. Accidental Irradiation at Place of Work. Nice, France. Apr. 25-29. (in press).

77. Court Brown, W.M. and R. Doll. 1957. Leukaemia and aplastic anaemia in patients irradiated for ankylosing spondylitis. Med. Res. Council Rept. Ser. No. 295. Her Majesty's Stationery Office. London.

78. Jablon, S., M. Ishida and M. Yamasaki. 1965. Studies of the mortality of A-bomb survivors. 3. Radiation Res. 25: 25-52.

79. Seigel, D.G. 1966. Frequency of live births among survivors of Hiroshima and Nagasaki atomic bombings. Radiation Res. 28: 278-288.

80. Bizzozero, O.J., Jr., K.G. Johnson and A. Ciocco. 1966. Distribution, incidence and appearance time of radiation-related leukemia in Hiroshima and Nagasaki, 1946-1964. New Engl. J. Med. 274: 1095-1102.

81. Bizzozero, O.J., Jr., K.G. Johnson and A. Ciocco. 1966. Observation on type-specific leukemia, survivorship and clinical behavior of radiation-related leukemia, Hiroshima and Nagasaki, 1946-1964. Atomic Bomb Casualty Comm. Tech. Rept. 6-66. Ann. Intern. Med. (in press).

82. Brill, A.B., Masanabu Tomanaga and R.M. Heyssel, 1962. Leukemia in man following exposure to ionizing radiation. Ann. Internal Med. 56: 590-609.

83. Fujisaku, S. 1957. Age adjusted death rates for leukemia and aleukemia in 16 countries. Tohoku J. Expt. Med. 65: 322.

84. Lewis, E.B. 1957, E.B. 1957. Leukemia and ionizing radiation. Science 125:965-972.

85. Upton, A.C., V.K. Jenkins, H.E. Walburg, Jr., R.L. Tyndall, J.W. Conklin and N. Wald. 1965. Observations of viral, chemical, and radiation-induced myeloid and lymphoid leukemias in RF mice. Natl. Cancer Inst. Monograph 22: 330-347.

86. Upton, A.C., A.W. Kimball, J. Furth, K.W. Christenberry and W.H. Benedict. 1960. Some delayed effects of atom-bomb radiations in mice. Cancer Res. 20: 1-62.

87. Lindop, P. and J. Rotblat. 1961. Long-term effects of single whole-body exposure of mice to ionizing radiation: Causes of death. Proc. Roy. Soc. 54: 332-349, 350-368.

88. Anderson, R.E. 1964. Malignant lymphoma in survivors of the atomic bomb, Hiroshima. Ann. Internal Med. 61: 853-862.

89. Loutit, J.F. 1962. Irradiation of Mice and Men. Univ. Chicago Press. Chicago, Ill.

90. Court Brown, W.M. and R. Doll. 1959. Irradiation of mice and men. Brit. Med. J. 1: 1063.

91. Ederer, F., R.W. Miller and J. Scotto. 1965. U.S. childhood cancer mortality patterns, 1950-1959. Etiologic implications. J. Am. Med. Assoc. 192: 97-100.

92. Court Brown, W.M. and R. Doll. 1961. Leukemia in childhood and young adult life. Brit. Med. J. 1: 981-988.

93. Kanamitsu, M., K. Morita, S.C. Finch, H. Kato and S. Onishi. 1966. Serologic response of atomic bomb survivors following Asian influenza vaccination. Atomic Bomb Casualty Comm. Tech. Rept. 4-66.

94. Davenport, F.M. and A.V. Hennessy. 1956. A serologic recapitulation of past experience with influenza A. Antibody response to monovalent vaccine. J. Exptl. Med. 104: 85-97.

95. Hollingsworth, D.R., H.B. Hamilton, H. Tamagaki and G.W. Beebe. 1963. Thyroid disease: A study in Hiroshima, Japan. Med. 42: 47-71.

96. Socolow, E.L., A. Hashizume, S. Neriishi and R. Niitani. 1963. Thyroid carcinoma in man after exposure to ionizing radiation: A summary of the findings in Hiroshima and Nagasaki. New Engl. J. Med. 268: 406-410.

97. Wood, J.W., et al. 1966. Radiation-related thyroid carcinoma. Draft, Atomic Bomb Casualty Comm. Tech. Rept. Aug. (to be published).

98. Wanebo, C.K. and K.G. Johnson. 1967. Pulmonary cancer in atomic bomb survivors. Draft, Atomic Bomb Casualty Comm. Tech. Rept. Jan. (to be published).

99. Angevine, D.M. and S. Jablon. 1964. Late radiation effects of neoplasia and other diseases in Japan. Ann. N.Y. Acad. Sci. 114: 823-831.

100. Beebe, G.W., M. Ishida and S. Jablon. 1962. Studies of the mortality of A-bomb survivors. I. Radiation Res. 16: 253-280.

101. Conkite, E. 1956. Pathologic effects of atomic radiation. Natl. Acad. Sci. (US) Publ. 452: 1-13.

102. Stewart, A., J. Webb, D. Giles and D. Hewitt. 1956. Malignant disease in childhood and diagnostic irradiation in utero. Preliminary communication. Lancet 2: 447.

103. Stewart, A., J. Webb and D. Hewitt. 1958. A survey of childhood malignancies. Brit. Med. J. 1: 1495-1508.

104. MacMahon, B. 1962. Prenatal x-ray exposure and childhood cancer. J. Natl. Cancer Inst. 28: 1173-1191.

105. Wood, J.W., K.G. Johnson and Y. Omori. 1967. Children exposed in utero to the atomic bomb in Hiroshima: Follow-up at 20 years. Pediatrics (in press).

106. Graham, S., M.L. Levin, A.M. Lilienfeld, L.M. Schuman, R. Gibson, J.E. Dowd and L. Hempelmann. 1966. Pre-conception, intrauterine, and postnatal irradiation as related to leukemia. In Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. W. Naenszel. pp. 347-371. Natl. Cancer Inst. Monograph 19.

107. Kato, H., W.J. Schull and J.V. Neel. 1966. A cohort-type study of survival in the children of parents exposed to atomic bombings. Am. J. Human Genet. 18: 339-373.

108. Hoshino, T., T. Itoga and H. Kato. 1956. Leukemia in the offspring of parents exposed to the atomic bomb at Hiroshima and Nagasaki: Preliminary report. Meeting Japan. Assoc. Hematol. Mar. 28-30.

109. Miller, R.W. 1966. Relation between cancer and congenital defects in man. New Engl. J. Med. 275: 87-93.

110. March, H.C. 1944. Leukemia in radiologists. Radiology 43: 275-278.

111. Ulrich, H. 1946. Incidence of leukemia in radiologists. New Engl. J. Med. 234: 45-46.

112. Burch, P. 1960. Radiation carcenogenesis: A new hypothesis. Nature 185: 135-142.

113. Kaplan, H.J. 1952. Mortality of mice after total-body irradiation as influenced by alterations in total dose, fraction and periodicity of treatment. Natl. Cancer Inst. 12: 765-775.

114. Greene, W. 1958. Role of a vicarious object in the adaptation to object loss. Psychosomatic Med. 20: 344-350; 1959. 21: 438-447.

114a. Greene, W.A. and G. Miller. 1958. Psychological factors and reticuloendothelial disease. IV. Observations on a group of children and adolescents with leukemia: An interpretation of disease development in terms of the mother-child unit. Psychosomatic Med. 20: 124-144.

115. Ciocco, A. 1965. JNIH-ABCC Life Span Study and ABCC-JNIH Adult Health Study. Mortality 1950-64 and disease and survivorship 1958-64 among sample members aged 50 years or older, 1 October 1950. Atomic Bomb Casualty Comm. Tech. Rept. 18-65.

116. Grahan, D. and K.F. Hamilton. 1957. Genetic variation in the acute lethal response of four inbred mouse strains to whole body X-irradiation. Genetics 42: 189-198.

117. Platt, J.R. 1964. Strong inference. Science 146: 347-353.

118. Murphy, D.P. 1928. Ovarian irradiation: Its effects on the health of subsequent children; review of literature, experimental and clinical, with a report of three hundred and twenty human pregnancies. Surg. Gynecol. Obstet. 47: 201-215.

119. Goldstein, L. and D.P. Murphy. 1929. Etiology of the ill-health in children born after maternal pelvic irradiation. II. Defective children born after postconception pelvic irradiation. Am. J. Roentgenol. Radium Therapy Nucl. Med. 22: 322.

120. Rugh, R. 1959. Vertebrate radiobiology (embryology). Ann. Rev. Nucl. Sci. 9: 493-522.

121. Rugh, R. 1952. Fetal X-irradiation and fertility. Proc. Soc. Exptl. Biol. Med. 80: 388-395.

122. Manson, M.M., W.P.D. Logan and R.M. Loy. 1960. Rubella and other infections during pregnancy. Ministry Health Publ. No. 101. 101 pp. Her Majesty's Stationery Office. London.

123. Lenz, W. 1964. Chemicals and malformations in man. In Congenital Malformations. pp. 263-276. Second Intern. Conf. Congenital Malformations. Intern. Med. Congr. Ltd. New York.

124. Harada, M. 1964. Neurophsychiatric disturbance due to organic mercury poisoning during the prenatal period. Psychiat. Neurol. Japan 66: 429-468.

125. Neel, J.V. and W.J. Schull. 1956. The effect of exposure to the atomic bombs on pregnancy termination in Hiroshima and Nagasaki. p. 241. Natl. Acad. Sci.-Natl. Res. Council Publ. 461. Washington, D.C. p. 241.

126. Stoller, A. and R.D. Collmann. 1965. Virus aetiology for Down's syndrome (mongolism). Nature 208: 903-904.

127. Robinson, A. and T.T. Puck. 1965. Sex chromatin in newborn: Presumptive evidence for external factors in human nondisjunction. Science 148: 83-85.

128. Uchida, I. and E.J. Curtis. 1961. A possible association between maternal radiation and mongolism. Lancet 2: 848-850.

129. Sigler, A.T., A.M. Lilienfeld, B.H. Cohen and J.E. Westlake. 1965. Radiation exposure in parents of children with mongolism (Down's syndrome). Bull. Johns Hopkins Hosp. 117: 374-399.

130. Schull, W.J. and J.V. Neel. 1962. Maternal radiation and mongolism. Lancet 2: 537-538.

131. Yamazaki, J.N., S.W. Wright and P.N. Wright. 1954. Outcome of pregnancy in women exposed to the atomic bomb in Nagasaki. Am. J. Diseases Children 87: 448-463.

132. Slavin, R.E., N. Kanata and H.B. Hamilton. 1967. A cytogenetic study of 92 cases of Down's syndrome in Hiroshima and Nagasaki. (in press).

133. Conard, R.A. and W. Hicking. 1965. Medical findings in Marshallense people exposed to fallout radiation. J. Am. Med. Assoc. 192: 457-459.

134. Conard, R.A., L.M. Meyer, J.S. Robertson, W.W. Sutow, W. Wolins and H. Hechter. 1959. Effects of fallout on a human population. Radiation Res. Suppl. 1: 280-295.

135. Nehemias, J.V. 1962. Multivariate analysis and the IBM 704 computer applied to ABCC data on growth of surviving Hiroshima children. Health Phys. 8: 165-183.

136. Wood, J.W., R.J. Keehn, S. Kawamoto and K.G. Johnson. 1966. The growth and development of children exposed in utero to the atomic bombs, Hiroshima and Nagasaki. Atomic Bomb Casualty Comm. Tech. Rept. 11-66. Am. J. Public Health. (to be published).

137. Hempelmann, L.H. 1967. Neoplasms in persons with x-rays in infancy for thymic enlargement. A report of the third follow-up survey. J. Natl. Cancer Inst. (in press).

138. Conti, E.A., G.D. Patton, J.E. Conti and L.H. Hempelmann. 1960. Present health of children given x-ray treatment to the anterior mediastinum in infancy. Radiology 74: 386-391.

139. Conard, R.A., et al. 1966. Thyroid nodules as a late sequele of radiation fallout in a Marshall Island population population exposed in 1954. New Engl. J. Med. 274: 1392-1399.

140. Sheline, G.E., et al. 1962. Thyroid nodule occurring late after treatment of thyrotoxicosis with radioiodine. J. Clin. Endocrinol. 22: 8-18.

141. Serra, J.A. 1965. Modern Genetics. Vol. 2, p. 553. Academic Press. New York.

142. Silverberg, S.G. and R.A. Vidone. 1966. Adenoma and carcinoma of the thyroid. Cancer 19: 1053-1062.

143. MacMahon, B. and M.A. Levy. 1964. Prenatal origin of childhood leukemia. New Engl. J. Med. 270: 1082-1085.

144. Modan, B. and A.M. Lilienfeld. 1965. Polycythemia vera and leukemia—The role of radiation treatment. Medicine 44: 305-344.

145. Wald, N., W.H. Borges, C.C. Li, J.H. Turner and M.C. Harnois. 1961. Leukaemia associated with mongolism. Lancet 1: 1228.

146. Miller, R.W. 1963. Down's syndrome (mongolism), other congenital malformations and cancers among sibs of leukemic children. New Engl. J. Med. 268: 393-401.

147. Schade, H., L. Schoeller and K. W. Schultze. 1962. D-trisomie (Patau syndrom) mit kongenitaler myeloischer leukamie. Med. Welt. 50: 2690-2692.

148. Zuelzer, W. W., R.I. Thompson and R. Mastrangelo. 1966. Chromatid exchange and breakage in the pedigree of a child with leukemia and partial D-trisomy: Evidence for a genetic factor in leukemogenesis and gential malformations. Blood 28: 1008.

149. Borgas, W.H., J.W. Nicklas and G. W. Hamm. 1967. Prezygotic determinants in acute leukemia. J. Pediat. 70: 180-184.

150. Engel, E., E.J. McGee, R.C. Hartmann and M. Engel-de Montmollin. 1965. Two leukemic peripheral blood stemlines during acute transformation of chronic myelogenous leukemia in D/D translocation carrier. Cytogenetics 4: 157-170.

151. Behrman, R.E., A.T. Sigler and A.S. Patchefsky. 1966. Abnormal hematopoissis in 2 or 3 siblings with mongolism. J. Pediat. 68: 569-577.

152. Sawitsky, A., D. Bloom and J. German. 1966. Chromosomal breakage and acute leukemia in congenital telangiectatic erythema and stunted growth. Ann. Internal Med. 65: 487-495.

153. Bloom, G.E., S. Warner, P.S. Gerald and L.K. Diamond. 1966. Chromosome abnormalities in constitutional aplastic anemia. New Engl. J. Med. 274: 8-14.

154. Tough, I.M. and W.M. Court Brown. 1965. Chromosome aberrations and exposure to ambient benzene. Lancet 1: 684.

155. Vigliani, E.C. and G. Siata. 1964. Benzene and leukemia. New Engl. J. Med. 271: 872-876.

156. Joneja, M.G. and H.F. Stich. 1965. Chromosomes of tumor cells. IV. Cell population changes in thymus, spleen, and bone marrow during X-ray-induced leukemogenesis in C57BL/6J mice. J. Natl. Cancer Inst. 35(3): 421-434:

157. Lea, D.E. 1946. Actions of Radiations on Living Cell. pp. xii and 416. Cambridge, England.

158. Russell, W.L., L.B. Russell and E.M. Kelly. 1959. Dependence of mutation rate on radiation intensity. Intern. J. Rad. Biol. Special Suppl. pp 311-320.

159. Mavor, J.W. 1922. The production of non-disjunction by X-rays. Science 55: 295-297.

160. Murphy, D.P. and L. Goldstein. 1929. Etiology of the ill-health of children born after maternal pelvic irradiation. Am. J. Roentigenol. 22: 207-219.

160a. Maurer, H. 1933. Untersuchungen an Kindern strahlen-behandelter Mütter Zentr. Gynäkol. 57: 819-820.

161. Schull, W.J. 1963. Hereditary effects. Nucleonics 21: 54-57.

162. Musil, J. 1962. Vliv opakovanych [illegible]unich rtg vysentreni zen na vyvoj pristi generace. Rozhledy Tuberk. 22: 16-169.

163. Lejeune, J., R. Turpin and M.O. Tethore. 1959. Les enfants nes de parents irradies (cas particulier de la sex ratio). Ninth Intern. Congr. Radiol. pp. 1089-1095. G. Thieme Verlag. Munich.

164. Gentry, J.T., E. Parkhurst and G.V. Bulin. 1959. An epidemiological study of congenital malformations in New York State. Am. J. Public Health 49: 497-513.

165. Gianferrari, L., A. Serra, G. Monganti, A. Cresseri, A. Bonino, W. Gualandri and G. C. Arena. 1961. A genetic survey in some Italian areas with different background radiation levels. Second Intern. Conf. Human Genet. (Rome). Abst. 103. Amsterdam. Excerpta Med.

166. World Health Organization. 1959. Effects of radiation on human heredity: Investigations of areas of high natural radiation. Tech. Rept. No. 166. pp. 1-47.

167. Schull, W.J. and J.V. Neel. 1958. Radiation and the sex ratio in man. Science 128: 343-348.

168. Schull, W.J., J.V. Neel and A. Hasizume. 1966. Some further observations on the sex ratio among infants born to survivors of the atomic bombings of Hiroshima and Nagasaki. Am. J. Human Genet. 18: 328-338.

169. Cox, D.W. 1964. An investigation of possible genetic damage in the off-spring of women receiving multiple diagnostic pelvic X-rays. Am. J. Human Genet. 16: 214-230.

170. Report of the United Nations Scientific Committee on the Effects of Atomic Radiation. 1966. Suppl. No. 14 (A/6314).

171. Abrahamson, S., H.U. Meyers, E. Himoe and G. Daniel. 1966. Further evidence demonstrating germinal selection in early premeiotic germ cells of Drosophila males. Genet. 54: 687-696.

172. Lefevre, G. and U.-B. Jonsson. 1964. The effect of mating intensity on mutation frequency patterns detected after irradiation of Drosophila melanogaster males. Genet. 50: 879-890.

173. Oster, I.I. 1958. Frequency dosage relations for mutations following X-irradiation of sensitive and resistant germ cells. Proc. 10th Intern. Genet. Congr. 2: 210-211.

174. Oftedal, P. 1964. Radiosensitivity of Drosophila spermatogonia. I. Acute doses. Genet. 49: 181-193

175. Crow, J.F. and R.J. Temin. 1964. Evidence for the partial dominance of recessive lethal genes in natural populations of Drosophila. Am. Naturalist 98: 21-33.

176. Parker, D.R. 1959. The induction of recessive lethals in Drosophila oocytes. Genet. 45: 135-138.

177. Himoe, E. 1966. Dose-rate independence of radiation induced mutation in Drosophila females. (abstr.). Genet. 54: 339.

178. Muller, H.J., I.I. Oster and S. Zimmering. 1963. Are chronic and acute gamma irradiation equally mutagenic in Drosophila? In Repair from Genet. Rad. Damage. F.H. Sobels, Ed. pp. 275-304. Pergamon Press. New York.

179. Oftedal, P. 1964. Radiosensitivity of Drosophila spermatogonia. II. Protracted doses. Hereditas 51: 13-30.

180. Oftedal, P. 1964. Radiosensitivity of Drosophila spermatogonia. III. Comparison of acute and protracted irradiation efficiencies in relation to cell killing. Mutation Res. 1: 63-76.

181. Abrahamson, S. and L.D. Friedman. 1964. X-ray induced mutations in spermatogonia cells of Drosophila and their dose-frequency relationship. Genet. 49: 357-361.

182. Russell, W.L. 1965. Studies in mammalian radiation genetics. Nucleonics 23: 53-56, 62.

183. Russell, W. L., L. B. Russell and E.M. Kelly, 1959. Dependence of mutation rate on radiation intensity. In Immediate and Low Level Effects of Ionizing Radiations. A.A. Buzzati-Traverso, Ed. pp. 311-320. Taylor and Francis, Ltd. London.

184. Russell, W.L. and E.M. Kelly, 1965. Mutation frequency in female mice exposed to small X-ray dose at high dose rate. (Abstr.) Genet. 52: 471.

185. Russell, W.L. 1965. Effect of the interval between irradiation and conception on mutation frequency in female mice. Proc. Natl. Acad. Sci. (US) 54: 552-554.

186. Russell, W.L. and E.M. Kelly. 1966. Mutation frequency in female mice exposed to high-intensity X-irradiation delivered in small fractions. Science 154: 427-428.

187. Purdom, C.E. 1963. The effects of intensity and fractionation on radiation induced mutation in Drosophila. In Repair from Genet. Rad. Damage. F.H. Sobels, Ed. pp. 219-235. Pergamon Press. New York.

188. Tazima, Y. 1965. Mechanisms controlling two types of dose-rate dependence of radiation-induced mutation frequencies in silkworm gonia. Japan. J. Genet. Suppl. 40: 68-82.

189. Baldwin, W.F. 1965. Visible mutation frequencies in Dahlbominus oogonia produced by acute X-rays and chronic gamma-radiation. Mutation Res. 2: 55-59.

190. Benedict, Ruth. 1964. The Chrysanthemum and the Sword. Houghton Mifflin. Boston.

191. Takeo, Doi L. 1962. Amae: A key concept for understanding Japanese personality structure. In Japanese Culture, Its Development and Characteristics. R.J. Smith and R.K. Beardsley, Eds. Viking Fund Publ. Anthropol. No. 34. p. 421.

192. Sargent, W. 1957. Battle for the Mind. p. 38. Doubleday. Garden City, N.Y.

193. Glass, A.J. 1957. Observations upon the epidemiology of mental illness in troops during wartime. In Symposium on Preventive and Social Psychiatry. pp. 185-198. Walter Reed Army Inst. Res. U.S. Govt. Printing Office. Washington, D.C.

194. Shizume, E.Y. and D.J. de S. Price. 1962. Letter to the Editor. Bull. Atomic Sci. 18: 29.

195. Tsuzuki, M. 1947. Report on the Medical Studies of the Effects of the Atomic Bomb, in General Report, Atomic Bomb Casualty Comm. pp. 67-111. Natl. Res. Council. Washington, D.C.

196. Tomonaga, Masanobu. 1966. Genbakushō (atomic bomb disease). Nagasaki Bunka 19: 1.

197. Frank, J.D. 1946. Emotional reactions of American soldiers to an unfamiliar disease. Am. J. Psychol. 102: 631-640.

198. Hollingsworth, James W. and Paul S. Anderson, Jr. 1961. Adult Health Study, Hiroshima. Preliminary report. 1958-59. ABCC Tech. Rept. 11-61.

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13. ABSTRACT

These proceedings on the selected effects of a general war present the results of the first conference which was held at Princeton, January 18-21, 1967.

Topics discussed were: Somatic Effects from Blast, Thermal, Acute Radiation and Chronic Radiation; Genetic Effects on Children of Hiroshima and Nagasaki, Ionizing Radiation and Chromosomal Changes, and Genetic Studies of Drosophila in Mice and Humans; and Psychosocial Effects including Psychosocial Response in Hiroshima. Social Impact on Hiroshima and Nagasaki, and Attitudinal implications for other populations. The conference was sponsored by the Defense Atomic Support Agency under the auspices of the New York Academy of Sciences. Documentation of subsequent conferences on the same subject are planned.

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