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CUMULATIVE EFFECTS OF BORANE TOXICITY AS REVEALED BY A CLINICAL TEST

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Biomedical Laboratory

Aerospace Medical Division

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WRIGHT AIR DEVELOPMENT DIVISION

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Project No. 7165

Task No. 71836

WRIGHT AIR DEVELOPMENT DIVISION
AIR RESEARCH AND DEVELOPMENT COMMAND
UNITED STATES AIR FORCE
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

FOREWORD

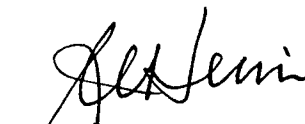
This work was performed under Project No. 7165, "Health Hazards of Materials and Radiation," Task No. 71836, "Evaluation and Control of Toxic Chemical Materials," administered by the Toxic Hazards Section, Physiology Branch, Biomedical Laboratory, Aerospace Medical Division, of the Wright Air Development Division. The assistance of Capt. F. Kriewaldt, USAF, VC, Capt. L. Doerr, USAF, VC, and Capt. James Prine, USAF, VC, for veterinary services is gratefully acknowledged.

Animal experimentation was performed in accordance with the Rules for Animal Care as established by the American Medical Association.

ABSTRACT

A series of studies on various aspects of the toxicology of the boron-derivative, high-energy fuels is presented. These studies, made possible by a new technique, supply information on the absorption and transport of boranes in the body. Evidence for the chronic buildup of boranes in the body is presented. The animal data is correlated with studies and observations of accidental human exposures and the analytical technique is evaluated.

PUBLICATION REVIEW



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INTRODUCTION

In May 1952, work was begun with boron hydrides (or boranes) as a source for more powerful high-energy fuels (HEF) and propellants. This new class of compounds was toxic and presented a definite health hazard to manufacturing and handling personnel. In spite of care and observance of general principles in handling toxic substances, the number of cases of borane intoxication recorded in the medical literature has been mounting steadily. Rapid absorption by inhalation or through the skin, the marked susceptibility of the nervous system, and the lack of immediate warning symptoms are major factors in borane toxicology. Because the theoretical energy yields are so promising, a much wider application of the boranes is expected and the number of potential exposures will increase.

Although a number of reports have appeared on borane toxicity (refs. 1, 3, 5-9) experimental data on serum borane levels and their correlation to the severity of intoxication has not been available. In addition, the lack of data on the absorption and elimination of boron hydrides has been responsible for many diagnostic problems in the past. This lack of data was due to no simple and specific test being available for the detection of boron hydrides in blood or biological material.

This technical report describes a series of studies on the toxicity of the boron hydrides, possible with the development of a test for boron hydrides in serum. As described in WADC TR 59-123 (ref. 4), these studies were designed to augment our knowledge of borane toxicity and explain some observations of accidental human exposures. Caution should be exercised against rigid extrapolation of the absolute values from animal experiments to humans. However, we feel that the general principles illustrated by these studies apply to humans and contribute to better understanding of the basic problems.

EXPERIMENTAL DESIGN

The studies are reported in the order performed. This order needs brief explanation:

A. Acute-Lethal Studies

The acute-lethal studies were initiated to determine the rate of absorption of HEF-3 and to relate serum borane levels to the clinical states of the animals.

B. Acute-Sublethal Studies

Acute-sublethal studies were designed to determine the duration and extent to which the boranes are present in the serum following acute exposure.

C. Subacute-Symptomatic Studies

The subacute-symptomatic studies were undertaken to establish the range of dosage levels of HEF-3 which must be absorbed on successive days to produce various states of toxicity but, on a single occasion, would produce no manifestations.

D. Subacute-Asymptomatic Studies

The subacute-asymptomatic studies were undertaken to establish the limits of borane absorption which would not cause overt toxicity.

E. Rate-of-Recovery Studies

Finally, to clarify the rate of recovery from borane toxicity, first to a state of freedom from symptoms and second to freedom from all toxic effects, we designed two separate studies.

F. Serum from Accidental Exposure Cases

Serum from 27 accidentally exposed workers was analyzed.

All dogs weighed between 10 and 21 kg. They were quarantined for 21 days and tested for leptospirosis and histoplasmosis. Their diet consisted of commercial dried dog food and horsemeat. Rabbits were the New Zealand white strain, weighing between 2.2 and 4.0 kg., and were fed Purina rabbit chow. Tuberculin-negative rhesus monkeys, weighing between 7.1 and 7.8 kg., were fed fresh fruit, vegetables, dairy products, and Purina monkey chow.

The boranes used in these studies were decaborane and its derivative fuel, HEF-3, both of high purity. Olive oil was used as a vehicle for injecting the boranes. Various concentrations of boranes in olive oil, an individual dose averaging about 0.5 ml., were administered intraperitoneally. Some skin applications were attempted, but due to the additional exposure through inhalation they were abandoned.

METHODS AND RESULTS

A. Acute Lethal Studies

We designed the acute-lethal studies to correlate the clinical picture and serum borane levels in acute exposures to boranes. We hoped good correlation could be found between the serum levels and symptomatology. Due to the difference in symptoms

between acute and chronic exposures, adequate dosage was assured to produce severe acute effects without causing instantaneous death. The dosage administered was approximately LD₁₂₅. Three dogs were subjected to 15 mg./kg. of HEF-3 in oil injected intraperitoneally. Venous blood samples of 3 to 4 ml. were drawn at stated times (figure 1) and analyzed as described in an earlier publication (ref. 4).

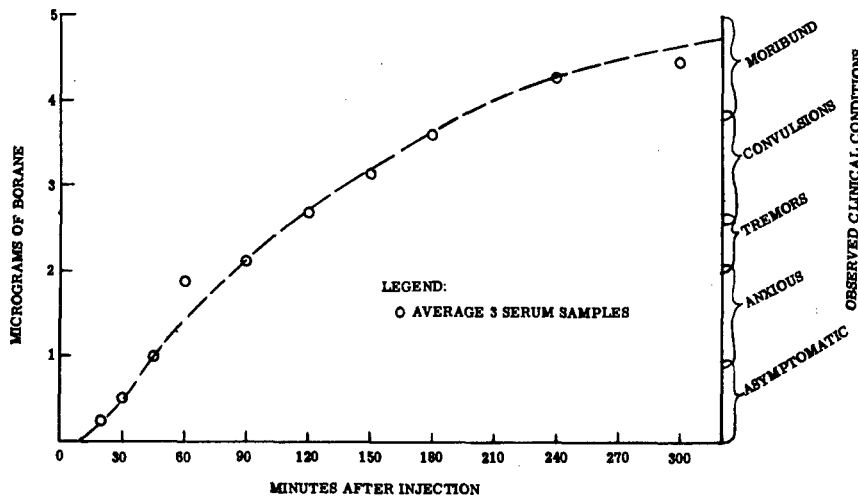


Figure 1. Serum Borane Levels and Observed Clinical Condition.
Acute-Lethal Dose of HEF-3

The results of the serum borane analyses are illustrated in figure 1. The animals exhibited a series of clinical states of increasing severity. For the first 30 minutes they were friendly and inquisitive, seeming completely well. Detectable borane levels were first observed about 15 minutes after injection, and reached 0.85 $\mu\text{g.}/\text{ml.}$ before signs of toxicity were noted. After approximately 45 minutes, as the serum borane level reached 1 $\mu\text{g.}/\text{ml.}$, they became anxious and uneasy. All animals vomited and suffered irregular tremors but at no time appeared in pain. Within 1 hour the serum borane levels ranged between 1.5 and 3 $\mu\text{g.}/\text{ml.}$ At this time tremors and muscular incoordination developed. Seizures began with increasing severity 90 to 120 minutes after exposure. The convulsions lasted from 60 to 90 seconds each, interrupted temporarily by apnea and resumed with renewed respiration. The blood levels within this stage ranged from 2.4 to 3.8 $\mu\text{g.}/\text{ml.}$ No drugs were given to modify the seizures and the animals soon became moribund for periods of 1 to 5 hours before death. The serum borane now ranged from 4 to 4.8 $\mu\text{g.}/\text{ml.}$

B. Acute-Sublethal Studies

We designed the acute-sublethal studies to investigate the duration of serum borane levels following a single intraperitoneal injection of HEF-3. We sought information on how high the serum concentration would be and how long detectable levels would be maintained.

Groups of 3 dogs and 3 rabbits each were subjected to 1.0, 3.0, 5.0, and 7.0 mg./kg. HEF-3. Venous samples were drawn preceding exposure and at intervals of 4, 24, 48, 72, and 96 hours.

The results of this experiment are presented in figures 2 and 3 and illustrate several new points about single, acute exposures. The presence of boranes in the circulating blood is temporary, the duration varying with the magnitude of the dosage. Following injections of larger amounts, the boranes were detected for periods of 6 to 7 days, whereas small asymptomatic doses are cleared from the blood in 24 to 36 hours.

C. Subacute-Symptomatic Studies

The subacute-symptomatic studies were designed to establish the range of dosage levels of HEF-3 absorbed on successive days to produce various states of toxicity but, on a single occasion, would produce no symptoms. We realized that dosage levels producing signs of toxicity might be well above the levels producing symptoms. However, well defined objective signs were the only available end point. In the acute-sublethal studies described above, 1 mg./kg. did not produce any signs although an elevated, blood borane level was noted.

Therefore, several species of laboratory animals were injected with 1 mg./kg. of HEF-3 in olive oil on successive days until a state of intoxication was produced. The

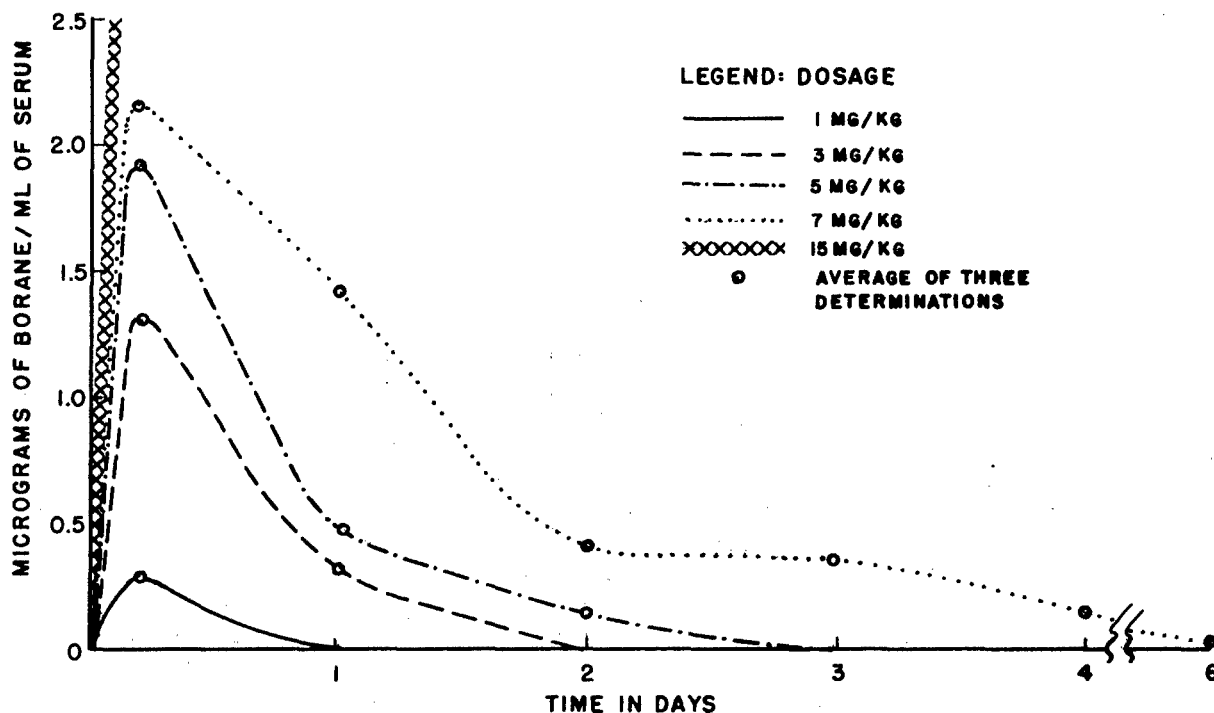


Figure 2. Serum Borane Levels in Dogs Following Acute Absorption of HEF-3

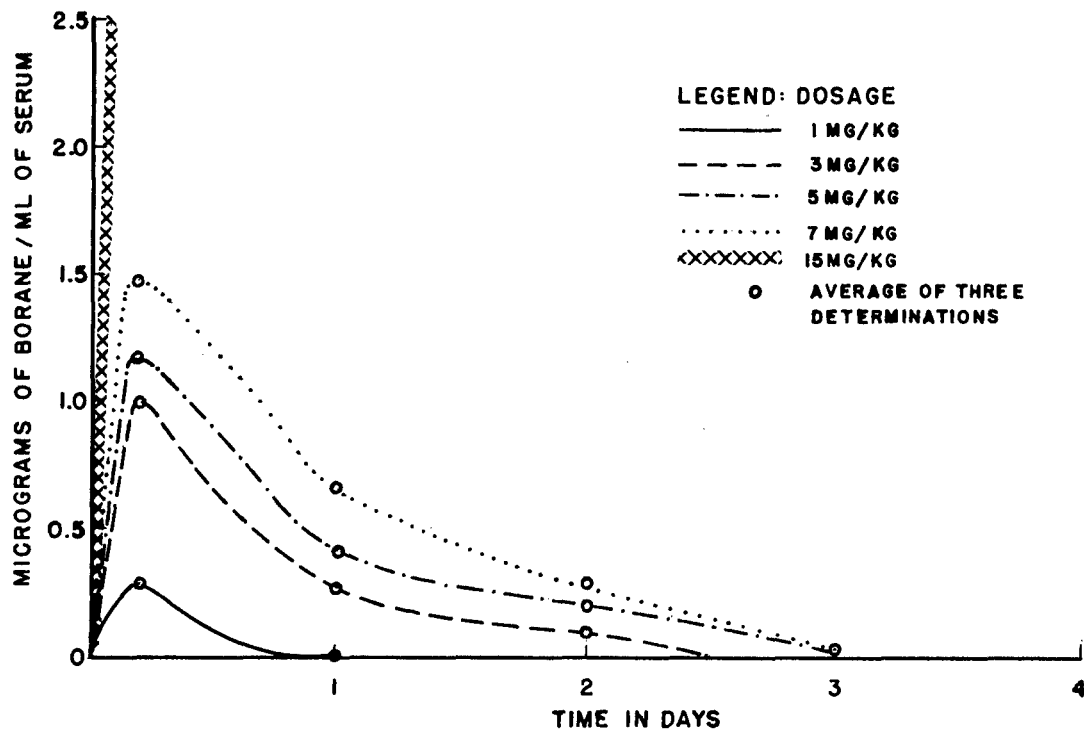


Figure 3. Serum Borane Levels in Rabbits Following Acute Absorption of HEF-3

injections were continued for 1 day after the onset of the first questionable signs of toxicity, so that doubtful information would not be recorded. Similarly, other animals were injected at the same dosage level but for a period shorter by 2 days. The animals were observed by several individuals throughout the period of injection and for 1 week following, in case latent effects of toxicity occurred. Animals in this series included 6 rhesus monkeys, 12 dogs, and 12 rabbits. The results are shown in figure 4. The only signs of toxicity observed were related to central nervous system (CNS) depression. Regardless of the species, the signs of toxicity included decreased motor activity, decreased interest in surroundings, and a stuporous state. None of the rabbits injected for 7 successive days developed signs of toxicity but the 6 rabbits injected for 9 days did. Likewise, the dogs injected for 5 days remained clinically well throughout the experiment while the 6 dogs receiving two additional injections became depressed. Although the monkeys appeared well after several injections, the onset and degree of toxicity could not be accurately determined because the monkeys quickly learned to "play sick" following the injection of even 1 ml. of physiological saline.

D. Subacute-Asymptomatic Studies

Repeated small doses of boranes can have an accumulative toxic effect. After preliminary trials the following tissue analysis method was followed:

The animals were sacrificed by exsanguination under ether. About 10 gm. of tissue was removed from the central portion of the liver. This was homogenized with equal parts of distilled water in modified blenders (ref. 3). The homogenate was the starting material and 2 ml. was extracted and treated. The brain tissue was analyzed by boiling 1 gm. with 10 ml. curcumin reagent (ref. 4). This was dried and treated in the same fashion. Brain and liver tissue analyses of 10 control animals, 5 rabbits and 5 dogs, treated in the manner were negative.

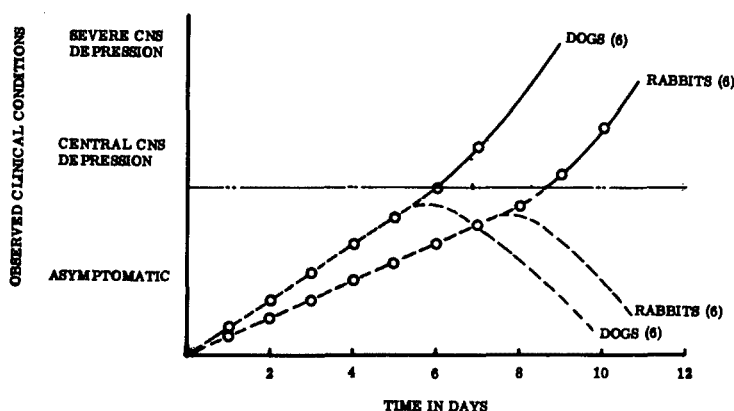


Figure 4. Effect of Continued Daily Absorption of 1 mg./kg. of HEF-3

These preliminary tissue-borane studies suggested that the boranes were not immediately excreted from the body but remained there for several days or longer. This and the results of the previous experiments suggested a cumulative effect. Therefore, we attempted to define the ability of the body to cope with these materials.

The first problem was to establish threshold levels of borane hydrides that could be absorbed daily without cumulative chronic buildup or overt signs of toxicity. To do this, 6 dogs and 6 rabbits, in groups of 3 each, were subjected for 1 month to constant daily doses of 25, 100, and 250 $\mu\text{g.}/\text{kg.}$ of HEF-3. Weekly borane serum levels and daily observed symptoms were recorded. In addition, brain and liver tissues were analyzed as described above.

The results are listed in table I. The animals appeared well throughout the experiment. Both rabbits and dogs showed slight weight gain and were alert, friendly, and inquisitive for the entire period. Serum borane levels at weekly intervals were negative. Analyses for boranes in liver and brain tissue of the animals receiving 25 $\mu\text{g.}/\text{kg.}$ of HEF-3 daily were negative. However, the liver and brain tissue of animals subjected to 100 or 250 $\mu\text{g.}/\text{kg.}$ of HEF-3 were slightly positive, ranging from 0.1 to 0.32 $\mu\text{g.}/\text{gm.}$ wet tissue, while the serum borane was negative.

E. Rate-of-Recovery Studies

Due to the chronic buildup in the subacute studies we questioned how long the boranes affected the body. It was pertinent to know whether the cumulative effects were to be measured in days or weeks. The problem was more complex due to the fact a normal-appearing animal might have substantial levels of boron hydrides or derivatives present in various tissues. Our question was now extended to include not only how long

TABLE I
TISSUE ANALYSIS

Animal	Daily Dosage	Serum Borane Levels	Clinical State	µg. Borane per gm. Wet Tissue	
				Brain	Liver
Rabbit	0.025 mg./kg.	0	Well throughout	Negative	Negative
Rabbit	0.025 mg./kg.	0	Well throughout	Negative	Negative
Rabbit	0.025 mg./kg.	0	Well throughout	Negative	Negative
Rabbit	0.250 mg./kg.	0	Well throughout	Negative	0.10
Rabbit	0.250 mg./kg.	0	Well throughout	0.10	0.12
Rabbit	0.250 mg./kg.	0	Well throughout	Negative	0.11
Dog	0.100 mg./kg.	0	Well throughout	Negative	0.18
Dog	0.100 mg./kg.	0	Well throughout	Negative	0.21
Dog	0.100 mg./kg.	0	Well throughout	Negative	0.18
Dog	0.250 mg./kg.	0	Well throughout	0.12	0.25
Dog	0.250 mg./kg.	0	Well throughout	0.15	0.32
Dog	0.250 mg./kg.	0	Well throughout	0.12	0.30

it took to detoxify or eliminate boranes to the point of no clinical manifestations, but how long latent toxicity might persist. The studies on the rate of recovery were performed in two series:

First Series:

The first series established the normal rate of recovery. HEF-3 was administered in various dosage schedules to a number of animals, causing moderate to severe sickness. The HEF-3 was then withheld and the animals were allowed to recover without benefit of therapy. The clinical state of the animals, the dosage, and the time for recovery were recorded to establish the natural speed of recuperation.

HEF-3 dosage schedules were designed to cover acute, massive intoxications as well as subacute intoxications with a gradual buildup of boranes in the body:

- a. Six monkeys were injected with 1 mg./kg. on 7 of 9 successive days.
- b. Six dogs were injected with 1 mg./kg. for 12 successive days.
- c. Three dogs were injected with 3 mg./kg. for 2 successive days.
- d. Three dogs were injected with 7 mg./kg. as a single exposure.

Observations on Rate of Recovery - First Series:

The monkeys subjected to 1 mg./kg. of HEF-3 for 7 of 9 successive days were left severely depressed. Two monkeys were unconscious for several hours. Twenty-four hours after the last injection the monkeys lay huddled in corners showing neither concern nor response to stimulation. We noted no spontaneous movements and little interest in food. At 48 hours, the animals sat motionless for long periods. At 4 days, the monkeys again became interested in their environment but moved only cautiously and guardedly. Eating habits were normal. On the sixth day motor activity was spontaneous and quick. Interest in their environment returned but total activity was reduced and the animals remained relatively inactive for occasional periods. By the eighth day after the last injection the animals appeared normal.

The six dogs, subjected to 1 mg./kg. of HEF-3 daily for 12 days, became unconscious and unresponsive to mild stimulation. Following the final injection the dogs remained unconscious for about 24 hours. No convulsions were noted. Forty-eight hours later the dogs were stuporous. Little attention or interest in food was observed and a mucous diarrhea developed. On the fourth day the dogs were somewhat more responsive, occasionally moving about the runs in guarded fashion. The diarrhea ceased. On the sixth day the dogs were more alert, friendly, responsive to attention, and ate well. By the eighth day the dogs appeared normal.

The three dogs subjected to 3 mg./kg. of HEF-3 for 2 successive days developed signs commensurate with moderate CNS depression. For 48 hours following the last injection, the animals were stuporous and showed little interest in their environment. At no time did we note tremors, convulsions, or loss of consciousness. Little spontaneous activity was noted. The dogs stood or lay still for hours at a time. On the third day the animals moved about more freely but were still guarded in their actions. On the fourth day the animals appeared normal. No residual effects were noted.

The three dogs subjected to a single exposure of 7 mg./kg. of HEF-3 developed convulsions within 12 hours of injection. They were deeply depressed but conscious 24 hours later. A moderate diarrhea lasting about 36 hours developed. Forty-eight hours after injection a little spontaneous movement was noted. The dogs lay or stood essentially motionless for hours. On the fourth day they resumed eating regularly, moved about quietly and slowly, and acknowledged the observer with little interest. On the sixth day the dogs were alert, friendly, and inquisitive, but less active than normal. By the seventh day the dogs appeared normal.

Second Series:

The second series simulated repeated, frequent (occupational) exposures. HEF-3 (1 mg./kg.) was administered daily to the animals, causing intoxication. They were then allowed to recover to the point where they appeared clinically well to all observers. Then we resumed the injections of HEF-3 (1 mg./kg.) and continued until the animals were again brought to the same general level of illness from which they had just recovered. At this point we withheld the HEF-3 for 2 weeks, when the daily administration of 1 mg./kg. was started for the third time. We continued these injections until the animals were again brought to the same general level of intoxication. The time, dosage schedule, and clinical state were recorded carefully.

Observations on Rate of Recovery - Second Series:

The six monkeys were allowed to recover from moderate intoxication as indicated by the pattern of the first series. They then received two successive daily injections of HEF-3. They remained well and, after 2 days, two additional injections were given.

They again became severely ill, four being unconscious for 4 to 8 hours. On the third day following this exposure the animals were stuporous and depressed. By the fifth day they were again interested in their environment but were cautious and exhibited little spontaneous movement. On the seventh day, the animals appeared normal. After 7 additional days, daily injections were resumed. After the fourth injection, the animals developed signs of nervous system depression. Injections were now withheld and the animals again began to recover. They were free of signs of toxicity 4 days later, suggesting no change in rate of recovery.

The dogs recovering from severe intoxications were injected with 1 mg./kg. of HEF-3 on 2 successive days beginning on the first day they appeared completely well. Following the second injection, they became extremely toxic, and four animals were completely unresponsive for almost 24 hours. The recovery pattern observed here was uneventful. With the injections of HEF-3 after the 2-week period for 5 successive days, a state of mild intoxication again developed. All animals recovered from this final injection and appeared normal within 5 days.

F. Serum from Accidental Exposure Cases

A limited number of serum samples was available for analysis from 27 accidentally exposed workers. Two workers were acutely exposed to milliliter amounts of HEF-3 by skin contamination. Exposed areas were washed within minutes. Serum samples were taken 4 hours after exposure and at daily intervals. Serum was drawn from seven workers suffering from acute exposure to pentaborane and from 18 workers moderately sick from what was considered chronic pentaborane and decaborane exposure.

Serum determinations of the workers acutely exposed to HEF-3 reached 0.5 µg./ml. and 0.6 µg./ml. 4 hours after exposure. Serum boranes diminished over the following 2 days and were undetectable on the third day. These men remained free of symptoms during the whole episode. The 7 workers suffering from acute exposure to pentaborane had values of 0.7 µg./ml. to 1.3 µg./ml. during the early course of their illnesses. Samples collected from the 18 chronic exposure cases yielded 15 negative values and 3 positive values, ranging from 0.15 to 0.22 µg./ml.

Pathology

Examination of animal tissues revealed no gross or microscopic changes specifically attributable to HEF exposure. A bacterial pneumonia was noted in several animals rendered unconscious by HEF-3. In animals recently injected, minute fat droplets compatible with olive oil were noted on peritoneal surfaces.

SIGNIFICANT FEATURES OF BORANE INTOXICATION

The acute-lethal studies illustrate the rapid absorption of HEF-3 and establish a correlation between the serum borane level and observed clinical condition. The clinical appearance of the animals could be consistently related to a range of serum borane values. Detectable borane levels appeared within 15 minutes after exposure and rose rapidly to a peak in 4 or 5 hours. These studies emphasize the need for rapid decontamination of HEF spills. Skin application studies performed by this laboratory indicate rapid penetration of intact skin. Detectable serum levels were also reached within 15 minutes, again indicating the need for immediate skin decontamination.

The observation that serum borane levels are elevated for 3 or 4 days after moderate exposures and up to 7 days after more severe exposures indicated that cumulative effects

are possible. The subacute-symptomatic studies augmenting the work of Svirbely (refs. 7, 9) demonstrate that daily administration of small doses of HEF produces no overt toxicity but, if continued for a sufficient period, results in a gradual build-up of toxic substances in the body ultimately producing death. The subacute-asymptomatic studies reveal the presence of boranes in liver and central nervous system tissue following repeated exposures to amounts of boranes insufficient to cause signs of toxicity. Immediately following recovery from the acute toxic effects, there still may be a residual or subthreshold level of toxic materials. During this period re-exposure to even a small amount of HEF, normally of no consequence, results in severe intoxication. However, when animals were given sufficient time to recover, in our studies 7 additional days, no residual susceptibility was noted. Men repeatedly exposed to boranes have quickly developed signs and symptoms following relatively minor exposures normally of no consequence. To the authors, these observations indicate that the boranes do have a cumulative effect, a significant feature of borane toxicity.

We questioned whether the delayed appearance of toxic signs in the subacute-symptomatic studies were due to a clear-cut accumulation of fuel or to some particular degradation product, requiring a certain amount of time to form. The 12 animals subjected to the same dosage schedule but for a slightly shorter period developed no signs of toxicity. We therefore felt that the boranes were indeed being absorbed but were not present in sufficient concentration to precipitate overt toxicity. In our experience, the onset of clinical toxicity was never delayed more than 24 hours after the last exposure.

CONCLUSIONS AND RECOMMENDATIONS

The diagnostic value of the test in acute exposure (ref. 4) is well substantiated by both animal experimentation and limited human experience. Including the borane test as a routine procedure in the medical examination of all production and handling personnel may help to detect acute asymptomatic exposure cases, directing attention to faulty handling techniques.

A negative test does not exclude the presence of chronic borane intoxication, especially when low-level, daily occupational exposures have led to tissue saturation with boranes.

Since a cumulative mechanism appears to exist sufficient time for complete recovery should be allowed before returning moderately symptomatic exposure cases to borane-handling areas.

Due to demonstrated temporary deposition of borane in the liver and central nervous system, the pre-employment and periodic physical examinations should include electroencephalograms and liver profiles.

Careful long term followup studies of exposure cases are clearly indicated to preclude unnoticed development of late organic impairments. Subjective symptomatology may be the only sign of chronic intoxication. Thus, careful case histories should be maintained.

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