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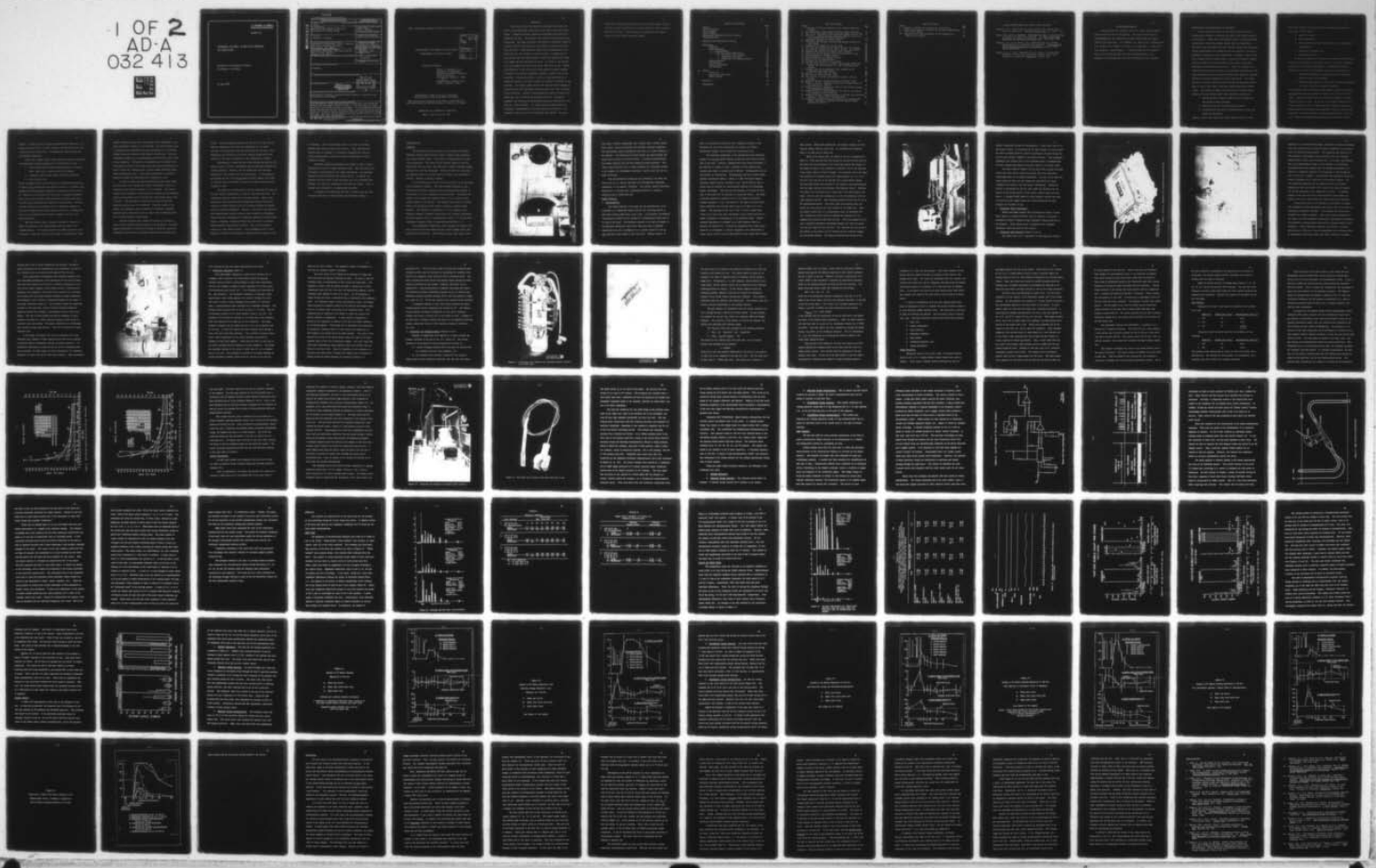

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INTRAVENOUS GAS EMBOLI IN MAN AFTER COMPRESSED AIR OCEAN DIVING

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Non-symptomatic venous gas emboli (vge) in man after ocean diving was studied with the Doppler "Bubble Detector." Curves of vge score post-dive after several dive profiles is presented. The relationship between at-depth exercise and post-dive vge was studied. Lower levels of vge were found with moderate exercise underwater than with either resting or heavy exercise. Exercise-induced perfusion increases during diving can increase N_2 uptake and, thus, vge formation.

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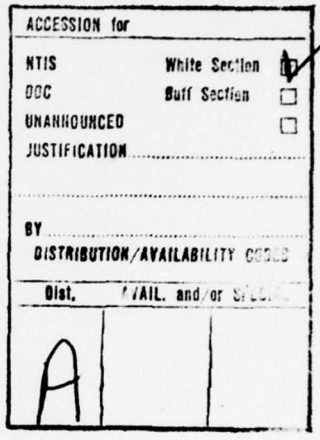

INTRAVENOUS GAS EMBOLI IN MAN AFTER COMPRESSED AIR OCEAN DIVING

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ABSTRACT

This 3-year project was directed at defining the presence and extent of non-symptomatic venous gas emboli (vge) in man after ocean diving. A Doppler ultrasonic flowmeter (precordial) was used for obtaining the vge data. The post-dive time course of vge occurrence was documented. Vge appeared within a few minutes of surfacing, peaked in numbers within an hour post-dive, and declined to control levels by 3 hours post-dive. When monitored during water decompression on the 100 ft./40 min. dive profile, no vge were found. It appears that in these studies vge were alvays present if tissue N_2 tensions were equal to or higher than the theoretical M values. If, however, the tensions were even slightly less than the M values, there were no vge. Significant differences in vge scores were found between individual subjects. In addition, each subject consistently exhibited a specific level of vge occurrence. Human ocean dives, as well as, dog experiments in a hyperbaric chamber were done to define the influence of exercise on vge formation. The animal studies showed that exercise induced changes in tissue perfusion can substantially effect N_2 uptake and, thus, the extent of vge occurrence. Exercise during hyperbaric exposure increases N_2 uptake and, thus, increases vge formation post-dive. Specialized equipment was developed for quantifying human work performance in the open sea. These included: 1) a subject-worn recording system for acquisition of physiological and environmental data underwater, 2) an underwater ergometer and 3) an underwater gas sampler. The open

ocean human studies showed that there was a trend for higher levels of vge after moderate exercise levels at depth than after either resting or heavy levels of work. These results are explained on the basis of exercise and cold induced perfusion changes.

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PUBLICATIONS RESULTING FROM THIS CONTRACT

Pilmanis, A.A.: Intravenous gas emboli in man after selected open ocean air scuba dives. Undersea Biomed. Res. 1(1), pA18, 1974.

Dwyer, H.J. and A.A. Pilmanis: Physiological Studies of Divers Working at Depths to 99 FSW in the Open Sea. In: Proc. of the Sixth Symposium on Underwater Physiology, 1975. San Diego, Calif.

Pilmanis, A.A., R.D. Rader, J.K.C. Henriksen and J.P. Meehan: Instrumentation for underwater data acquisition. Biotelemetry II 2nd International Symposium on Biotelemetry, May 21, 1974, Davos, Switzerland.

Pilmanis, A.A., J.K.C. Henriksen and J.H. Dwyer: An underwater ergometer for diver work performance studies in the ocean. Accepted for publication, Ergonomics, January 1975.

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BACKGROUND AND OBJECTIVES OF PROJECT

It has long been accepted that the clinical condition known as decompression sickness is primarily the result of gas bubble formation in the body (1). While direct experimental evidence is generally lacking concerning the link between bubble location and the manifestations, inferences from observations of bubble locations and the well established clinical manifestations have established that this condition is the direct pathological consequence of gas embolization. In addition, there is now sound evidence that gas emboli trigger a series of biochemical changes which are additive to the direct effects of gas emboli in the pathophysiology of decompression sickness (2). Thus, the condition of decompression sickness stems from, and is primarily dependent upon, the inert gas partial pressure gradients developed after hyperbaric exposure between the ambient breathing gas and the body tissues. The degree and rates of various tissue inert gas saturation and desaturation determine if, and to what extent, tissue gas emboli formation and growth occurs. The primary variables associated with the evolution of gas bubbles in the body during and/or after hyperbaric exposure are:

1. ambient pressure (depth of water) and the resulting inert gas partial pressure gradients;
2. bottom time and the resulting inert gas uptake;
3. decompression time and the resulting balance of inert gas uptake and elimination.

However, under actual open ocean diving conditions there are other

factors that influence inert gas uptake and elimination by alterations in circulation. These include:

1. degree of exercise
2. water temperature
3. water immersion and the associated state of cardiovascular weightlessness
4. constrictive equipment worn
5. psychological factors

In the past definition of decompression sickness has been entirely based on overt symptoms in the intact subject or on direct visualization of bubbles in acute animal preparations. Decompression tables for returning to sea level from hyperbaric exposures are derived from:

1. mathematical modeling of hypothetical tissue compartment inert gas uptake and elimination;
2. statistical interpretation of occurrence of subjective symptoms in extensive hyperbaric exposure experience.

The relatively recent development of non-invasive methods of detecting in vivo intravascular gas emboli has opened a new and, for the first time, a relatively objective field of study for the problems of decompression sickness in man. By the use of the Doppler ultrasonic flowmeter, modified for use as a bubble detector, definitive evidence has been obtained that circulating non-symptomatic venous gas emboli (vge) exist after certain dive profiles previously considered "safe" (3, 4, 5, 6, 7, 8). These non-symptomatic gas emboli have been termed "silent

bubbles." Despite the lack of apparent symptoms from these vge, it is highly probable that there is tissue damage to working divers over even a relatively short diving career. Circulating gas emboli of any size are potentially dangerous.

There was a definite need to fully explore the potential usefulness and applicability of bubble detection from several points of view:

1. early warning method during working exposures in chamber dives, ocean dives, caisson work, and to altitude;
2. monitoring system during clinical treatment procedures;
3. basic research tool.

Recent investigators have used primarily the transcutaneous flowmeters to detect in vivo intravascular gas emboli in man (6). In addition, both implanted and transcutaneous placements in animals have been used (3, 6). Almost all of the studies have been done in hyperbaric chambers. Thus, many of the inert gas uptake/elimination variables listed above for open ocean diving were not considered in these studies. The task of bubble detection in an immobile man in a hyperbaric chamber is much less complex than the task of bubble detection in a working diver in the ocean environment where pressure change is but one of the influencing factors.

Data was required from a variety of hyperbaric conditions to outline the presence of the "silent bubbles" and the extent of the resulting damage. The overall purpose of this ONR contract has been to attempt to define the occurrence and extent of decompression "silent

bubble" formation in man after ocean diving and the importance of the above influencing variables on vge formation. The initial objective was to demonstrate the feasibility of in vivo intravascular bubble detection during the post-dive period of open ocean air scuba dives. This was immediately followed by a characterization of the post-dive time course of vge occurrence. Development and modification of the Doppler unit was needed in order to record vge during open water decompression in order to define the "starting point" of venous bubble formation during stage decompression dives. These goals required that an objective method for bubble data quantification be developed and used.

In order to define any detected intravascular gas emboli within the framework of the external and internal environments of man, an underwater data acquisition system was to be utilized. Two earlier models of this underwater recording system developed by this laboratory had been successfully used in previous open ocean diving studies (9,10, 11,12,13). The development, fabrication and testing of the third generation system were to be completed. From the results of the studies, the feasibility could be explored of developing a simplified feed-back device which when worn by a diver could immediately alert him to emboli formation in his tissues.

The next objective was to define the influence of exercise at depth on the occurrence and time course of vge formation. The physiological adjustments to exercise are thought to modify the uptake and elimination characteristics of nitrogen during and after hyperbaric ex-

posure. The gas transport dynamics for any dive as a whole may be different depending on the extent and combination of 1) at depth exercise, 2) decompression exercise and 3) post-dive exercise. How extensively vge occurrence is varied by exercise during these three phases of a dive may be important to any decompression schedule.

Theoretically, at depth exercise would increase N_2 uptake by the tissues because of muscle vasodilitation and the resulting increased rate of blood perfusion. However, the few studies that have addressed themselves to this question have been inconclusive. It was proposed that through the use of the in vivo bubble detector method, data would be collected which would define the effect of at depth exercise on gas evolution in blood.

The effects of at depth exercise must be separated from those of exercise during decompression, as well as, from post-dive work. Decompression exercise results in two opposing effects. Early it was thought that mild exercise during decompression should decrease the incidence of bubble formation since the resulting increased circulation increases inert gas elimination. However, in the field it was observed that decompression exercise increased bends incidence by facilitating bubble formation, probably due to mechanical agitation of supersaturated tissues. Similarly, mechanical effects may also result in increased bubble formation with post-dive exercise. This is very noticeable during use of the bubble detectors. If an investigator wants to elicit bubbles to be heard by the Doppler he typically asks the subject to flex

an extremity. The act will usually result in a burst of precordial bubbles where there had been none with rest. Thus, although little objective data is available, it is generally recognized that exercise during decompression or post-dive should be discouraged to reduce incidence of bends.

Additional equipment development was needed in order to define objectively the levels of energy expenditure used. This was done by the development and fabrication of two unique instruments: the underwater ergometer and the underwater gas sampler (9,14). In addition, the incidence and time course of vge occurrence was to be correlated with Workman's M values for surfacing for nine half-time tissues. Thus, a program was developed for a programmable calculator.

A chamber study using dogs was done to further define the role of muscle blood flow on decompression induced bubble formation.

METHODOLOGY

Facilities

All experimental work was done at the University of Southern California Catalina Marine Science Center, located at Big Fisherman Cove, Santa Catalina Island. This facility is ideally located for open ocean experiments. The waters off the Catalina Island lee side are historically calm and clear, with underwater visibility normally ranging between 50 and 100 feet horizontal. Within one-fourth of a mile from the laboratory there are a great variety of bottom types and depths to 200 feet. Weather is rarely a problem.

At the waterfront are located the pier and dock, the diving locker and the hyperbaric chamber. The newly installed double-lock, man rated 100 psi hyperbaric chamber serves several purposes (Figure 1). Its primary use is as a hyperbaric research facility. In addition, the County of Los Angeles has contracted with U.S.C. to treat all local diving accidents at this facility. The chamber also serves as a safety feature and training tool of the Center's extensive scientific diving program. The chamber is a large (24 ft. x 9 ft.) diameter steel cylindrical chamber originally constructed by the Vacudyne Corporation. The total chamber volume is approximately 1,000 cu. ft. with the inner compartment having a volume of approximately 600-700 cu. ft.

The experimental preparations, data reductions and support work were carried out from the Marine Science Center building located 100 yards from the waterfront. This building includes office space, labor-

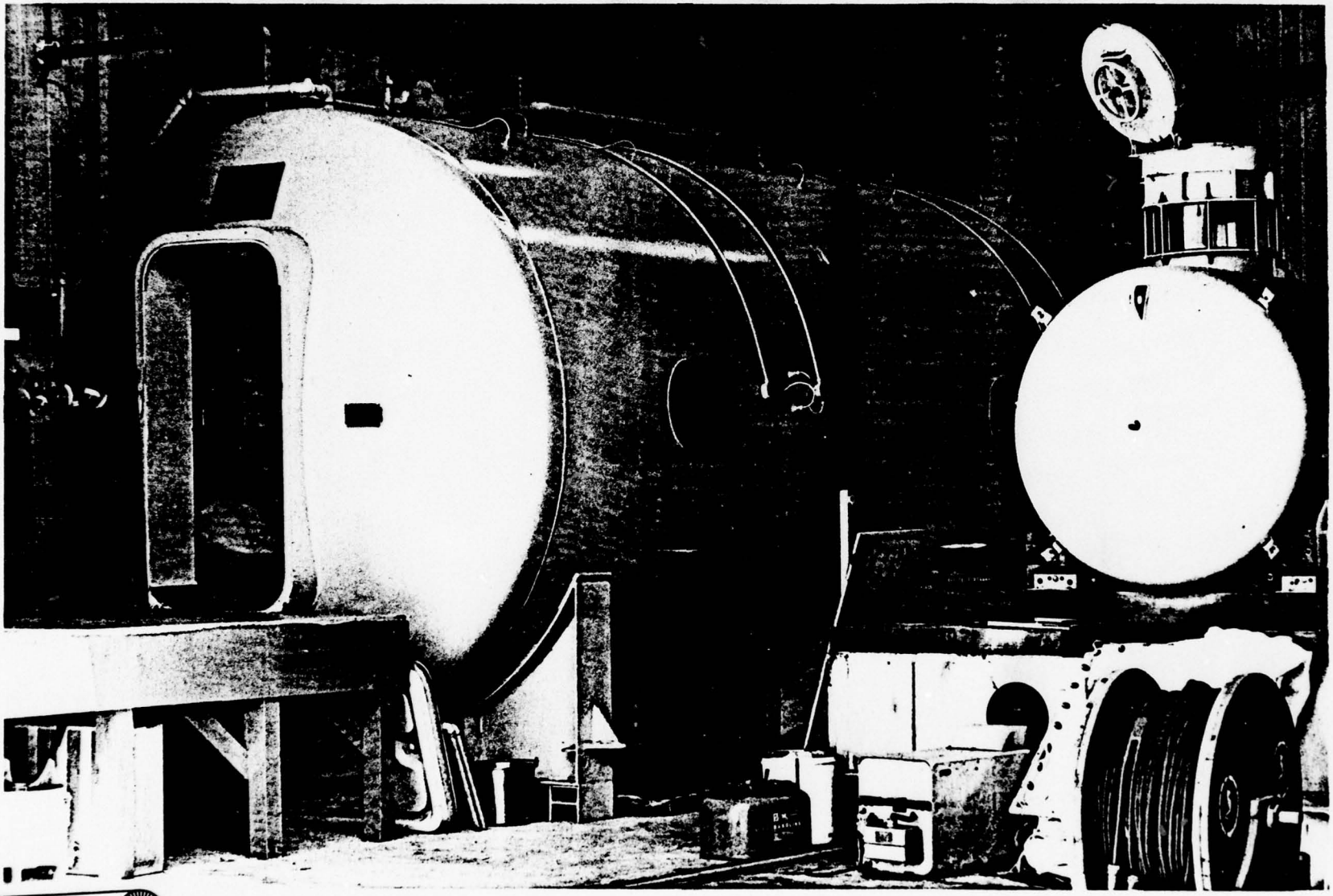

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Figure 1. University of Southern California Santa Catalina Hyperbaric Chamber.

atory space, library, photographic lab, machine shop, constant temperature rooms, electron microscope and moderate electronic equipment. The experimental animal preparations were done in a trailer adjacent to the chamber. The diving site for these studies was approximately 300 yards from the dock in 100 feet of water. A powered diving platform was always anchored over the site during the diving operations. This "open ocean diving test site" is being developed as a permanent facility to be available for investigators requiring a control ocean test site to a depth of 200 feet.

The instrumentation development and assembly of the study was done at the U.S.C. Human Centrifuge and Environmental Physiology Laboratory in Los Angeles, California. This facility contains electronics laboratories, a machine shop, photography laboratory, chemistry laboratory and an immersion tank.

Bubble Detection

a. Instrumentation

The initial objective of the study was the demonstration of the feasibility of a reliable gas emboli detector and recording system in man after working scuba dives in the ocean. An atraumatic non-surgical transcutaneous method for the detection of circulating gas emboli was to be used in the study. The ultrasonic sensors to be used are similar to the ultrasonic flowmeters which had at that time been successfully demonstrated by other investigators to be reliable sensors for the in vivo detection of gas emboli in man ((4, 5, 6, 8). Similar sensors, as

well as the associated electronics were originally developed in this laboratory for use in the measurement of vascular and cardiac dimensional changes, and for blood flow measurement (15).

The electronic modification of the miniaturized blood flowmeter system to an in vivo bubble detection system was done during the first year of the study. However, although bench work proved successful in the detection of bubbles in water, attempts to detect injected intravascular gas emboli in animals proved difficult. Developmental work on this system was discontinued. Simultaneously with the in-house bubble detector development efforts, a Model A 5 MHz Precordial Doppler Ultrasonic Bubble Detector developed by Dr. Merrill Spencer was acquired from the Institute for Environmental Medicine and Physiology, Seattle, Washington. This unit was successfully used during the first year of the project for the detection of venous gas emboli. The large precordial transducer consisted of two $\frac{1}{2}$ inch square piezoelectric crystals separated 1.3 cm. and tilted at a 13^{o} angle so that the ultrasonic transmitter and receiver beams cross in a region 3 to 4 cm distant. The advantage of this unit is that it covers a large tissue volume at its focus and, thus, positioning is less critical and there is higher probablility of detecting vge in the pulmonary blood. Further technical information is available in the ONR Technical Report by Spencer and Johanson (6). It should be emphasized that it takes many hours for an investigator to become competent in the identification of proper sensor location and the differentiation of gas emboli from normal

heart sounds. Within this qualification, the Doppler technique of intravascular bubble detection works well. Its portability and simplicity make it an ideal unit for field work.

Early in the second year, the Model A unit was exchanged for a Model B. This unit was then used during the second and third years of the project for the acquisition of all venous gas emboli data (Figure 2). The Model B detector was modified for underwater use by the extension of the sensor cable to 50 feet in length. The connector end of the cable was enclosed in a pressure proof case (approximately $1\frac{1}{2}$ x 3 x 4 in.). The lid of the case has an O-ring seal and 2 quick-release clamps. The precordial sensor was attached to the subject's chest approximately over the right ventricle with Beckman ECG adhesive collars. Elasticon was placed over the sensor and a strain loop of the cable taped to the waist. The wet suit was carefully put on over this placement with the cable exiting at the hip. This attachment method worked well for all of the decompression dives. The coiled cable and pressure-proof connector box were worn by the diver snapped to his weight belt. When the subject reached his first decompression stop, he unsnapped this unit, released the coiled cable and snapped the connector box to a weighted line lowered to him from the boat. From the weighted line he took a miniature underwater microphone/speaker unit which he placed over his ear inside his wet suit hood. The connector box was pulled to the surface by the tender, the lid removed and the connector plugged into the bubble detector. The topside technician then monitored the

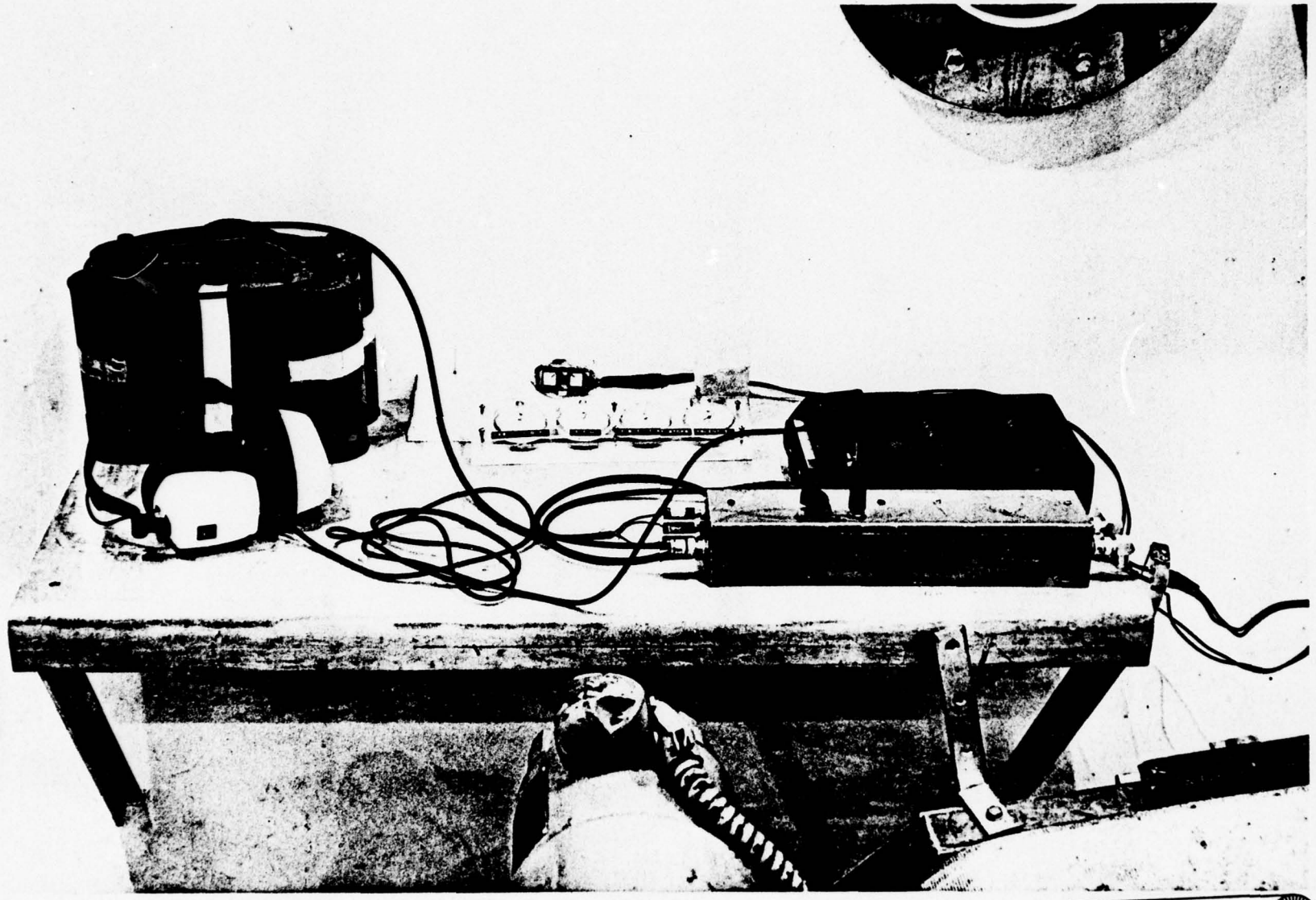

Figure 2. The Precordial 5 MHz Doppler Ultrasonic Bubble Detector

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subject continuously during his decompression. If the sensor was not in the proper location, as determined by the heart sounds, the tender asked the diver to simply adjust the sensor by grasping it directly through the wet suit and moving it slightly in the proper direction. This underwater adjustment was common since in the submerged state of weightlessness the heart changes both shape and location relative to its topside mode.

The same Model B Doppler and the large deep focusing precordial sensor were also used to locate and monitor vge at the pulmonary artery/right heart in the animal experiments. An area on the dog's left chest behind and below the left foreleg was kept shaved for better coupling of the sensor to the skin using a coupling gel. During the periods of monitoring for vge the audio signal was monitored by earphones so that proper position of the sensor could be found and maintained. A modified SONY Model TC-110A Cassette recorder was used to record the audio signal during each monitoring period for later analysis and "scoring" of vge.

b. Underwater Work Performance

Before meaningful Doppler data concerning the effects of underwater exercise on bubble formation could be acquired, an accurate quantitative method of defining energy expenditure during diving had to be developed. Three unique pieces of equipment were designed, fabricated, tested and used for this purpose.

1. Underwater Data Recorder (Figures 3 and 4)

The staff of the U.S.C. Department of Physiology has extensive

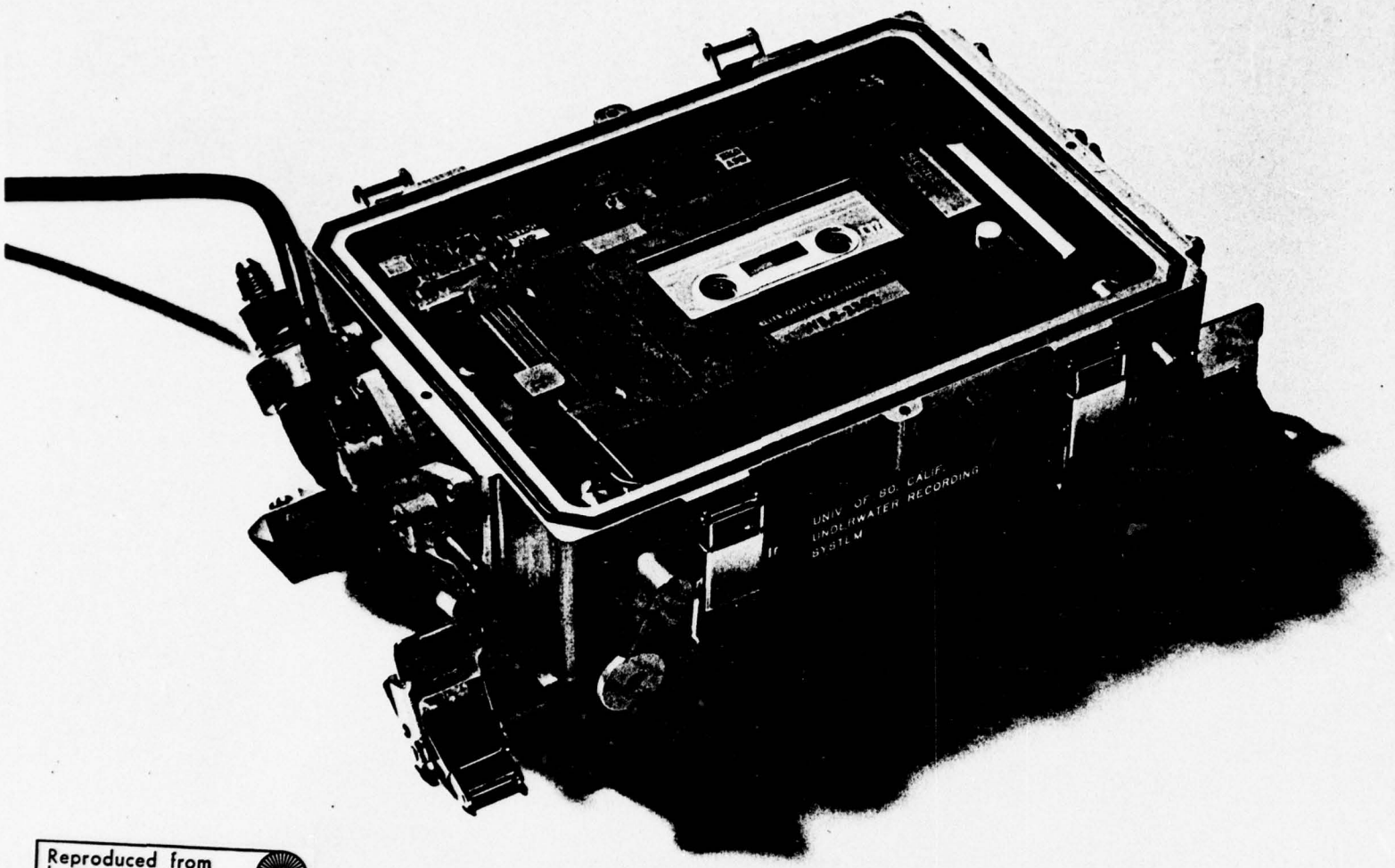

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Figure 3. The U.S.C. Underwater Data Acquisition System with lid off.

Figure 4. The U.S.C. Underwater Data Acquisition System in use on a diver pushing the Underwater Ergometer.

experience in environmental physiology, particularly aerospace physiology. From this experience has evolved an advanced capability in the development of biomedical instrumentation. This capability was directed at a new technique of physiological data acquisition from free-swimming subjects under normal ocean conditions (12). In the past, experimental data on the physiological changes in man exposed to an underwater environment have been obtained either by simulating underwater conditions in a hyperbaric chamber or by acquisition of data through the use of "hard wire" methods. The described data acquisition system has obvious advantages over a "hard wire to the surface" system. The free-swimming limitations and the potential hazard of recording physiological data from divers in the ocean by the hard-wire method are *entirely eliminated* by the "underwater recorder." In addition, because of the short and direct wiring involved in this system, the quality of the data is much better than with the hard-wire technique.

Several years prior to the initiation of this project our laboratory attempted to obtain physiological data from divers in the ocean via miniaturized underwater telemetry. The technique was discarded as unreliable for research purposes. Since many research situations do not require real time underwater monitoring of physiological parameters, the underwater recording system has proven a practical method of data acquisition. Unlike underwater telemetry, this system is not hampered by loss of data due to loss of transmission and is therefore able to consistently record good quality data without limiting its boundary.

Physical data, such as water temperature and pressure, and physiological parameters can be simultaneously and continuously recorded on tape during the dive and recovered and analyzed after the dive.

Three generations of underwater data acquisition systems have been undergoing development in this department for the past ten years. They have been funded by and used for a variety of projects at U. S. C. The current system employs a small subject-carried, pressure-proof aluminum case containing a small audio tape recorder and a specially built miniaturized electronics package consisting of signal conditioners and oscillators, each of which is frequency-modulated by the output of one signal conditioner (Figure 3). The oscillator outputs are added and recorded on the audio tape, and the recorded frequency-modulated signals are played back through a discriminator bank for strip chart display. The unit is self-contained and has the capability of simultaneously recording up to eleven separate channels of data during unrestricted open ocean diving. The signal conditioners are interchangeable and have variable gain controls. This unit is described in detail elsewhere (12).

In this study, the system was used in conjunction with a new underwater gas sampler to define objectively the work levels employed to study the effect of underwater exercise on bubble formation. The parameters continuously recorded by the unit on free-swimming scuba divers included: (1) water depth, (2) water temperature, (2) ventilation frequency, (4) heart rate and (5) scuba tank pressure. These parameters

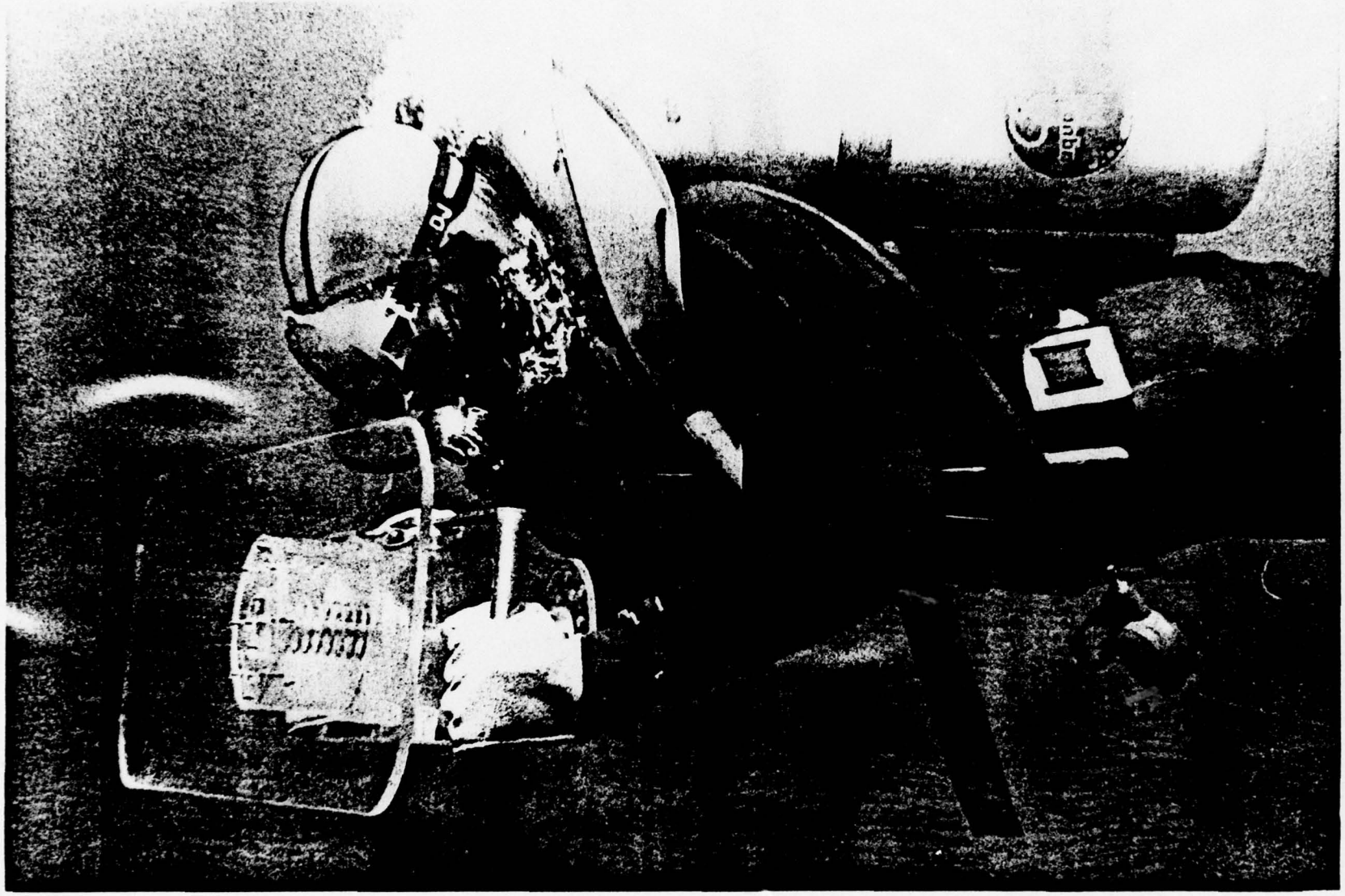

Figure 5. Underwater Ergometer in use by diver.

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were required for the work output determination (see below).

2. Underwater Ergometer (Figure 5)

This light-weight, inexpensive, mobile device provides the investigator with an objective and reproducible method of imposing variable exercise levels on diving subjects of open water experiments. The unit was designed to enable accurate regulation of the work output of fin-swimming scuba divers (14). It was designed and constructed by Mr. John Henriksen. The underwater ergometer is basically a drag board which a diver works against as he moves it through the water. The device is made of clear plexiglass and consists of a resistance plate, a spring-balanced piston assembly and a handlebar assembly. The resistance plate measures 92 cm by 31 cm and is 1 cm thick. The piston assembly consists of two clear cylinders closed at one end. The smallest of the cylinders is attached at its closed end to the resistance plate and measures 20 cm long and 15 cm in diameter. The larger cylinder is slipped over the small one and is 16.5 cm in diameter and 20 cm long. A metal rod extends from the resistance plate through a hole in the end of the large cylinders. The rod holds a spring in place which resists compression of the piston assembly and guides the cylinders as they slide together. Guide slots and bolts on each side of the piston assembly hold the cylinders in proper alignment. The handle bar assembly consists of two pieces of metal pipe 2.5 cm in diameter and 2.5 cm wide. This assembly is attached to the piston assembly by screws imbedded in plastic blocks which are curved on one side and

glued to the outer cylinder. The ergometer weighs six kilograms on land and one kilogram negative underwater.

The diver works on the ergometer by grasping the handle bars with both hands and pushing it through the water. He kicks so that the resistance plate is perpendicular to this course and depth plane. The apparatus is held so that the piston assembly is twenty-four to thirty centimeters below the chin, the arms are flexed ninety degrees, and the resistance plate slightly in front of the diver. As the ergometer is pushed through the water a substantial drag is produced by the resistance plate. At a particular speed a diver must produce a force, by kicking, which exceeds the resistance offered by his body and life-support equipment by an amount equal to the drag on the resistance plate. This additional drag is indicated by the degree to which the spring in the piston assembly is compressed. A scale marked in kilograms is located on the ergometer to indicate to the diver the added resistance he is working against. This spring can be calibrated in the laboratory by placing the resistance plate on a flat surface and adding sets of lead weights to the closed end of the large cylinder. The scale is then marked on the side of the small cylinder where the edge of the large cylinder rests at each set of calibration weights. The diver reads this scale while swimming by simply glancing down at the cylinder wall just below and in front of him. Since the resistance of the ergometer is a function of the water speed, constant work outputs may be obtained by simply having the subject maintain the resistance indicator at a

particular level. The diver work output of moving the resistance plate through the water may be calculated by multiplying the resistance indicated on the ergometer scale times the diver's swimming speed. The work of moving the diver and his life-support system through the water cannot be measured by this method. However, this factor can be standardized. Drag measurements of different individuals have shown that differences between dissimilarly equipped divers are greater than differences between similarly equipped divers who vary greatly in height and weight (9, 16). All the test subjects wore identical scuba equipment.

A reliability analysis of diver swim speed and kick-rate while working against several resistances was carried out (9). The Pearson Product-Moment correlation coefficients for test-retest reliability analysis were found to exceed 0.97 in all except one case. Analysis of the data with a t-statistic showed that there was no significant difference between consecutive swims at each respective ergometer resistance ($p < .05$).

3. Underwater Gas Sampling System (Figures 6 and 7)

Samples of exhaled gas were obtained by a unique multiple gas sampler developed in this lab by Dr. H. J. Dwyer (9). The system operates on the principle of vacuum sampling and consists of:

a) a brass V-shaped manifold which interrupts the exhaust hose of a standard two-hose scuba regulator, and

b) ten evacuated steel cylinders connected to the manifold.

Unlike some previous underwater samplers, this unit does not require

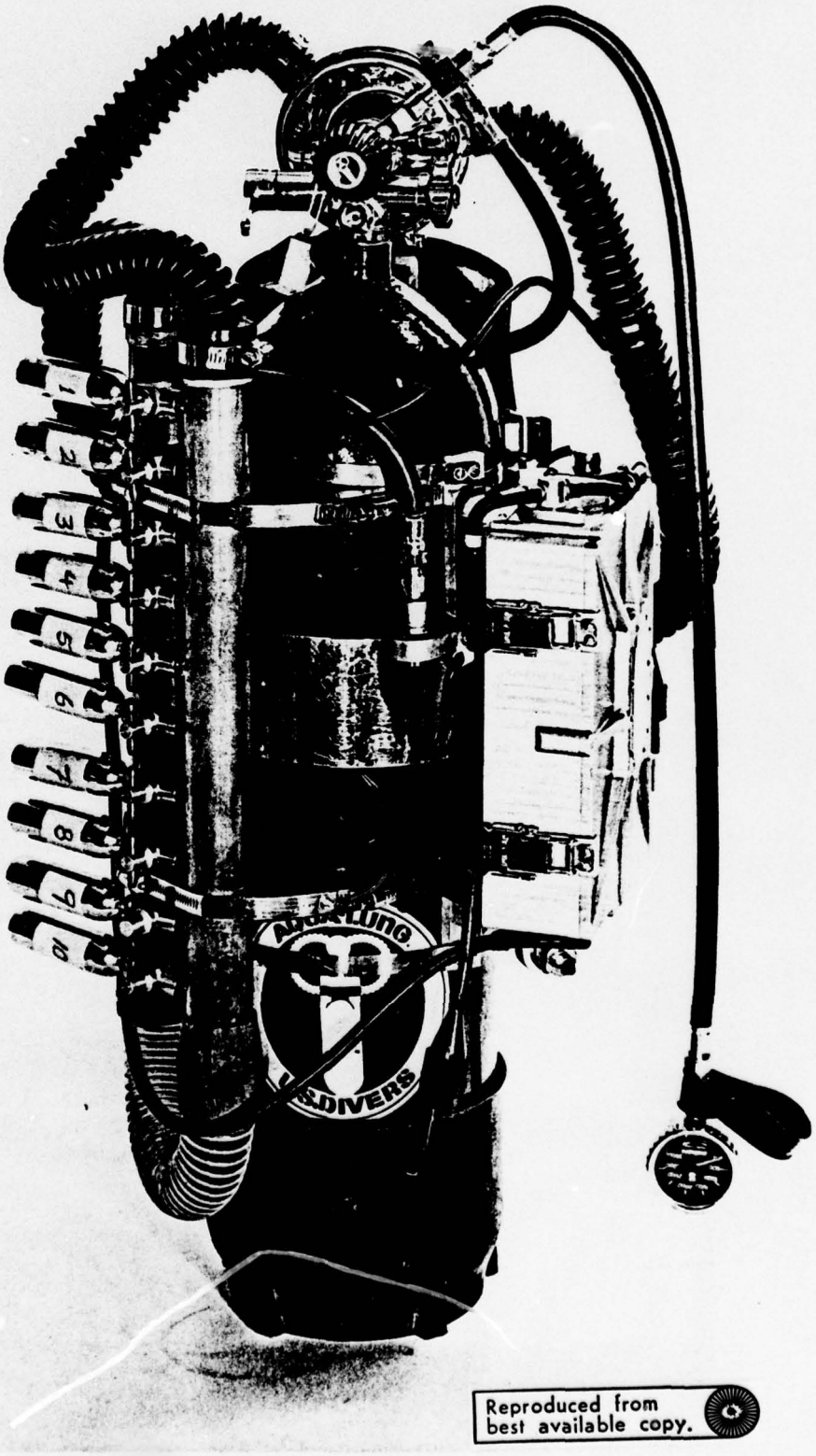

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Figure 6. Underwater Gas Sampler and Recording System mounted on a scuba tank.

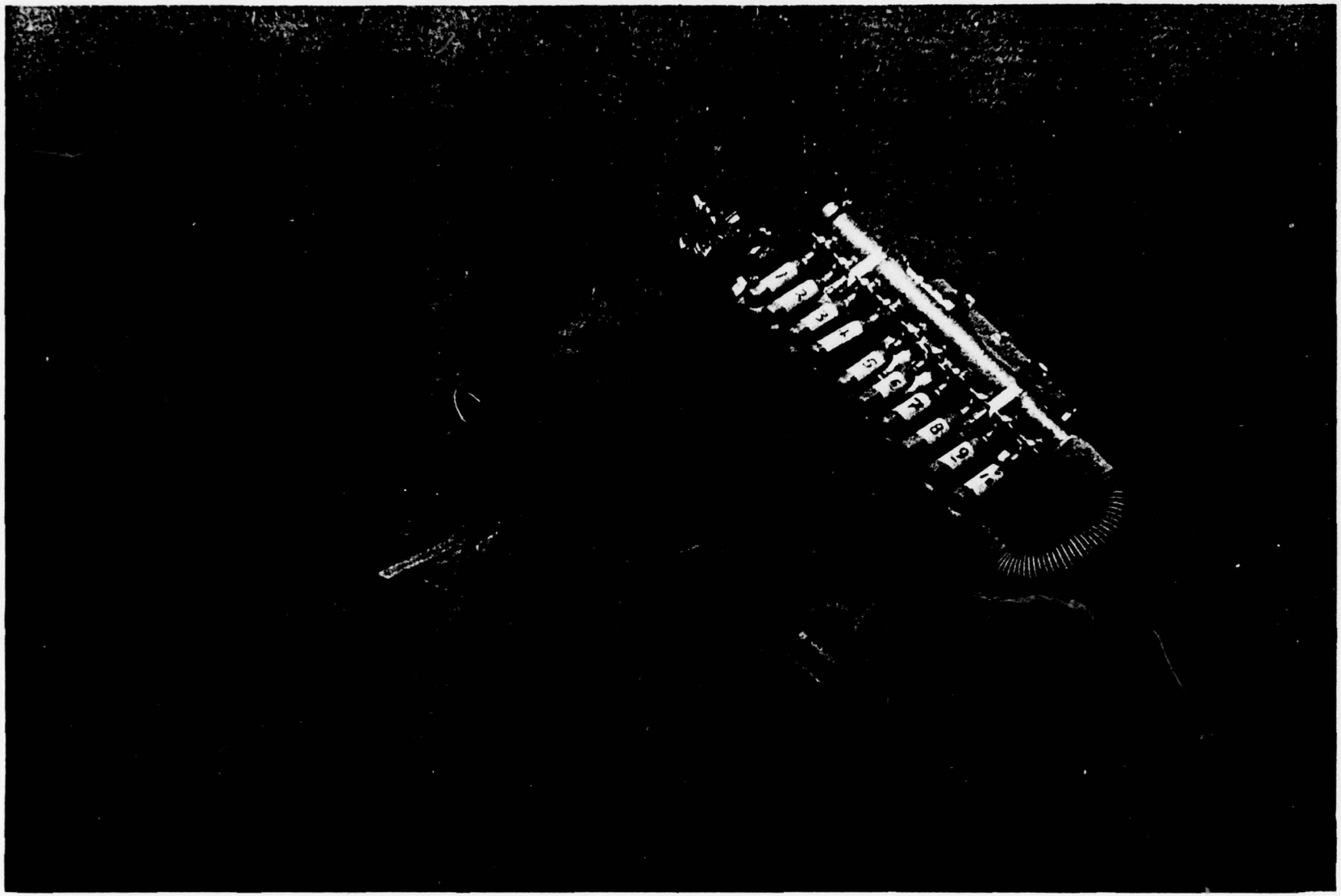

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Figure 7. Underwater Gas Sampler in use on diver.

the diver's gas to be vented to the surface for analysis nor is the unit limited to one sample per dive. The system allows as many as ten samples to be taken at different levels of metabolic activity during a single dive. Furthermore, it is self-contained, easily attached to the scuba tanks, and the cylinder valves can be operated by the researcher swimming beside the subject. As the subject exhales through the sampling manifold, samples of the gas are obtained by opening the cylinder valves during various experimental conditions. The pressure differential fills the cylinders with exhaled gas. The amount of gas collected is directly proportional to the absolute pressure.

The gas samples are transferred from the vacuum cylinders to a 50 cc syringe through a special low-volume fitting. The gas samples are analyzed for O_2 and CO_2 content by a Beckman Model E2 Oxygen Analyzer. The accuracy of this method, as well as the analyzer was checked and calibrated with reference gases.

The VO_2 and VCO_2 were calculated by the following formulas:

1. $VO_2 = (V_I \cdot F_IO_2/100) - (V_E \cdot F_EO_2/100)$
2. $VCO_2 = V_E/100\ (F_ECO_2 - .03)$

The volume of air removed from the scuba tank, over one-minute periods was calculated by the formula:

$$V = \Delta P(K)$$

where ΔP is the tank pressure differential in psi and K is the number of cubic feet of air contained in the tank for 1 psi. For the tanks used in this study K = .02876 cubic feet. The result was a measure of

inspired volume (V_I), in L/min., which could be corrected to STPD by simply correcting for the ambient temperature since ambient pressure was not a factor in the ΔP. However, in order to express the V_I in L/min. BTPS it was necessary to introduce an appropriate correction factor for the ambient pressure at which the ΔP was recorded. The Depth Correction Factor (DCF) was calculated by the formula:

$$DCF = Pb \; / \; Pb + (D \cdot 23)$$

where Pb is the barometric pressure expressed in mm Hg, D is the depth in feet of sea water, and 23 is the pressure equivalent, in mm Hg, of one foot of sea water. The volume of air actually inspired by the subject, corrected to BTPS, was then determined by the formula:

$$V_{I_{BTPS}} = (V_I \cdot DCF)\ FBTPS$$

It was assumed that the temperature of the air delivered to the subject was equal to the ambient water temperature. Thus, the water temperature data was used to correct the air consumption volume (V_I) to BTPS and STPD. The tidal volume (V_t) was calculated by dividing the minute volume, V_E BTPS, by the ventilation frequency. The air consumption ($V_I + V_E$) was determined by monitoring the pressure change of the scuba tanks during the dives.

Upon arrival on the bottom the recorder was turned on and the subject assumed the resting position on his knees with his trunk at a slight forward incline. Four minutes after the beginning of rest gas samples were taken. At the end of the fifth minute of rest the subject was handed the ergometer and signaled to begin swimming against a

resistance of 1.5 Kg. for four minutes. After three minutes of work, during which the subjects traveled an average of sixty meters, gas samples were taken. The swim was terminated after four minutes, the recorder was turned off, and the ergometer was taken from the subject. Four-minute swims at a resistance of 2.4 Kg were done on separate dives. A two-minute rest period was given between each work period. Gas samples were taken at the same point in time during all working swims.

A series of preliminary dives was done utilizing these three items of equipment in order to establish the specific exercise levels to be used during the bubble detection dives. The dives were at 100 feet of depth and involved six subjects. Rest and three levels of exercise were used. The final parameters obtained from these dives included:

1. heart rate
2. oxygen consumption
3. CO_2 production
4. pulmonary ventilation
5. tidal volume
6. ventilation frequency, and
7. energy expenditure.

Diving Procedure

During the course of the 3-year study, 18 resident scientific divers of the U.S.C. Catalina Marine Science Center were used as subjects. These people routinely perform working dives and are

thoroughly familiar with the diving station. These divers were certified by the U.S.C. Catalina Marine Science Center to specific depths and strictly adhere to the U.S. Navy Standard Compressed Air Decompression Tables. Thus, the divers were tested for bubble formation while performing work in familiar underwater surroundings and adhering to the depth and time limits currently accepted as safe by the diving community.

All dives were free-swimming air scuba dives. The dive team consisted of 1) the subject, 2) the underwater monitor and 3) the topside monitor. The dive platform was moored to a vertical descent line which was permanently attached to an earth anchor in the bottom. In most cases a wake of a boat was the largest wave condition encountered. A large diameter weighted decompression line with a tank and regulator at the 10 ft. and 20 ft. depths was put over the side. With longer duration dives, extra sets of double tanks with regulators and depth gauges were lowered to the bottom with a line. These were suspended just off the bottom so that sand, etc. did not enter the regulators. After extensive experience with several brands of depth gauges (in a test chamber and in the ocean) it was concluded that none of them had the reliable accuracy required for these experiments. Thus, a steel cable lead-line was used as the primary depth sounder and was dropped and left suspended during all dives. Markers on this line every 10 ft. permitted reasonably accurate ascent rates. The subject and the underwater monitor each carried 2 depth gauges and one watch. The depth gauges were calibrated on the bottom with the lead-line and followed when not

in visual distance of the lead-line. Since in this area the maximum tidal changes are approximately 6 feet, it was important to schedule these dives during the times of the tidal cycles when the subject's chest could be maintained exactly at the prescribed depth while resting (kneeling on bottom) and while exercising with the ergometer. Chlorox bottles anchored to the bottom with earth anchors acted as navigational aids. Visibility was between 40 and 80 ft. horizontal. Water temperature was between 11 and 13°C. Currents were occasionally present but did not create any major problems. The subjects' descents were totally passive. For the longer dives, both the subject and the underwater monitor switched to the extra sets of double tanks when their tank pressure reached 600 psi. The switch required no more than 2 minutes. During the resting dives, the subject knelt motionless on the bottom for the full bottom time.

The underwater exercise was intermittent: 2 minutes of rest followed by 2 minutes of exercise. The 100 ft./25 min. dives entailed 4 rest periods and 4 exercise periods; the 100 ft./40 min. dives had 8 rest and 8 exercise periods. The ascents were at 60 ft./min. and, as with the descents, were passive and controlled through buoyancy regulation.

The Doppler recordings were made on the diving platform and at the marine laboratory. Two-minute control recordings were made prior to each dive. After the Model B was converted for use underwater, continuous recordings were made during decompression periods. Due to

the time required for boarding the dive platform and the removal of diving gear, the earliest bubble detection recordings were made 3-5 minutes after the point of surfacing.

Eight two-minute post-dive recordings were made at 5, 15, 30, 45, 60, 90, 120 and 180 minutes after surfacing. The data was simultaneously recorded on tape (Sony Model TC-110A cassette recorder) and monitored with earphones. Subjects were seated and motionless during the recordings.

Dive Profiles

During the first year of the study, the following 3 dive profiles were used:

	Depth (ft.)	Bottom Time (min.)	Decompression (min./ft.)
1.	100	25	none
2.	100	30	3/10
3.	190	10	2-3/20 4-5/10

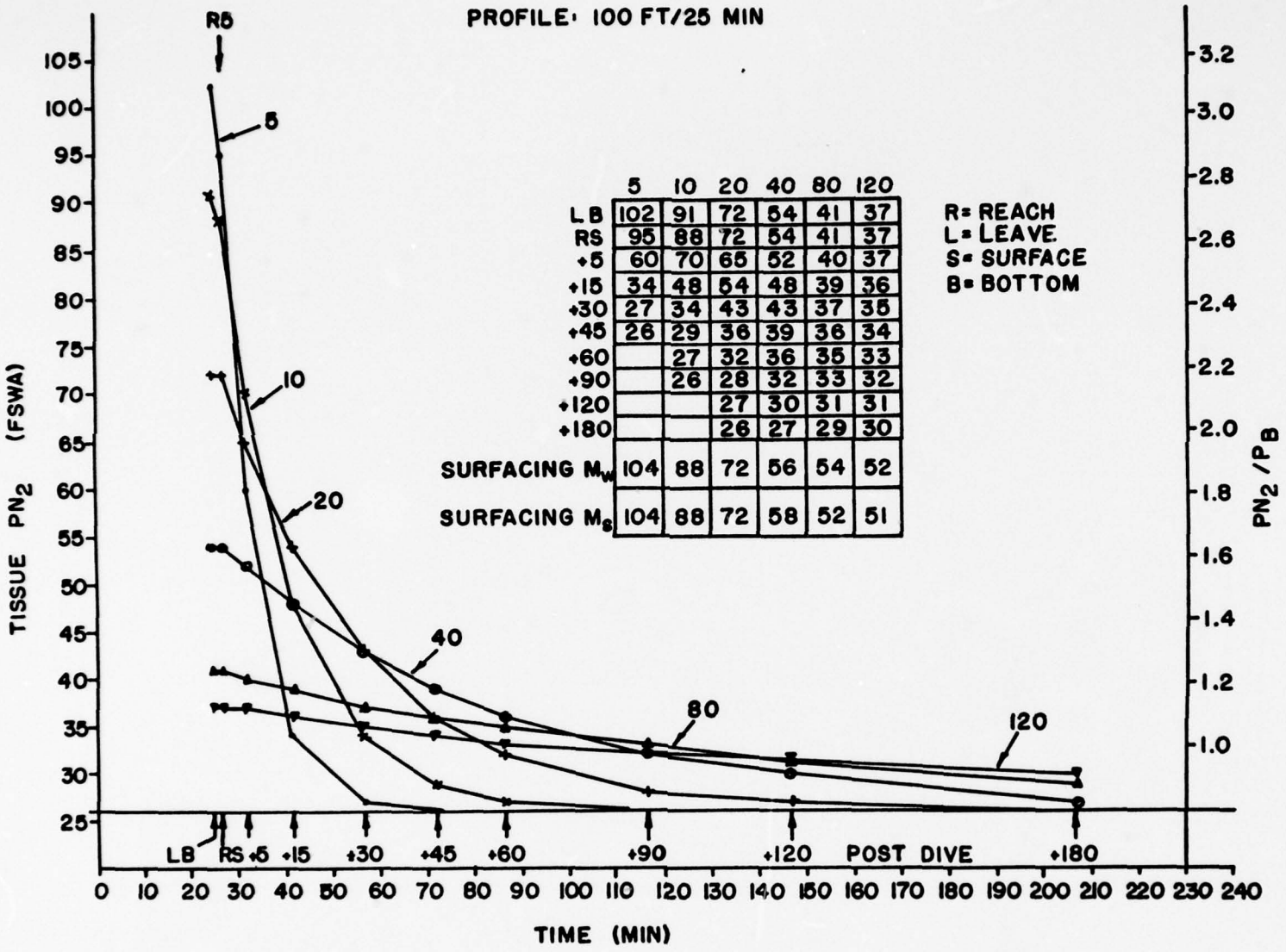
During the second and third years the dive profiles were the following:

	Depth (ft.)	Bottom Time (min.)	Decompression (min./ft.)
1.	100	25	none
2.	100	40	15/10

The primary factor determining the choice of these dive profiles was a practical one. The realities of cold exposure, air consumption, diver experience and diver safety were strongly considered.

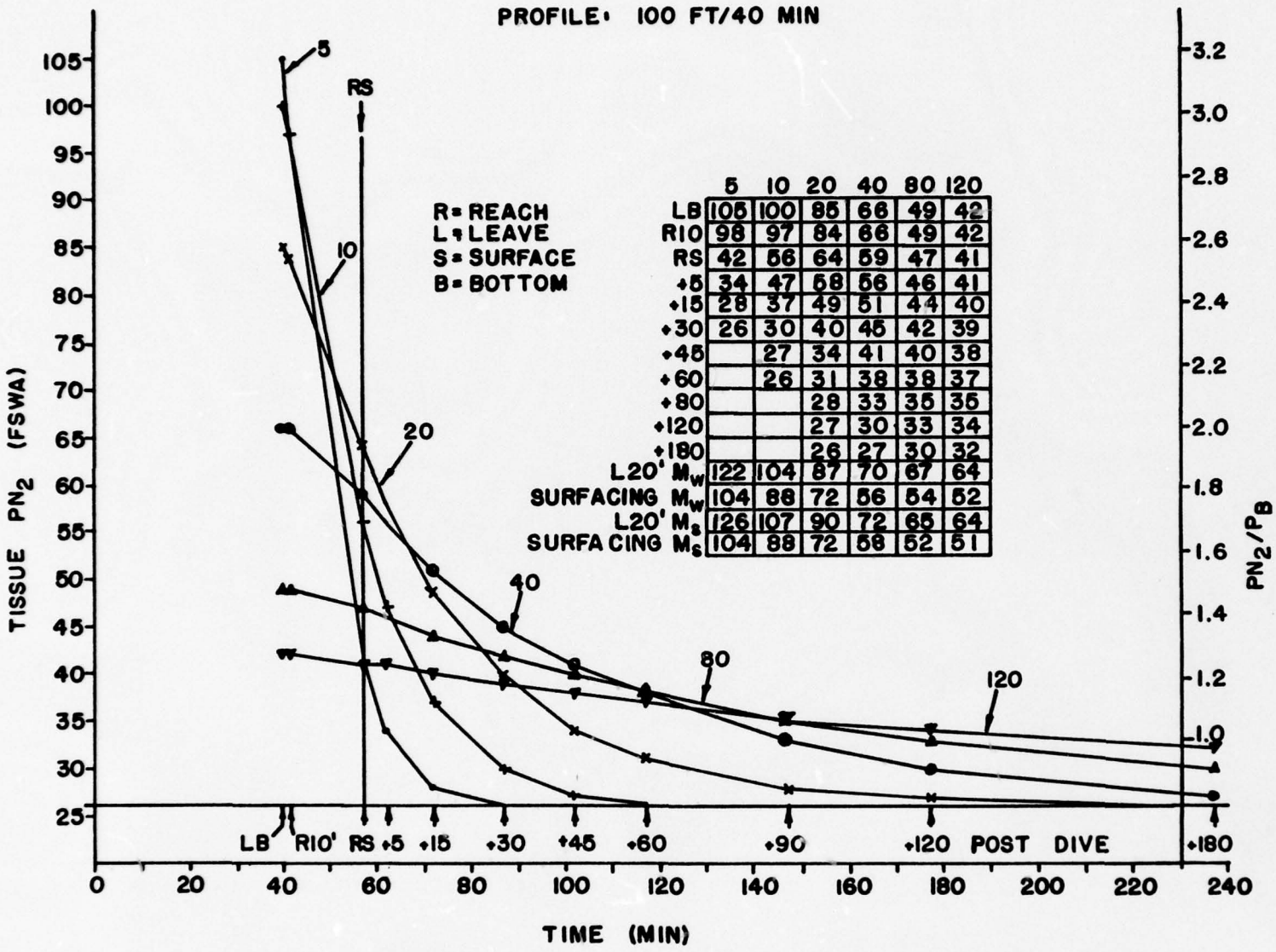
From the early work of this contract it was evident that non-symptomatic vge are present after several ocean dive profiles when carried out to the exact maximum "no-decompression" limits. It was of interest to define the critical half-time tissues during the immediate post-dive periods. Thus, a method was developed for fast calculation of tissue inert gas tensions for any hyperbaric exposure. The Haldane method of decompression calculations, modified by the U.S. Navy and described by Workman (18) and outlined by Braithwaite (17), which is ordinarily performed either by hand or by a computer program, was used for these calculations.

A program for the Hewlett Packard HP-65 Programmable Pocket Calculator which allows accurate and rapid calculation of tissue inert gas tensions, decompression stops required and length of stops for any exposure parameters (depth, time, gas mixtures) was developed by Dr. Bruce Bassett and Ms. Sharon Christopherson. The program is constructed so that hand calculations can be performed in a fraction of the time required using an ordinary calculator or slide rule and with far less chance of error. For each phase of exposure, the depth, time, inert gas fraction, and starting tissue tension are entered and stored. With subsequent entry of each half-time tissue value, the computer calculates the respective inert gas tension. To calculate decompression, the half-time of each tissue is entered along with the limiting tissue tension (M value) for that half-time, and its new starting tissue tension. The greatest depth calculated by the computer is selected as the decompres-



	5	10	20	40	80	120
LB	102	91	72	54	41	37
RS	95	88	72	54	41	37
+5	60	70	65	52	40	37
+15	34	48	54	48	39	36
+30	27	34	43	43	37	35
+45	26	29	36	39	36	34
+60		27	32	36	35	33
+90		26	28	32	33	32
+120			27	30	31	31
+180			26	27	29	30
SURFACING M_W	104	88	72	56	54	52
SURFACING M_S	104	88	72	58	52	51

Figure 8. The inert gas tensions for the 100 ft./25 min. dive profile.



	5	10	20	40	80	120
LB	105	100	85	66	49	42
RIO	98	97	84	66	49	42
RS	42	56	64	59	47	41
+5	34	47	58	56	46	41
+15	28	37	49	51	44	40
+30	26	30	40	45	42	39
+45		27	34	41	40	38
+60		26	31	38	38	37
+80			28	33	35	35
+120			27	30	33	34
+180			26	27	30	32
L20' M_w	122	104	87	70	67	64
SURFACING M_w	104	88	72	56	54	52
L20' M_s	126	107	90	72	65	64
SURFACING M_s	104	88	72	58	52	51

Figure 9. The enert gas tensions for the 100 ft./40 min. dive profile.

sion stop depth. The time required at the stop is similarly calculated by the program. The inert gas tensions for the dive profiles used in conjunction with the Doppler ultrasonic bubble detection experiments have been calculated by use of this program (Figures 8 and 9). Also a complete laboratory file of inert gas tensions resulting from all standard, as well as several non-standard dive profiles is being generated using this programmable calculator.

This method was also used to determine the dive profile for a marine biologist requiring extensive bottom time (5 hours and 40 minutes) between 50 and 80 feet (19). Since calculation of decompression requirements for such a multilevel non-standard profile constitutes experimental diving, the Doppler was used as a safety measure. The diver was monitored during the last 90 minutes of his dive as well as for two hours post-dive. No bubbles were recorded at any time, thus, proving the safety of this non-standard profile for that particular person. The sensor placement functioned perfectly and was still effectively attached to the chest when he surfaced.

Animal Experiments

An adult male mongrel dog weighing 35 Kg was used to study the effect of exercise on gas exchange during and following hyperbaric exposure (20).

Prior to implantation, the subject dog received five chamber exposures to depth from ten to one hundred feet of sea water (fsw) to

determine his response to pressure change, isolation, noise and adiabatic temperature changes encountered in the hyperbaric chamber. Prior to and following implantation, but prior to the experimental series of exposures the subject dog received eight sessions on the treadmill for training and/or baseline vena caval flow determinations (Figure 10). Implantation of the vena caval blood flow probe was performed using i.v. Serutal for initial anesthesia followed by Halothane to maintain anesthesia for the duration of the surgery (Figure 11). The flow probe was implanted around the abdominal vena cava posteriorly to the junction of the renal vein. The flow probe lead with the connector capped was, after suturing the muscle layers, located in a subcutaneous pocket for later surgical retrieval. Approximately one month after implantation the flow probe lead was exteriorized anteriorly from the pocket through a bluntly dissected subcutaneous tunnel using Serutal and Halothane anesthesia. A specially designed jacket was placed on the animal at this time with a zippered pocket attached into which the exteriorized lead was tied. A small inflated inner-tube was placed around the dog's neck and tied to the jacket to prevent the animal from reaching the pocket and/or exteriorized lead. Prior to closure of the incision the function of the flow probe was checked electronically.

The treadmill used in the exercise studies consisted of a rotating reinforced canvas belt, 11.5 ft. length, driven by a 100 v electric motor connected to an in-line variable speed control. The incline of the treadmill could be varied from 0^{o}, horizontal, to 12^{o}, full incline, and

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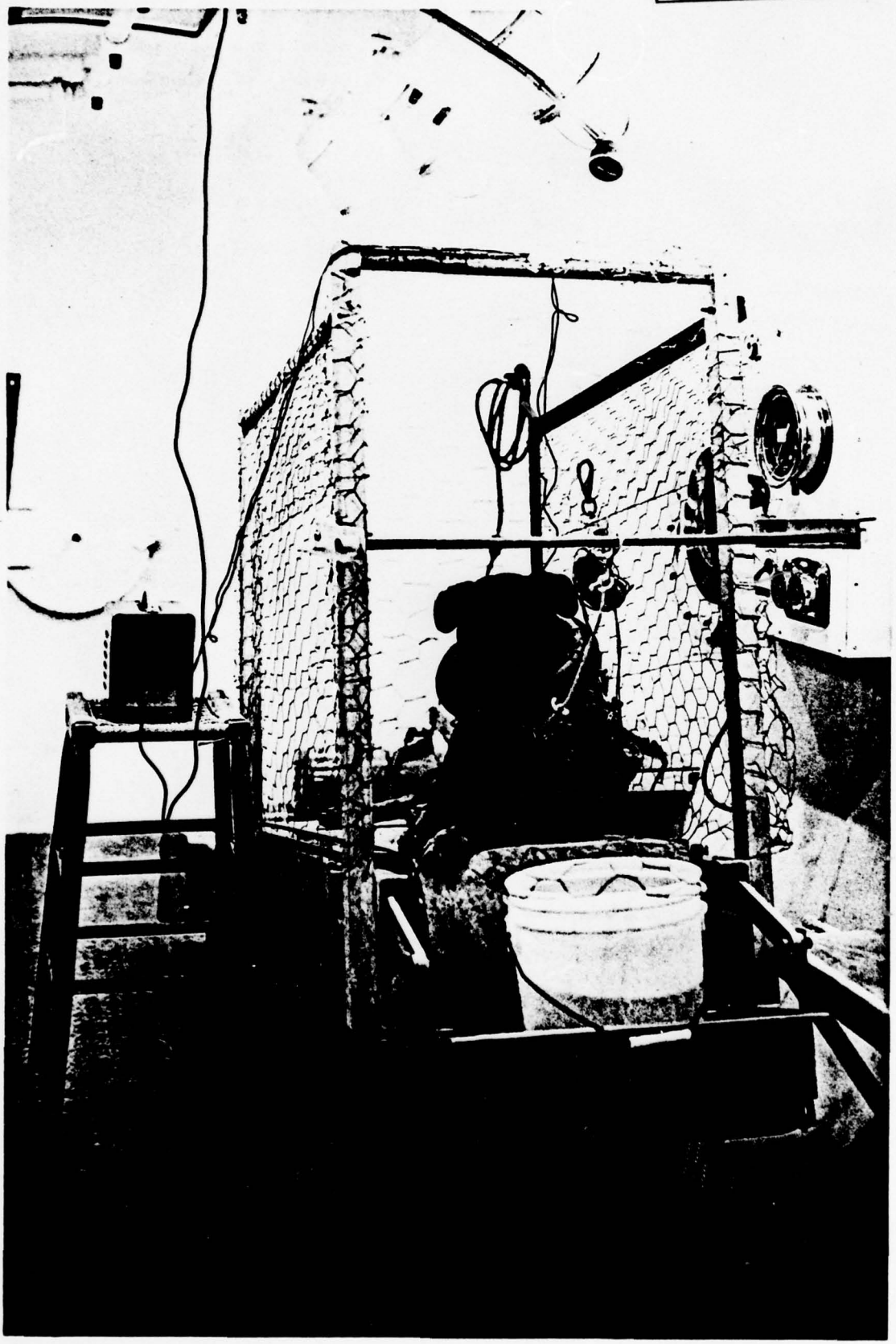


Figure 10. Implanted dog resting on treadmill inside chamber.

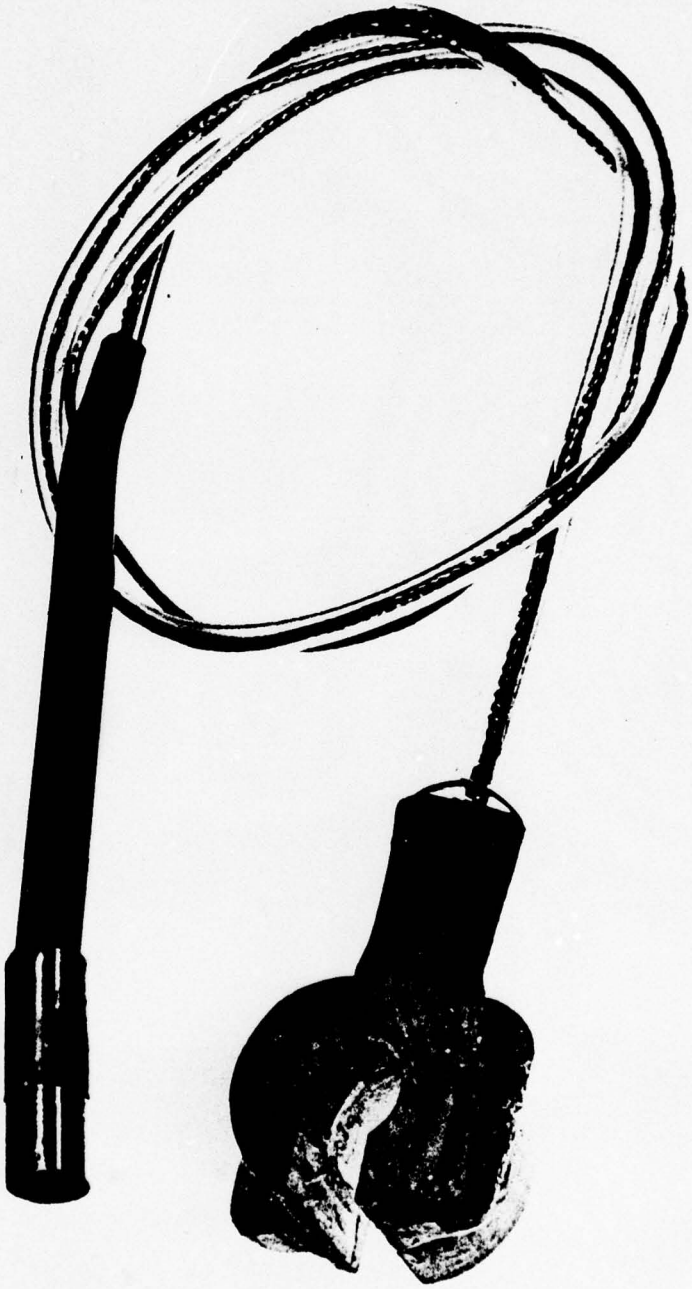


Figure 11. Flow probe used to monitor vena caval blood flow in dog.

the speed varied up to 5.2 mph at full speed. All exercise was conducted at 3.4 mph at 12° incline. The treadmill was modified with a wire mesh cage with a removable lid and rear portion so the animal was somewhat restrained while in the chamber, however he could stand, run, sit or lie down completely.

The dog was monitored for vge while lying on the inclined treadmill on his right side, head at the elevated end of the treadmill, and with all four legs extended comfortably out from the body. The dog became quite unconcerned with this frequent procedure and therefore cooperated completely, remaining in this position at complete rest for all of the five-minute recording periods. Five-minute vge recording periods were conducted as follows: ten to fifteen minutes prior to the dive; five to ten minutes post-dive, except in the series which involved exercise during and following decompression; fifteen to twenty minutes, thirty to thirty-five minutes, forty-five to fifty minutes, sixty to sixty-five minutes, ninety to ninety-five minutes, 120 to 125 minutes, and 180 to 185 minutes post-dive. Abdominal vena caval blood flow was monitored by the implanted ultrasonic interferometric flow probe developed by Rader et. al. (21). The probe crystals were excited by a continuous-wave 5-MHz signal generated by a battery operated signal conditioner located next to the animal enclosure in the chamber. The flow signal was recorded on one channel of a Brush Mark 280 two-channel recorder, located outside the chamber, via a through-hull instrumentation connector panel. Vena caval blood flow was monitored continuously from

ten to fifteen minutes prior to the dive until 180 minutes post-dive except during the five minute vge recording periods. Flow could not be monitored during these periods because of interference with the flow signal by the Doppler ultrasonic vge detector. Failure of the flow probe following completion of the experimental series precluded a determination of the zero flow signal and therefore precluded the determination of absolute flow values.

Isoxuprine HCl (VASODILAN, Mead Johnson Laboratories) was the skeletal muscle vasodilator used in this study. 0.1 mg/Kg animal weight was chosen as the dosage based on animal studies with a similar vasodilator (22). The vasodilator was administered intramuscularly by injection. Test exposure times at 100 fsw were selected based on the calculated nitrogen tensions in the five, ten, twenty, forty, eighty and one hundred twenty-minute half-time tissues. The exposure times evaluated were 15, 20, 25, 35, 45, and 60 minutes. The animal was tested in the chamber at all of these exposures. A threshold exposure time at 100 fsw, in terms of post-decompression "silent" vge detection, was determined to be twenty minutes for the resting experimental animal used in these studies.

Using the silent bubble threshold exposure, the following 5 test conditions were done:

1. Resting Exposures

2. Exercise during exposure - The exercise period lasted 15 minutes: 6 minutes during descent and 9 minutes at the bottom.

3. Exercise during decompresion - The 15 minute exercise period included 30 seconds at depth, the entire decompression time and the initial 12 minutes of post-dive time.

4. Vasodilation during exposure - This profile duplicated the resting exposure except that 3.5 mg Isoxuprine-HCl (0.7 cc) was injected i.m. in the left hind leg prior to the start of the exposure.

5. Vasodilation during decompression - This profile also duplicated the resting exposures except 3.5 mg Isoxuprine-HCl was injected as described above at one minute prior to the start of decompression.

Data Analysis

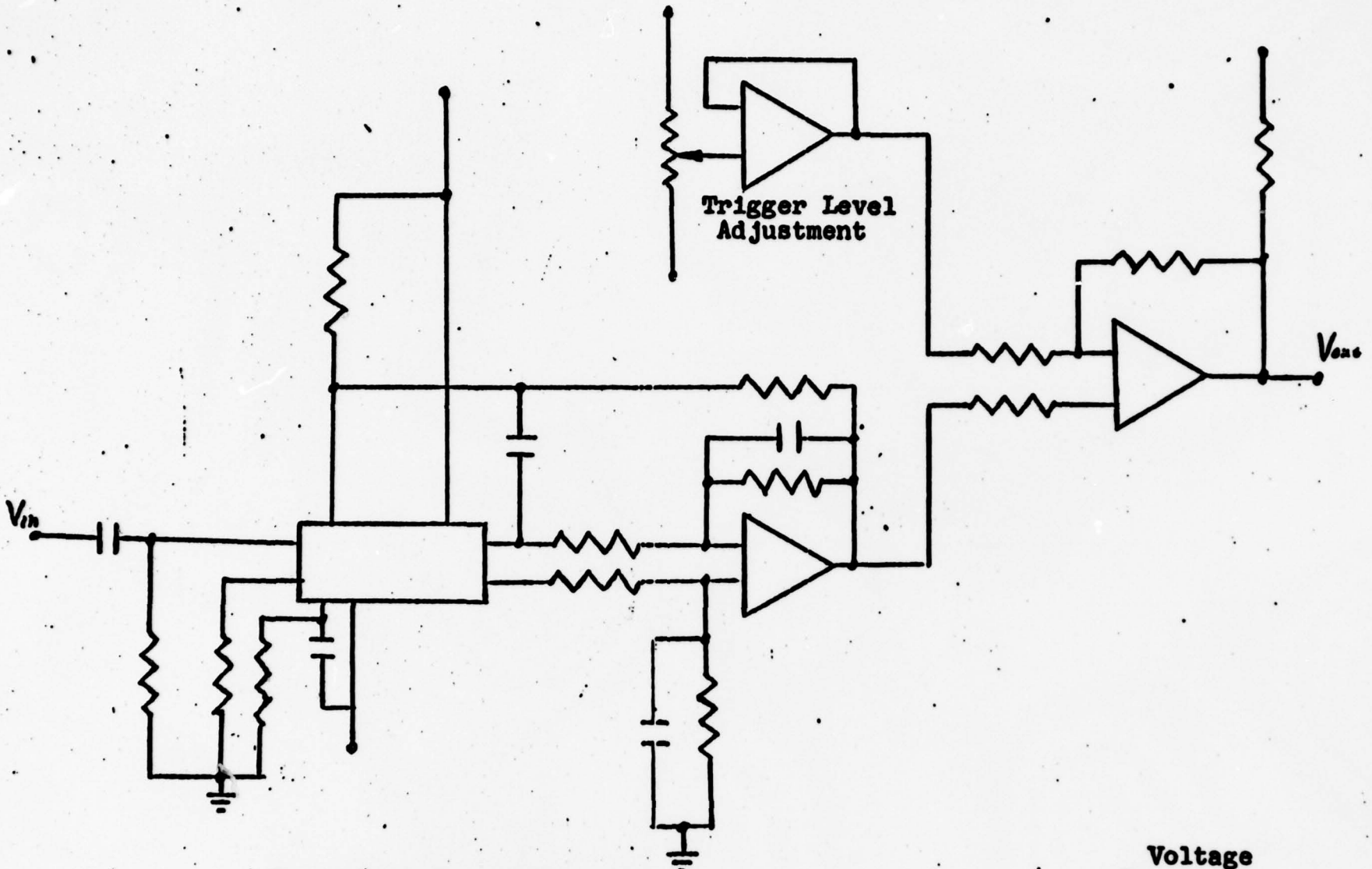
We feel that still the most pressing requirement in the field of human intravascular bubble detection is the development of a reliable and standardized method for quantifying the data.

Animal studies were done the first year to verify the electronic characteristics of the intravascular bubbles as recorded by the bubble detector. The animals and human data were subjected to audio and oscillograph analysis. A method was then developed for quantification of this type of data. Intravascular bubbles when visualized by an ultrasonic device, functioning on the Doppler principle, induce a variation in signal level and frequency of the conditioned signal. The voltage change induced by bubble formation is similar to that induced by normal flow velocity variations; however, the frequencies appear to be slightly higher then that induced by normal flow variations. The device we have

developed takes advantage of this higher frequency to acquire a first order measurement of bubble formation. The circuit consists of three stages: a high pass filter which removes the lower frequency components normally associated with the flow variation, a phase locked loop discriminator adjusted to respond maximally to the frequency content produced by bubble formation, and a trigger circuit which produces a pulse each time its input is forced above a specified level by the discriminator output. These pulses are then counted by an electronic counter and visually displayed (Figure 12). Figure 13 shows an example Brush recording. A manual frequency selector is set at a level at which no events are evident during the 2 minute control recording. In this case, that level was 1190 Hz. The post-dive recordings are then played back through the system and any input frequencies above this pre-set control level are simultaneously displayed on the Brush Recorder and a digital counter. It is important to note that these signals are termed "events" not bubbles. Occasionally there are cardiac events which also elicit above-control level frequencies. However, the majority of these electronic events are interpreted to represent gas emboli passing through the right heart. The events are tabulated for each 2-minute period and compared with the audio counts made for the same period.

There were two problems encountered with this method of bubble quantification. The sensor placement had to be very critical, since it was found that signals produced by valve closures and/or peak flow were

Figure 12. Circuit Diagram of the "Bubble Counter".



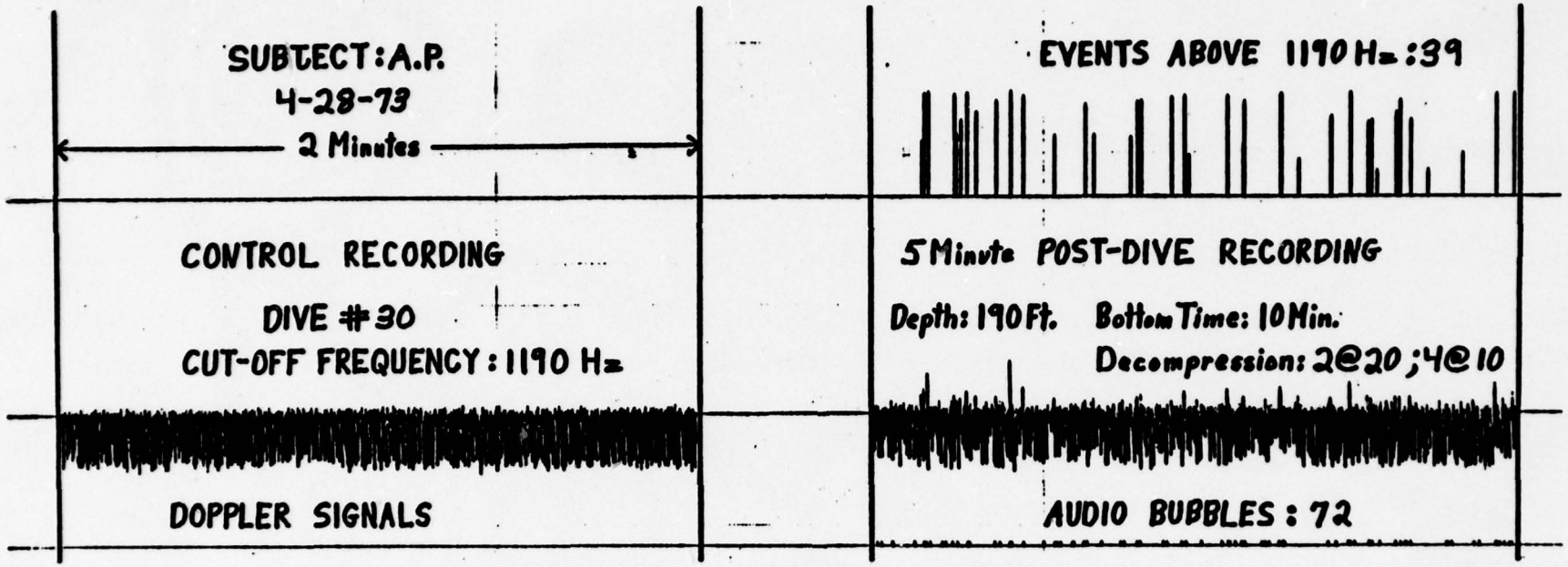


Figure 13. Brush Recording from the "Bubble Counter."

sometimes as high as those produced by bubbles and, thus, masked the data. These closure and flow sounds were identified and avoided on placement. Secondly, a substantial portion of the bubbles that were audible in the earphones did not display above control frequencies. In addition, during the second and third years the "bubble counter" became increasingly unstable electronically until it had to be taken out of service. Thus, much of the data presented in this report are entirely audio counts.

Work was continued on the improvement of the bubble quantification technique. This work was aimed at the identification of an electronic signature of bubbles. To aid in this research an extremely simple working model of pulsating heart flow was devised (Figure 14). At certain velocities of water flow, the thin latex pulsates at fixed rates. Gas emboli can then be injected into this dynamic model and picked up by the Doppler sensor. Thus, electronic Doppler bubble signals can be created at will for analysis. However, the contract was completed before an accurate quantification method was found.

The audio analysis of Doppler signals in the human experiments was done in the following manner. The monitor listened to all of the 2-3 minute tape recordings of a series to eliminate any with gross interference. He then listened to random 1 minute (2 minutes during the first year) segments at least three times counting individual events which he interpretted as bubble sounds. This is a very time-consuming effort requiring total attention. The monitor did not spend more than

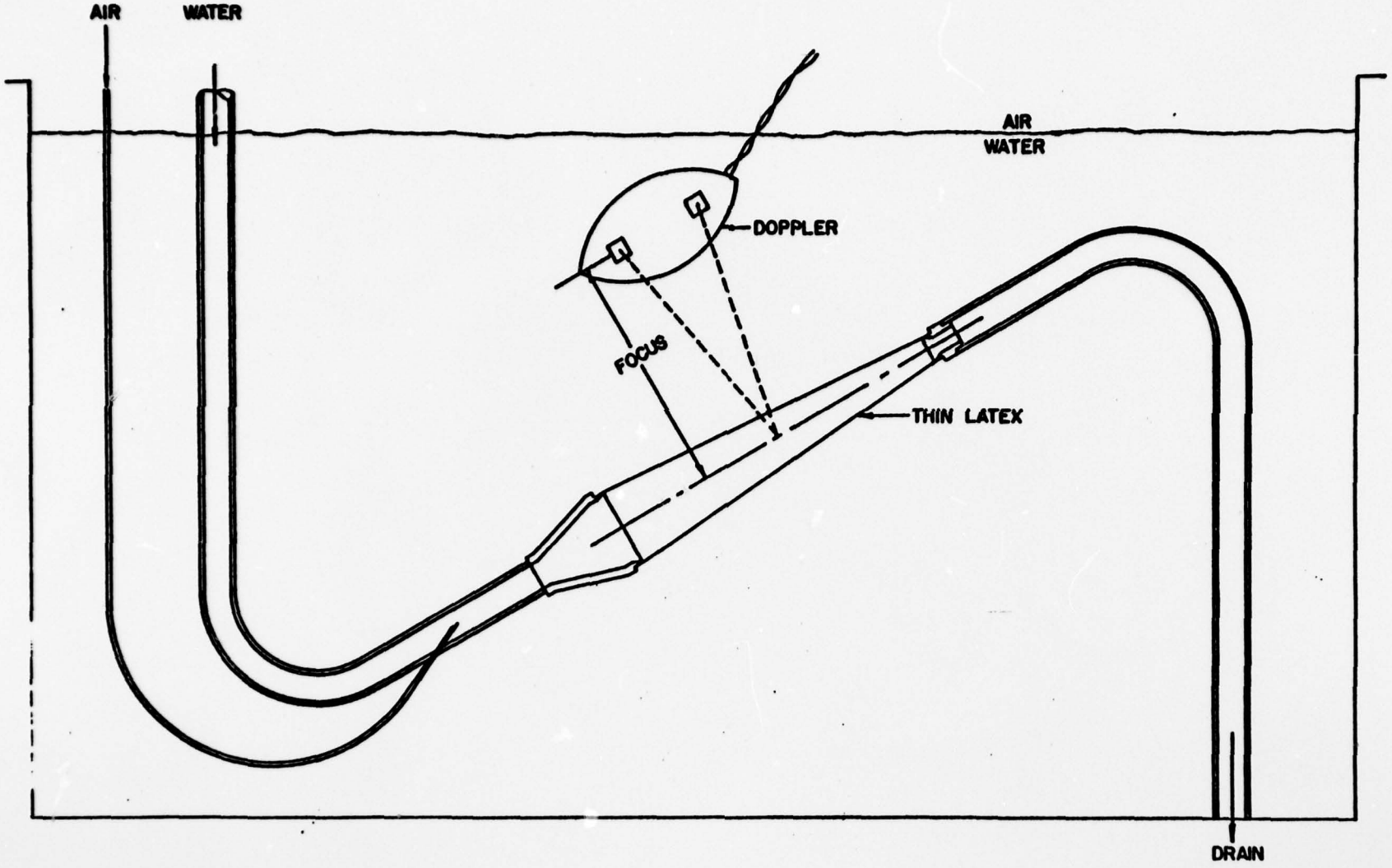


Figure 14. Model of flow in pulsating heart.

one hour at any one time listening to the tape since it was found that accuracy drastically decreased for longer periods. Periods on the tape which had so many bubble sounds that it was impossible to make individual counts were marked "continuous."

There was an attempt made to analyze the bubble data from the animal experiments in a slightly more objective manner. The cassette recordings from each exposure had all voice references erased from the tapes as to the type of experiment, date or recording period. A disinterested individual then covered all written references to the date or type of exposure involved with a label containing a code number randomly assigned to the tapes. The record of the code numbers versus the date and type of exposure was maintained in a sealed envelope by the disinterested party until all tapes had been analyzed for vge counts. Each one minute section of recording was played from four to six times. The first playback was used to count heart beats, to assess the quality of the recording, and to assess the positioning of the sensor according to the blood flow sounds heard. The subsequent three to five playbacks were used to count the abnormal sounds (hereafter called events) produced by vge (described as clicks, chirps, whistles, etc.). When the same number of events were counted repeatedly in three playbacks no further counts were performed. If there were differences in the number of events counted during the first three playbacks then a total of five separate counts were made. Beyond 25 events/minute the number could only be estimated and the following estimations were used: 50% of the

heart cycles contained one event, 75% of the heart cycles contained one event, 100% of the heart cycles contained 1, 2, 3, 4 or 5 events. The estimated vge count per minute was, in these cases, obtained by multiplying the recorded number of heart beats in that one minute segment by 0.5, 0.75, 1, 2, 3, 4 or 5. When there were an estimated three to five events per beat the heart sounds were nearly obliterated, being replaced by a continuous cyclical roaring sound. The mean number of events counted or estimated for each one minute segment were then averaged over the five minute recording period to obtain a mean and standard deviation of the events occurring per minute during that observation period. The mean counts, as events/minute, for each recording period were converted to a "vge score" as follows: a mean count of from 0 to 0.99 events/minute was scored as 0. It was felt that a mean count of less than 1.0 represented sufficient doubt on the part of the listener as to the discreteness of the sound heard to discount it as an artifact or observer error. A score of 1.0 was assigned to mean counts from 1.0 to 17.0. Seventeen point zero was chosen as an approximation of $\frac{1}{4}$ of the number of heart beats/minute in the resting animal (70 bpm), and represents a best estimate of what is referred to by Spencer (6) as an "occasional event" in his scoring system. A mean of 17.1 to 70.0 events per minute was scored as 2.0 to coincide with Spencer's estimate of frequent events but less than half of the heart cycles containing vge sounds. Three point zero was the score assigned to mean estimates from 70.1 to 140.0 events/minute and 4.0 was the score for mean esti-

mates greater than 140.1 (2 events/heart cycle). Finally, the means and standard deviation of the assigned scores for each observation period for all the exposures in the specific experimental profile were calculated and used for the statistical comparisons between profiles.

Mean heart rates were calculated for each of the observation periods from the one minute counts. The means and standard deviations of the heart rates for each observation period for all the exposures in the specific experimental profile were calculated and used for the statistical comparisons between profiles.

Continuous recording of vena caval blood flow was performed. The recordings were analyzed visually for abnormal signals possibly caused by vge.

The nitrogen tensions at the time of reaching surface pressure were calculated for six theoretical tissues having half-times of 5, 10, 20, 40, 80 and 120 minutes using the exposure time parameters recorded for each exposure. The mean and S.D. were calculated for the surfacing nitrogen tensions in each of the six theoretical tissues for the five experimental exposure types.

RESULTS

One hundred ten subject-dives in the open ocean for the purpose of vge monitoring during the 3-year study were made. A separate series of 28 dives was done for the ergometer evaluations and 57 dives for the work output determinations.

First Year

No symptoms of decompression sickness were seen as a result of any of the diving. Intravascular "silent bubbles" were present, to some degree, after all of the dives reported. The averaged vge occurrence time-courses of the three dive profiles are seen in Figure 15. "Silent bubbles" were present within a few minutes after surfacing from the dives. The number of events generally peaked within an hour post-dive, declined and was close to control levels by three hours post-dive. Table I gives the levels of significance of each averaged recording to the control levels. Significant differences occur at the 5, 15, 30 and 45 minute post-dive recordings. In all cases, except one, there were significant differences between the means of individual subject-dives, i.e., the degree of occurrence of bubbles significantly varied between all of the subject-dives of each of the 3 dive profiles (Table II). There were also significant differences (except in one case) between the means of the 9 sets of recordings for each of the 3 dive profiles. A great range of individual variability was seen. Furthermore, each individual showed a relatively consistent degree of bubble formation on various dive profiles and repeated dives. In particular, the subject in

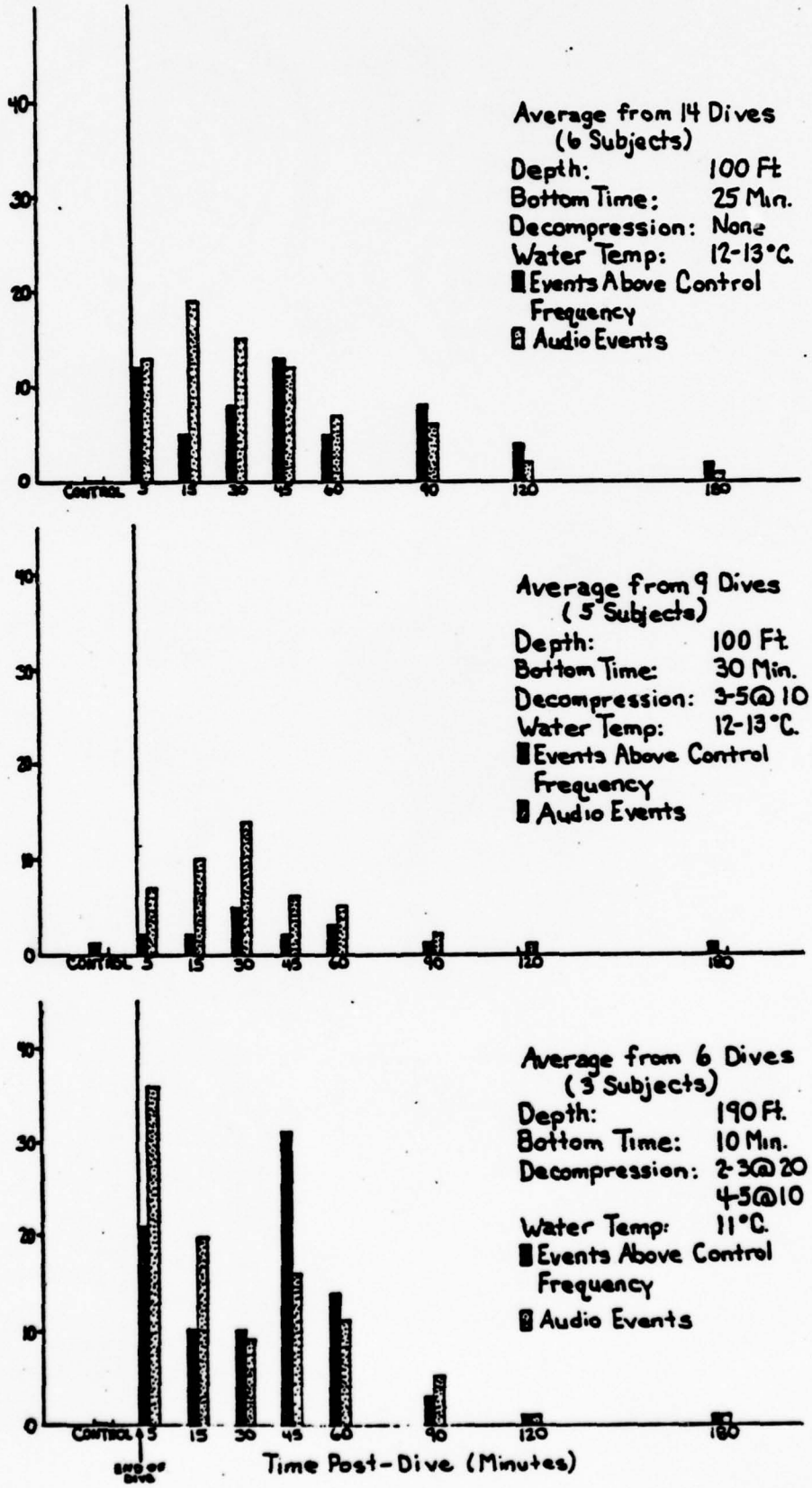


Figure 15. Averaged vge data from 3 dive profiles.

TABLE I

Dunnet's t Statistic

Dive Profiles	Recordings Min. Post-dive							
	5	15	30	45	60	90	120	180
1. 100 ft-25 min No Decompression								
Audio	**	**	**	**	NS	NS	NS	NS
Electronic	**	NS	NS	NS	NS	NS	NS	NS
2. 100 ft-30 min 3 min @ 10 ft								
Audio	NS	*	**	NS	NS	NS	NS	NS
Electronic	NS	NS	*	NS	NS	NS	NS	NS
3. 190 ft-10 min 2 min @ 20 ft 4 min @ 10 ft								
Audio	**	*	NS	NS	NS	NS	NS	NS
Electronic	**	NS	NS	NS	NS	NS	NS	NS

NS = not significant
* = P less than 0.05
** = P less than 0.01

TABLE II

Single Factor Analysis of Variance for Repeated Measurements

		Between Subject-Dives		Between Recordings	
1.	100 ft-25 min No Decompression	F			
	a. Audio	11.00	.01	5.55	.01
	b. Electronic	10.33	.01	2.00	NS
2.	100 ft-30 min 3 min @ 10 ft				
	a. Audio	3.31	.01	3.34	.01
	b. Electronic	1.33	NS	2.82	.01
3.	190 ft-10 min 2 min @ 20 ft 4 min @ 10 ft				
	a. Audio	5.83	.01	3.77	.01
	b. Electronic	3.25	.01	2.00	.01

Figure 16 consistently produced large numbers of events, even after a relatively "safe" dive profile. A bottom time of 25 minutes is the "no-decompression limit" for a depth of 100 feet according to the U.S. Navy Standard Air Decompression Tables. Yet, this subject always exhibited large numbers of events after such an exposure. However, when relatively short decompression periods were added to the dive profile, the number of post-dive events was drastically reduced. On one occasion, immediately after this individual surfaced from a dive and demonstrated excessive numbers of events (but no symptoms), he was put on 100% oxygen breathing by mask for 15 minutes. The number of events had significantly decreased at the end of this 15 minute period and there was no count by 3 hours post-dive.

Second and Third Years

The preliminary study was directed at the objective definition of work levels to be used during the bubble detection dives. Measurements made with the similarly equipped divers indicated that at resistances of 1.5 and 2.4 Kg on the underwater ergometer the swim speed is 21.5 and 24.2 m/min., respectively, with very small intra-and inter-individual differences. Thus, the work of moving the ergometer through the water at two of the resistance levels was calculated to be 32.37 and 58.24 Kg m/min. (5.112 and 8.946 Kcal/min/m^2), respectively. True physiological differences were found to exist between three resistance levels (Table III). An example of the data obtained by the underwater recording system is shown in Figure 17.

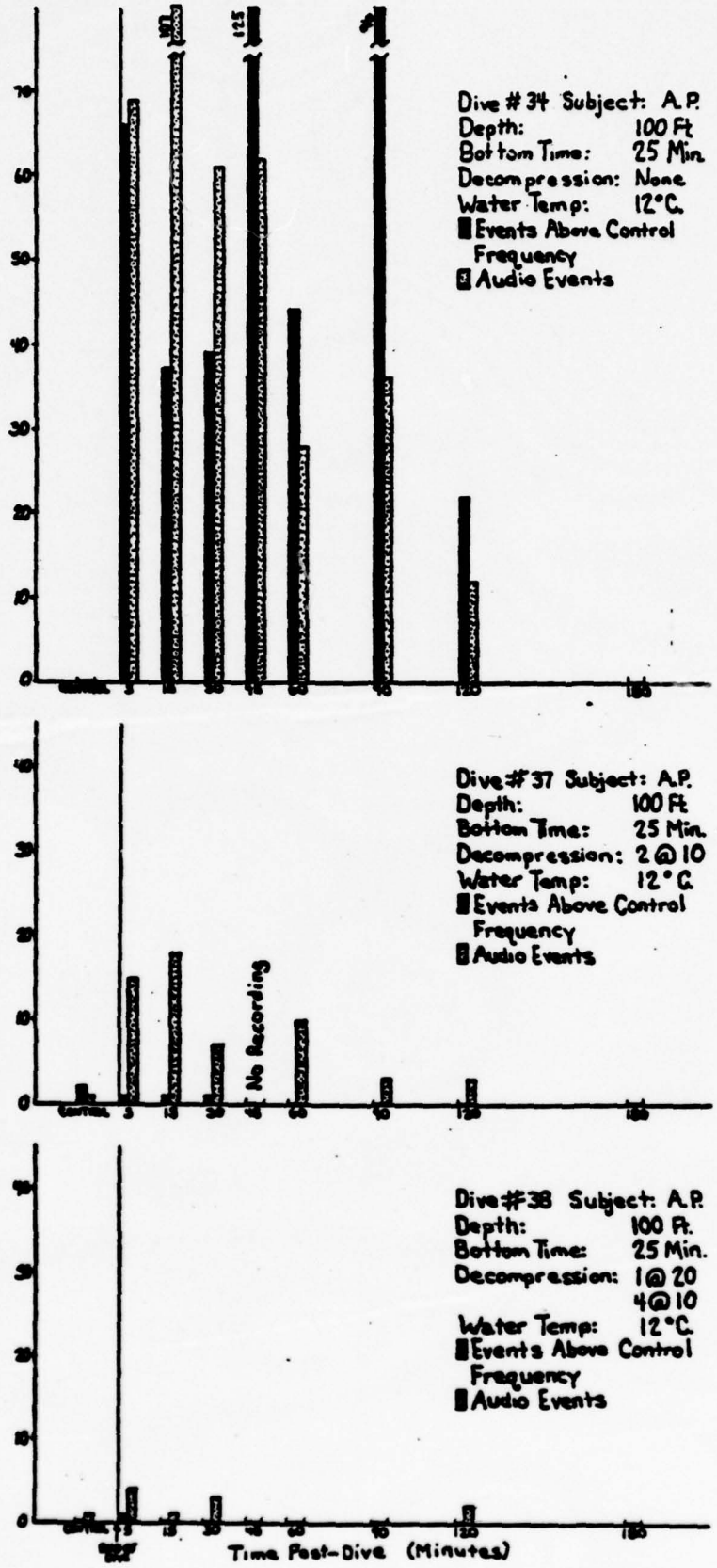


Figure 16. Vge data from subject A.P. after 3 dive profiles; depth and bottom times were identical, only the decompression was changed.

TABLE III

Mean Values and Standard Deviations for Ventilatory Volume (V_E), Respiratory Frequency (Vf), Tidal Volume (Vt), Oxygen Consumption (VO_2), Heart Rate and Energy Expenditure from Three Work Resistances on the Ergometer at 100 fsw for Six Subjects

	Ergometer Resistance (Kg)			
	Rest	1.5	2.0	2.4
V_E (L/min., BTPS)				
MEAN	11.363	43.478	58.919	70.905
SD	4.249	11.194	3.112	8.878
Vf (Breaths/min.)				
MEAN	10.167	23.833	24.833	27.667
SD	2.483	9.600	4.119	4.474
Vt (L/Breath)				
MEAN	1.147	1.928	2.413	2.584
SD	0.395	0.519	0.311	0.129
VO_2 (L/min., STPD)				
MEAN	0.429	1.982	2.715	3.465
SD	0.173	0.345	0.281	0.310
HR (bpm)				
MEAN	75.333	138.83	151.16	163.50
SD	12.987	18.70	16.30	14.88
Energy Expenditure (K cal/min./sq. m)				
MEAN	1.103	5.112	7.024	8.946
SD	0.437	0.826	1.021	1.025

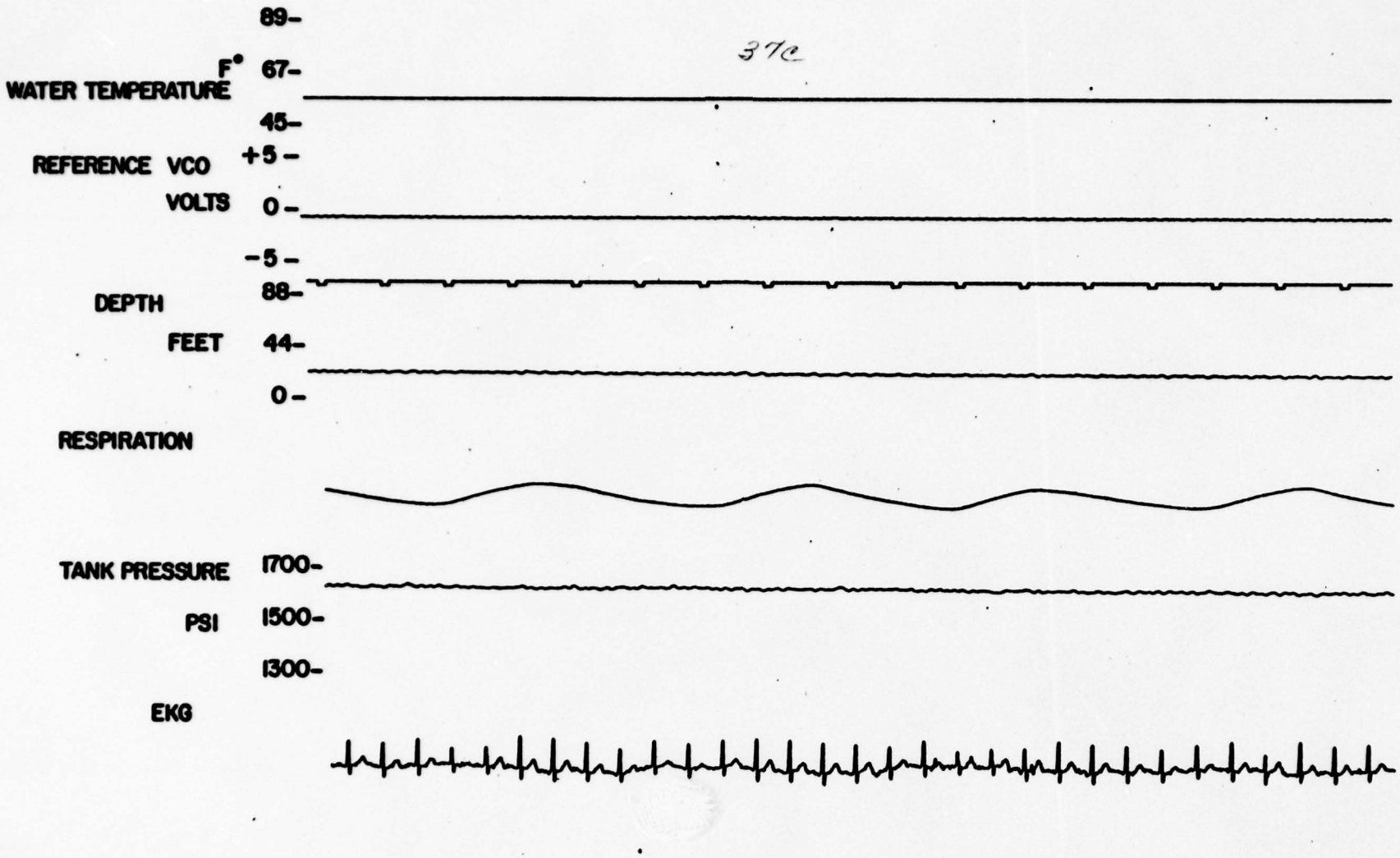


Figure 17. Example strip chart recording of data obtained by the Underwater Recording System.

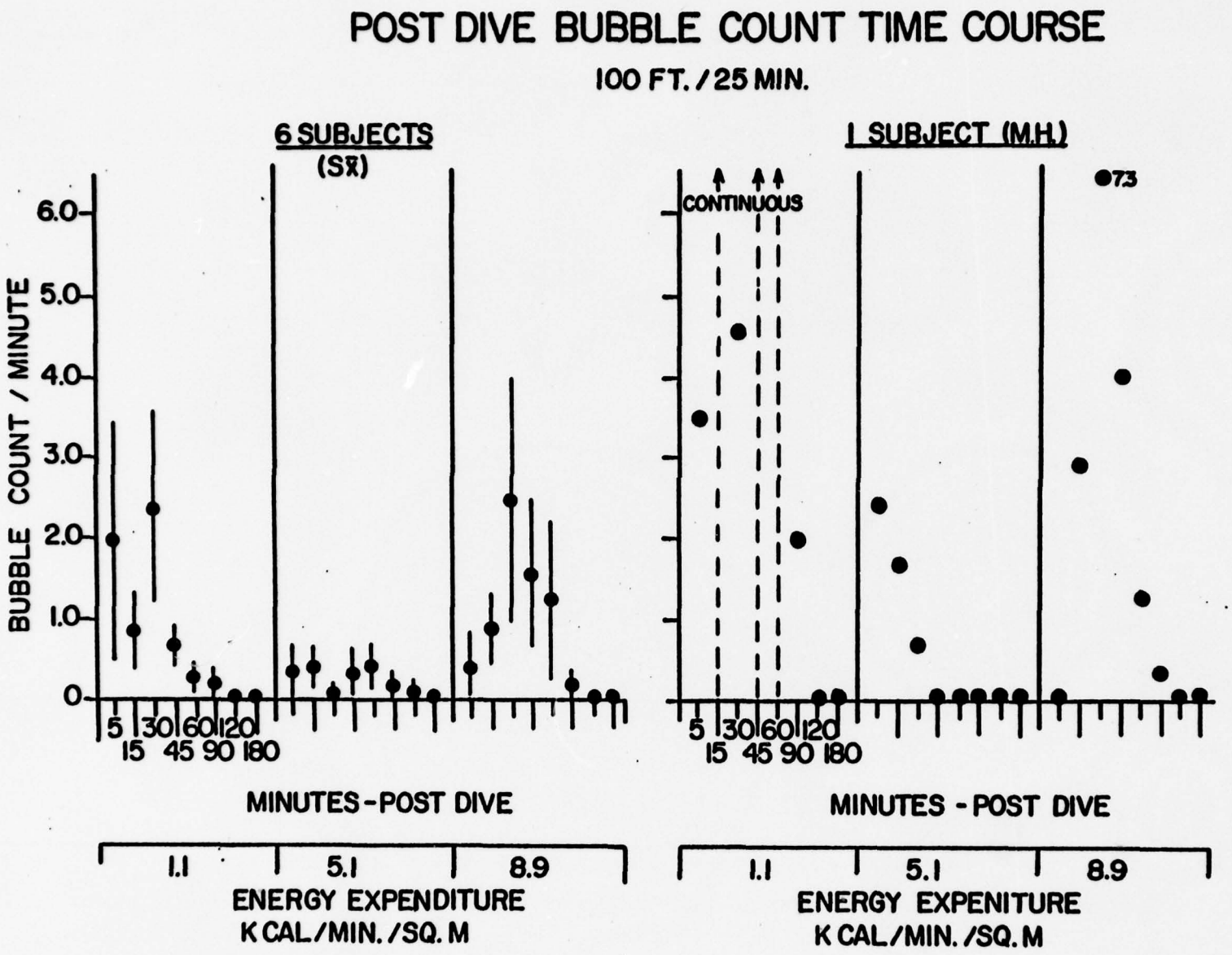


Figure 18. Vge data for 100 ft. /25 min. dives.

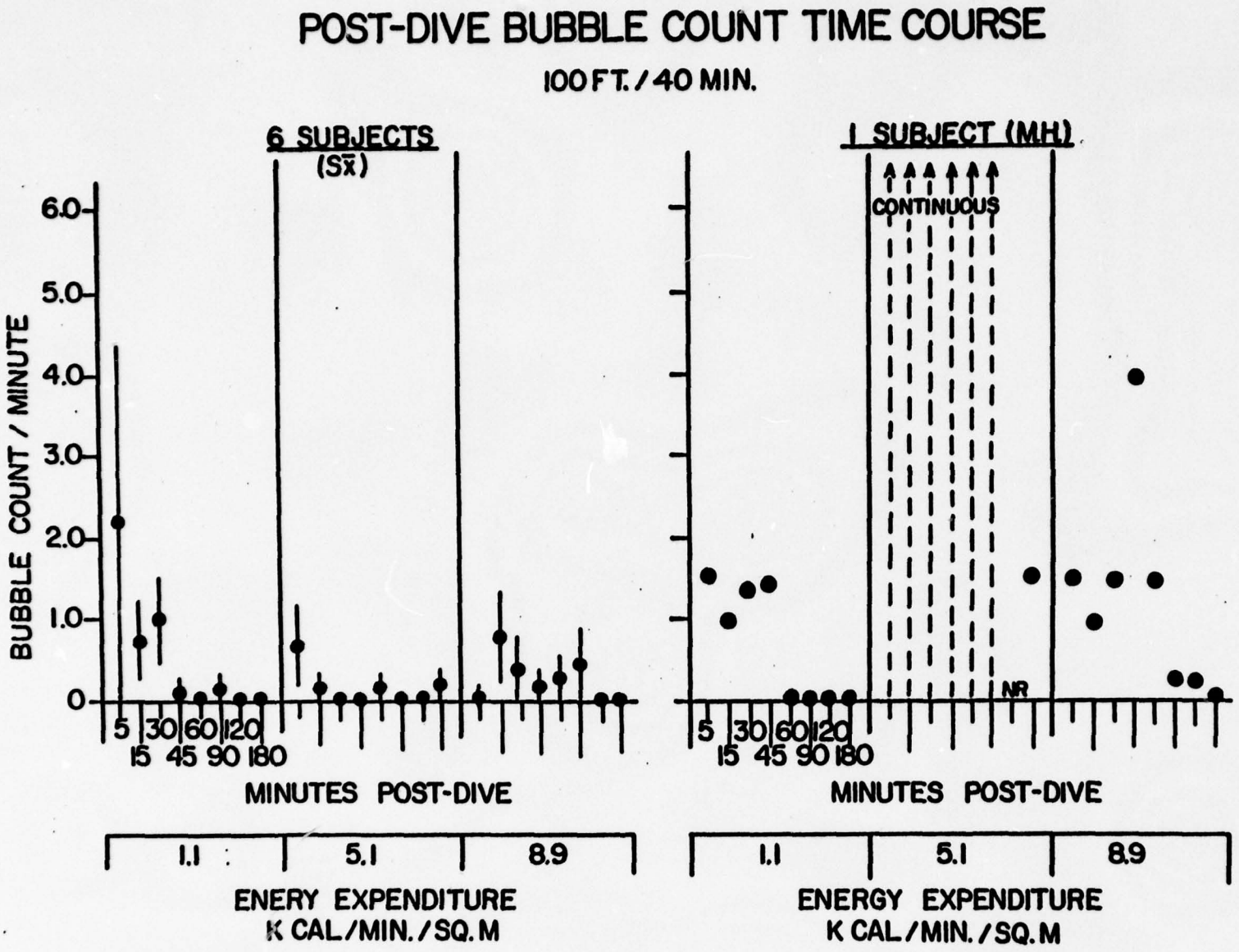


Figure 19. Vge data for 100 ft. /40 min. dives.

The cabling method of monitoring a decompressing submerged subject in the sea with the Doppler worked well. The dive profile used for this part of the study was 100 feet of depth, bottom time of 40 minutes with 15 minutes of decompression at 10 feet. The diver was monitored for vge during the entire 15 minutes of decompression. Data from 21 such dives (seven subjects) are included here. No vge sounds were heard during any of these nine decompressions. However, when monitored immediately after surfacing, all individuals had vge (Figure 19). The post-dive time course of bubble occurrence was consistent with the previous year's results. Typically, the bubbles appear within five minutes after surfacing, a peak level is reached within one hour and the numbers gradually decline to control levels by three hours post-dive (Figures 18 and 19). The data also further confirmed that individual subjects show a relatively consistent degree of bubble formation when compared to other subjects. In addition, the inter-subject variation of vge formation was as great as described earlier.

One case of symptomatic decompression sickness occurred. Within minutes of surfacing from an experimental dive, the subject developed pain in the right hip (data from this dive is not included here). When monitored with the Doppler, continuous "showers" of bubbles were heard precordially. The subject was treated within the hour at a nearby hyperbaric chamber on U.S. Navy Treatment Table 5 and the symptoms, as well as, the vge were quickly resolved. This investigator monitored the subject prior to, during and after the chamber

treatment with the Doppler. Just prior to compression there were continuous "showers" of vge in the subject. Upon compression to 30 fsw, a few individual vge were heard. When 60 fsw was reached no vge and no symptoms were found. No vge were heard during or after the treatment. The cause of this accident was a misunderstanding of the dive profile by the subject.

Figures 18, 19 and 20 show the data obtained in the attempt to relate "at-depth" exercise to the occurrence of vge. Data from seven subjects are shown. Six of these are grouped and one (M. H.) is shown separately. The reason for this is that this subject on several occasions had such large quantities of vge present that a count could not be made. Thus, his data are shown separately and labelled "continuous" where quantification could not be done. There were no significant differences in vge occurrence between the three levels of exercise. However, the trend toward fewer bubbles after the moderate exercise dives (5.1 Kcal/min/sq m) than either the resting or the heavy exercise dives is apparent.

Animal Studies

A total of 32 dog exposures were done in the chamber to 100 fsw. As has been mentioned, the exposure time of 20 minutes at 100 fsw was selected as the standard vge threshold exposure. This selection was based on two factors: 1) the calculated surfacing values for nitrogen tensions in the 10, 20 and 40 minute half-time tissues were close to the human values without exceeding them, and 2) the numbers

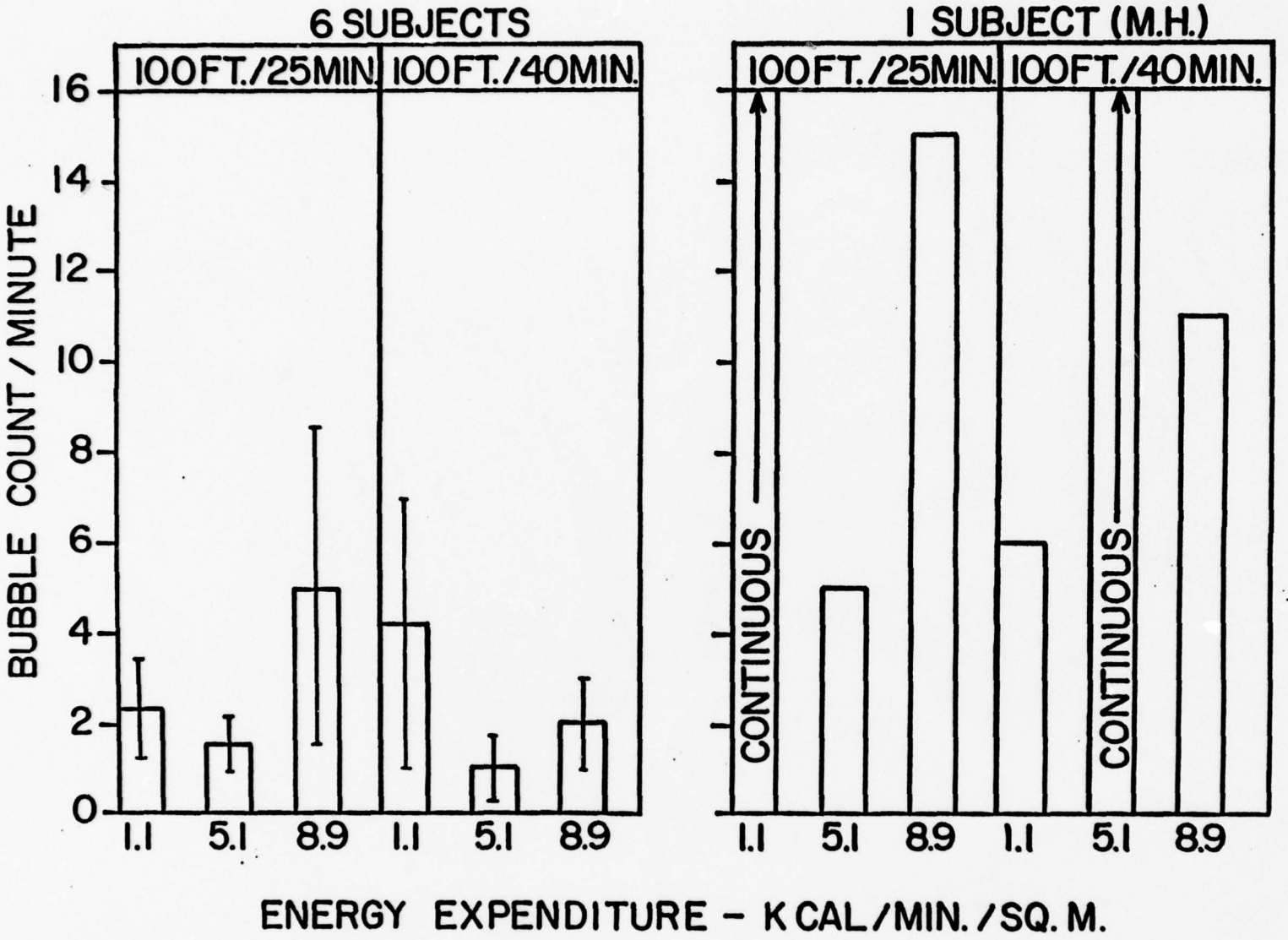


Figure 20. Vge count vs. 3 levels of underwater exercise; two dive profiles.

of vge detected were more than after the 15 minute exposure, but not as many as after the 25, 35, 40 and 60 minute exposures which were of the magnitude that would make quantification difficult and symptoms likely. No symptoms were seen in the dog after any of the experimental dives.

1. Resting Exposures. The data for the resting exposures are presented in Figure 21. Bubbles were detected between 15 and 35 minutes at their highest level (1.0 $\pm$0), declined to 65 minutes and were absent beyond that time. The mean vena caval blood flow was not significantly different from the pre-dive control values.

2. Exercise during exposure. As seen in Figure 22, mean vge scores displayed an increasing trend through the first 2 post-dive periods, reached a maximum (4.0) through the next 3 periods (to 60 minutes) and then declined during the last 3 periods. The mean vena caval blood flow was significantly greater than pre-dive controls prior to exercise, during exercise, just after exercise and in six of the 9 post-dive periods. The maximum value was reached at the end of the exercise period and was a difference of +12.25 flow units. Using the data provided with the flow probe, this represents an increase in flow of 4,322 ml/min. Calculations showed that this represents a three-fold increase of total cardiac output.

3. Exercise during decompression. The maximum mean vge score (1.75 $\pm$1.5) was observed during the second post-dive period (Figure 23). The mean scores then declined and reached zero after 120 minutes post-dive. Mean vena caval blood flow was significantly

Figure 21

Results of 20 Minute Resting

Exposures at 100 fsw

A. Mean vge scores

B. Mean vena caval blood flow

C. Mean heart rate

Vertical bars indicate standard deviations

*** Indicates no significant difference when compared to pre-dive control values of flows and heart rates**

Unmarked means indicate $P < 0.05$ for flow and heart rate

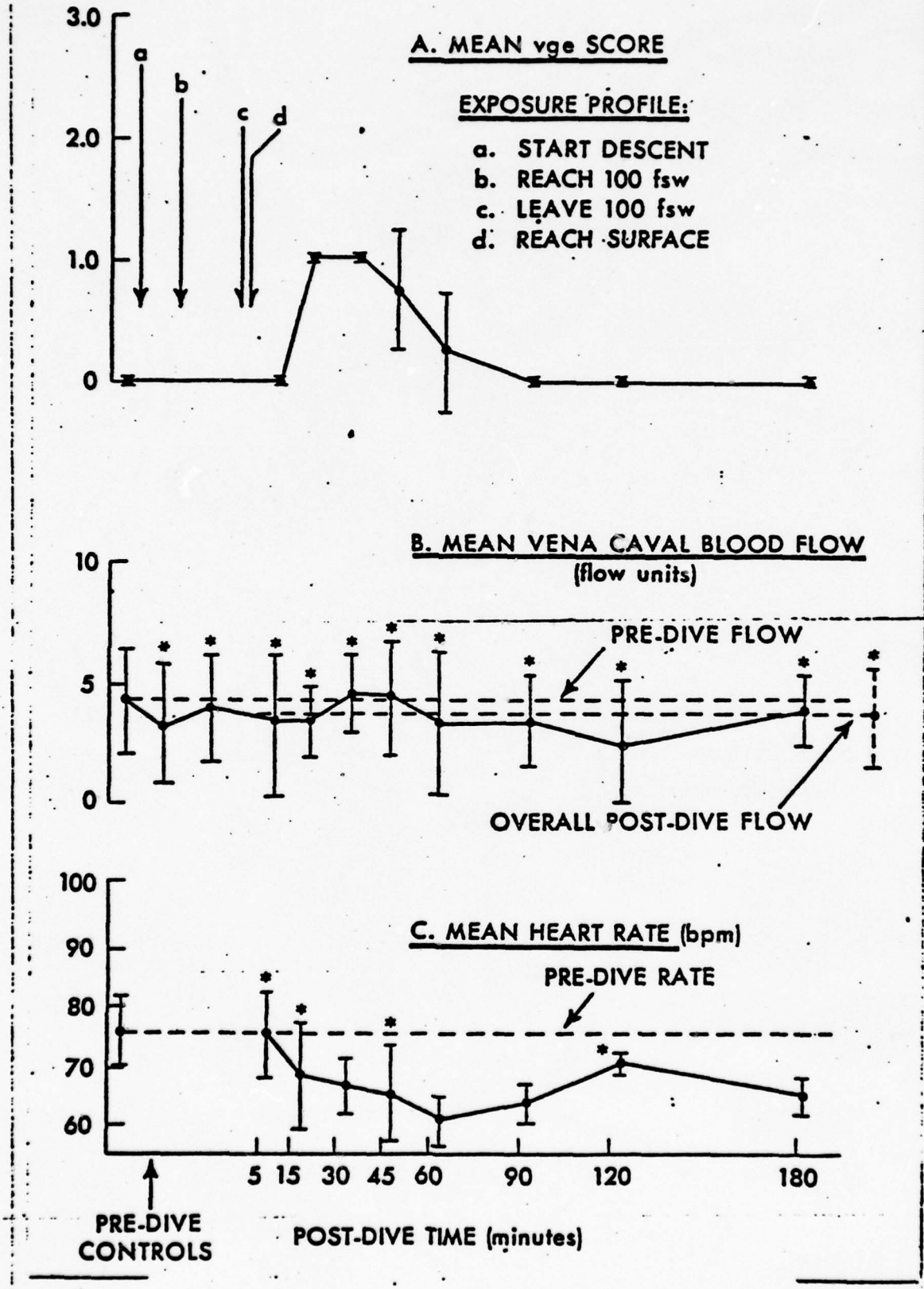

3.0
2.0
1.0
0
a
b
c
d
A. MEAN vge SCORE
EXPOSURE PROFILE:
a. START DESCENT
b. REACH 100 fsw
c. LEAVE 100 fsw
d. REACH SURFACE
10
5
0
B. MEAN VENA CAVAL BLOOD FLOW
(flow units)
PRE-DIVE FLOW
OVERALL POST-DIVE FLOW
100
90
80
70
60
C. MEAN HEART RATE (bpm)
PRE-DIVE RATE
5 15 30 45 60 90 120 180
PRE-DIVE CONTROLS
POST-DIVE TIME (minutes)

Figure 22

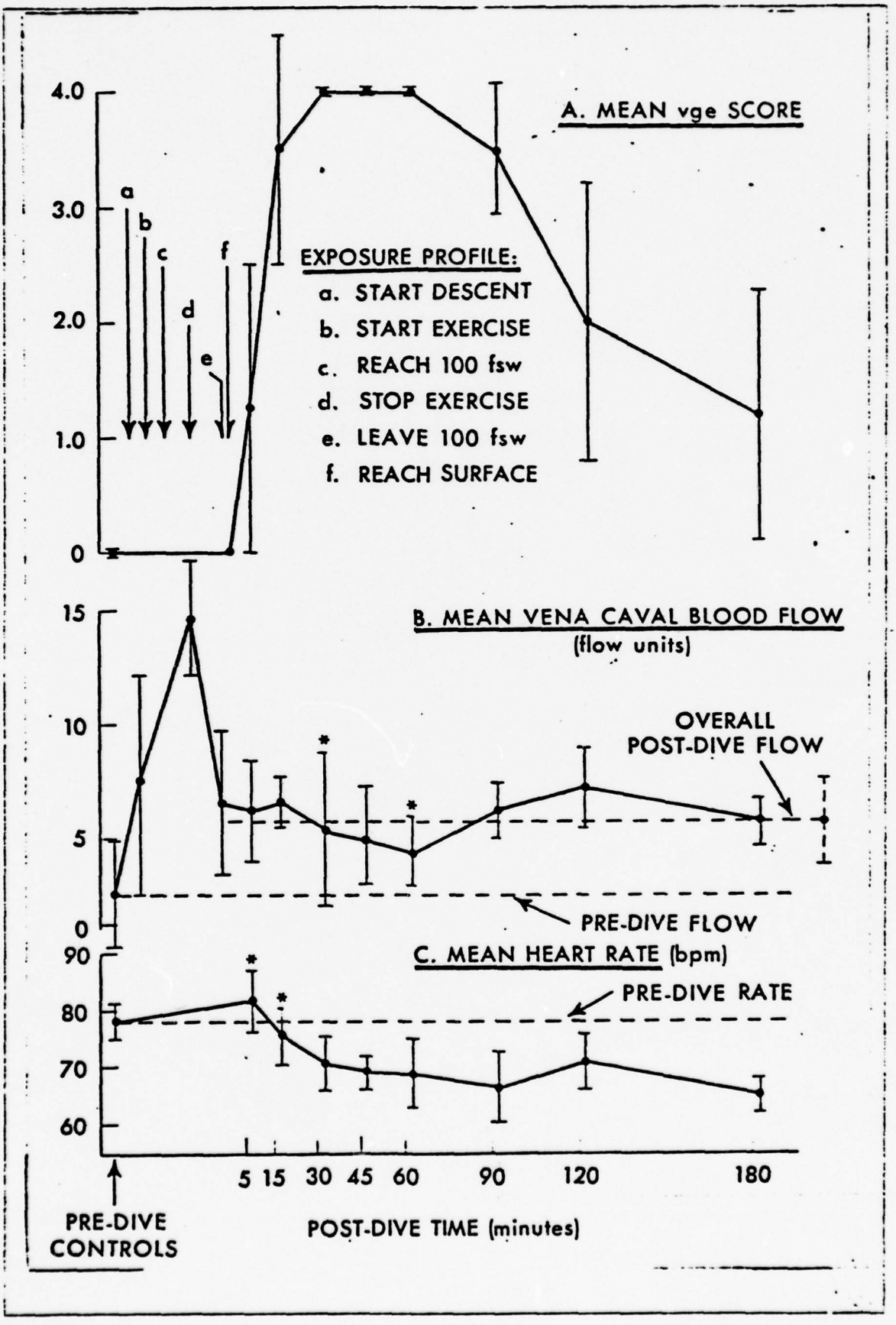
Results of 20 Minute Exposures with Exercise during Descent to, and Exposure at, 100 fsw

A. Mean vge scores

B. Mean vena caval blood flow

C. Mean heart rates

(see Figure 21 for legend)


A. MEAN vge SCORE
4.0
3.0
2.0
1.0
0
a
b
c
d
e
f
EXPOSURE PROFILE:
a. START DESCENT
b. START EXERCISE
c. REACH 100 fsw
d. STOP EXERCISE
e. LEAVE 100 fsw
f. REACH SURFACE
B. MEAN VENA CAVAL BLOOD FLOW
(flow units)
15
10
5
0
OVERALL
POST-DIVE FLOW
PRE-DIVE FLOW
C. MEAN HEART RATE (bpm)
90
80
70
60
PRE-DIVE RATE
5 15 30 45 60 90 120 180
PRE-DIVE
CONTROLS
POST-DIVE TIME (minutes)

greater than pre-dive values only during the exposure period and at the end of the exercise period.

4. Vasodilation during exposure. The vena caval blood flow data revealed that maximum values were attained during descent and during the time spent at 100 fsw. As seen in Figure 24 maximum (3.33 $\pm$0.58) vge scores were reached during the second and third periods, declined and were absent after 60 minutes post-dive. Mean vena caval blood flows were significantly greater during descent, exposure and six out of eight post-dive periods. The maximal flow increase was +7.41 flow units which represents a flow of 2,614 ml/min. or approximately 60% of the increase attained with exercise.

5. Vasodilation during decompression. As with the resting situation no vge were detected in the first period (Figure 25). The maximal vge (0.75 $\pm$0.5) score was seen in the second period. The scores declined and were absent after 50 minutes. Mean vena caval blood flows were significantly greater than pre-dive levels during five of the eight post-dive periods. Maximal flow was 4.31 flow units which represented 1,521 ml/min. or 35% of the increase with exercise.

Figure 26 presents a comparison of the mean vge scores as a function of the post-dive time for all five exposure series and for a 60 minute resting exposure at 100 fsw. In terms of both quantitative and temporal relationships the 20 minute exercising exposure mean vge scores are most closely correlated with the 60 minutes resting exposure, while the 20 minutes vasodilation during decompression series correlates

Figure 23

Results of 20 Minute Exposures at 100 fsw

with Exercise during and following Decompression

A. Mean vge scores

B. Mean vena caval blood flow

C. Mean heart rates

(see Figure 21 for legend)

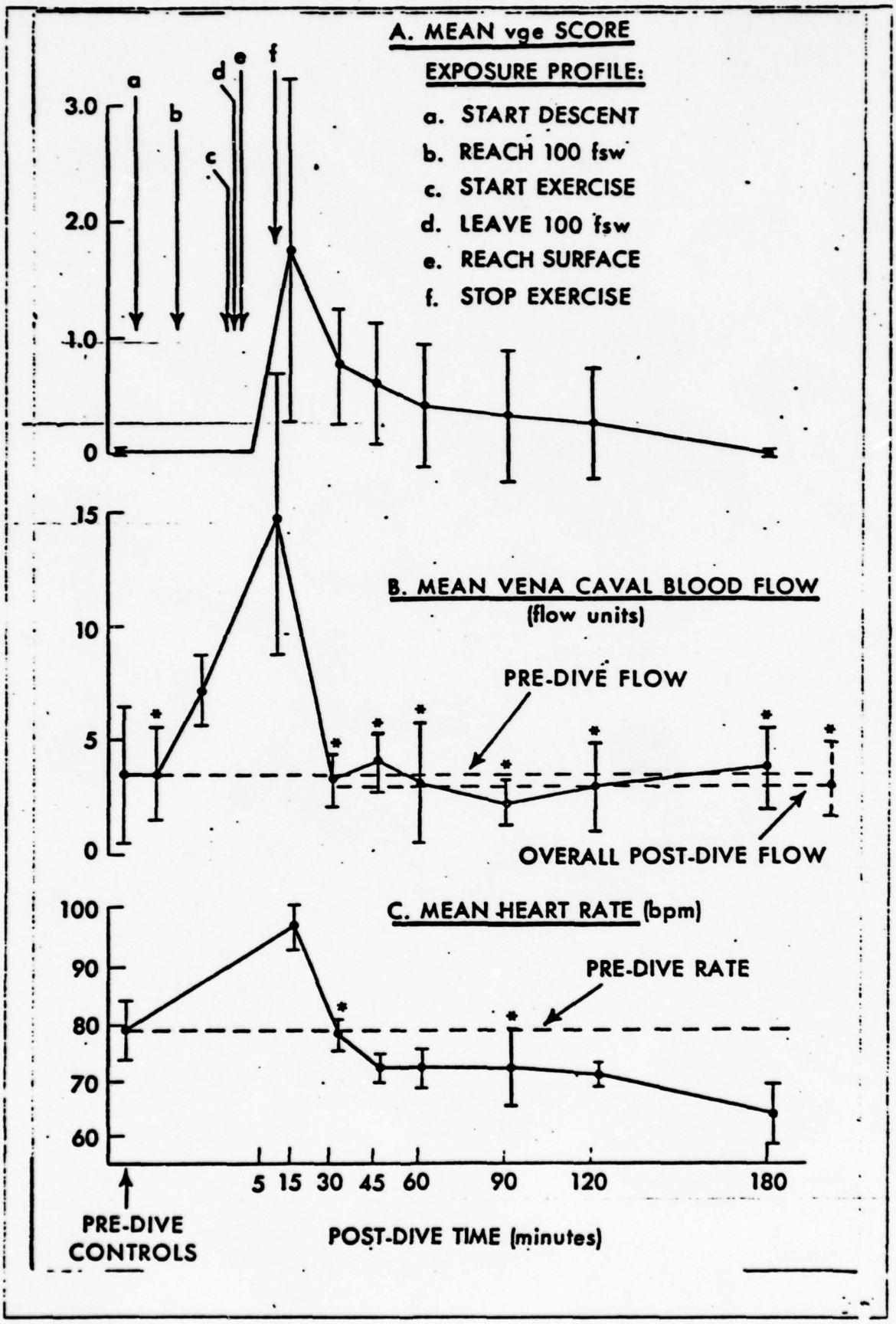

A. MEAN vge SCORE
EXPOSURE PROFILE:
a. START DESCENT
b. REACH 100 fsw
c. START EXERCISE
d. LEAVE 100 fsw
e. REACH SURFACE
f. STOP EXERCISE
3.0
2.0
1.0
0
B. MEAN VENA CAVAL BLOOD FLOW
(flow units)
15
10
5
0
PRE-DIVE FLOW
OVERALL POST-DIVE FLOW
C. MEAN HEART RATE (bpm)
100
90
80
70
60
PRE-DIVE RATE
5 15 30 45 60 90 120 180
PRE-DIVE CONTROLS
POST-DIVE TIME (minutes)

Figure 24

Results of 20 Minute Resting Exposures at 100 fsw

with Injection of Vasodilator Prior to Exposure

A. Mean vge scores

B. Mean vena caval blood flow

C. Mean heart rates

(see Figure 21 for legend)

NOTE: Heart rates during the dive profile (dashed line) represent heart rates measured during 2 to 3 control experiments at sea-level pressure

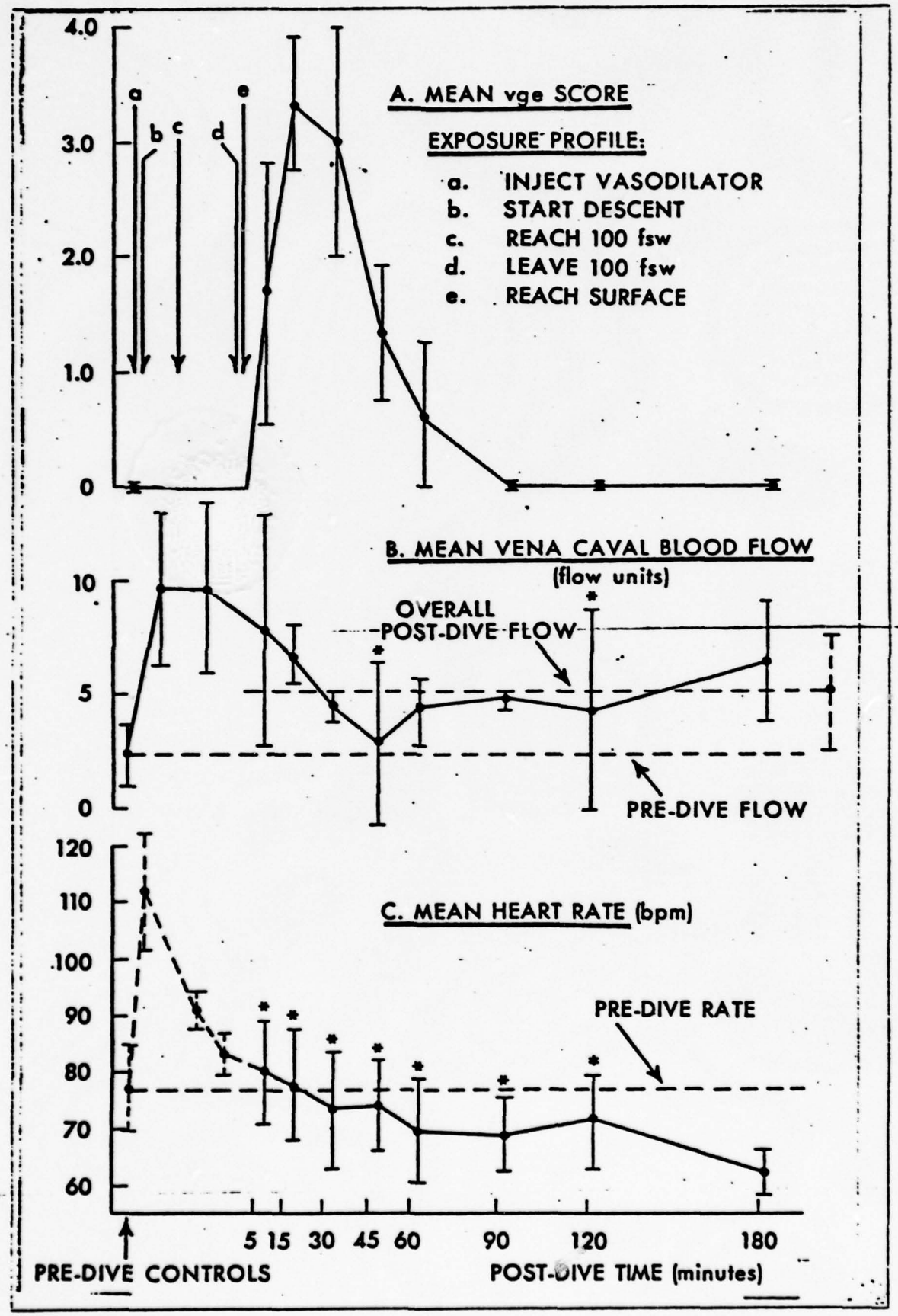

A. MEAN vge SCORE
EXPOSURE PROFILE:
a. INJECT VASODILATOR
b. START DESCENT
c. REACH 100 fsw
d. LEAVE 100 fsw
e. REACH SURFACE
4.0
3.0
2.0
1.0
0
B. MEAN VENA CAVAL BLOOD FLOW
(flow units)
OVERALL POST-DIVE FLOW
PRE-DIVE FLOW
10
5
0
C. MEAN HEART RATE (bpm)
PRE-DIVE RATE
120
110
100
90
80
70
60
5 15 30 45 60 90 120 180
PRE-DIVE CONTROLS
POST-DIVE TIME (minutes)

Figure 25

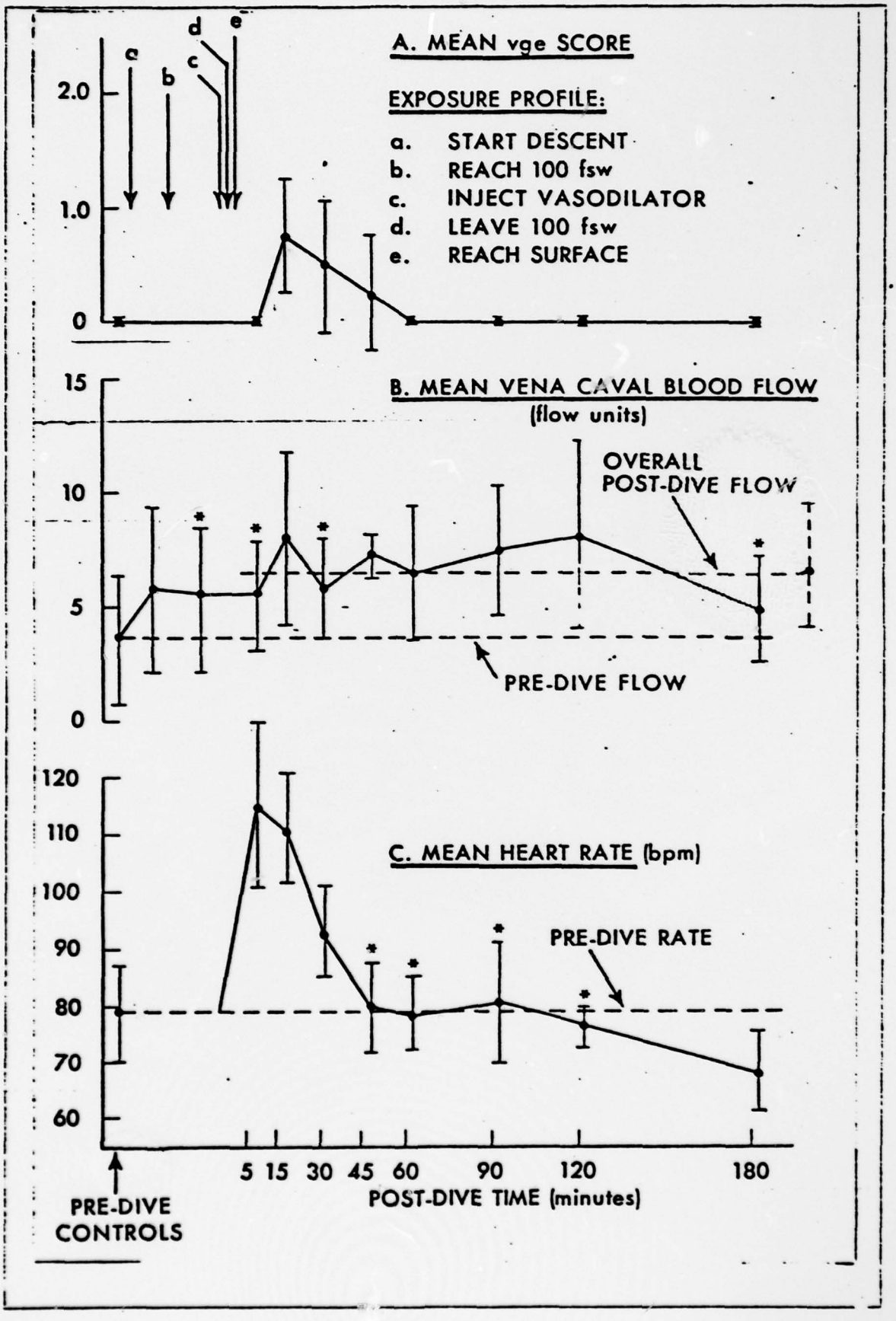
Results of 20 Minute Resting Exposures at 100 fsw with Vasodilator Injected 1 Minute Prior to Decompression

A. Mean vge scores

B. Mean vena caval blood flow

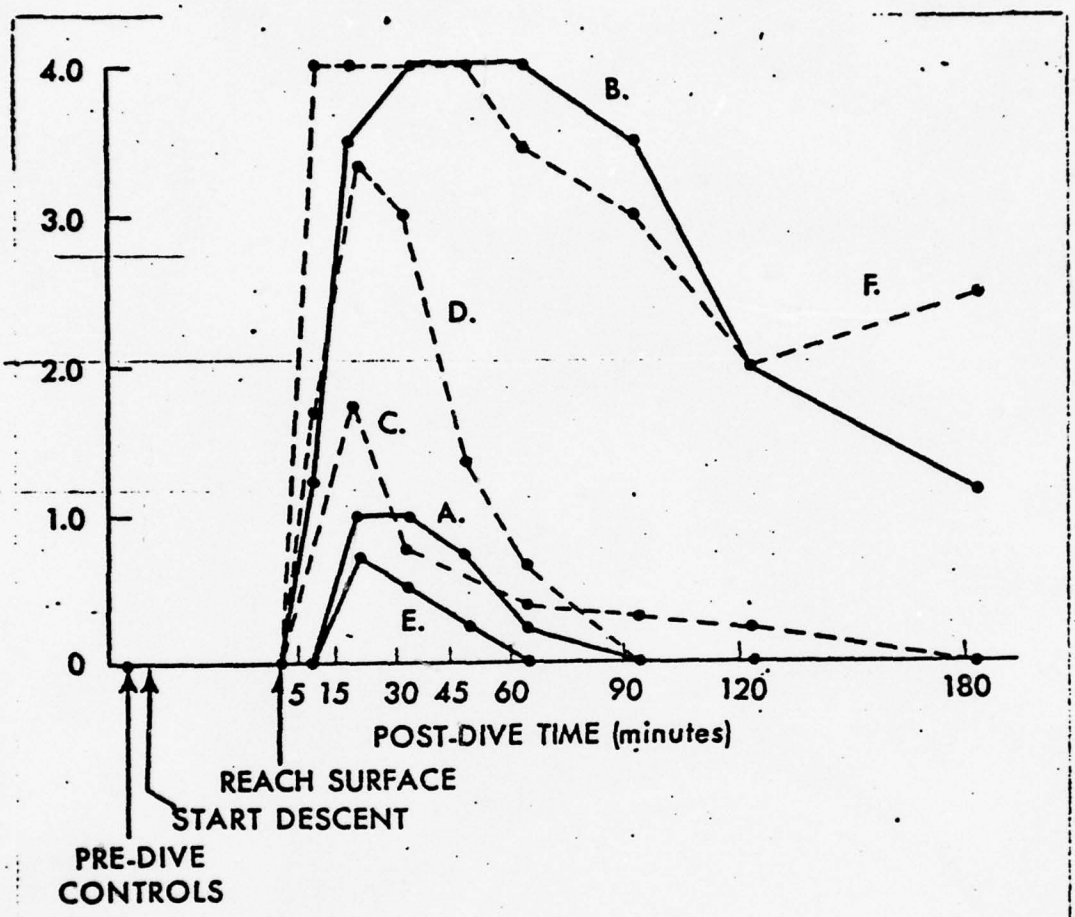
C. Mean heart rate

(see Figure 21 for legend)


A. MEAN vge SCORE
EXPOSURE PROFILE:
a. START DESCENT
b. REACH 100 fsw
c. INJECT VASODILATOR
d. LEAVE 100 fsw
e. REACH SURFACE
2.0
1.0
0
B. MEAN VENA CAVAL BLOOD FLOW
(flow units)
OVERALL POST-DIVE FLOW
PRE-DIVE FLOW
15
10
5
0
C. MEAN HEART RATE (bpm)
PRE-DIVE RATE
120
110
100
90
80
70
60
PRE-DIVE CONTROLS
5 15 30 45 60 90 120 180
POST-DIVE TIME (minutes)

. Figure 26

Comparison of Mean VGE Scores Obtained in all Experimental Series, Including a Comparison with 60 Minute Resting Exposures at 100 fsw



A. RESTING EXPOSURE, 20 min. AT 100 fsw
B. EXERCISING EXPOSURE, 20 min. AT 100 fsw
C. EXERCISING DURING DECOMPRESSION, 20 min. AT 100 fsw
D. VASODILATION DURING EXPOSURE, 20 min. AT 100 fsw
E. VASODILATION DURING DECOMPRESSION, 20 min. AT 100 fsw
F. RESTING EXPOSURE, 60 min. AT 100 fsw

most closely with the 20 minutes resting exposure vge scores.

DISCUSSION

The full extent of the pathophysiological complexity of decompression sickness has recently become more and more apparent. At the same time, many of the basic mechanisms of action that lead to the varied and interrelated clinical manifestations of decompression sickness remain elusive. This laboratory was one of several which a few years ago initiated studies aimed at elucidating part of the physiological puzzle of this diving ailment through the use of ultrasonic in vivo bubble detection. From these studies has emerged the concept of intravascular "silent bubbles." The existence of these asymptomatic venous gas emboli is now generally accepted. However, the pathophysiological significance of these bubbles has still not been adequately defined.

It is clear from this study, as well as others, that man can tolerate and eliminate at the lungs relatively large quantities of gas emboli from the venous system without developing clinical symptoms of decompression sickness. It is also clear that pre-symptomatic bubbles are present in large numbers after open ocean dives which strictly adhere to the limits of the U.S. Navy Standard Air Decompression Tables. It would appear that silent bubble formation and clinically symptomatic bubble formation are not two distinct conditions, but rather, the same condition at various levels of gradation. The point of development of obvious symptoms is not necessarily synonymous with the start of tissue damage. The pathology from vge may simply be a milder form of symptomatic tissue damage. Bubbles are known to

trigger pathologic reactions indirectly through surface activity at the gas-blood interface. Thus, any gas emboli in the tissues are potentially harmful. The complex hematological changes associated with circulating gas emboli have been extensively described (2).

Bove, Hallenbeck and Elliott (23) have shown in dogs that extensive venous gas embolization can result in a complex series of hematological and cardiovascular changes terminating in spinal infarction. The extent of these venous bubbles required for manifestation of spinal symptoms is not clear. If this hypothesis can be applied to man, one wonders at what level of vge occurrence, as determined by the Doppler, a spinal "hit" could occur.

Dysbaric osteonecrosis can be induced experimentally in animals with intravascular bubbles (24). There vascular bubbles basically obstruct and disrupt blood flow and cause wall damage to the blood vessels in bone. It would appear likely that chronic exposure to even sub-symptomatic diving which is capable of eliciting vge could result in chronic bone damage. In addition, the circulating gas emboli may only be the <u>detectable</u> indicators of the presence of bubbles in other tissues. The presence and effect of "silent" gas bubble formation in non-vascular tissue has not been investigated.

It is evident from the results of this study that silent bubbles are present to some extent in all individuals after exposure in the open ocean to the particular dive profiles described. It is also clear that even very small extensions of the decompression times for these

profiles will substantially reduce, if not eliminate, the occurrence of vge post-dive (Figure 16). These data point out the accuracy of the U.S. Navy Standard Air Decompression Tables used. Since the basis of these tables is the presence of overt symptoms in a large population sample in conjunction with theoretical model calculations, which are also ultimately based on symptomatology, the occurrence of silent vge at these limits is not surprising. If one accepts that only overt clinical manifestations define decompression sickness then the results of this study testify to the accuracy of the Tables. This author prefers to look upon the condition of decompression sickness as being defined by the first gas bubbles formation in the body whether they elicit overt symptoms or not. However, such a definition is academic since, currently, only intravenous silent bubbles can be detected, and then only if one has a Doppler unit available on site and has been trained in its use.

The time course post-dive of vge occurrence is evident in the results (Figures 15, 18, 19, 21 and 22). The emboli appear within a few minutes after surfacing, peak in numbers within an hour post-dive, and then decline to control levels by 3 hours post-dive. This was true in the human exposures in the open sea, as well as animal exposures in a chamber. These data indicate that if a Doppler type unit is to be used for clinical confirmation of decompression sickness, it must be used within one or two hours of surfacing. This was confirmed by two of the clinical cases brought to the Catalina facility for recompression therapy of Type II (spinal) symptoms. In both cases the delay to the

chamber was in excess of 6 hours and in neither case were vge found when the Doppler was used. In contrast, it has been noted in the literature that non-symptomatic animals exhibit vge up to 72 hours post-dive (6).

Development of the HP-65 program for rapid computations of tissue inert gas tensions enables us to: 1) define which half-time tissues are limiting for each dive profile, 2) determine N_2 elimination curves for the post-dive periods, and thus, 3) compare the occurrence of vge with the theoretical tissue N_2 tensions. Figure 8 shows that after a 100 ft./25 min. dive the 10 and 20 minute half-time tissues are limiting. Both of these tissues have tensions exactly equal to the M values. On the other hand, after the 100 ft./40 min. (Figure 9) dive, the P_{N_2} of the 40 minute half-time tissue was limiting and, in fact, higher than either M value. Vge are present almost always at surfacing from either of these dives. If even two minutes of decompression at 10 feet are added to the 100 ft./25 min. profile, the vge numbers are drastically reduced (Figure 16). If two minutes at 20 feet and four minutes at ten feet are added, no vge are recorded. Thus, with at least the dive profiles tested, at the M values there is definite intravenous bubble formations. It can be speculated that there is also bubble formation in extravascular tissues. The above data are in agreement with the findings of Spencer (25).

The described method for open ocean bubble detection during underwater decompression worked well. With the one dive profile used

(100 ft./40 min.), there were no vge detected at the 10 ft. stop. Figure 9 shows that at reaching the 10 ft. stop of this dive, no tissues were limiting. Here again, the data indicates if the tissue N_2 tensions are only slightly less than the M values, bubble formation does not occur.

One of the original objectives of this study was to investigate the feasibility of using the bubble detection method during decompression to determine individual decompression profiles. This investigator feels that underwater ultrasonic monitoring for vge routinely by divers themselves in order to gauge their decompression is not a fruitful approach for several reasons. Most important, it is an after-the-fact approach. Obviously, it is much easier to prevent bubble formation than to resolve bubbles and associated tissue damage. Secondly, with currently available equipment it would be highly impractical for divers in the field to reliably identify vge. It would be extremely difficult to do this underwater. Thirdly, although vge have been recorded during decompression in a chamber, the consensus of data indicates that it is in the post-dive period that silent bubbles reliably manifest themselves.

In agreement with other published data (6), the results of this study confirmed the individual person variability of vge formation. In all cases, except one, there were significant differences between the means of individual subject-dives, i.e., the degree of occurrence of bubbles significantly varied between all of the subject-dives of each of the 3 dive profiles (Table II). Furthermore, each individual showed a relatively consistent degree of bubble formation on the various dive

profiles. Since individuals are consistent in the degree of bubble formation from hyperbaric exposures, it is suggested that standardized chamber tolerance tests using a bubble detector could be of great value to people regularly exposed to this environment. An individual found to be highly susceptible to bubble formation could then routinely adjust his decompression schedules accordingly. In this way, the risk of development of acute decompression sickness symptoms, as well as potential chronic bone problems, could be reduced.

The final objective of this study was the attempt to define the relationships between decompression bubble formation and exercise. The animal experiments (Figures 21, 22 and 23) demonstrated that increased blood flow to actively exercising muscle increased the N_2 loading of these tissues when generalized sustained exercise was performed during exposure to a vge threshold dive profile. The result is an increased production of vge following decompression. The effect of 15 minutes of exercise during exposure to 100 fsw was equivalent to a 3-fold increase in exposure time at rest in terms of post-dive vge score. This was directly related to the 3-fold increase in blood flow produced by the exercise. On the other hand, exercise <u>during decompression</u> did not result in any significant change in the mean vge scores found after the resting exposure. This demonstrates that, at least with the type of exercise and dive profile used, the mechanical factors of exercise during decompression are of relatively little importance to vge formation. That the primary influence of exercise on vge occurrence

is perfusion changes rather than mechanical factors was further confirmed by the results of the drug-induced vasodilation experiments (Figures 24 and 25). Here there was no exercise and, thus, no mechanical factors present. It was again found that with increased tissue perfusion during exposure, i.e., increased N_2 loading, there were significant increases in vge formation post-dive. With increased perfusion during decompression, post-dive vge scores were not significantly different from resting exposures scores.

An interesting observation was made during these animal experiments regarding blood flow results. The mean blood flow remained significantly greater when compared to the resting values in the exercise during exposure series for as long as three hours post-dive. However, these sustained post-dive flow elevations were not seen in the exercise during decompression series. In this series the mean vena caval flow returned to resting levels within 20 minutes after cessation of exercise. The only difference between the two series was the greatly elevated and sustained vge score following the exercise during exposure series. Do intravascular bubbles modify vascular tone? Can vge elevate and sustain venous blood flow? If so, does that modify N_2 elimination?

In contrast to the chamber animal experiments, all human exercise experiments were done in the open ocean. A substantial series of preliminary experiments were required prior to the bubble detection dives. A method for determining and standardizing levels of exercise underwater in the ocean was developed. The Underwater Data Recorder,

Underwater Ergometer and Underwater Gas Sampler provided an objective and quantitative means of defining the exact level of a diver's exercise underwater. This was necessary in order to correlate the extent of vge occurrence with various levels of physical work. Exercise during bottom exposure only was used; all decompression was done at rest.

From Figure 20 we can see that with both dive profiles there was a trend, not statistically significant, for more vge to form after the resting and the heavy exercise at depth dives than after the moderate work dives. Subjectively, the 5.1 K cal/min/m^2 (moderate) level of underwater activity was found to be comfortable by the divers, both from the standpoint of physical exertion as well as cold stress. The heavy exercise was found to be very close to maximal. There was no cold stress with either the moderate of heavy exercise dive. The resting level was somewhat uncomfortable due to cold stress and boredom.

Spencer (6) found that in divers actively using one arm during their bottom time, more vge occurred in the non-exercising arm than in the exercising arm. He postulated that in the exercise arm post-exercise hyperemia could result in greater N_2 elimination than in the non-exercising arm and, thus, fewer bubbles post-dive. This concept might also explain the above results. In the resting exposure, the subject probably has close to normal resting perfusion during the early part of the dive. Progressively, during the bottom time, due to vaso-constriction from cold stress, blood flow in the muscle bed diminishes. This lower level of blood flow does not immediately reverse upon

surfacing from the dive. Thus, there is "restricted" N_2 elimination post-dive predisposing the subject to vge formation. With moderate exercise at depth, there is little cold stress and an exercise-induced increase in perfusion and N_2 uptake. Since there is a relatively short time period between termination of at depth exercise and surfacing (approximately 5 minutes with the 100 ft./25 min. profile and approximately 20 minutes with the 100 ft./40 min. profile) post-exercise hyperemia is probably still in effect and N_2 elimination is also at a higher level post-dive. Likewise, with heavy exercise at depth there is increased perfusion and N_2 uptake during the exposure (greater than at moderate exercise). There is also undoubtedly post-exercise hyperemia present after surfacing and, thus, increased N_2 elimination. However, since vasodilation in muscle during and after exercise is primarily determined by local metabolic factors and not N_2 gradients, the excess N_2 taken up by the heavily exercising muscle tissues may not be eliminated fast enough by the increased perfusion of the post-exercise hyperemia and vge formation may be accelerated. In contrast, the moderate exercise situation may coincide with an optimum blood flow level for maximizing N_2 elimination.

It should be noted that the results of this study support the instructions in the U.S. Navy Diving Manual (26) which state that if heavy exercise and/or cold conditions are encountered during diving, there should be a compensatory increase in decompression time.

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