TABLE I

Subjects as indicated by roman numerals	Cc of tidal air, with Schafer method, reduced to 0° C. and 760 mm Hg., dry	Cc of tidal air, with combined method, reduced to 0° C. and 760 mm Hg., dry	Percent efficiency of combined method compared to Schafer	Subjects as indicated by roman numerals	Cc of tidal air, with Schafer method, reduced to 0° C. and 760 mm Hg., dry	Cc of tidal air, with combined method, reduced to 0° C. and 760 mm Hg., dry	Percent efficiency of combined method compared to Schafer
I	754	1,126	149.3	VI	811	1,252	154.3
II	858	1,279	149.1	VII	711	1,128	158.6
III	914	1,392	152.2	VIII	827	1,217	147.1
IV	914	1,395	152.6	IX	835	1,265	151.4
V	991	1,427	143.9				

Modifications of the Schafer prone pressure method have been frequently proposed. Investigations of many of these methods have revealed no real advantage. The method introduced by Holger Nielsen (3) has recently received publicity in the daily press. We have not been able to demonstrate that the Holger Nielsen method affords any greater ventilation than the Schafer prone pressure method. In view of the fact that the Schafer method has been standardized by an immense experience, it would be unfortunate to introduce any new details for no real advantage. It is believed that the Schafer method should be continued as the standard when only a single operator is available. On the arrival of an assistant, however, lifting the arms upon release of pressure and lowering the arms when pressure is reapplied should afford a simple means of appreciably increased ventilation.

SUMMARY

1. A combination of the Schafer method and arm lift is evaluated and advocated because of the increased ventilation it affords.
2. The value of the arm lift is emphasized.

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THE USE OF OXYGEN IN THE TREATMENT OF COMPRESSED-AIR ILLNESS[1]

By ALBERT R. BEHNKE, Lieutenant, Medical Corps, United States Navy, and LOUIS A. SHAW

The early investigators (Bert, 1878; Zuntz, 1897; and Heller, Mager, and von Schrötter, 1900) concluded largely as a result of theoretical considerations that recompression combined with oxygen inhalation provided a rational and effective treatment for compressed-air illness. Oxygen inhalation, however, has been neglected probably

[1] From the Department of Physiology, Harvard School of Public Health, Boston, Mass. Received for publication Sept. 6, 1935. This research was aided by the Miriam Smith Rand fund.

for the following reasons: conclusive experimental evidence as to its value was lacking; man's tolerance for oxygen was not known; and facilities were not available for its economic administration. Thus, the treatment for compressed-air illness outlined in the Naval Diving Manual (1924) is essentially recompression with the patient breathing air. While this type of treatment affords relief in mild cases, it often fails in serious cases to prevent disability or death. It seemed worth while, therefore, to evaluate oxygen therapy on an experimental basis, and to determine the limits within which oxygen could be safely inhaled. In this paper are summarized the results of these studies.

The problem.—Rapid decompression after sufficient exposure to increased air pressure may result in the formation of nitrogen bubbles in the blood stream. These bubbles when sufficiently large and numerous mechanically obstruct blood flow, and deprive the tissues of their normal blood supply. Deprivation of blood supply gives rise to the characteristic symptoms of compressed-air illness, namely, asphyxia, paralysis, and pain. These symptoms occur either singly or in combination, and indicate that the areas for bubble formation and predilection are the right side of the heart and pulmonary vascular bed, the spinal cord, and probably the bone marrow. Treatment aims at the removal of bubbles from these areas in the shortest possible time in order to minimize injury particularly with reference to the spinal cord and the right ventricle.

Theoretical considerations.—The routine treatment of compressed-air illness, recompression with the patient breathing air, relieves symptoms immediately by reducing the size of bubbles, but accomplishes little in the way of bubble removal. This will be made clear by an example. If a dog in equilibrium with a gauge pressure of 60 pounds is suddenly decompressed to atmospheric pressure, the dissolved nitrogen in the blood will exist in a state of supersaturation, and diffusion of nitrogen from the blood stream into the lungs will proceed with an initial pressure head of 60 pounds (3,040 mm.). Should bubbles form in the blood, the tension of nitrogen drops rapidly since the pressure in the bubbles is that of the surrounding medium (1 atmosphere) in addition to their surface energy. Since the percentage of nitrogen in a bubble is about

$$82\frac{(760-(47\text{(water vapor)}+\rightarrow 45\text{ (carbon dioxide tension)}}{760}$$

$$\frac{+40\text{ (oxygen tension)}}{}\cdot 100),$$

and in the alveolar air,

$$75\frac{(760-(47\text{ (water vapor)}+40\text{ (carbon dioxide tension)}\rightarrow}{760}$$

$$\frac{+100\text{ (oxygen tension)}}{}\cdot 100),$$

nitrogen diffusion proceeds at a pressure head (disregarding surface energy) of about 56 millimeters (7.3 percent .760). Nitrogen from the tissues, meanwhile, is diffusing into the peripheral capillaries at an excess pressure of 60 (—) pounds. Under these conditions the blood stream is rapidly filled with gas in bubble form. By the time that recompression is applied the quantity of nitrogen in bubble form is well in excess of the capacity of the blood to dissolve nitrogen at practicable pressures. Nitrogen in bubble form must be eliminated by passage either into the tissues through the peripheral capillaries, or into the lungs through the pulmonary capillaries. When air (79 percent nitrogen[2]) is breathed nitrogen diffusion from a bubble (82 percent nitrogen) proceeds with a negligible pressure head.

The futility of eliminating large quantities of nitrogen from bubbles in the pulmonary capillaries when air is breathed, is further emphasized by the results of clinical experience. Thus, Haldane (1927) states that bubble elimination is a slow process, and that it may be necessary to keep a patient in the pressure chamber 24 hours or more. Keays in 1912 reporting on 3,692 cases of compressed-air illness states that recompression, while an efficient means of treatment in mild cases often fails to prevent disability and death in severe ones. In the experiments of Boycott, Damant, and Haldane (1908) bubbles were found in the blood stream of a goat 2 days following decompression, and in the spinal cord 27 days after decompression.

The inhalation of oxygen, on the other hand, reduces the tension of nitrogen in the inspired air to a value approaching zero, and ensures not only a maximum elimination of nitrogen, but also an immediate relief of asphyxia (oxygen lack). At atmospheric pressure oxygen inhalation raises the pressure head for nitrogen diffusion from 7.3 percent to about 80 percent of 1 atmosphere, or an 11-fold increase as compared with air inhalation.

The questions are whether experimental evidence supports these theoretical considerations, and over what range of pressure oxygen breathing is feasible?

Experimental data.—Experiments, heretofore showing the advantage of oxygen inhalation compared with air, have been too few in number to be conclusive. In order to provide more extensive data with reference to the absorption of nitrogen bubbles, 26 experiments were performed on anesthetized dogs decompressed in 10 seconds from a pressure of 65 pounds after an exposure of 1 hour and 45 minutes. The results in detail and their physiologic implications are reported by Behnke and Shaw (1935). For this paper the essential findings, which apply specifically to the problem under discussion, are summarized.

[2] Includes argon.

The diagram shown in figure 1 represents the procedure in a typical experiment. The 10-second decompression from an excess pressure of 65 pounds led to the development of massive intravascular formation of bubbles in a period of 15 to 60 minutes unless recompression supervened. During period II (fig. 1) bubbles could be detected at the onset of symptoms in cutaneous arteries and veins. Pathognomonic of bubble formation was an increase in respiratory rate, a temporary rise followed by a fall in blood pressure, and a slowing of the pulse rate (fig. 2). Accompanying these symptoms were manifestations of critical arterial anoxemia and a slowing of the circulation. The tongue and mucous membranes and blood withdrawn from the femoral artery were cyanotic. Analysis of the oxygen content of arterial blood showed a reduction from the initial values as high as

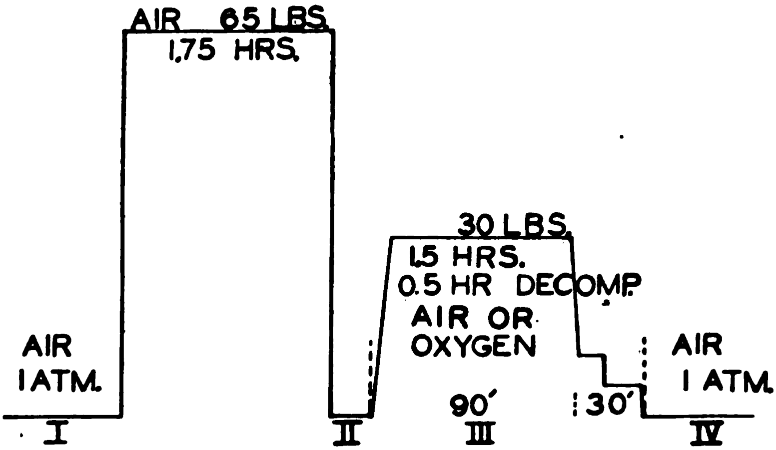

FIGURE 1.—Experimental procedure in the compression and recompression of anesthetized dogs. I, control period; II, asphyxial period following a 10-second decompression from 65 pounds gauge pressure after 1.75 hours exposure; III, recompression to 30 pounds pressure with the dog breathing either oxygen or air; IV, period following recompression, dog breathing air.

66 percent. The reduction of mixed venous blood withdrawn from the right side of the heart was relatively greater, giving a 50 percent or more increase in the arterial-venous oxygen-content difference. The low oxygen content of the venous blood indicated a remarkable slowing of the blood flow through peripheral capillaries. In addition, there was usually a concentration of red blood cells evidently as a result of plasma loss.

The clinical picture was, therefore, acute asphyxia with symptoms indicative of shock. The high degree of oxygen deficit in arterial blood was attributed to the blockage of the pulmonary vascular bed with nitrogen bubbles. These bubbles, presumably, not only interfered with pulmonary ventilation by limiting alveolar expansion but also restricted the circulating blood to comparatively few channels so that the volume of blood flowing through the lungs was diminished in relation to alveolar diffusion surface. Without recompression death occurred rapidly from respiratory, followed by circulatory, failure, usually with an interval between the two of a few seconds to several minutes.

Recompression to a gage pressure of 30 pounds (fig. 1, period III) with the dog breathing either air or oxygen relieved the asphyxia. Respiratory rate returned to normal, blood pressure was improved, pulse rate increased (fig. 2), and the oxygen content of arterial blood was as high or higher than the initial values. It is important to note that at this stage apparent recovery was as rapid with either air or oxygen inhalation. It is during the period following recompression, however, that striking differences are noted.

One hour following recompression with air (fig. 1, period IV) the asphyxial symptoms (increased respiratory rate, anoxemia) reached

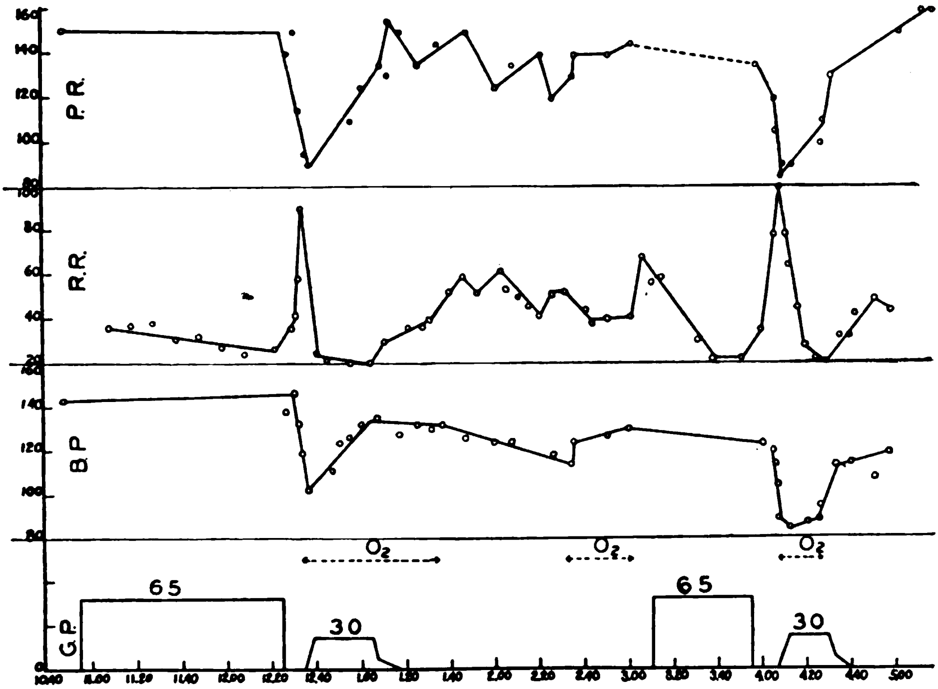

FIGURE 2.—Alterations in blood pressure, respiratory rate, and pulse rate of a dog decompressed in 10 seconds from a gauge pressure of 65 pounds after 1.5 hours exposure followed by recompression (interval of 10 minutes) to a pressure of 30 pounds (oxygen) for 25 minutes. Pressure was then lowered to atmospheric in 12 minutes, and oxygen inhalation continued for 17 minutes.

Preceded by a period of oxygen breathing (30 minutes) compression of the dog was again carried out at a pressure of 65 pounds for a period of 45 minutes followed by a 10-second decompression. After an interval of 12 minutes the dog was recompressed to a pressure of 30 pounds for 20 minutes (oxygen inhalation).

their former degree of severity. The low oxygen values were almost the same as those of period II. Bubbles reappeared in cutaneous vessels, and were always present in large quantities in mixed venous blood. At autopsy this finding was verified by the presence of bubble accumulations in the large veins, right side of the heart, and pulmonary arteries. In the peripheral arterial bed bubbles were usually present in the skin and in the extremities. The greatest amount of gas, however, collected in the venous side of the circulatory system.

In contrast with air recompression the period following oxygen breathing at a pressure of 30 pounds was characterized by a constant or only slight increase in the respiratory rate and by the maintainance of normal values for oxygen saturation of arterial blood. Bubbles when present at autopsy were confined to vessels of the extremities where the blood flow had been at a standstill during the recompression period. While recovery was usually not complete (subnormal blood pressure and circulatory rate) the advantage of oxygen over air was convincingly demonstrated by the permanent relief from asphyxia, and by the complete absorption of nitrogen bubbles from the circulating blood as determined by autopsy examination.

While the experimental evidence shows conclusively the value of oxygen in relieving asphyxia and in promoting bubble absorption it

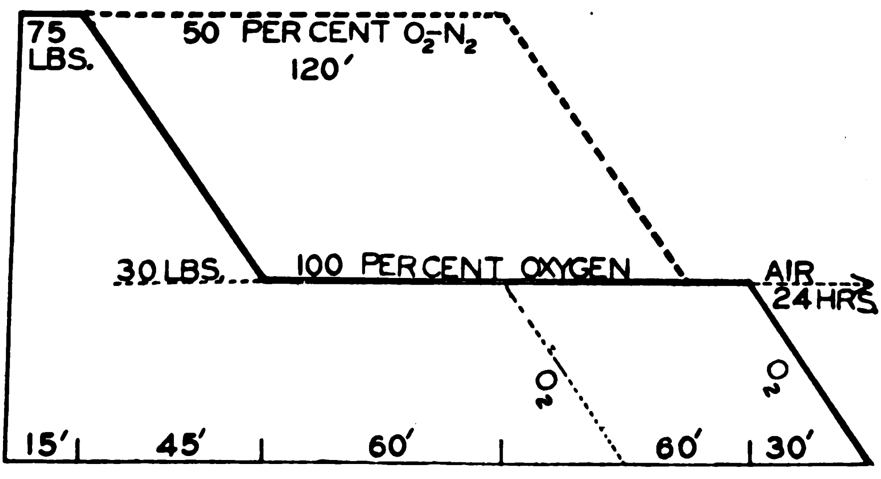

FIGURE 3.—The treatment of a serious case of compressed-air illness with oxygen and an oxygen-nitrogen mixture. Pure oxygen is breathed at or below a pressure of 30 pounds gauge, while the 50 percent oxygen-nitrogen mixture is inhaled between pressures of 30 and 75 pounds. The course of the treatment and the alternatives are clearly outlined.

is essential to determine whether pressures higher than 30 pounds are necessary in recompression. Were asphyxia the only factor to consider, an excess pressure of 30 pounds combined with oxygen inhalation would be sufficient for all cases of compressed-air illness. Whether paralysis could be prevented by a pressure as low as 30 pounds, could not be determined in experiments on the anesthetized dog.

In order to determine the adequacy of a pressure of 30 pounds in preventing paralysis a second series of experiments was performed on intact dogs subjected to the same experimental conditions as were the anesthetized dogs. The results of these experiments are summarized in table 1. Dog 1, for example, was decompressed from 65 pounds excess pressure in 10 seconds after an exposure of 1 hour and 45 minutes. Three minutes following decompression the dog became excited and showed signs of pain (one of two experiments in which pain was a definite symptom). Four minutes later the dog was recompressed in an oxygen atmosphere to a pressure of 30 pounds for a period of 90 minutes. When the pressure was again lowered to 1 atmosphere (in 1 minute) paralysis of the hind legs was manifest. At autopsy bubbles were not visible in the blood stream. From this experiment it appeared that while bubbles were absorbed, recompression to 30 pounds pressure was insufficient to prevent the development of paralysis. This experiment showed the necessity of reestablishing the blood supply to the spinal cord in the shortest possible time. As a result of several similar experiments (dogs 2, 3, table 1), it was concluded that while the asphyxia was relieved and while the bubbles were absorbed, paralysis could develop during the breathing of oxygen at a pressure of 30 pounds.

In subsequent experiments the treatment was altered by raising the pressure to 65 pounds and then lowering it 5 pounds every 10 minutes until the level of 30 pounds was reached. Air was breathed during this period because a 50-percent oxygen-nitrogen mixture was not available. When the pressure was lowered to 30 pounds the chamber was filled with pure oxygen and the pressure maintained for 1 hour. A 1-minute decompression to atmospheric pressure completed the treatment. With this method of therapy paralysis either did not develop (during the recompression period), or the progress of the paralytic symptoms was stopped (dog 6, table 1). An exception to this statement may be the fact that in dog 5 paralysis of the hind legs developed after 24 hours following an apparently slight injury to the spinal cord.

TABLE 1.—*Treatment of compressed-air illness*

Dog	Date	Exposure	Following decompression (10-second decompression)	Recompression	Results
1	Apr. 23	1.75 hours, 65 pounds.	3 minutes, excitement, pain, outstanding symptom, no paralysis or asphyxia, 7 minutes, recompression.	30 pounds O_2 for 90 minutes.	Paralysis of hind legs. No bubbles in the blood stream.
2	Apr. 24	1.5 hours, 65 pounds.	No symptoms..........	....	
2	Apr. 29	1.75 hours, 65 pounds.	5 minutes, very active; 8 minutes, heart pounding; 9 minutes, slow heart rate (68); 10 minutes, languid, limping, left foreleg raised from floor; 14 minutes recompression.	30 pounds for 90 minutes (O_2). After 10 minutes, 30 pounds for 90 minutes (O_2).	Foot-drop. Dog able to stand but drags hind feet in walking. Unimproved by second recompression. Next day, paralysis and anesthesia of hind legs.
3	May 10	......do..........	6 minutes, rigidity, left hind leg. Recompression.	30 pounds for 60 minutes (O_2). After 20 minutes 30 pounds for 60 minutes (O_2).	Limping; left hind leg rigid. Complete recovery after second recompression.

TABLE 1.—*Treatment of compressed-air illness*—Continued

Dog	Date	Exposure	Following decompression (10-second decompression)	Recompression	Results
3	May 14	1.75 hours, 65 pounds.	9 minutes, limping, left foreleg off floor; 10 minutes recompression.	30 pounds, 60 minutes. After 2 minutes (O_2), 30 pounds, 60 minutes (O_2). After 9 minutes, 30 pounds, 60 minutes (O_2).	Right hind leg flexed. Spastic gait. Weakness hind legs. Spasticity and weakness increased. Autopsy, no bubbles.
4	May 22	......do.........	4 minutes, collapsed....	65 pounds air.......	Died in 26 minutes under 50 pounds pressure. Autopsy, bubbles in all blood vessels.
5	May 24	......do.........	5 minutes, left hind leg raised off floor. Recompression.	65 pounds air [1] 70 minutes; 30 pounds O_2, 1 hour.	Recovery.
5	May 28	......do.........	5 minutes, left foreleg flexed. Recompression.	......do.[1]............	Do.
5	June 11	......do.........	8 minutes, stretched out, unable to move. Recompression.	......do.[1]............	Do.
5	June 18	......do.........	12 minutes, no control over hind legs; 13.5 minutes, dyspnea. Recompression.	......do.[1]............	Weakness left hind leg. Ataxia left hind leg. Next day, dog able to walk but developed paralysis (hind legs) on the second day.
6	June 25	......do.........	24 minutes; limping Recompressed.	O_2 30 pounds, 1 hour.	Recovery.
6	June 26	......do.........	15 minutes, drags right hind foot; 17 minutes lying on side, hind legs useless.	65 pounds air [1], 70 minutes; 30 pounds O_2, 1 hour.	Right hind leg ataxic; 2 days later no improvement. 14 days later, recovery.
7	...do....	1.75 hours, 65 pounds.	20 minutes, all extremities drawn up, dog in pain. Recompression.	65 pounds air [1], 30 pounds O_2 1 hour, 30 pounds O_2, 1 hour.	In good condition for 10 minutes, then vomiting, diarrhea. Active, in good condition.
7	June 28	1.77 hours, 65 pounds.	17 minutes, rigidity hind legs; 18 minutes, hind legs paralyzed, extended, priapism, dyspnea. Recompression.	65 pounds air [1], 30 pounds O_2, 1 hour.	Spastic paralysis of the hind legs. Reflex arc intact. Autopsy, distended bladder. spinal cord, lungs, normal. No bubbles. Petechial hemorrhages in fat.
8	June 29	......do.........	27 minutes, cyanosis of mucous membranes and tongue. Left leg raised off floor.	65 pounds air [2] to O gage in 4 hours, 43 minutes.	Weakness, hind legs. Autopsy, a few bubbles in cutaneous vessels.

[1] Air pressure raised to 65 pounds, then lowered to 30 pounds at the rate of 2 minutes per pound; dog then breathed oxygen at 30 pounds pressure for 1 hour.
[2] Recompression essentially as outlined by the Diving Manual.

From these experiments it can be concluded that whenever bubble formation is massive (i. e., after a 10-second decompression from 65 pounds, 1.75 hours exposure) application of pressure to 65 pounds is necessary to prevent or to arrest the progress of incipient paralysis. While it is extremely doubtful if fully developed paralysis at the time of recompression will improve with the application of pressure, it is imperative in every case to compress the bubbles to a small size, and to secure their rapid absorption or removal from blood vessels in the spinal cord. The application of these findings clinically now depends upon the tolerance of man for oxygen at high pressures.

Tolerance of man for high oxygen pressure.—At atmospheric pressure healthy men can breathe oxygen for a period of 6 hours without symptoms indicative of pulmonary irritation. At higher pressures the effects of oxygen on the nervous system supersede those with reference to the lungs. At a pressure of 3 atmospheres, for example, pure oxygen can be breathed for a period of 4 hours without producing pulmonary injury (Behnke, Forbes, and Motley, 1935). It would appear that insofar as lung damage was concerned there was no direct relationship between the oxygen tension and the duration of exposure. The nervous symptoms, however, bear a direct relationship to the oxygen tension and the duration of exposure. At atmospheric pressure nervous manifestations of a minor character may accompany the breathing of oxygen (Behnke, Johnson, Poppen, and Motley, 1935). At a pressure of 3 atmospheres definite and sometimes apparently alarming symptoms occur during the fourth hour in every experiment. Preceded by a period of normality and with fairly abrupt onset, a rise in blood pressure, increase in pulse rate, and contraction of the visual fields with diminution in visual acuity point to the action of oxygen on the nervous system. Rapid and complete recovery invariably follows when air is again breathed. At a pressure of 4 atmospheres the limit of oxygen breathing is about 45 minutes. At this pressure convulsions or fainting may occur.

The mechanism underlying the action of oxygen on the nervous system is not known, but the significant fact is the reversibility of the nervous phenomena since complete recovery invariably follows the removal of oxygen. In experiments on 12 healthy men subjected (4 or 5 times) to oxygen tensions up to 4 atmospheres residual injury was not detected. While an oxygen tension of 4 atmospheres is to be regarded as a potentially convulsive level and hence to be avoided, a level of 3 atmospheres definitely represents a subconvulsive tension. The fact that pure oxygen at this pressure can be breathed for 3 hours permits oxygen therapy to form an essential part of the treatment for compressed-air illness. It should be remembered that a patient may tolerate oxygen for periods in excess of 3 or 4 hours in view of the probable anoxemia and slowed circulation, symptoms which in dogs were associated with large quantities of bubbles in the blood stream.

Recompression based on oxygen therapy.—The fundamental principle underlying the treatment of compressed-air illness consists in the application of the fact that pure oxygen at a pressure of 30 pounds (3 atmospheres absolute) can be breathed for a period of 3 hours. If pressures higher than 3 atmospheres are used, the partial pressure of oxygen can be maintained at 3 atmospheres by adding air or nitrogen. For convenience, a 50 percent oxygen-nitrogen mixture could be made available for respiration between 3 and 6 atmospheres absolute. For pressures of 3 atmospheres or less pure oxygen would be breathed.

It is extremely difficult in view of the undetermined quantity of nitrogen in bubble form and of its undetermined distribution in the vascular beds of different organs to draw up a rigid outline of treatment for all cases of compressed-air illness. The condition of the patient, of course, is the criterion for guidance in treatment. The dog experiments (massive bubble formation in the blood stream), however, demonstrate the necessity of using comparatively high pressures (65 pounds) in order to prevent paralysis. These experiments in addition give an approximation of the time necessary for the absorption of bubbles.

For the serious cases of compressed-air illness in which the previous degree of pressure and duration of exposure, and in which the symptoms (asphyxia, paralysis, loss of consciousness) indicate extensive formation of bubbles, the reapplication of the pressure to 75 pounds with the patient breathing a 50 percent oxygen-nitrogen mixture ensures the immediate relief of asphyxia and the arrest of incipient nerve lesions. The pressure is then maintained at 75 pounds for a minimum period of 15 minutes with the understanding that the time can be extended to 2 hours for the moribund or paralyzed patient.

It may be well to consider what this initial stage in treatment will accomplish. The bubbles of nitrogen according to Boyle's law will be reduced to one-sixth of their volume at atmospheric pressure; as a result of compression the surface area of the bubbles in proportion to their volume is almost doubled, hence diffusion of nitrogen into the surrounding blood will be increased; the capacity of the blood and the tissues to absorb nitrogen will be increased six-fold; and if a 50 percent oxygen-nitrogen mixture is breathed, nitrogen will be eliminated from the body at a pressure head of 3 atmospheres. The quantity of nitrogen eliminated at 75 pounds pressure (with the respiration of a 50 percent oxygen-nitrogen mixture) can be approximately calculated as follows: With a pressure head of 1 atmosphere 35 to 50 cubic centimeters of nitrogen (NTP) are eliminated by a man at rest, weighing 60 kilograms. With a pressure head of 3 atmospheres and a normal circulatory rate a minimum of 105 cubic centimeters (NTP) of nitrogen per minute would be eliminated from the body as long as bubbles maintained nitrogen saturation in the blood stream (large veins and right side of the heart). Under these conditions it would be reasonable to expect the elimination of all or nearly all of the nitrogen in bubble form.

The second and final stage in treatment (patient conscious, respiratory rate normal) following 15 minutes' exposure to 75 pounds pressure, or at least 2 hours' exposure if paralysis is present, consists in the reduction in pressure to 30 pounds at the rate of 1 pound per minute. At this pressure pure oxygen is substituted for the oxygen-nitrogen mixture. The breathing of pure oxygen for a period of 1 to 2 hours serves to eliminate completely any residual bubbles following the first stage in treatment. This statement is based on the observation that in dogs dying from massive nitrogen bubble formation recompression to a pressure of 65 pounds and decompression in 70 minutes (in air) to 30 pounds pressure followed by 1 hour of oxygen inhalation resulted in the elimination of bubbles visible to the unaided eye. Since the circulatory rate in man is one-half that of the dog, the time for oxygen breathing is doubled (i. e., 2 hours at 30 pounds for the treatment of a previously moribund patient). If the patient is in good condition after 1 hour of oxygen breathing the pressure is lowered to that of the atmosphere over a period of 30 minutes. If, on the other hand, the condition of the patient indicates that bubbles are still present after the completion of oxygen breathing at a pressure of 30 pounds or its equivalent partial pressure for 3 hours, or if the return to atmospheric pressure is attended by increased respiratory rate, difficulty in breathing, and pain, air is substituted for oxygen and a pressure of 30 pounds maintained for 24 hours. At the end of 24 hours the inhalation of oxygen for 2 hours followed by a 30-minute decompression to atmospheric pressure completes the treatment. In figure 3 the therapy is represented in graphic form.

In order to summarize the main points in the treatment of a serious case of compressed-air illness it can be stated that recompression is simplified to two stages and utilizes oxygen breathing. In the first stage the patient breathing a 50-percent oxygen-nitrogen mixture is recompressed to 75 pounds pressure for a minimum period of 15 minutes. Symptomatic recovery and the absorption of all or nearly all of the nitrogen bubbles are the objectives in this procedure. Treatment at a pressure of 75 pounds can be prolonged for 2 hours, if necessary. In the second stage after the pressure has been reduced to 30 pounds, pure oxygen is breathed for a period of 1 to 2 hours. Treatment is completed by decompression in 30 minutes to atmospheric pressure. Unrelieved or partially relieved symptoms require treatment by prolonging the compression at 30 pounds (patient breathing air). At the end of 24 hours oxygen is breathed for 2 hours. Toxic symptoms from oxygen should not occur (contraction of the visual fields, rise in blood pressure and pulse rate) with 3 hours of oxygen breathing in any 24-hour period.

Since 90 percent of the cases of compressed-air illness are mild and exhibit symptoms designated as "bends" (pain in the extremities), recompression to a pressure of 30 pounds with the inhalation of oxygen for 1 hour, followed by a 30-minute decompression, should be sufficient.

Concluding notes.—During the first stage in the recompression treatment the body should be maintained in a horizontal position with the head and shoulders inclined slightly downward. This procedure will protect the blood supply to the brain since bubbles tend to accumulate in the most elevated parts of the body (Van Allen, Hrdina, and Clark, 1929).

The follow-up treatment of the severe cases is designed to protect the heart and to prevent pneumonia. In patients afflicted with spinal cord injury retention of urine and the formation of decubitus ulcers are to be prevented. The concentration of red blood cells (plasma loss) associated with massive bubble formation (dogs) suggests the value of isotonic glucose-saline solution preferably administered subcutaneously, or if intravenously, very slowly (500 cubic centimeters in 30 minutes) and only after the completion of compression treatment.

The facilities for oxygen administration consists of cylinders and a spirometer to which is attached either a helmet or a mask. By adding a soda lime cannister, cooling coil, and blower oxygen can be rebreathed with a minimum loss of gas.

SUMMARY

On the basis of theoretical considerations and experimental evidence oxygen inhalation combined with recompression comprises the essential treatment for compressed-air illness.

Since pure oxygen can be breathed without discomfort by healthy men for 3 hours at a gage pressure of 30 pounds (3 atmospheres absolute), it follows that an equivalent tension (50-percent oxygen-nitrogen mixture) can be breathed at a pressure of 75 pounds (6 atmospheres absolute) for a corresponding period of time. These values for oxygen tolerance make possible the use of oxygen over the whole recompression period.

In the treatment of the severe cases of compressed-air illness (prostration, asphyxia, and paralysis) recompression consists of two stages (fig. 3). During the first stage the pressure is raised to 75 pounds and maintained for a period of 15 minutes to 2 hours. At this pressure a 50-percent oxygen-nitrogen mixture is breathed. The second stage consists in lowering the pressure at the rate of a pound per minute from 75 to 30 pounds gage, and substituting oxygen for the oxygen-nitrogen mixture. Oxygen is breathed at a pressure of 30 pounds for a period of 1 to 2 hours, and treatment is completed by a 30-minute decompression to atmospheric pressure. Unrelieved symptoms require a prolonged stay at 30 pounds pressure for 24 hours and a second period of oxygen inhalation.

In the treatment of mild cases of compressed-air illness ("bends") oxygen inhalation at a pressure of 30 pounds for 1 hour followed by a 30-minute decompression should effect permanent relief.

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PATHOLOGICAL FRACTURES

By Foster H. Bowman, Lieutenant Commander, Medical Corps, United States Navy, retired

Allen states that spontaneous fractures are not rare, but that published information on their diagnosis and treatment is extremely brief and scanty. The primary cause, or pathological condition, is of great importance, while the immediate cause is usually of little importance.

These fractures are indeed quite common and it is surprising that more has not been written about them.

A good definition of a spontaneous fracture might be "A fracture occurring from apparently insufficient trauma." The bone gives way under apparently trifling stress, because of defective development, constitutional disease, local or general bone disease.

The fractures from incoordinated but powerful muscle contraction, as occur in tabes or syringomyelia, hardly belong to this group. Also in old age it is quite usual to have absorption of bone tissue in both