Cardiorespiratory Responses of Anesthetized Dogs to Compression Therapy Following Experimental Decompression Sickness

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ABSTRACT

Decompression sickness was studied by use of a special technic for the production and recognition of the disorder in a laboratory animal. Objective evidence is presented for substantiating the use of compression therapy in the treatment of both caisson and altitude types of decompression sickness.

There is suggestive evidence that even very small overpressures may be of benefit in treating or transporting some cases of severe altitude decompression sickness.

The importance of rapid recognition and treatment is emphasized, as well as careful scrutiny of all patients who appear to be "hyperventilating" after decompression, especially those patients with other signs or symptoms of decompression sickness.

TREATMENT OF REFRACTORY altitude decompression sickness by increasing the ambient pressure has been a matter receiving considerable speculation in the past few years. Experience in utilizing compression therapy for altitude decompression sickness has been limited, however.

Three cases have been reported in which compression was utilized as the primary form of therapy in patients who continued to show symptoms of decompression sickness after recompression to ground level had been accomplished.

Donnell and Norton[8] reported the case of a 39-year-

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old pilot who had been in an altitude chamber at reduced pressure for a total time of 50 minutes, with time above 20,000 feet probably near 35 minutes. The patient noted a tingling sensation on the medial aspect of the left arm and hand, together with difficulty in moving the left hand. He also had a dryness in the throat and substernal area and an itching and pricking sensation in the interscapular area. At ground level during the physical examination, cyanosis of the nail beds was noted, but the blood pressure and pulse rate were normal. Eighty minutes after the first appearance of symptoms at altitude, the patient was unable to follow a conversation.

The disorder progressed so that by the time he reached a recompression chamber, 5 hours after the initial onset of symptoms, his blood pressure was less than 90 mm. Hg and he had paralysis of the left upper extremity, blindness, and severe disorientation.

Compression to 6 atmospheres absolute pressure was followed in 15 minutes by circulatory improvement. The patient was gradually decompressed in 38 hours and showed gradual, but sustained, improvement so that by the time he left the chamber, he had only "mild difficulty with his ability to deal with symbolic abstractions."

Coburn[7] reported the case of a 47-year-old pilot who was exposed to a simulated altitude of 37,000 feet for 1 hour. On descent to ground level, the pilot showed loss of motor function in the left lower leg; and 75 minutes postrun, he began showing evidence of circulatory problems. On arrival at the recompression chamber 2 hours postrun, the patient appeared moribund, with blood pressure 50/0 and pulse rate 100 to 120 beats per minute. He was compressed to 3 atmospheres absolute pressure in 1 minute, at which time his blood pressure was 100/60 and pulse rate 100. Within 3 minutes, the blood pressure and pulse rate were normal, and he had regained almost 100 per cent of the lost motor function. Decompression was accomplished from 3 atmospheres in 59 minutes, and recovery was uneventful.

A third case of altitude decompression sickness treated by compression was reported from Edwards Air Force Base[5] in which a photographer developed decompression sickness during an actual flight at 40,000 feet cabin altitude. Compression therapy began less than 6 hours after the onset of the disease. Shoulder pain, numbness, and chokes were the main symptoms these were fully relieved at 3 atmospheres absolute pressure, with partial relief obtained at 1.75 atmospheres absolute pressure. Compression treatment was initiated using the U. S. Navy Treatment Table III[16] from 6 atmospheres absolute, but the patient suffered a recurrence of symptoms at 3 atmospheres absolute pressure. Recompression to 5 atmospheres gave relief, so the patient was decompressed from 5.4 atmospheres using U. S. Navy Treatment Table IV[16] with an uneventful recovery.

A fourth patient was treated early this year in Sweden; the case was reported at the 1963 Aerospace Medical Association Convention, but details have not as yet been published. It was a case of neurocirculatory collapse which did not respond to a very short exposure to compression therapy; there was a one-day delay in initiating the therapy, however.

The present study was accomplished in an effort to objectively substantiate the rationale for using compression therapy in severe decompression sickness, both caisson and altitude types.

METHODS

Large mongrel dogs were anesthetized and instrumented for recording physiologic data. Proximal systemic aortic pressure (SAP), central venous pressure (CVP), pulmonary artery pressure (PAP), ECG, respiratory frequency (RF), and flowing femoral venous and arterial blood were observed throughout the procedures. Aortic pressure and pulmonary artery pressure were taken in via polyethylene catheters through Statham P 23 Db transducers to a Sanborn recorder. The pulmonary artery catheter was equipped with an inflatable balloon at the tip to facilitate placement.[11] Central venous pressure was recorded via a polyethylene catheter through a Statham P23BB transducer to the recorder. Flowing femoral arterial and venous bloods were observed by means of plastic viewing cuvettes inserted into the femoral vessels.[12] In some[17] instances, catheter tip manometers described by Wetterer and Pieper[17] were used for SAP, PAP, and CVP, and recordings were made on an Electronics for Medicine oscillograph recorder.

Unobstructed respiration was assured by utilizing large endotracheal catheters (size 42-44F). Animals using pressure breathing had tracheostomy tubes in place to prevent leakage. In the altitude experiments, the dogs taken to 40,000 feet equivalent (140.7 mm. Hg) breathed 100 per cent oxygen through a diluter-demand regulator (A14) while those taken to 43,000 feet equivalent (122 mm. Hg) breathed 100 per cent oxygen through a pressure-demand regulator (D-2) utilizing the valves from an M-2200 mask (22 to 25 mm. Hg positive pressure at 43,000 feet). The pressure chamber which was used had an absolute pressure range of 8.0 mm. Hg to 90 p.s.i. (4,560 mm. Hg).

Severe, acute decompression sickness was produced in all 21 dogs; in 10 dogs the sickness was the caisson-type, and in 11 dogs the sickness was the altitude-type. Each dog served as his own control in evaluating physiologic changes. Ground level control values were used for the dogs taken to altitude. In the animals given caisson disease, we used control values taken after decompression but before the development of decompression sickness.

Criteria for the diagnosis of severe decompression sickness were the appearance of gross bubbles in the venous circulation, pulmonary hypertension, and tachypnea.[12]

Procedure:—Unpremedicated dogs weighing over 40 pounds were anesthetized with I. V. pentobarbital sodium (30 mgm./kg.) and given I. V. heparin (5 mgm./kg.). The pulmonary artery catheter was placed through the right external jugular vein. The systemic artery catheter and central venous pressure catheter were inserted through the femoral vessels of the same

hind leg. The femoral vessels of the other leg were used to observe the blood flow. Respiratory frequency was monitored by means of a pneumograph. Lead II of the ECG was used in calculating pulse rate and observing the heart rhythm.

After instrumentation and heparinization were accomplished, the animal was placed in the pressure chamber and the atmospheric pressure altered in one of two ways.

Caisson Disease:—The chamber pressure was increased to 6 atmospheres absolute pressure (75 p.s.i. gage) and maintained at this level for 60 minutes. Decompression to 1 atmosphere absolute (ground level) produced caisson disease in the animals after 8 to 30 minutes. Four animals were then decompressed to 522 mm. Hg absolute pressure (10,000 feet equivalent) to show a further increase in the signs of decompression sickness. Recompression to 3 atmospheres absolute (30 p.s.i. gage) pressure was then accomplished, followed by return to 1 atmosphere absolute (ground level). Two animals were then recompressed again to 3 atmospheres and returned to 1 atmosphere to show that the signs were reproducible.

All hyperatmospheric chamber pressure changes were accomplished at the rate of 7 p.s.i./minute (350 mm. Hg/minute); decompressions to less than 1 atmosphere were at a rate of 5,000 feet/minute.

Altitude Decompression Sickness:—The animals were placed in the pressure chamber and the atmospheric pressure was then reduced to 140.7 mm. Hg (40,000 feet equivalent). Four animals were taken to 122 mm. Hg pressure (43,000 feet equivalent) using positive pressure breathing. After the appearance of the signs of decompression sickness, the atmospheric pressure was increassed to 750 mm. Hg (ground level). One animal which had persistent mild pulmonary hypertension and tachypnea at this point was overcompressed to 3 atmospheres absolute pressure.

Chamber pressure changes were accomplished at the rate of 5,000 feet equivalent/minute.

RESULTS

Caisson Disease:—The appearance of gross bubbles in the femoral venous viewing chamber was associated with a rise in both pulmonary artery pressure and respiratory rate in all animals. The pulmonary artery diastolic pressure rose from a mean of 6.11 ($\pm$ 1.03) * mm. Hg to a mean of 30.44 ($\pm$ 2.32) mm. Hg, and respiratory frequency rose from a mean of 14.3 ($\pm$ 4.36) to 49.8 ($\pm$ 10.99) breaths per minute. Recompression to 3 atmospheres absolute pressure brought the diastolic PAP mean from 30.44 ($\pm$ 2.32) to 4.89 ($\pm$ .70) mm. Hg, and the respiratory frequency from 49.8 ($\pm$ 10.99) to 21.3 ($\pm$ 5.97). Diastolic pressure readings were used, since diastolic pressure reflects the circulatory runoff through the pulmonary vascular bed. Dog C8 inadvertently expired early, and dog C9 had no PAP recorded.

When animals were returned to ground level, the pulmonary hypertension was again evident; however, the increase in respiratory frequency was not significant. Tables I and II indicate the values for the individual animals.

The decreases in diastolic PAP and respiratory frequency observed during treatment with increasing

TABLE I. CAISSON DISEASE—PULMONARY ARTERY DIASTOLIC PRESSURE

Subject	Control	Decompression sickness	Recompression	Ground
C_1	4	33	3	17
C_2	3	30	4	18
C_3	12	30	7	17
C_4	6	23	1	42
C_5	5	30	6	26
C_6	10	35	8	25
C_7	3	36	5	42
C_{10}	7	17	5	8
C_{11}	5	40	5	37
$\bar{X}$	6.11	30.44	4.89	25.77
$S_{\bar{X}}$	1.033	2.315	.696	4.054
Significance of differences between means		.05	.05	.05
		n.s.		
			n.s.	

TABLE II. CAISSON DISEASE—RESPIRATORY FREQUENCY

Subject	Control	Decompression sickness	Recompression	Ground
C_1	46	136	70	58
C_2	5	29	12	17
C_3	5	19	8	12
C_4	9	38	9	37
C_5	12	38	11	14
C_6	14	18	12	10
C_7	13	56	34	46
C_9	6	72	16	43
C_{10}	26	36	16	16
C_{11}	17	56	25	35
$\bar{X}$	14.3	49.8	21.3	28.8
$S_{\bar{X}}$	4.362	10.985	5.972	5.389
Significance of differences between means		.05	.05	n.s.
		n.s.		
			.05	

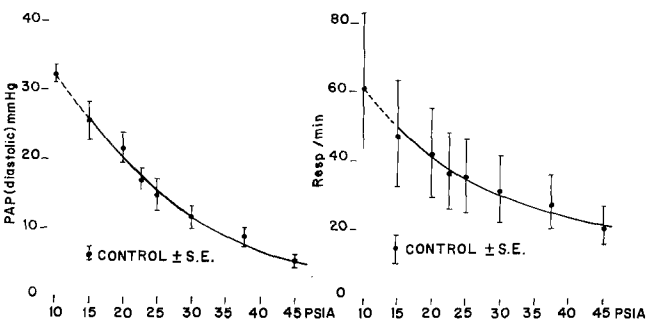

Fig. 1. Recompression effects on pulmonary artery diastolic pressure and respiratory frequency for caisson disease.

*Values in parentheses represent standard deviation of the mean.

atmospheric pressure are described for all animals in Figure 1. The 10 p.s.i.a. values are for 4 animals only.

During recompression, the bubbles in the venous viewing cuvette diminished markedly in size but never completely disappeared. No new bubbles formed after recompression.

Altitude Decompression Sickness:—As in the caisson study, the appearance of bubbles in the venous circulation was followed shortly by pulmonary hypertension and tachypnea. The pulmonary artery diastolic pressure rose from a mean of 8.82 (± 1.11) mm. Hg to 24.09 (± 1.23) mm. Hg, and the respiratory frequency increased from 10.64 (± 2.99) to 43.91 (± 8.74) breaths per minute (Tables III and IV). Upon recompression to 750 mm. Hg pressure, the animals showed a decline in pulmonary artery pressure from 24.09 (± 1.23) mm. Hg to 8.54 (± 1.52) mm. Hg and a decline in respiratory frequency from 43.91 (± 8.74) to 21.27 (±5.02). The decreases in diastolic PAP and respiratory frequency observed during recompression are shown for all animals in Figure 2. The 123 mm. Hg values are for 4 animals only.

TABLE III. ALTITUDE DECOMPRESSION SICKNESS—PULMONARY ARTERY DIASTOLIC PRESSURE

Subject	Control	Decompression sickness	Recompression
A_1	15	27	12
A_2	11	20	8
A_3	14	60	20
A_4	8	28	10
A_5	8	18	7
A_6	10	20	6
A_7	2	9	3
A_8	5	19	8
A_9	8	23	13
A_{10}	8	20	3
A_{11}	8	21	4
$\bar{X}$	8.82	24.09	8.54
$S_{\bar{X}}$	1.110	1.230	1.523
Significance of differences between means		.05 .05 n.s.	

TABLE IV. ALTITUDE DECOMPRESSION SICKNESS—RESPIRATORY FREQUENCY

Subject	Control	Decompression sickness	Recompression
A_1	8	60	42
A_2	25	78	40
A_3	34	92	54
A_4	9	80	22
A_5	6	30	10
A_6	6	10	4
A_7	4	19	12
A_8	3	12	6
A_9	7	40	20
A_{10}	12	27	12
A_{11}	3	35	12
$\bar{X}$	10.636	43.909	21.273
$S_{\bar{X}}$	2.986	8.738	5.018
Significance of differences between means		.05 .05 n.s.	

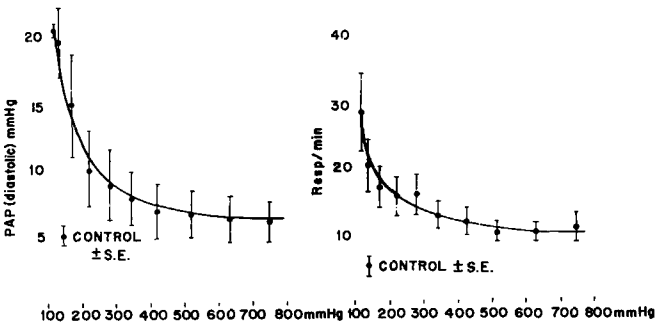

Fig. 2. Recompression effects on pulmonary artery diastolic pressure and respiratory frequency for altitude decompression sickness.

As in the caisson disease experiments, recompression did not cause the bubbles to completely disappear, although the bubbles decreased markedly in size. Apparently, no new bubbles were being formed at ground level, as observed through the viewing cuvette.

One animal (Fig. 3) which had persistent pulmonary hypertension and tachypnea after recompressing to 1 atmosphere absolute, was treated with overcompression therapy to observe the effects. Overcompression to 3 atmospheres absolute was accomplished, at which point the pulmonary artery diastolic pressure and respiratory frequency were normal.

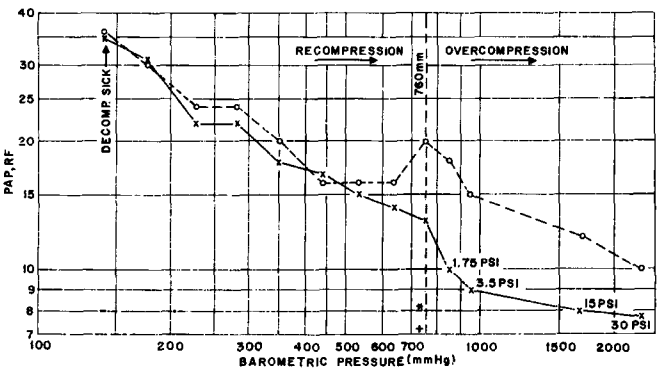

Fig. 3. Effect of increasing pressure on pulmonary artery diastolic pressure (X) and respiratory frequency (O) for altitude decompression sickness. (Experiment A-9.)
*PAP control, 8 mm. Hg
+RF control, 7/min.

In neither group of experiments did bubbles appear in the arterial system until the animal was near circulatory collapse.

Statistical management:—Tests of significance of differences between means of pulmonary artery diastolic pressures and between means of respiratory frequencies were accomplished using Student's t-test at a level of significance of .05. The variances used in the t-tests were calculated by analysis of variance technics; but since normality in these experiments very likely does not hold, the sign test, a nonparametric procedure, was thought to be more applicable. The two procedures gave the same results for these data.

The responses of the pulmonary artery pressures and respiratory frequencies to recompression therapy assume a linear appearance when plotted on log-log graph paper. The slope of the caisson recompression curve probably should not be compared to the slope of the altitude recompression curve, since the rates of recompression for the two groups were not comparable.

Figures 1 and 2 show plots of actual values. Very similar curves are obtained from plotting per cent increase above the control values against increasing barometric pressures.

Figure 3 shows plotted results of a single experiment. The beginning of overcompression indicates changes in the pulmonary artery pressure which would appear to be significant, since the standard deviation for control values taken on individuals over a period of time was 0.4429; but values after treatment were sometimes considerably lower than the original control values; thus, such a test may not be valid.

DISCUSSION

The objective signs of decompression sickness in anesthetized dogs were reversed in all animals by increasing the barometric pressure. Since the amount of increased barometric pressure necessary to reverse the signs varied for the individual animals, humans could be expected to vary in response to different levels of compression therapy. Small increases in ambient pressure are sometimes very effective in reducing the most severe pulmonary hypertension.

It is thought that the pulmonary hypertension and tachypnea are produced by mechanical blockage of the pulmonary vascular tree by the bubbles, since the bubbles appear in the venous circulation before the pulmonary hypertension and tachypnea are evident. During recompression therapy, the pulmonary artery pressure appears to follow more closely the predicted values for each barometric pressure than does the respiratory frequency. The pulmonary artery pressure is not influenced by the level of anesthesia nor the mild (10,000 feet equivalent) hypoxia produced in the animals at simulated high altitude, since it has been shown that inspired oxygen concentrations less than 8.5 per cent at ground level are necessary to produce pulmonary hypertension.[2] Marked changes in respiratory frequency can be utilized as a sign of decompression sickness; however, this sign is not as reliable as the presence of intravascular bubbles or pulmonary hypertension.

Pulmonary hypertension and tachypnea are signs of pulmonary emboli in general[4, 13, 14, 15] and are not specific for decompression sickness. Injection of small bubbles into the venous circulation also causes pulmonary hypertension and tachypnea.[10, 12] Two animals treated for caisson decompression sickness by recompression were then re-decompressed, and then re-recompressed. Each time the animals were decompressed, the signs of decompression sickness appeared without delay; and each time they were recompressed, the pulmonary artery pressure and respiratory frequency returned to normal, with a marked decease in size of the bubbles. The presence of intravascular bubbles and the reversibility of the signs with increased barometric pressure would indicate strongly that the bubbles are causing the signs in these experiments.

The term *decompression sickness* is generally used to mean any form of the disease caused by expanding gases. The syndrome which we have reported in these experiments probably more closely resembles "chokes" than any other form of decompression sickness. Of course, "chokes" is a severe form of the disease which frequently progresses to neurocirculatory collapse. The tachypnea which we report has been observed in humans in association with "chokes;"[1, 3] this rapid, shallow breathing is frequently described as being due to the fact that respiration is painful. The rapid, shallow breathing observed in anesthetized animals with decompression sickness would give the impression that pain may not be the only factor involved.

Intravascular bubbles, pulmonary hypertension, and tachypnea occurred in every one of the animals in both groups; however, there was a marked difference in the severity of the illness. The mere occurrence of bends pain in humans indicates the presence of bubbles in the body, and the probable presence of intravascular bubbles also. In most cases, the intravascular bubbles are apparently eliminated by external respiration. When the bubble buildup is high in the pulmonary bed, pulmonary hypertension and tachynea can result to a varying degree. This may occur even in the absence of typical "chokes" symptoms. In this event, the appearance of the rapid, shallow respiration may be the only obvious sign of early, severe decompression sickness. Indeed, Behnke states, "rapid, shallow respiration (tachypnea), [is] a sign pathognomonic of bubbles in pulmonary vessels following decompression."[3] This possibility should be considered in each case before a diagnosis of "hyperventilation" is made, especially if there are other symptoms of decompression sickness.

The fact that bubbles did not appear in the arterial system until near circulatory collapse can be explained on the basis that the intra-arterial pressure is sufficiently high to keep the bubbles invisible (if they are present); thus, the arterial system with its high pressure is a naturally occurring protection against arterial bubble emboli.

In experiment A9 (Fig. 3), the trend in reducing the PAP and RF is noted from the slopes of the lines. Even an overpressurization of 3.5 p.s.i.g. reduced the residual pulmonary hypertension, and both PAP and RF were normal at 30 p.s.i.g. overpressure. Although all of these latter changes are not statistically significant, certainly the trend would indicate that some individuals with altitude decompression sickness may benefit from small overpressures of 5 to 10 p.s.i.

Since pressures up to 14.7 p.s.i.g. could be utilized without the necessity for backup systems (Haldane's 2 : 1 rule), consideration should be given to this possibility; but inadequate treatment can sometimes be worse than no treatment at all, so the adequacy of this type of treatment must be carefully evaluated before it is utilized. Until this is accomplished, use of the same treatment tables which the U. S. Navy has found so effective in the management of diver's bends is recommended. In utilizing this animal preparation for the evaluation of therapy, caution must be exercised. In our caisson disease experiments, the pulmonary hypertension and tachypnea were reversed by only 3 atmospheres absolute pressure, while it is well known that 6 atmospheres absolute pressure are necessary for the best results.[16] More definitive experiments in this category are presently being accomplished; however, findings in humans must be evaluated at the recompression facilities throughout the world. Since the severe form of the disease is so rare, it is imperative that all cases of altitude decompression sickness treated by overcompression be reported in the literature, with special attention to the exact pressure at which the signs and symptoms are alleviated. The establishment of the USAF SAM Decompression Sickness Management Team is an attempt to centralize data and treatment of the disorder in the United States Air Force.

Rapid recognition and treatment cannot be overemphasized; regardless of the etiology of delayed decompression sickness, the sooner the pulmonary hypertension, tachypnea, bubble formation, and bubble size are reduced, the better should be the chances of complete recovery.

Availability of treatment facilities is a major problem in the utilization of this form of therapy. Portable chambers are available, but they have disadvantages as well as advantages. A lightweight portable chamber capable of producing 6 atmospheres absolute pressure could be utilized in transporting patients to treatment facilities. Of course, isolation of the patient may be undesirable in a rare instance.

The rapidity with which the previously mentioned signs can occur deserves some comment. After decompression, the animal may appear perfectly stable for 5 to 30 minutes before showing any sign of decompression sickness, then suddenly develop all three signs to a severe degree within 1 minute. One should remember this possibility when there is a differential diagnosis of either myocardial or cerebral ischemia. As a matter of fact, coronary and cerebral vessels usually show a great many bubbles in decompression sickness; thus, one would expect symptoms of myocardial or cerebral ischemia. The ECG may not be too helpful in distinguishing chokes from myocardial infarction, since the early changes may sometimes be identical. High oxygen pressure therapy has recently been suggested for many hypoxic disorders, and could be beneficial regardless of the etiology of the myocardial or cerebral disorder.

The fact that bubbles do not disappear completely with recompression was observed in this study and has been reported by Downey[9] in vitro. Complete resolution of the bubbles may not be necessary; reduction in size of the bubbles and prevention of new bubble formation would appear mandatory, however, thus allowing the respiratory system, internal and external, to rid the circulatory system of the remaining bubbles.

The exact nature of the neurovascular damage caused by bubbles, spasm, sludging, fat emboli, and even bone marrow emboli[6] is not known, but compression therapy should be of benefit in any case.

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