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Pressure in the treatment of spinal cord decompression sickness

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Leitch DR, Hallenbeck JM. Pressure in the treatment of spinal cord decompression sickness. Undersea Biomed Res 1985;12(3):291–305.—Previous work had shown that a P_{O_2} of about 2.0 bar was the optimal P_{O_2} for the treatment of spinal cord decompression sickness (DCS). With 20 anesthetized dogs the hypothesis was tested that pressures in excess of a threshold, taken as 3 bar, did not enhance recovery of spinal cord DCS. Dogs were subjected to a 15-min air dive at 10 bar (300 ft) and decompressed over 5.5 min. At the surface, spinal cord evoked potentials (SEP) were observed for changes indicating DCS. Fifteen minutes after DCS was first detected the dogs were recompressed to 3, 5, 7, or 2.8 bar breathing 66, 40, 29, or 100% oxygen which gave a P_{O_2} of 2.0 bar except in the 2.8 bar group. The recovery of the SEP over 2 h was observed. Group mean recoveries at 67, 62, 29, and 42% were not significantly different after 120 min. As the hypothesis was supported, a tentative proposal for changing current therapy was made.

decompression sickness
brain
spinal cord
evoked potentials
nitrogen narcosis
therapy
oxygen
pressure
recompression

It is believed that Pol and Watelle in 1854 first suggested that recompression was the treatment of choice for decompression sickness (DCS). They observed that workers with DCS had discovered that reentering the workings cured their pain (1). Bert's experiments led him to the same conclusion (2). Although Smith, who coined the term "caisson disease" while associated with the building of the Brooklyn Bridge in 1873, had no idea of the mechanism of DCS, he recommended that a medical treatment lock should be provided (3). The first such lock was probably that of Sir Ernest Moir during the building of the Hudson River Tunnel in 1893 (4). Certainly by 1895 Snell was using a medical lock on the Blackwall Tunnel site (5). His general policy was to recompress to the depth of relief for up to 30 min and not to exceed working pressure.

Keays reporting in 1909 recommended returning to working pressure. Although many cases recovered at lower pressures, there was a high recurrence rate if the higher pressure was not used (4). In 1912 Ryan recommended only using ⅔ of the working pressure with a wait of up to 60 min.

In 1939 Yarborough and Behnke (6) summarized U.S. Navy experience and the available treatment options around a discussion of Boyles Law, as recompression to:

a. Pressure of relief
b. Pressure of relief plus an arbitrary amount
c. Pressure of the causative dive
d. Pressure greater than the pressure of the causative dive.

Behnke and Shaw demonstrated that 3 bar (66 ft) was adequate to stop the cardiopulmonary DCS arising in dogs after long dives at 5.4 bar (7). Breathing air did not however present recurrences. Yarborough and Behnke practiced recompression of depth of relief plus 1 bar. They developed guidelines within a minimum pressure of 4 bar (100 ft) and a maximum pressure of 6 bar (165 ft). They also set a minimum time at maximum pressure of 30 min (8). They observed "that those patients who respond to pressure treatment, do so rather promptly." The approach to those with a poor response was empirical in "that as long as improvement occurs the maximum pressure should be maintained for a period of at least 2 hours." They modified their Haldanian type decompression to use oxygen from 2.8 bar (60 ft) back to the surface. In the event of recurrence they recompressed to the depth of relief which was invariably less than 3 bar and stayed 12 to 24 h.

These treatment practices had about a 50% recurrence, as did modified versions (9). In 1945 Van der Aue et al. produced treatment outlines embodying the Yarborough and Behnke principles in formal tables (10). These U.S. Navy tables continue in worldwide use in various forms to this day. The available treatment pressures recommended for use in air DCS range from 2.8 bar breathing oxygen to 10.7 bar breathing air or mixtures (9).

Little experimental work has been reported in support of this range of treatment pressures. Barnard (11) cites Russian experiments which claim to show that increasing the maximum treatment pressure increased the effectiveness of treatment in animals with severe DCS. This is contrary to his own findings (12). Mice recompressed to depths between 5 and 80 m after surfacing from dives at between 125 and 225 m, survived best in an optimum pressure range of 15 to 40 m irrespective of the depth of the causative dive. They believed the range was probably 20 to 25 m.

Recent studies of the optimum pressure for treating cerebral arterial gas embolism, where Boyles Law may be considered to be particularly relevant, showed that pressure in excess of 2.8 bar (60 ft) did not improve recovery in the model used (13). In the previous study in this series, an optimal oxygen pressure for treatment of spinal cord DCS has been established as 2.0 bar (14). The purpose of this study was to see whether increasing treatment pressure with a fixed oxygen pressure of 2.0 bar would improve recovery while at pressure.

MATERIALS AND METHODS

The method using spinal cord evoked potentials (SEP) to study DCS has been fully described in the previous paper (14). Twenty adult male mongrel dogs weighing between 10.0 and 14.5 kg were used in the final data pool.

Dogs were assigned to one of four treatment groups according to their weight, so that there were 5 in each group. This ensured that the group mean weights were similar (range 11.3–12.1 kg). The treatments included three with a P_{O_2} of 2.0 bar and ambient pressures of 3, 5 and 7 bar and a fourth group breathing oxygen at 2.8 bar. The last group was included to provide a comparison with the generally accepted standard treatment. The desired P_{O_2} was obtained by providing 66, 40, 29, or 100% oxygen in nitrogen mixtures to the ventilator. The experiments were cycled so that after each series of four treatments the next replication began with a dog from a different group. The basic statistical analysis was a one-way analysis of variance.

The dive protocol was standardized and all dogs were compressed to 10.0 bar (90 m, 300 ft) at a rate of 75 ft (22.7 m)·min^{-1} while breathing air. They remained at 10 bar for 15 min before being decompressed at 18 m·min^{-1} to 18 m and at about 14 m·min^{-1} from there to the surface. Dogs that did not show SEP changes within 30 min of surfacing were given a further 9 min at 10 bar. One dog in each group required a second dive. Mean ascent time was about 5.5 min (range 5.0–5.7 min). Recompression for treatment began 15 to 17 min after the diagnosis of spinal cord DCS was made.

Dogs developing hypotension during the pretreatment surface interval were maintained by giving 8% sodium bicarbonate to correct the acidemia, followed by lactated Ringer's solution. The previously described six exclusion criteria (14) were applied in this experiment and led to the elimination of 12 cases from the final data pool.

After the dogs were decompressed at the end of the experiment, the spinal cords were removed within 30 min of death from those dogs in the low pressure groups. The cords from the 3 and 2.8 bar groups were fixed in 10% phosphate buffered formalin (4% wt/vol formaldehyde). The low pressure groups alone were used because there was little risk of DCS from the final decompression. Thus, any changes could reasonably be attributed to the initial decompression and have some bearing on recovery during treatment.

The cords were cut into seven segments, cervical, four thoracic, and two lumbar segments. These in turn were sectioned and three or four sections stained with hematoxylin and eosin. The sections were scrutinized for gross changes and for extent of hemorrhage. Each section was scored by the number and size of hemorrhages. Each hemorrhage was approximately graded 1 to 4: 1- <10 RBC; 2- 10 to 20 RBC; 3- 20 to 40 RBC; and 4- >40 RBC. Each section was then scored for gray and white matter by multiplying each grade by its frequency. The mean section score was then calculated for the four main cord segments: cervical, upper thoracic, lower thoracic, and lumbar.

RESULTS

Evoked potentials

The cortical evoked potentials (CEP) were similar to those seen previously. Peroneal CEP P_1,N_1, and P_2 latencies were 15.8 ms (SE 0.5), 22.8 ms (SE 0.5), and 34.2 ms (SE 0.7), respectively, and the median CEP latencies were 12.8 ms (SE 0.3), 19.3 ms (SE 0.4), and 31.9 ms (SE 1.0). Exposure to 10 bar of air barely altered the latencies to 15.8 ms (SE 0.5), 23.1 ms (SE 0.5), and 34.4 ms (SE 1.0) for the peroneal

CEP, and to 12.9 ms (SE 0.3), 19.3 ms (SE 0.4), and 31.9 ms (SE 0.8) for the median CEP.

The overall mean control amplitudes for peroneal CEP, P_1N_1 and N_1P_2, were 31 pV(SE 5) and 43 pV(SE 6), respectively. These were depressed by 10 bar of air to 83% (SE 3) and 86% (SE 3), respectively. Similarly, for the median CEP, 40 pV(SE 5) and 50 pV(SE 6) amplitudes were depressed to 71% (SE 2) and 76% (SE 2), respectively.

The summated amplitudes for left and right peroneal-lumbar and for the left median-cervical SEP were marginally affected by 10 bar of air, being 97% (SE 2), 95% (SE 2), and 95% (SE 2) of their controls. All these findings conform with earlier work (14, 15).

Systemic changes

The onset times of the systemic changes are shown in Table 1. The earliest change indicating DCS was again a reduction in EEG amplitude. However, only 50% of cases were affected in this series (Table 1). The range of onset times was 2 to 17 min after surfacing. Cerebrospinal fluid pressure (CSFP) rose in 13 cases to levels shown in Table 2. There were 5 cases where CSFP rose in the absence of EEG changes and 2 cases where EEG changed without a CSFP rise. In only one case did the CSFP rise begin before the EEG change. The onset time for the CSFP rise was 3 to 18 min. There was no systematic association between CSFP rise and reduction in SEP amplitude.

Thirteen cases showed a rise in right ventricular pressure (RVP) to as high as 170/0. In no cases did this precede the CSFP rise, in 4 cases the rises were coincident and in 4 cases the RVP rise was later. The remaining 5 were not associated with

TABLE 1
EVENT TIMES DURING DECOMPRESSION SICKNESS

Pressure (bar)	3.0	5.0	7.0	2.8	*F* ratio
Event					
Surfacing to D	15	18	10	16	0.80
	±3	±5	±3	±4	
D-1 to D	4	4	4	5	0.86
	±1	±1	±1	±1	
Last SEP to Tr	1	2	2	2	0.22
	±1	±1	±1	±1	
RVP ↑	11(3)	9(3)	14(2)	12(5)	—
CSFP ↑	15(4)	8(2)	9(4)	8(3)	—
EEG ↓	10(3)	8(1)	6(4)	3(2)	—
$\overline{BP}$ ↑	23(3)	−(0)	−(0)	24(2)	—
$\overline{BP}$ ↓	−(0)	−(0)	−(0)	−(0)	—

Times in minutes shown as mean ± standard error. Figures in parenthesis indicate number of dogs. D = diagnosis of DCS in the SEP, D-1 = last normal SEP. Tr = start of treatment, RVP = right ventricular pressure, CSFP = cerebrospinal fluid pressure, $\overline{BP}$ = mean blood pressure, *arrows* indicate a rise or fall in pressure or amplitude.

TABLE 2
CARDIOVASCULAR VARIABLES

Pressure (bar)	3.0	5.0	7.0	2.8	*F* ratio
Mean Blood Pressure (mmHg)					
Postdive	118 ± 5	122 ± 10	112 ± 5	120 ± 8	0.39
Interval range	88 – 185	82 – 162	80 – 127	90 – 203	—
Pre-Tr	132 ± 16	112 ± 8	103 ± 7	117 ± 4	1.41
Change on Tr	– 36(5)	– 11(2)	– 9(2)	– 29(4)	1.39
Tr 15 min	130 ± 8	144 ± 12	133 ± 4	121 ± 11	1.16
Tr 120 min	114 ± 4	119 ± 5	123 ± 7	115 ± 7	0.53
Right Ventricular Pressure (mmHg)					
Postdive	43/0	18/2	48/2	29/0	1.72
Interval range (Systolic)	20–170	12–34	10–110	18–115	—
Pre-Tr	61/4	25/1	54/1	53/1	0.81
Tr 15 min	72/1	20/2	50/3	31/2	1.37
Tr 120 min	29/4	19/3	42/2	24/0	1.70
Cerebrospinal Fluid Pressure (mmHg)					
Postdive	12 ± 4	14 ± 3	11 ± 5	19 ± 5	0.72
Interval range	1–22	2–22	1–28	5–56	—
Pre-Tr	10 ± 4	11 ± 4	10 ± 3	20 ± 4	1.50
Tr 15 min	10 ± 3	11 ± 5	21 ± 8	17 ± 7	0.80
Tr 120 min	10 ± 3	18 ± 9	23 ± 8	18 ± 7	0.67
Cerebral Perfusion Pressure (mmHg)					
Postdive	106 ± 7	108 ± 9	101 ± 8	101 ± 5	0.27
Pre-Tr	122 ± 13	101 ± 6	93 ± 8	98 ± 6	2.00
Tr 15 min	120 ± 7	133 ± 8	112 ± 8	104 ± 14	1.76
Tr 120 min	104 ± 6	101 ± 12	100 ± 8	97 ± 13	0.08

Pressures in mmHg shown as ± 1 standard error. Mean blood pressure = diastolic BP + ⅔ pulse pressure. Figures in parentheses indicate number of dogs.

a significant CSFP rise. The pattern of change in RVP and CSFP during the pretreatment interval and treatment was similar to that seen previously (14) (Table 2).

During the pretreatment interval only 4 dogs had an increase in systolic blood pressure of more than 20 mmHg above any prediagnosis pressure. They all developed pressure in excess of 200 mmHg but none reached 300 mmHg. While these pressures were transient, in no case did the systolic pressure in these or other cases fall even transiently below 100 mmHg during the pretreatment interval. Compression caused a transient fall in mean blood pressure of up to 70 mmHg in 13 cases.

Cerebral perfusion pressure calculated as the difference between CSFP and mean blood pressure is shown in Table 2. Heart rate increased by up to 45 beats·min^{-1} in 13 cases and was unchanged in 7 during the pretreatment phase. It returned to control levels during treatment. The mean rise was 12 beats·min^{-1}.

Variable amounts of fluid were given after the diagnosis of DCS (Table 3). They were effective in maintaining blood pressure and reducing the expected rise in hema-

TABLE 3
HEMATOCRIT AND FLUID BALANCE

Pressure (bar)	3.0	5.0	7.0	2.8	*F* ratio
Hct control (%)	44 ±2	44 ±1	43 ±1	45 ±1	0.66
Pre-Tr	47 ±3	47 ±2	43 ±2	47 ±1	1.02
Tr 40 min	49 ±3	47 ±2	43 ±1	45 ±2	1.36
Tr 80 min	46 ±2	45 ±2	44 ±1	45 ±2	0.26
Overall fluid balance (ml)	77 ±71	140 ±51	108 ±26	4 ±48	1.27
Fluid-in in Tr (ml)	190 ±46	186 ±42	96 ±20	100 ±27	2.15
Urine output (ml)	178 ±55	104 ±12	146 ±40	140 ±30	0.65

Data given as mean ±1 standard error.

tocrit to a mean of 2.2% (Table 3). In only 3 cases did the increase exceed 5% and 15 cases showed a rise of only 3% or less.

A marked respiratory acidemia occurred during the pretreatment phase (Table 4). Five dogs had an arterial pH of less than 7.3 which was caused by an elevation in arterial P_{CO_2}. In 6 dogs Pa_{CO_2} exceeded 39 mmHg and in 3 of these it was 45 mmHg or more. The 6 hypercapric dogs were also hypoxemic with a Pa_{O_2} less than 85 mmHg before treatment. The infusion of bicarbonate and compression resolved these conditions.

Up to the start of treatment the only variables that were significantly different between the group means were the arterial P_{CO_2} and P_{O_2}. This indicated a significant pulmonary gas exchange problem in the 2.8 bar treatment group, largely the influence of 2 dogs.

Spinal evoked potentials

The first identified SEP lesions in the 20 dogs in the final data pool included 13 left lumbar and 7 right lumbar lesions (Table 6). These SEP were used for diagnosis and assessment of recovery. The loss of SEP amplitude ranged between 29 and 100% by the start of treatment. At the start of treatment there was no significant difference in severity between the groups (Tables 5 and 6).

The time from surfacing at which SEP change was identified ranged between 2 and 30 min. There was no significant difference between the group means in spite of the shorter mean onset time in the 7 bar group (Table 1). The extent of the change leading to diagnosis (D-1 to D) ranged between +8 and −45% of control (Table 5). Occasionally an increase in amplitude was seen as inhibitory fibers ceased to function before there was a generalized loss of amplitude. The interval between the last normal

TABLE 4
ACID-BASE AND GAS ANALYSIS

Pressure (bar)	3.0	5.0	7.0	2.8	*F* ratio
Arterial pH					
Control	7.39 ± 0.01	7.37 ± 0.01	7.39 ± 0.02	7.38 ± 0.01	0.62
Pre-Tr	7.35 ± 0.02	7.34 ± 0.02	7.34 ± 0.03	7.31 ± 0.03	0.49
Tr 40 min	7.34 ± 0.02	7.41 ± 0.02	7.38 ± 0.05	7.38 ± 0.02	0.94
Tr 80 min	7.37 ± 0.02	7.40 ± 0.02	7.37 ± 0.04	7.37 ± 0.01	0.47
Arterial P_{CO_2} (mmHg)					
Control	33 ± 1	34 ± 1	33 ± 2	35 ± 1	0.92
Pre-Tr	32 ± 2	38 ± 2	38 ± 3	43 ± 3	3.32*
Tr 40 min	36 ± 3	34 ± 2	40 ± 5	38 ± 2	0.72
Tr 80 min	37 ± 3	35 ± 3	41 ± 4	36 ± 1	0.64
End-Tidal F_{CO_2} (%)					
Control	3.4 ± 0.3	3.8 ± 0.4	3.3 ± 0.3	3.7 ± 0.3	0.30
Pre-Tr range	2.2 – 4.1	2.1 – 5.3	1.8 – 4.1	2.5 – 4.6	—
Pre-Tr	3.7 ± 0.1	3.6 ± 0.4	3.4 ± 0.4	3.7 ± 0.3	0.22
Tr 40 min	4.4 ± 0.3	4.4 ± 0.3	4.7 ± 0.3	4.6 ± 0.2	0.31
Tr 80 min	4.5 ± 0.3	4.6 ± 0.1	4.5 ± 0.3	4.2 ± 0.1	0.61
Arterial P_{O_2} (mmHg)					
Control	94 ± 3	96 ± 2	92 ± 3	90 ± 3	1.02
Pre-Tr	108 ± 10	99 ± 6	89 ± 4	74 ± 9	3.33*

Data shown as mean ±1 standard error. *Under *F* ratio indicates a significance of $P < 0.05$.

TABLE 5
CHANGES IN SPINAL EVOKED POTENTIALS

Pressure (bar)	3.0	5.0	7.0	2.8	*F* ratio
Time Period					
D-2 to D-1	+3	+3	−1	−4	1.42
	±4	±2	±1	±3	
D-1 to D	−27	−7	−17	−4	2.08
	±7	±5	±3	±11	
D to Pre-Tr	−54	−47	−60	−65	0.29
	±15	±14	±15	±14	
Pre-Tr loss	86	70	84	81	0.45
	±7	±13	±12	±8	

Change in SEP as a percent of control shown as mean ±1 standard error. D-2 = penultimate normal SEP, D-1 = last normal SEP, D = diagnostic SEP.

SEP and the diagnostic SEP was less than 5 min in all cases (Table 1). The check on diagnostic perception of comparing the penultimate normal SEP (D-2) with the last presumed normal SEP (D-1) showed no difference between the group means

TABLE 6

THE EXTENT AND SEVERITY OF CNS DECOMPRESSION SICKNESS AS INDICATED BY SPINAL EVOKED POTENTIAL AMPLITUDE

Dog No. (EP)	LPL	LPT	LPC	RPL	RPT	RPC	LMC	EEG (Time)
200	100	?	100	[100]	?	100	0	—
201	[100]	100	100	52	?	?	0	14
202	[65]	>41	>29*	0	24	14*	>20*+	9
203	75	69	47	[76]	?	?	0	8
204	[90]	100+	100	86	>67+	100	0	—
211	[98]	85	100	11	>23	?	66	7
213	0	0	?	[52]	58	?	0	—
216	[95]	?	96	85	100	100	0	—
217	[77]	68	50	0	0	?	0	—
218	[29]	100	100	0	100	100	0	9
220	24	13	27*	[36]	35*	36	0	3
221	90	83	82	[100]	?	?	0	—
222	[99]	?	100*	89	?	?	0	17
224	[90]	100	95	76*	71*	100	0	2
225	84	97	100	[93]	100	100	0	3
231	[100]	100	100	85	?	?	0	—
232	>69	?	>46	[90]	?	100	0	—
233	[65]	?	55	37	?	>58	>20*	4
234	[59]+	?	68	74+	?	?	94*+	2
235	[93]	?	100	89	?	100	0	—

Data are presented as percent of control SEP lost at the start of treatment. [ ] = principal and controlling SEP, ? = no record, * = no recovery at 120 min, + = cord hemorrhage at postmortem. Evoked potentials are LPL = left peroneal—lumbar, LPT = left peroneal—thoracic, and LPC = LP—cervical. Similarly RPL, RPT, RPC = right peroneal—lumbar, thoracic and cervical, LMC = left median-cervical. Figures in the EEG column are times in minutes of the start of a reduction in amplitude.

(Table 5). In 2 cases in the 7 bar group onset time was very short so the prediagnostic SEP were recorded at pressure.

The progression of the SEP amplitude loss between diagnosis and start of treatment (D to Pre Tr) is shown in Table 5. Three cases showed a stable SEP loss before treatment; 1 in the 3 bar group and 2 in the 7 bar group. The remainder showed a progressive deterioration. The interval between the last pretreatment SEP and the start of compression therapy varied between 0 and 6 min (Table 1). Four dogs showed a further SEP loss over compression. The additional losses were 36% (3 bar), 21% (5 bar), 2 and 11% (2.8 bar) with respective intervals before compression of 1, 6, 2, and 2 min.

Recovery of SEP amplitude was generally rapid in the first 15 min of treatment (Table 7) and ranged between 1 and 100%. Although the 3 bar group led the recovery at 15 min there was no significant difference between the group means. The 3 and 2.8 bar groups showed a further improvement in group means by 40 min. This led to a significant difference between the groups means ($P < 0.05$). The significant difference was preserved at 80 min but lost by 120 min, as the 3 bar group entered a slow deterioration while the 5 bar group continued to improve. The 7 and 2.8 bar groups remained stable. The decline in the 3 bar group was largely due to one case which lost 70% of its 100% peak at 15 min. The rest of the group all showed a small reduction in amplitude. Two dogs contributed to the continued improvement of the 5 bar group, and 1 dog to the late fall in the 2.8 bar group. Ten dogs did not show more than a 4% deterioration from their peak recovery. The 120 min of treatment ended with no significant difference between the groups.

The individual peak recovery times covered the entire treatment period. Using all the available SEP data showed the group mean peak recovery times to be 54, 46, 60, and 81 min (F ratio 1.13). The mean SEP recovery at these times was 91, 80, 54, and 63% (SE 6 to 12 and F ratio 3.46). The subsequent deterioration was similar in the 4 groups at 24, 18, 25, and 20%.

The extent of CNS involvement as far as it can be assessed is shown in Table 6. This includes the onset times for EEG changes as well as the pretreatment SEP

TABLE 7
SEP RECOVERY AS PERCENT OF LOSS

Pressure (bar)	3.0	5.0	7.0	2.8	F ratio
Time Period					
at 15 min	75	55	31	32	3.11
	±11	±12	±13	±9	
at 40 min	89	53	31	46	4.20*
	±10	±14	±13	±8	
at 80 min	80	58	27	50	3.89*
	±10	±13	±11	±9	
at 120 min	67	62	29	42	2.39
	±13	±13	±8	±12	

Recovery of SEP during treatment expressed as percent of loss shown as mean ±1 standard error. *Under F ratio shows significance at $P < 0.05$.

amplitude loss. Expressing the number of left and right lumbar and left cervial SEP affected as a percentage of the available recordings gave group means of 50, 40, 50, and 60%, thus demonstrating that the extent of cord DCS was similar in all four groups.

Discarded data

Twelve dogs were discarded because they did not satisfy the criteria for inclusion. Three required second dives and four required controlling dives to prevent cardiovascular collapse. The diagnosis was missed in 4 cases which resulted in excess delay to treatment, 4 cases showed no recovery, the remainder experienced one each of prolonged hypotension as low as 50/34 for 13 min, physiological instability, spontaneous recovery, and random SEP changes.

Pathology

Three dogs had macroscopic hemorrhages in the cord. Number 202 had gross hemorrhage over the left lateral column in the cervical cord, No. 204 had petechial hemorrhages over the thoracic cord, and No. 234 had gross hemorrhage over the cervical cord and petechial hemorrhages over the lumbar cord (Table 8).

Cords were removed from 9 dogs in the experiment. A control dog which was prepared in the same way for a control study but was not dived was added, as was the cord from a case of untreated DCS.

TABLE 8

FREQUENCY AND DISTRIBUTION OF HEMORRHAGE IN SPINAL CORD

Site	Lumbar		Lower Thoracic		Upper Thoracic		Cervical	
Distribution	G	W	G	W	G	W	G	W
Dog								
Control	0	0	1	0	3	0	0	0
DCS No Tr	0	0	5	5	2	20	37	3
201	3	0	7	0	0	0	0	0
202	0	0	10	7	6	11	3	0+
203	4	0	9	1	11	0	12*	4*
204	41	43	13	41+	12	9+	16	153
231	1	0	1	0	4	1	0	0
232	1	2	1	0	2	0	0	0
233	1	0	2	0	8	0	33	2
234	3	11+	1	3	3	7	14	41
235	0	1	0	5	4	0	2	0

Mean section hemorrhage expressed as the product of frequency and grade for each main cord segment, divided into gray (G) and white (W) matter. Dogs were undived control, untreated DCS, 4 dogs from the 3 bar group, and 5 dogs from the 2.8 bar group. + = Macroscopic subarachnoid hemorrhages, * = unilateral hemorrhages.

Sections showed a spectrum of hemorrhages from nil to frequent and macroscopic, as seen in No. 204 (Table 8). Additional changes seen were instances of vascular congestion and a teased appearance. An estimate of the extent of hemorrhage is shown in Table 8.

A small number of hemorrhages were seen in the thoracic cord of the control dog. This suggests a need for a greater number than 2 or 3 per section to indicate pathology. For the most part the majority of hemorrhages occurred in the gray matter except in dogs 204 and 234.

Three dogs (202, 233, 234) experienced a loss of cervical SEP which did not return during treatment (Table 6). The cervical cords of 202 and 234 showed gross subarachnoid hemorrhage, while those of 233 and 234 showed extensive microscopic hemorrhages. Comparing the amount of lumbar SEP recovery at 120 min (Table 9) with the extent of lumbar cord hemorrhage showed only a correlation between the poorest recovery in each group and the greatest amount of hemorrhage.

DISCUSSION

At the start of treatment the four treatment groups were physiologically similar except that 2 dogs in the 2.8 bar group displayed a respiratory acidosis with hypoxemia. This was indicative of a pulmonary perfusion problem although it was not particularly reflected in the pretreatment RVP. It did not result in any particular lack of recovery, as it occurred in both the worst and the best dogs in the group. The groups had similar degrees of spinal cord DCS. During treatment a large proportion of the recovery had occurred by 15 min with a peak of recovery at around 60 min. Twenty minutes either side of this peak there was a significant difference between the groups (ANOVAR). By 120 min the difference was no longer significant. The

TABLE 9
INDIVIDUAL SEP RECOVERY AT 120 MIN

Dog	SEP Site	% SEP Lost	% Loss Recovered
		3 Bar Treatment	
201	LPL	100	84
202	LPL	65	97
203	RPL	76	82
204	LPL	90	44
		2.8 Bar Treatment	
231	LPL	100	55
232	RPL	90	18
233	LPL	65	68
234	LPL	59	10
235	LPL	93	60

Data shown as percent of SEP loss recovered at 120 min. The sites of the principal lesions were LPL—left peroneal lumbar, RPL—right peroneal lumbar.

hypothesis that additional pressure would not improve recovery once a pressure threshold had been passed and an optimum P_{O_2} established was supported. The data also suggested that the rate of recovery when using a P_{O_2} greater than 2.0 bar might be slower although the same end point might be reached.

Only 3 dogs recovered their SEP loss completely but none of the recovery was sustained. Comparison of this and the previous study (14) suggested that better control had been established over the model. The systemic DCS now seemed less severe. The rate of EEG loss fell from 72 to 55%, and the rate of involvement of the three principal SEP fell from 71 to 50%. However, the mean amplitude loss of the controlling SEP rose from 64 to 80%. There was a lower incidence of hypertension, down from 36 to 25%, and none of these approached a systolic pressure of 300 mmHg where previously 3 dogs had exceeded that pressure. There was less acid-base disturbance, the hematocrits were largely contained, and there were no cases of hypotension in the pretreatment period in spite of a smaller volume of fluid being infused. The number of instances of transiently reduced blood pressure during compression fell from 88 to 65% possibly indicating less free systemic gas. The level of recovery gained by the best groups in each study was similar in spite of a greater SEP loss in this study and more severe systemic disturbance in the former study. The fact that 10 dogs did not show more than a 4% reduction from peak recovery coincides with the observation in the previous study that those that did not materially deteriorate were confined to the 2.0 bar or higher oxygen groups. There was some deterioration in all cases in this study and the reduction from peak amplitude was similar in the four groups.

There was only 1 case where a rise in RVP preceded EEG amplitude loss, providing a possibility of transpulmonary bubble passage to cause cerebral DCS. The remaining 9 cases of cerebral DCS were presumably due to authochthonous bubble formation.

Although there was no significant difference between the group mean onset times it is notable that the 7 bar group with the shortest onset time had the lowest recovery. There was again a correlation between onset time and recovery. The least squares regression equation was:

$$Y = 15.50 + 2.35t$$

where Y was the percent recovered and t was onset time in minutes ($R = 0.51$, $P < 0.001$). Separating recovery into those achieving less than 40% and those achieving more than 60% at 120 min gave mean onset times of 6.7 and 20.2 min (Student's t test $P < 0.001$). So in this study where all treatments are expected to be equally effective it is confirmed that early onset time militates against good recovery where there is at least a 15-min delay before recompression. Again there was no difference in the percent of SEP loss at 75 and 78%, respectively. There was no identifiable association with systemic changes, cerebral DCS, hemorrhages, or extent of cord DCS to account for this relationship.

The possibility of autochthonous bubble formation must be raised. It may be responsible for the cerebral DCS which is of early onset, and if treatment is delayed, as here, it may lead to a greater amount of permanent damage as it progresses.

Again the physiological presentation tended to support the model of epidural vertebral venous obstruction as the mechanism of spinal cord decompression sickness as outlined by Hallenbeck and coworkers (16, 17). However, there were four instances of unilateral cord involvement which rest uneasily in that hypothesis although those

authors did observe a similar finding (17). In addition, the distribution of hemorrhages in the cords of 9 dogs with DCS showed partial gray matter sparing in only 8 out of 34 cord segments showing any degree of hemorrhage (Table 8). Gray matter sparing is said to be compatible with venous obstruction while arterial occlusion affects gray matter particularly in the watershed regions (18). The cord sections presented a picture apparently dominated by evidence of arterial obstruction with hemorrhages concentrated in central gray matter. This presentation is compatible with general ischemia and embolic phenomena. It can only be concluded that epidural vertebral venous system obstruction is only one part of a more varied mechanism causing spinal cord damage. The apparent differences in findings probably represent the varied nature of the causative dives that govern the form of the DCS presentation. There was no correlation between frequency of hemorrhage and previous hypertension.

The use of Boyle's Law to determine what would be an appropriate treatment pressure founders on not knowing the nature and location of the offending bubbles. In lozenge-shaped bubbles, volume must be reduced before reduction in diameter can occur as it does in spherical bubbles.

Compression to 3 bar reduces volume to 33% and diameter to 69%. At 5 bar these values are 20 and 58%, respectively, and at 7 bar they are 14 and 52%. Evidently the law of diminishing returns becomes relevant for diameter before it does so for volume. Volume is dramatically reduced by moderate compression but it takes much greater pressures to similarly reduce diameter.

Kunkle and Beckman in discussing bubble resolution conclude that because of the increased inert gas tension inherent in breathing air at 6 bar, the enhanced reduction in bubble size is countered by the reduced rate of gas clearance (19). This results in a similar rate of bubble clearance to that found while breathing oxygen at 2.8 bar. The advantage of greater pressures is supposed to be the rapid initial reduction in bubble size for the early restoration of circulation.

The objectives in treatment are to stop further bubble growth, restore oxygenation and circulation, and to increase inert gas clearance. It is axiomatic that compression and a raised P_{O_2} are necessary to achieve these aims. It has now been established that an optimal P_{O_2} is about 2.0 bar (14). This has the advantage of being less toxic than the currently used P_{O_2} of 2.8 bar. The degree of vasoconstriction will be less which must be advantageous and it can be breathed for longer periods without break. No effort has been made to establish a pressure threshold in these studies but it has been shown that pressures above 3 bar do not improve recovery in this model. This conforms with the findings of Barnard and Hanson (12) and Leitch et al. (13). A major remaining question is whether a failure of recovery at a lower pressure can be reversed by further compression. In view of the probable difference in pathology it seems unlikely. Certainly there is little clinical evidence that further compression produces a step improvement (20).

The disadvantage of breathing mixtures that include nitrogen is that the nitrogen gradient is reduced. However, breathing 34% nitrogen at 3 bar only gives a P_{N_2} of 1.02 bar; little more than breathing room air at atmospheric pressure. In practice, using 66% oxygen at 3 bar does not appear to have any disadvantages when compared with 2.8 bar of oxygen, in spite of the additional nitrogen. Although the outcome was the same the rate of recovery at the higher pressure with the lower oxygen may be better. The reduction in risk from oxygen toxicity would be the main reason to move

away from the 2.8 bar oxygen treatment. The possibility of an increased rate of response would also be a sound reason. If a new treatment was introduced it might take the form of breathing 66% oxygen at 20 m (66 ft) for 1 or 2 h with a 10 min air break per hour followed by a bleed to 10 m (33 ft), with a switch to oxygen before completing a table similar to USN 6 (RN 62). The obvious disadvantage is the supply of a 66% oxygen mixture.

This model of spinal cord DCS now lends itself to testing chemotherapy and some of the more contentious treatment ploys being suggested such as oxyhelium for treating air DCS.

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The experiments reported herein were conducted according to the principles set forth in the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Animal Resources, National Research Council, DHHS, Pub. No. (NIH) 78-23.

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Leitch DR, Hallenbeck JM. La pression dans le traitement de la maladie de décompression de la moëlle épinière. Undersea Biomed Res 1985; 12(3): 291–305.—Un travail antérieur avait démontré qu'une Po_2 d'environ 2.0 bars était la Po_2 optimale pour le traitement de la maladie de décompression (DCS) de la moëlle épinière. L'hypothèse que les pressions supérieures à un seuil, fixé à 3 bars, n'augmentaient pas le recouvrement de la DCS de la moëlle épinière fut vérifiée avec 25 chiens anesthésiés. Les animaux furent soumis à une plongée à l'air de 15 min à 10 bars (300 pieds) et décomprimés en 5.5 min. Au retour à la surface, les potentiels évoqués somatosensoriels (SEP) de la moëlle épinière furent observés pour les changements indicatifs de la DCS. Quinze minutes après que la DCS fut premièrement détectée, les chiens furent recomprimés à 3, 5, 7, ou 2.8 bars en respirant de l'oxygène à 66, 40, 29, ou 100% pure, résultant dans une Po_2 de 2.0 bars, excepté dans le groupe à 2.8 bars. Le recouvrement du SEP fut observé pendant 2 h. Les moyennes du recouvrement de 67, 62, 29, et 42%, respectivement, dans les groupes n'étaient pas significativement différentes après 120 min. Puisque l'hypothèse était supportée, une proposition tentative pour changer le mode courant de thérapie fut faite.

maladie de décompression
cerveau
moëlle épinière
potentiels évoqués
thérapie
oxygène
recompression
narcose à l'azote

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