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Oxygen in the treatment of spinal cord decompression sickness

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Leitch DR, Hallenbeck JM. Oxygen in the treatment of spinal cord decompression sickness. Undersea Biomed Res 1985;12(3):269–289.—Twenty-five anesthetized dogs were used to find the optimum P_{O_2} for the delayed treatment of spinal cord decompression sickness (DCS). They were instrumented for the measurement of physiological variables and somatosensory spinal evoked potentials (SEP) given an air dive of 15 min at 10 bar (300 ft) and decompressed in under 6 min. At the surface SEP were observed for signs of DCS. Fifteen minutes after cord DCS was observed in the SEP, the dogs were compressed to 5.0 bar breathing one of 5 gas mixtures giving a P_{O_2} of 1.0, 1.5, 2.0, 2.5, or 3.0 bar. At the start of therapy all groups were in a similar physiological state with a similar loss of SEP. Between 40 and 120 min, recovery was significantly different ($P < 0.05$) between the groups, most SEP recovery having occurred within 15 min. The treatments ended with 22, 32, 70, 66, and 42% recovery, respectively. It would appear that the optimum P_{O_2} is around 2.0 bar.

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

Early treatment of spinal cord decompression sickness (DCS) is usually effective and problem free (1, 2). Delay in treatment leads to poor results (1, 2). The standard treatments are based on little experimental evidence and mostly on empirical applications of theoretical concepts (3). The purpose of this study was to find the lowest effective partial pressure of oxygen for the delayed treatment of spinal cord DCS. As early as 1854 Pol and Watelle considered using oxygen to treat DCS (4). This was resurrected with the consideration of the use of pressure by Bert in 1878 (5).

Behnke and Shaw (6) observed that dogs with severe cardiopulmonary DCS responded well with recompression to 3.0 bar regardless of whether they breathed air or oxygen. However, on decompression those treated with air had a recurrence of cardiopulmonary DCS whereas those treated with oxygen did not, indicating better gas clearance with oxygen.

The standard air tables using compression to 6.0 bar (50 m, 165 ft) were developed by Van der Aue et al. (7). These were highly effective until the number of delayed treatments increased (1). Subsequently, Goodman and Workman introduced the minimal oxygen compression therapy used at 2.8 bar (18 m, 60 ft) (8). This forms the mainstay of current treatment for all DCS occurring after surfacing.

The use of oxygen at high partial pressures is not without hazard. Sustained exposure to 2.8 bar of oxygen causes cerebral oxygen toxicity. The problem is alleviated by interrupting oxygen exposure with short intervals of air breathing (9). However, Hart (10) reported successful treatment of DCS with continuous oxygen breathing beginning with 30 min at 3 bar followed by 60 min at 2.5 bar before decompression.

Balentine has reported central cord necrosis in rats exposed to 3.0 bar of oxygen for 5 h (11). He suggested that it probably occurs between 2.5 and 5 h. It has also been demonstrated that high levels of oxygen cause vasoconstriction (12). Intuitively this must be a disadvantage in treating a disease believed to be largely the result of impaired perfusion (13). In preparation for this study we developed an electrophysiological model using somatosensory evoked potentials in anesthetized dogs (14–18). The diagnosis of cord DCS was made when significant changes were seen in spinal cord evoked potentials (SEP) recorded from three cord sites and with three peripheral nerve inputs. Together with the cortical evoked potentials (CEP), this montage permitted regional localization of the lesions. It has been demonstrated that SEP amplitude is altered by changes in spinal cord blood flow caused by DCS (16).

MATERIALS AND METHODS

Conditioned adult male mongrel dogs weighing 9.1 to 13.6 kg were sedated with xylazine (2.2 $mg \cdot kg^{-1}$ s.c.) and atropine (0.05 $mg \cdot kg^{-1}$ s.c.) before being anesthetized with intravenously administered pentobarbital sodium (13.5 $mg \cdot kg^{-1}$). The anesthesia was continued with half the initial dose at 20 min and thereafter with 4 $mg \cdot kg\ h^{-1}$ given in divided doses at 20 min intervals. All experiments were terminated with an intracardiac injection of saturated potassium chloride solution.

The dogs were intubated, and ventilation was maintained with a modified BIRD® Mark 7 ventilator using air. A catheter was placed in the left forepaw cephalic vein for anesthesia maintenance and infusions. The right and left femoral arteries were catheterized with polyethelene tubing (o.d. 2.42 mm) for arterial pressure (AoP) monitoring and blood sampling. A 7F gauge preformed catheter (Becton-Dickinson No. 9423) filled with heparinized saline was advanced from the right femoral vein to the right ventricle for pressure (RVP) measurement. Pressures were measured using Gould Statham P23 1D physiological transducers with matching transducer amplifiers and recording on a Gould recorder (Model 2800).

A five-lead ECG harness was connected and a Lead II recording made continuously. Rectal temperature was maintained between 37.5 and 39.0°C. A urinary catheter was inserted into the bladder and connected to a collection bag.

The dog was placed prone in a rigid head holding stand which incorporated a hot water plate. The skull was exposed by reflecting skin, muscle, and periosteum over the right somatosensory cortex. Skin from over the distal part of the nasal bones was

also excised. The skull and nasal bones were drilled and a pair of stainless steel electrodes inserted.

Pairs of insulated 1-mm diameter stainless steel wire electrodes were introduced percutaneously in a bipolar configuration into adjacent interspinous spaces at L1, T8, and C7. They were tapped into the spinal lamina of adjacent vertebrae until their points were securely embedded about 2 mm into the bone. Impedance of the cortical and spinal electrodes was less than 2 and 6 kohm, respectively (14).

Stimulating electrodes were pairs of stainless steel needles carried on double banana plugs. The peroneal nerve electrodes were inserted percutaneously astride the neck of the fibula where the peroneal nerve was palpable. The median nerve electrodes were inserted on the medial aspect of the distal end of the humerus where the median nerve was palpable. A stimulus of 100 to 140 V (3 × motor threshold) with a duration of 0.3 ms and a current of 10 mA was given at a rate not exceeding 2.5 s^{-1}.

The stimulator (Grass S88) and two Nicolet Computers of Average Transients (CAT) (Models 1074 and 1072) were driven by a Nicolet Stimulus Pulse Generator (NIC-502). The stimulus was delivered through a Grass Photoelectric Stimulus Isolation and Constant Current Unit (PISU 6C) and directed through a fourway switching box on the outside of the chamber. The output from all four evoked potentials (EP) sites was averaged simultaneously (n = 128) on the two CATs; the three SEP by the 1074 with a 26–30 ms span, and the CEP by the 1072 with a 110 ms span. The outputs were observed on two Tektronix oscilloscopes (5110) and recorded on three Hewlett-Packard X-Y plotters (HP 7045 A and B). On leaving the chamber through a CONAX high pressure penetrator, the signals went to differential amplifiers (NIC-200A) (gain 10^4) before being further amplified (gain 40) and filtered on a 30 to 3000 Hz bandpass (NIC-501 A). The signal for the CEP was tapped between the amplifier and filter, amplified and filtered (1 to 30 Hz) by a Gould Universal amplifier, and recorded on the chart recorder to give a single site EEG.

When preparation was complete the dog and stand were moved into the compression chamber (Bethlehem Corporation Size 66 × 30 in.) and all connections made. The heating plate was connected to a hard-piped hot water system supplied from outside the chamber. End-tidal CO_2 was continuously monitored by a Beckman LB2 carbon dioxide analyzer, and maintained at 3.0 to 4.5% of surface equivalent by altering cycle time and inspiratory flow rate. The ventilator could also be manually operated from outside the chamber. Pentobarbital anesthesia was provided through a port in the chamber wall. One of the arterial lines was also connected to a port so that blood samples could be drawn when the chamber was pressurized. After the EP system was tested a needle was inserted percutaneously into the cisterna magna and connected to another transducer for the measurement of cerebrospinal fluid pressure (CSFP).

Control data were collected including arterial blood gases, pH, and hematocrit. Up to five sets of EP were collected from each peripheral nerve input before diving commenced. The EP were recorded with the positive up convention beginning 2 ms before stimulation. Figure 1 shows the form of peroneal and median CEP with three principal waves—P_1, N_1, and P_2—relating to near-field cortical potentials. In addition, as many as 11 far-field potentials (FFP) relating to subcortical events were observed between the stimulus and P_2. The SEP (Fig 2) produce complex polyphasic wave forms giving up to 10 easily identifiable pairs of peaks. The more rostral along the

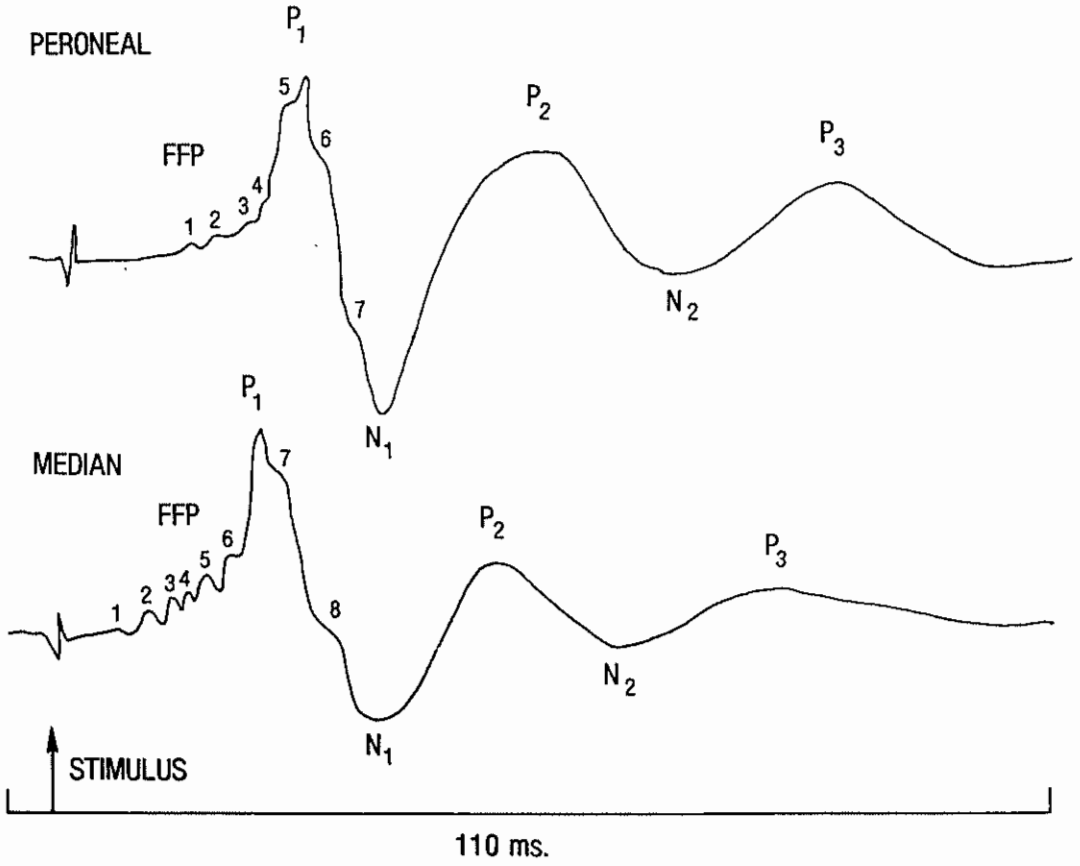

Fig. 1. Representative cortical evoked potentials. Near field principal cortical peaks are marked P_1, N_1, etc; and the far-field potentials are numbered 1 to 8.

spinal cord the recording was from the stimulus, the longer the latency, the greater the spread, and the smaller the amplitude of the traveling waves. Records from close to the root input were dominated by a large negative wave and a later slow positive wave at 8 to 10 ms, as is shown in the peroneal L4 and median C7 recordings.

During the development of the model it was seen that in DCS most early SEP changes were related to amplitude and not to latency. This allowed the use of a simple summing of peak-to-peak amplitudes in SEP as a means of quantifying events (14, 17, 18). The mean of the control values was calculated and all subsequent recordings were expressed as a percent of control. The CEP were similarly quantified using the three principal peaks. It had previously been demonstrated that SEP were unaffected by the air pressures to be used (15), so correction factors were not required between the treatment groups.

Dogs were assigned to one of the five treatment groups according to their weight. This ensured that the group mean weights were similar (range 11.1 to 12.6 kg). All treatments were at a pressure of 5.0 bar (132 ft). Oxygen mixtures of 20, 30, 40, 50, and 60% in nitrogen were supplied to the ventilator. These gave a P_{O_2} of 1.0, 1.5, 2.0, 2.5, and 3.0 bar, respectively, at 5.0 bar. The experiments were cycled so that after each series of five treatments the next replication began one place to the right.

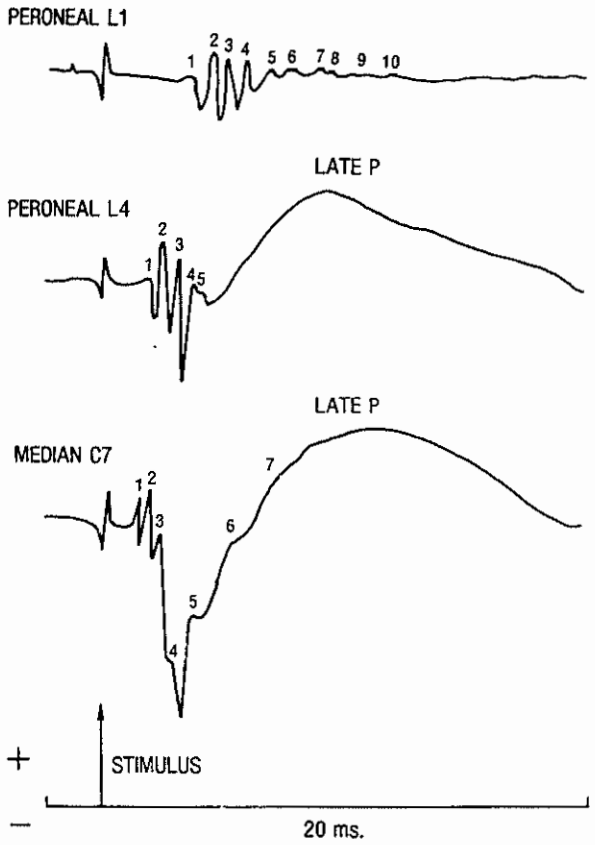

Fig. 2. Representative spinal evoked potentials. Records are labeled with the stimulus and recording sites. The two peroneal SEP show the difference between SEP near the site of root input (L4) and a more rostral site (L1). The median-C7 SEP shows the SEP at the root input. Successive traveling waves are marked in sequence at the positive peak.

Early in the preparation the hematocrit was checked. All dogs with a hematocrit below 35% were rejected. All dogs with a hematocrit above 47% were given normal saline or lactated Ringer's solution sufficient to drop the hematocrit to 42% to correct any dehydration. If excess fluid was given it was cleared and the hematocrit rose again by the time the control period was reached. The urine bag was emptied at the end of the control period.

After satisfactory control measurements were made the chamber was compressed with air to a pressure of 10.0 bar (300 ft) at a rate of 23 $m \cdot min^{-1}$. At pressure, duplicate EP recordings of the left peroneal and median nerve inputs were made. After a bottom time of 15 min (the first 4 dogs had shorter dives, 3 had 12 min and one 14 min before settling on the 15-min bottom time) the chamber was decompressed at a rate of 18 $m \cdot min^{-1}$ to 18 m and at about 14 $m \cdot min^{-1}$ from 18 m to the surface. This resulted in ascent times between 5.0 and 6.0 min. In practice the chamber was held with 0.5 m of pressure at the surface to make the ventilator oxygen dump bag vent from the chamber.

Continuous EP recordings were made after arrival at the surface. Left and right peroneal recordings were alternated on a 3- to 4-min cycle. This allowed observation

of the whole length of the cord. As soon as a minor change was observed which was seen to progress on the next recording, the diagnosis of DCS was made. Compression for treatment began 14 to 17 min after the diagnosis was made. If a lesion was identified in the cervical region during a peroneal recording the next SEP measured would be the median.

If after a 30 to 40 min surface interval no SEP changes were seen, the dog was given another dive with a bottom time of 9 to 13 min. This was necessary in 7 of the 25 dogs. When any dog with DCS developed hypotension during the surface interval this was corrected with lactated Ringer's solution. Additional fluid was given to correct the hemoconcentration commonly seen. Hypotension and hemoconcentration were usually associated with an acidosis which was corrected with 8% sodium bicarbonate. Fluid balance was recorded over the period beginning with the dive until the end of treatment.

There were six criteria used to eliminate dogs from the study group. These were:

a. A missed diagnosis resulting in the pretreatment interval being too long.
b. Insufficient loss of SEP amplitude (<10% loss).
c. Hypotension resulting in systolic blood pressure being below 100 mmHg for more than 5 min.
d. Failure to show more than 5% recovery at any stage in the treatment.
e. Any degree of spontaneous recovery during the pretreatment interval.
f. Sudden cardiovascular collapse during treatment.

Immediately before compression for treatment, a last recording was made of the SEP reflecting activity in the damaged segment of cord ("controlling SEP"). The correct gas was switched to the ventilator and the chamber compressed to 5.0 bar at 23 $m \cdot min^{-1}$. A repeat of the controlling SEP plus the other possible evoked potentials were measured on arrival at pressure. The SEP recordings were repeated at 15 min. Thereafter all SEP demonstrating lesions were checked every 15 min and the rest at 30 min until 120 min of treatment were completed. Previous work on control dogs had shown that continuous exposure to 2.8 bar of oxygen for 2 h did not affect the SEP (15), so the treatment gases were breathed continuously throughout the treatment.

The pretreatment SEP were expressed as the percent of control that was lost. If there was a further loss of SEP amplitude during compression the lower value was taken as the loss. Because the severity of the lesions was both unpredictable and uncontrollable, a normalizing procedure was applied. All recovery was expressed as a percent of what was lost. As an example, if the pretreatment SEP was 40% of control this would be a 60% loss. At the end of treatment if the SEP have recovered to 80% of control, the increase of 40% is then expressed as a percent of the original 60% loss showing a 66% recovery of the loss. This procedure did not alter the trends observed when looking at straight percentages of control, but did reduce the variance of the groups. The reported treatment data include the nearest recording to the 15, 40, 80, and 120 min points. All results were expressed as the mean $\pm$ 1 SE. The basic statistical analysis was a one-way analysis of variance.

After the experiments were terminated, the spinal cords were removed from 11 dogs and fixed in 10% phosphate buffered formalin (4% wt/vol formaldelyde). These were sectioned and stained with hematoxylin and eosin for light microscopy. The cord was cut into seven gross segments and three or more specimen sections from each segment were observed for frequency of hemorrhage. The objective was to see

whether there was any relationship between SEP recovery and the frequency of hemorrhage in the cord.

RESULTS

Evoked potentials

The CEP recorded were similar to those seen previously. Peroneal CEP P_1, N_1, and P_2 latencies were 16.9 ms (SE 0.4), 25.0 ms (SE 0.7), and 37.7 ms (SE 1.0) and the median CEP latencies were 12.8 (SE 0.2), 19.5 (SE 0.3), and 32.4 (SE 0.8) ms, respectively. Exposure to air at 10 bar generally increased the latency. The increased latencies were, respectively, for peroneal CEP 17.2 (SE 0.4), 25.6 (SE 0.7), and 38.2 (SE 1.0), and median CEP 12.9 (SE 0.2), 19.5 (SE 0.4), and 32.8 (SE 0.7) ms. Only the increases in peroneal P_1 and N_1 latencies were significant ($P < 0.001$ by paired t test).

The overall mean control amplitudes for peroneal CEP P_1 N_1 and N_1 P_2 were 33 μV (SE 4) and 46 μV (SE 6), respectively. These were both depressed by 10 bar of air to 81% (SE 3). Similarly, for the median CEP, P_1N_1 and N_1P_2 were 48 μV (SE 8) and 58 μV (SE 9). They were depressed to 75% (SE 3) and 81% (SE 4). The summated amplitudes for left and right peroneal—lumbar and for the left median—cervical SEP were not affected by 10 bar of air, being 98% (SE 2), 99% (SE 2), and 99% (SE 1) of the controls.

Conduction velocities were calculated from interelectrode distances and peak latencies. The peroneal first traveling wave had a conduction velocity between stimulus and lumbar sites of 65 $m \cdot s^{-1}$ (SE 3). In the cord, velocity between lumbar-thoracic and thoracic-cervical sites was similar at 71 $m \cdot s^{-1}$ (SE 5) and 69 $m \cdot s^{-1}$ (SE 5), respectively. The median stimulus-cervical velocity was 79 $m \cdot s^{-1}$ (SE 4). The velocities from cervical site to cortex were 18 $m \cdot s^{-1}$ (SE 1) and 16 $m \cdot s^{-1}$ (SE 1). A later identified traveling wave had a much slower conduction velocity of 36 $m \cdot s^{-1}$ (SE 2) and 32 $m \cdot s^{-1}$ (SE 1) in the stimulus to first electrode segment for peroneal and median SEP. In the peroneal SEP this was reflected by a widening interval between the two waves as they passed rostrally. At the lumbar, thoracic, and cervical sites the peak-to-peak intervals were 3.14 ms (SE 0.20), 3.86 ms (SE 0.22), and 4.38 ms (SE 0.26), respectively.

Systemic changes

The onset times of the systemic changes are shown in Table 1. The earliest change indicating DCS in this model was a reduction in EEG amplitude. The reduction ranged from about 15% to the EEG being isoelectric. Eighty percent of dogs were affected (Table 1). This occurred 1 to 16 min after surfacing, as did the commonly seen rise in CSFP, although the two events were not necessarily synchronous. Cerebrospinal fluid pressure rose to levels shown in Table 2. Two dogs that did not show a rise in CSFP did not experience a reduction in EEG amplitude. Fifteen dogs experienced a rise in RVP to as high as 120/20 mmHg (Table 2). In 8 this rise occurred later than the CSFP rise. Toward the end of the pretreatment period, the raised RVP began to fall in 20 dogs and the raised CSFP began to fall in 15 dogs. In the remainder

TABLE 1
EVENT TIMES DURING DECOMPRESSION SICKNESS

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Event						
Surfacing to D	12 ±3	15 ±3	19 ±2	17 ±3	12 ±3	1.24
D-1 to D	4 ±1	5 ±1	5 ±1	8 ±3	5 ±1	0.80
Last SEP to Tr	2 ±2	1 ±1	2 ±1	3 ±1	3 ±2	0.53
RVP ↑	9(5)	9(4)	12(3)	5(2)	5(3)	—
CSFP ↑	4(5)	6(5)	8(4)	5(4)	4(5)	—
EEG ↓	2(4)	3(4)	11(3)	4(3)	5(4)	—
$\overline{BP}$ ↑	11(2)	14(4)	−(0)	13(1)	14(2)	—
$\overline{BP}$ ↓	−(0)	18(2)	−(0)	6(1)	10(2)	—

Times in minutes shown as mean ±1 standard error. Figures in parenthesis indicate numbers 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.

it remained constant or rose further. Compression therapy caused variable changes in CSFP but generally caused RVP to stabilize at around control levels (Table 2).

During the pretreatment interval 9 dogs had an increase in systolic blood pressure of more than 20 mmHg above any prediagnosis pressure. In 3 cases this exceeded 300 mmHg. In most cases of hypertension there was a precipitate fall in pressure within a few minutes. When pressure rose rapidly and leveled off, infusion of lactated Ringer's solution was started to prevent the rebound hypotension where systolic blood pressure fell below 100 mmHg. Of the 5 dogs in the final data pool that were transiently (< 5 min) hypotensive only 3 had been hypertensive. Cerebral perfusion pressure calculated as the difference between mean blood pressure and CSFP is shown in Table 2. Heart rate generally increased at some stage before treatment, then returned to pre-DCS levels. Ventilation rate was controlled completely in most dogs. During the DCS there were occasional episodes of tachypnea attributable to "chokes."

In spite of the volumes of fluid given before treatment began, 15 dogs still had an increase in hematocrit of more than 3% (Table 3). The greatest increase seen was 16%. Overall the increase was significant ($P < 0.001$ by paired *t* test) with a mean rise of 6% (SE 1). At the pretreatment point there was a significant difference between the groups ($P < 0.05$) for both the increase and the absolute level. The group mean increases were 6, 12, 5, 0, and 4%, respectively. The urine outputs in Table 3 were from the start of the dive until the end of treatment, a period of about 3 h.

A marked respiratory acidemia occurred during the pretreatment phase (Table 4). Nine dogs had an arterial pH of less than 7.3 which was caused by an elevation in arterial P_{CO_2}. In 12 dogs Pa_{CO_2} exceeded 39 mmHg and in 4 of these Pa_{CO_2} exceeded 45 mmHg. There was no independent change in bicarbonate ion concentration coincident with this. There was however a slight hypoxemia in 12 dogs which had an

TABLE 2
CARDIOVASCULAR VARIABLES

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Mean Blood Pressure (mmHg)						
Postdive	114 ± 4	16 ± 6	126 ± 5	108 ± 4	114 ± 9	1.08
Interval range	77–220	70–257	87–150	70–152	70–147	—
Pre-Tr	89 ± 3	103 ± 14	112 ± 8	100 ± 9	92 ± 8	1.02
Change on compression	−26(5)	−18(4)	−12(4)	−17(5)	−12(4)	0.49
Tr 15 min	127 ± 11	137 ± 24	116 ± 10	113 ± 4	125 ± 9	0.53
Tr 120 min	110 ± 16	108 ± 9	118 ± 3	105 ± 10	120 ± 113	0.34
Right Ventricular Pressure (mmHg)						
Postdive	18/1	25/0	18/1	25/5	20/0	1.73
Interval range (Systolic)	6–120	20–105	14–60	18–85	16–60	—
Pre-Tr	33/7	40/4	29/1	31/6	27/0	0.92
Tr 15 min	21/3	34/1	21/1	27/5	30/1	3.23*
Tr 120 min	16/0	20/0	16/1	23/4	21/1	0.93
Cerebrospinal Fluid Pressure (mmHg)						
Postdive	11 ± 2	10 ± 3	5 ± 1	16 ± 6	13 ± 3	1.07
Interval range	5–36	1–82	2–28	2–38	4–32	—
Pre-Tr	18 ± 3	23 ± 8	12 ± 4	13 ± 4	18 ± 5	0.68
Tr 15 min	27 ± 8	20 ± 5	9 ± 3	21 ± 4	24 ± 6	1.15
Tr 120 min	19 ± 8	21 ± 8	6 ± 3	22 ± 6	18 ± 3	1.21
Cerebral Perfusion Pressure (mmHg)						
Postdive	104 ± 5	106 ± 7	120 ± 5	92 ± 7	101 ± 8	2.32
Pre-Tr	71 ± 4	79 ± 12	100 ± 7	87 ± 9	74 ± 9	2.12
Tr 15 min	101 ± 8	117 ± 22	108 ± 8	92 ± 7	102 ± 10	0.55
Tr 120 min	92 ± 16	87 ± 10	115 ± 8	83 ± 13	102 ± 13	1.01

Pressures in mmHg shown as mean ± 1 standard error. Mean blood pressure = diastolic BP + 2/3 pulse pressure. Figures in parentheses indicate numbers of dogs. *Under *F*-ratio shows significance at $P < 0.05$. Tr = treatment.

arterial P_{O_2} of less than 85 mmHg. This resolved on compression treatment with high oxygen mixtures.

It can be seen that except for CSFP all the pressures stabilized at a level a little below control values, during treatment. The hematocrit, arterial pH, and P_{CO_2} all returned to levels not greatly different from the controls. In only two instances were the groups statistically different: the pretreatment hematocrit and the RVP after 40 min of treatment ($P < 0.05$). Compression caused an at least transient drop in mean blood pressure. The 2 dogs that showed a rise in pressure were both hypotensive at the start of compression (Table 2).

Spinal evoked potentials

An example of SEP changes during an experiment is shown in Fig. 3 The first identified SEP lesions in the 25 dogs in the final data pool included, 16 left lumbar, 6

TABLE 3
HEMATOCRIT (HCT) and Fluid Balance

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Hct control (%)	46 ±1	45 ±1	43 ±2	46 ±1	45 ±1	0.98
Pre-Tr	52 ±3	57 ±3	48 ±2	46 ±1	49 ±2	3.81*
Tr 40 min	51 ±2	47 ±3	44 ±1	44 ±1	48 ±2	2.76
Tr 80 min	50 ±1	46 ±2	44 ±1	45 ±3	48 ±2	1.85
Overall fluid Balance (ml)	261 ±48	204 ±8	94 ±56	155 ±64	216 ±78	0.92
Fluid-in in Tr (ml)	230 ±89	252 ±79	196 ±38	109 ±23	194 ±47	0.57
Urine output (ml)	126 ±22	195 ±60	170 ±31	103 ±18	144 ±22	1.11

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

right lumbar, 1 right thoracic, and 2 left cervical lesions (Table 6). These SEP were used for diagnosis and assessment of recovery. The loss of SEP amplitude ranged between 15 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 4 and 25 min. There was no difference between the group means (Table 1). The extent of the change leading to diagnosis (D-1 to D) ranged between 2 and 60% (Table 5). With the exception of 1 dog in the 2.5 bar group that had a 20-min interval between the last normal and diagnostic SEP, most intervals were 4 to 5 min (Table 1). In spite of the long interval in that case the SEP loss at diagnosis was only 4%. As a check on diagnostic perception, the penultimate normal SEP (D-2) was checked against the last presumed normal SEP (D-1) and no difference between the groups was seen (Table 5), nor was any large change found. Where the time to onset was short there were 9 cases where D-2 was measured at pressure in the dive.

The progression of the SEP amplitude loss between diagnosis and start of treatment (Tr) (D to Pre Tr) is shown in Table 5. The cord lesions were not stable at the start of treatment. All SEP were continuing to deteriorate at varying rates. The interval between the last pretreatment SEP and the start of compression therapy varied between 0 and 8 min. Two dogs showed a further loss of SEP during compression. One lost another 31% and the other 8%. They respectively had a 0- and 7-min interval between the last SEP and the start of treatment.

Recovery of SEP amplitude was rapid in the first 15 min of treatment (Table 7) and ranged between 5 and 100%. Although the 2.0 bar group led the recovery at 15 min there was no significant difference between groups. In the three groups with a P_{O_2} of 2.0 bar or more, mean group recovery continued out to 40 min. In the two lower P_{O_2}

TABLE 4
ACID-BASE AND GAS ANALYSIS

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Arterial pH						
Control	7.39±0.02	7.39±0.02	7.38±0.02	7.41±0.02	7.39±0.02	0.56
Pre-Tr	7.31±0.04	7.31±0.01	7.34±0.02	7.31±0.03	7.32±0.03	0.29
Tr 40 min	7.35±0.01	7.39±0.03	7.38±0.02	7.38±0.01	7.38±0.02	0.25
Tr 80 min	7.39±0.02	7.39±0.02	7.36±0.01	7.39±0.02	7.39±0.03	0.38
Arterial P_{CO_2} (mmHg)						
Control	34±2	34±2	35±2	32±1	34±1	0.55
Pre-Tr	42±5	38±1	40±3	39±3	40±3	0.22
Tr 40 min	35±1	35±2	37±6	36±3	34±2	0.07
Tr 80 min	38±2	38±4	39±3	34±2	36±4	0.33
End Tidal P_{CO_2} (%)						
Control	3.9±0.2	3.7±0.1	4.0±0.1	3.6±0.2	4.1±0.1	1.81
Pre-Tr range	2.6–4.7	2.6–5.0	3.2–4.9	2.9–4.4	2.5–5.2	—
Pre-Tr	3.8±0.2	4.0±0.3	4.1±0.3	3.8±0.3	4.1±0.3	0.32
Tr 40 min	4.7±0.3	4.6±0.3	4.5±0.4	4.4±0.3	4.4±0.2	0.18
Tr 80 min	4.4±0.5	4.6±0.2	4.5±0.1	4.2±0.4	4.5±0.3	0.27
Arterial P_{O_2} (mmHg)						
Control	94±2	92±3	92±1	95±3	94±2	0.27
Pre-Tr	80±11	80±6	90±5	94±6	89±6	0.71

Data shown as mean ±1 standard error.

groups, deterioration occurred between 15 and 40 min (7/10 cases) and the SEP amplitude stabilized for the remainder of the 2 h of treatment. The differences between the groups at 40, 80, and 120 min were significant ($P < 0.05$) with the 2.0 and 2.5 bar oxygen treatment groups having similar amounts of recovery (70 and 66%). Two dogs, one in each of the 1.0 and 1.5 bar groups ended with no recovery, in fact with a further SEP loss, having shown 74 and 53% recovery at 15 min. The peak recovery times covered the entire treatment period. Using all the available SEP data showed the group mean peak recovery times to be 60, 32, 54, 65, and 58 min (*F* ratio 0.44). The mean SEP recovery at these times was 63, 79, 91, 80, and 58% (SE 6 to 13 and *F* ratio 1.43).

The extent of CNS involvement as far as it was possible to assess it is shown in Table 6. This includes the onset times for EEG changes as well as the pretreatment SEP amplitude loss. As traveling waves are recorded more rostrally along the cord the reducing amplitude must lead to a greater error and therefore possible inconsistency in SEP loss. However, some local activity is included in the peroneal-lumbar SEP so it would be possible to see the local loss without affecting the traveling waves rostrally as possibly shown in dog 174. If the lumbar SEP loss is total then no assessment of thoracic cord can be made. Expressing the number of affected SEP as a percent of available recordings showed the proportion of affected cord segments in each group to be about 55, 59, 56, 39, and 67%, respectively. Taking only the left and

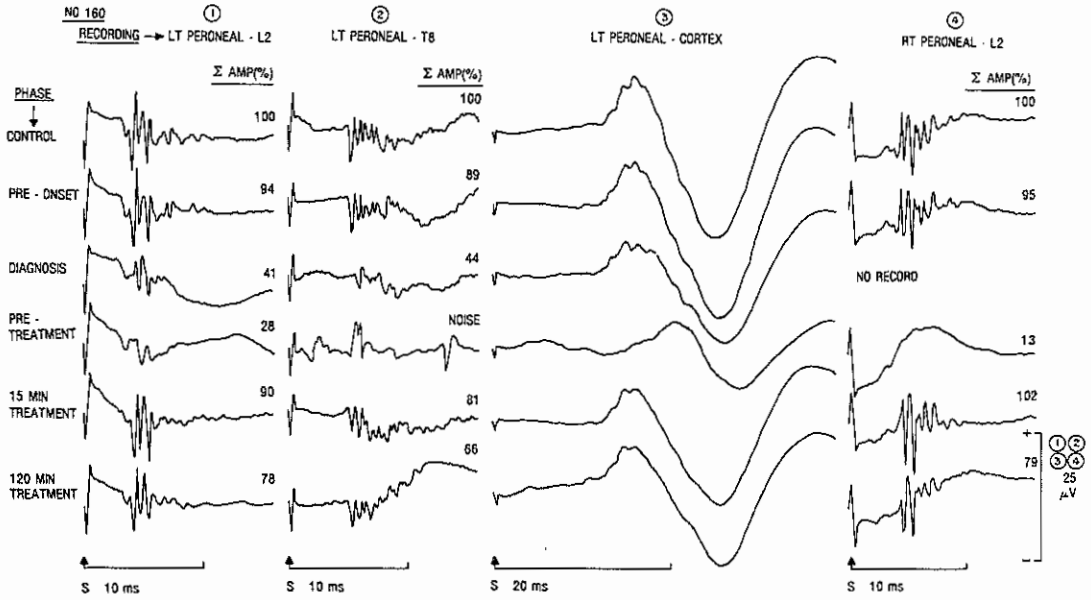

Fig. 3. Decompression sickness affecting the lumbar spinal cord. The experiment was controlled around the left lumbar SEP*(1)*. The development of DCS and its recovery through treatment are shown. The summated amplitude as a percent of control is given under Amp (%). Effect of the left lumbar lesion on the thoracic SEP and cortical evoked potential is also shown. Right lumbar cord was also involved in the DCS.

TABLE 5
CHANGES IN SPINAL EVOKED POTENTIALS

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Time Period						
D-2 to D-1	−1	−1	+3	−1	−2	0.94
	±2	±3	±5	±2	±4	
D-1 to D	−24	−11	−25	−25	−21	0.59
	±5	±4	±11	±11	±2	
D to Pre-Tr	−44	−28	−47	−41	−49	0.52
	±13	±8	±12	±15	±9	
Pre-Tr loss	67	52	69	68	65	0.52
	±11	±13	±9	±16	±11	

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.

right lumbar and the left cervical segments, those with large reliable SEP, the figures were 87, 80, 71, 53, and 60%, respectively. There was no consistent pattern of involvement related to recovery.

TABLE 6
THE EXTENT AND SEVERITY OF CNS DECOMPRESSION SICKNESS AS INDICATED BY SPINAL EVOKED POTENTIAL AMPLITUDE

Evoked Potential	LPL	LPT	LPC	RPL	RPT	RPC	LMC	EEG (time)
Dog								
140	[79]	?	86*	82*	?	100*	11	—
141	[28]*	?*	54*	11*	?*	33*	48*	1
142	[92]	100	100	90	100*	100*	65	4
143	[75]	100	100	20*	25	55	0	1
144	19	23	49	[62]	22*	37*	0	3
150	[18]	?*	?	14	?	100	0	—
151	[61]	47	57	47*	?	40*	0	3
152	17*	?*	0*	[29]*	26*	27	0	4
153	62	100	100	59	100	100	[89]	1
154	8*	?*	32*	41*	41*	100	[63]	6
160	[72]	56	?	86	?	?	0	—
161	59	?	?	[71]	?	?	?	—
162	0	0	0	[49]	31	48	0	16
163	[100]	?	?	100	?	?	0	7
164	[54]	100	100	66	100	100	58	11
170	?	38	64	11	[83]	100	27	3
171	[100]	100	100	100	100	?	0	—
172	[91]	80	100	81	100	100	0	6
173	[53]	36	?	0	0	?	0	—
174	[15]	0	?	0	?	?	0	2
180	[58]	78	100	0	0	0	33	1
181	68	70	100	[77]	64	?	0	—
182	[44]	28*	43	0	23	30	0	13
183	[100]	100	100	84	77	53	0	3
184	86	100*	100*+	[97]	?*	100*+	0+	3

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, LPT, LPC = left peroneal lumbar, thoracic, and cervical, 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.

Discarded data

Eight dogs were lost for various technical reasons. Another 4 completed treatment without any recovery of SEP at any time. Of these, 2 had a missed diagnosis which led to a pretreatment phase of more than 35 min. One experienced 9 min of profound hypotension (systolic BP 70 mmHg). The other developed an uncontrollable respiratory and acid-base problem which resulted in 80 min with a pH between 7.20 and 7.29 and a Pa_{CO_2} of 80 mmHg. These dogs were replaced in the data pool.

Pathology

A few petechial subarachnoid hemorrhages were seen in some dogs when the cords were removed. A major subarachnoid hemorrhage was present in the lumbar-thoracic cord of dog 184. There was poor SEP recovery in this case with a slow improvement reaching 12% at 120 min.

Many cord segments showed varying degrees of hemorrhage and occasional vascular congestion. Microscopic petechiae appeared in either or both gray and white matter. Most appeared in the central gray matter. The hemorrhages consisted of small clusters of red cells surrounding capillaries and venules to larger though still microscopic foci of bleeding. The appearance was compatible with hypoxia or embolic episodes. There was no other evident tissue damage, inflammatory infiltration, or edema. There was no detectable relationship between frequency or severity of hemorrhage and extent of SEP loss, its recovery, or subsequent deterioration. However, the one case (174) with the least SEP loss and the least cord involvement (left lumbar only) also had the smallest number of hemorrhages.

DISCUSSION

At the start of treatment the physiological condition of the five groups of dogs was similar except for a higher mean hematocrit in the 1.5 bar group. All groups had cord DCS of similar severity and similar development. During treatment a large proportion of the mean SEP recovery had occurred in all groups by 15 min. This was not sustained in the 1.0 and 1.5 bar groups. The higher P_{O_2} groups continued recovery until 40 min into treatment before stabilizing at a lower level between 40 and 120 min. Throughout the later part of treatment there was a significant difference between the groups with the 2.0 and 2.5 bar groups showing the best mean recovery of 70 and 66%, respectively.

The electrophysiological preparation performed within previously known limits. The CEP latencies and cord conduction velocities were similar to those reported elsewhere (19–21,). The narcotic effects of air on both CEP and SEP were the same as those previously seen in the same model (15).

The choice of 15 min for the interval between diagnosis and the start of treatment seems to have been correct. It resulted in only 4 dogs achieving a complete recovery although 2 of these failed to sustain it. The preliminary studies showed that complete recovery was unlikely after total ischemia lasting 15 min (18). The absolute level of recovery may have been enhanced by the use of pentobarbital anesthesia which suppresses neural metabolism, thus reducing oxygen demand (22, 23). As this was common to all treatments it cannot be considered to have influenced the outcome of the experiment. The range of P_{O_2} between 1.0 and 3.0 bar was selected because oxygen partial pressures outside this range were unlikely to be of practical use. The pressure of 5.0 bar allowed the use of air to provide the lowest P_{O_2} and fell in the middle of the range of treatment pressures in common use. There was a risk, if pressure were more effective than oxygen in treating DCS, that this pressure could mask any oxygen effect. This did not occur.

The physiological observations largely support those of Hallenbeck et al. (13, 24) as to the mechanism of cord DCS. All but 2 dogs showed a rise in CSFP compatible

with epidural vertebral venous system obstruction. This mechanism cannot be applied to cerebral DCS. Cerebral DCS occurred in 18 dogs, presenting as a loss of EEG amplitude. There were 4 cases where a rise in RVP preceded the loss of EEG amplitude. This indication of pulmonary vascular obstruction by bubbles points to the possibility of bubble break-through leading to arterial gas embolism (25). The presence of respiratory acidosis with hypoxemia also indicated pulmonary vascular obstruction. The remaining 14 dogs presumably had autochthonous bubble formation. The brain is a well-perfused organ with most of the blood flow in the gray matter, as is the cord. The high lipid content of the less vascular white matter would, with its higher nitrogen content, predispose to a greater risk of autochthonous bubble formation.

There was a strong indication of peripheral vascular obstruction by gas in that 22 dogs (88%) showed a transient step reduction in mean blood pressure during the compression phase of treatment. Compression of intra- or extravascular obstructing gas could lead to a sudden drop in peripheral resistance and a transient fall in blood pressure. There was no apparent reason why the SEP of 2 dogs should have deteriorated transiently on compression. The finding is similar to occasional clinical experience. It might result from vessel collapse on compressed bubbles or gas redistribution transiently worsening a local problem. No association with higher oxygen pressures occurred that would implicate oxygen uptake in bubbles, causing them to expand.

If the efficacy of specific treatments and speed of treatment are disregarded two major factors may govern the extent of recovery; first the severity and extent of cord involvement and second, aspects of systemic disturbance which will influence microcirculatory perfusion, such as perfusion pressure, hematocrit, and acid-base state.

When monitoring function using EP it is possible to obtain a crude estimate of the extent of cord involvement by differentiating between left and right and by having several interrogation points. To improve the localization, SEP from intercostal stimulation were made, but the technique was not sufficiently reliable to be used in this study. There were 5 cases of unilateral lumbar SEP loss. The median—cervical SEP showed the left cervical cord to be unaffected in 16 cases where the lumbar cord was affected. Separating the cases into those showing generalized cord involvement and those only "locally' affected gave respective mean recoveries at 120 min of 41 and 50% (t test $0.1 > P > 0.05$). This could have influenced the treatment group means as only 3 of the 8 cases affecting cervical cord occurred in the three high oxygen groups (Table 7). There was a smaller difference when unilaterally and bilaterally affected lumbar SEP were compared ($P > 0.6$). Within the treatment groups means there was no difference between the losses in the primary SEP. When all the primary SEP losses were divided between generalized and local cord DCS the SEP losses were the same at 68 and 65%, respectively. Severity of SEP loss therefore does not seem to be related to extent of cord involvement, but greater extent of injury may reduce recovery.

Although there was no significant difference in mean onset times between groups, the two best groups had longer onset times than the remainder. There was no relationship between onset time of SEP loss and the amount lost. However there was a relationship between onset time and amount of recovery. The least squares regression equation was:

TABLE 7
SPINAL EVOKED POTENTIALS RECOVERY AS PERCENT OF LOSS

P_{O_2} (bar)	1.0	1.5	2.0	2.5	3.0	*F* ratio
Time Period						
15 min	42 ±13	55 ±15	70 ±11	52 ±11	44 ±11	0.84
40 min	29 ±10	33 ±10	79 ±9	69 ±12	53 ±13	3.92*
80 min	29 ±14	30 ±11	68 ±6	71 ±11	39 ±8	3.79*
120 min	22 ±10	34 ±10	70 ±12	66 ±14	42 ±8	3.53*

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

$$Y = 12.92 + 2.18\,t$$

where Y was the percent recovered and t the onset time in minutes ($R = 0.48$, $P < 0.02$). Separating recovery into those with less than 40% and those with more than 60% gave mean onset times of 13.5 and 19.0 min (t test $P < 0.001$). Thus shorter time to onset may indicate a poorer chance of recovery. There was no difference between these two groupings for percent of SEP loss, 59 and 62%, respectively, so recovery was not related to severity.

What mechanisms could be related to speed of onset and a reduced recovery? No trend of more severe or frequent incidence of hemorrhage was indicated by microscopy. No particular systemic disturbance was notably associated with poor recovery. Discussion must then be speculative and relate to the possibility of some effect at a cellular level such as autochthonous bubble formation. There is a lag time of about 5 min between onset of ischemia and loss of SEP (18), but neuron compression as might be caused by autochthonous bubble formation would cause sudden SEP loss. In view of the number of dogs in which such an event may have occurred in the brain this becomes a strong possibility in the cord. The question of the relative damage caused by autochthonous bubbles vs. vascular obstruction arises. If hemorrhage does not occur with vascular obstruction then structural integrity is probably preserved. Recovery then becomes a matter of the duration and degree of ischemia. Function will cease below defined thresholds of blood flow, the so called "ischemic penumbra" (23). However, this does not cause cell death until blood flow falls further. Treatment of the totally ischemic cord must begin early to ensure complete recovery in cases not complicated by hemorrhage or autochthonous bubble formation. The maximum survivable period of ischemia for cord tissue is probably about 15 to 20 min (18). Successful late treatments suggest that the cord was not totally ischemic but was in the "ischemic penumbra." Transient recovery may indicate that oxygenation is sufficiently improved to restore function but that small reductions in perfusion or oxygen can revert tissue to a nonfunctioning state.

Intuitively, it seems likely that autochthonous bubble formation in neural tissue must disrupt neural integrity. If it damages cells or axons then recovery is unlikely. However, if the damage was confined to myelin then the possibility of repairable damage might exist. Such a proposition would allow for some of the long-delayed recovery now largely attributed to adaptation (18). Clearly, severe myelin disruption would damage axons thus causing permanent loss.

The fall in blood pressure and hemoconcentration reflects the severity of systemic disturbance. Fluid infusion is the essential method for correcting the hemoconcentration (26); this incidentally corrects the blood pressure. The fluid supplements over the period following onset of DCS were similar in each group but failed to completely correct the hematocrit in the 1.0 bar group. A markedly elevated hematocrit with the consequently increased viscosity must impair tissue perfusion.

Interpretation of the findings in the spinal cord sections was confounded by the history preceding their fixation. While changes could be attributed to the initial decompression insult and might be modified by the subsequent compression treatment, the fact of their final decompression also subjected them to a second insult. Two hours of air breathing at 5 bar (1.0 bar P_{O_2} group) could result in DCS, but breathing 60% oxygen (3.0 bar P_{O_2} group) makes DCS less likely. Therefore, without knowing the event that caused the lesions, no differentiation between groups would be valid because of the varied risk of DCS at the end.

Although systemically the presentation of DCS did not conflict with the venous obstruction model of Hallenbeck et al. (13), the frequency of cerebral involvement and the dominance of cord gray matter hemorrhages are at variance with that model. This reinforces the probability that arterial and autochthonous bubbles have more than an incidental role in the pathology of cord DCS in this model. Cockett et al. (26) reported examples both with and without gray matter sparing. Palmer et al. (27) saw extensive gray matter hemorrhages associated with white matter infarction in goat cords. The question then arises as to whether the different microscopic appearances represent cord DCS arising from different dive profiles and therefore of different severities.

There was an indication of brainstem involvement in possibly 9 cases. Such lesions could arise from any one of three possible mechanisms, arterial gas emboli, venous obstruction, or authochthonous bubble formation. The transient hypertension which in 3 cases reached very high levels could have been caused by a mechanism similar to that described in cats receiving arterial gas emboli to the brainstem as described by Evans et al. (28). Lesser rises may also result from increased peripheral resistance caused by vascular obstruction. The subsequent hypotension seen in some of these and other dogs may reflect a loss of peripheral sympathetic tone in association with the cord DCS or a reduction in cardiac output. Several cases showed ECG changes compatible with ischemia.

Having demonstrated a difference in efficacy for a range of oxygen partial pressures all at the same absolute pressure, it is necessary to attribute the difference to some property of the oxygen. As there were few systemic differences between the groups, only the nature of SEP recovery remains. The acute response to treatment suggested an optimum treatment of 2.0 bar P_{O_2}. Pressure was beneficial but a P_{O_2} between 1.0 and 1.5 bar did not produce much additional recovery, 1 and 2 dogs, respectively, showed further notable recovery with those treatments. The initial poor response could be attributable to inadequate tissue oxygenation because physically all groups

received the same compression. In addition, inert gas clearance would be slower in the low oxygen groups. These two factors could lead to a longer early hypoxic period which would lead to further cell death thus reducing longer term recovery.

At the higher P_{O_2} of 2.5 and 3.0 bar there would be vasoconstriction (29) even in the damaged tissue (12). While the increased inert gas gradient would hasten the clearance of bubbles, the vasoconstriction with reduced flow would retard bubble clearance and restoration of flow. However, the extended capacity for oxygen diffusion which probably meets the greatest need (23) would preserve hypoxic tissues for some time enabling them to recover later when perfusion was restored. Certainly 5 and 4 dogs, respectively, in the 2.5 and 3.0 bar groups showed further improvement during treatments.

Continuous exposure to high partial pressures of oxygen will lead to oxygen toxicity. If this were having a detrimental effect on the recovery of damaged cord tissue it should show as an increased deterioration in the high oxygen groups as exposure continued. The mean deteriorations for the 1.5 to 3.0 bar groups were: 11, 13, 11, and 11%, respectively (SE 2–5). The mean for the 1.0 bar group was distorted by 1 case with a 58% deterioration, otherwise the mean would have been the same as the others. Therefore there was no indication of oxygen toxicity.

As far as possible the physiological variables that could influence recovery were controlled. However, there was a tendency for factors such as blood pressure and thus cerebral perfusion pressure, hematocrit, and arterial P_{O_2} during the pretreatment and early treatment groups. The pretreatment group mean hematocrit was the only relevant variable significantly different between treatments. However, neither group means nor individual hematocrits correlated with recovery at 15 or 120 min [recovery at 120 min = 163.4–2.3 Hct with $R = 0.24$ ($P > 0.1$)]. With the exception of pretreatment, hematocrit analysis of covariance using various combinations of the physiological variables failed to alter the level of significance of between group differences for SEP recovery. Pretreatment hematocrit reduced the level of significance to $0.05 < P < 0.1$. It may be that the therapeutic measures taken during the surface interval and early treatment plus the exclusion criteria were effective in removing any clear association with lack of recovery.

If vascular obstruction is a major mechanism in cord DCS then a high perfusion pressure should be beneficial in treatment. However, in the normal cord, blood flow is constant within a range of mean blood pressure of 45 to 135 mmHg (30). Precisely how this affects focally damaged cord remains unclear. In stroke it is believed that hypotension should be avoided in order to maintain perfusion in the marginal areas (23).

Other factors play a part in secondary deterioration. Arterial air emboli cause endothelial damage in less than 3 min with resultant fluid and macromolecule leakage causing vasogenic edema (29). If the surrounding tissue has been ischemic sufficiently long then cytotoxic edema will also develop. When flow is restored within 60 min, cytotoxic edema lessens only to be replaced by vasogenic edema (31). The variable deterioration between cases may reflect such factors as the amount of intravascular gas and possibly the duration of its presence.

A second possibility is the development of delayed postischemic microcirculatory impairment. This arises from blood-damaged tissue interaction (32), and the products of blood-bubble interactions (33). Flow is initially restored but is then reduced as a form of local coagulopathy develops (32).

Either of these mechanisms may have caused the secondary deterioration, which exceeded what would have been caused by time alone in the model (15). There is however no indication that any of the treatments had a better preventive action than any other. It is noteworthy that the 5 dogs that did not deteriorate significantly were breathing 2.0 or more bar of oxygen. The main advantages of 2.0 to 2.5 bar of oxygen in treating spinal cord DCS appear to have been effective in the early stages of treatment. This implies that tissue oxygenation is probably the mainstay of oxygen therapy in the initial treatment of DCS. The next study will check whether altering the ambient pressure influences the efficacy of the lowest useful P_{O_2} of 2.0 bar.

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Leitch DR, Hallenbeck JM. L'oxygène dans le traitement de la maladie de décompression de la moëlle épinière. Undersea Biomed Res 1985; 12(3): 269–289.—Vingt-cinq chiens anesthésiés furent utilisés pour trouver la P_{O_2} optimale pour le traitement différé de la maladie de décompression (DCS) de la moëlle épinière. Ils furent équipés pour la mesure des variables physiologiques et des potentiels évoqués somatosensoriels (SEP), puis soumis à une plongée à l'air de 15 min à 10 bars (300 pieds) et décomprimés en moins de 6 min. Après le retour à la surface, les SEPs furent observés pour les signes de DCS. Quinze minutes après l'apparition de signes de DCS dans les SEPs, les chiens furent comprimés à 5.0 bars en respirant l'un de 5 mélanges gazeux donnant une P_{O_2} de 1.0, 1.5, 2.0, 2.5, ou 3.0 bars. Au début de la thérapie, tous les groupes étaient dans un état physiologique semblable avec une perte similaire du SEP. Entre 40 et 120 min, le recouvrement était significativement différent ($P < 0.05$) entre les groupes, la plus grande partie du rétablissement du SEP ayant survenu à l'intérieur de 15 min. Les traitements finirent avec 22, 32, 70, 66, et 42% de recouvrement, respectivement. Il apparaîtrait que la P_{O_2} optimale se situe autour de 2.0 bars.

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

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