Spinal decompression sickness: mechanical studies and a model

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Hills BA, James PB. Spinal decompression sickness: mechanical studies and a model. Undersea Biomed Res 1982; 9(3): 185–201—Six experimental investigations of various mechanical aspects of the spinal cord are described relevant to its injury by gas deposited from solution by decompression. These show appreciable resistances to gas pockets dissipating by tracking along tissue boundaries or distending tissue, the back pressure often exceeding the probable blood perfusion pressure—particularly in the watershed zones. This leads to a simple mechanical model of spinal decompression sickness based on the vascular "waterfall" that is consistent with the pathology, the major quantitative aspects, and the symptomatology—especially the reversibility with recompression that is so difficult to explain by an embolic mechanism. The hypothesis is that autochthonous gas separating from solution in the spinal cord can reach sufficient local pressure to exceed the perfusion pressure and thus occlude blood flow.

spinal cord
autochthonous gas
spinal decompression sickness

This study has been undertaken in two parts: a review of the clinical features of spinal cord decompression sickness (DCS) followed by a review of the proposed mechanisms and an experimental evaluation of several mechanical aspects of the spinal cord relevant to this disorder.

Clinical features

Boycott et al. (1) first observed that in deep air diving neurological symptoms commonly present before other forms of decompression sickness. Commercial air-diving experience in the southern sector of the North Sea (2), together with further experimental work (3, 4), has tended to confirm this finding. Heliox diving, by comparison, does not follow this tendency.

In 1938, following a series of deep, surface-orientated helium and oxygen dives in the U.S. Navy, Behnke and Yarbrough (5) commented on the rarity of unconsciousness and paralysis. They related the difference in incidence between equivalent air and heliox dives to the greater solubility of nitrogen in the lipids of the nervous system, especially in the large amount of white matter of the spinal cord.

Probably the largest series of cases of spinal cord decompression sickness was reported in 1909 by Blick (6). In more than 200 cases arising as a result of pearl diving off the northern coast of Western Australia there were 60 deaths, most of which occurred as the vessels were returning to port. At postmortem examination, Blick found that the deaths were associated with a gross macroscopic disruption of the cord in the lower cervical area. He described the section at this level as showing a characteristic teased appearance, as though the surface had been stippled with a needle. Often he found associated hemorrhages that extended transversely across the cord, while the deaths were attributed to respiratory impairment. In the survivors, Blick found that paraplegia was the dominant presentation, with many of the divers making an apparently full recovery from several episodes without recompression. Clinically he recorded the salient features as paralysis, usually of bilateral distribution, with loss of micturition, and he noted that the sensory nerves seemed to suffer much less than the motor. He observed that most divers carried a bladder catheter because of the frequency with which micturition was impaired following decompression.

The tendency for paraplegia to occur in preference to other CNS symptoms in both caisson workers and divers had been on record since the work of Paul Bert (7), while in pathological studies Heller et al. (8) have demonstrated that the white matter of the cord is principally affected.

Angiographic studies of the cord by Hassler (9) have shown an arterial architecture in which we feel that the lateral and posterior columns are particularly vulnerable to gas formation on decompression, when a high nitrogen loading has resulted from a deep air dive. This feature is especially apparent when the cord is sectioned longitudinally. Segments of the lower thoracic cord represent watershed areas of the spinal cord circulation—both laterally and vertically. It is singularly unfortunate in deep air diving that these areas of the cord with the highest ratio of white to grey matter and, hence, the greatest nitrogen content, have the poorest blood supply.

Mechanisms for spinal decompression sickness

Despite recording the formation of the gas in the cord as extravascular, Boycott et al. (1) supported the embolic mechanism first suggested by Bert (7). They believed that bubbles released from the lung lodged in the white matter of the cord, enlarging in situ with nitrogen derived from the tissue, although they also included the possibility of direct mechanical damage. Indeed, in a photomicrograph in the original Admiralty Report (10) they show gas distending a myelin sheath in the sections of the cord of a goat dying under pressure—a finding confirmed in later pathological studies (11). Even so, until quite recently it was generally accepted that spinal DCS was caused by arterial bubbles and considered as a part of one general category of neurological or Type II symptoms with a common physiological mediation of the basic insult.

It was then pointed out by Hallenbeck et al. (12) that spinal symptoms exceed cerebral symptoms of neurological DCS by about 3:1, and yet the brain receives some 75–85 times more blood flow than the spinal cord (13) and should therefore receive proportionally more arterial bubbles. They support this position by pointing out that in clinical disorders involving known systemic embolization, such as subacute bacterial endocarditis or fat embolism, the brain is the major target organ, spinal cord involvement being of the order of 0.4% in these diseases (14). Indeed, the cord is only listed in some neurological texts as a target organ because of the assumed role of arterial bubbles in decompression accidents, so that any reference to those in this discussion would involve circular reasoning.

Hallenbeck et al. (12) have therefore looked for a site of occlusion other than in the arterial system and have presented pathological evidence to support their contention that cord lesions arise by virtue of obstruction of the vertebral venous system by bubbles, platelet aggregates, and other products of blood-bubble interaction. They do, indeed, show strong evidence for the presence of the degradation products of blood-bubble interaction in the vertebral venous lakes of dogs decompressed from an exposure that causes spinal symptoms. However, it is difficult to tell whether such products are cause or effect. It can be argued that stasis of the circulation of the spinal cord by whatever mechanism could potentiate bubble formation in vertebral venous lakes and, hence, the products of the known interaction of any bubble with blood (15). It is also difficult to envisage how lakes drained by so many venous outlets, each devoid of the constrictive influence of a valve common in other venous systems, could be occluded to the point of causing dysfunction of the cord. Such a compromise of flow should cause dysfunction of the whole area draining into that lake, so that it becomes hard to explain the preponderance (5) of lesions in the white matter alone by this mechanism. Moreover, the current proponents of arterial embolism (16, 17) have studied the lesions in the spinal cord exhaustively and are convinced that the pathological findings are consistent with compromised arterial blood flow. In at least one of these studies (17), however, it is difficult to ascertain that the same lesions could not have been caused by compromised venous flow.

In the "vertebral venous" model (12) it has been proposed that obstruction is caused by both bubbles and the products of blood-bubble interaction that are incompressible and therefore offer a convenient explanation for the odd case of spinal DCS that is not relieved by recompression. Most cases, however, are resolved by pressurization—a fact compatible with both the "vertebral venous" and "arterial embolism" models if it were not for one further feature of recompression. This is the observation that when a diver is returned to normal atmospheric pressure soon after a compression affording complete relief from spinal DCS—as has occurred by accident or for operational reasons—the spinal symptoms return with the same distribution and intensity as before; i.e., symptoms are readily reversible in toto (18). It is very difficult to explain this basic fact by any embolic mechanism, since arterial emboli are known to be cleared when blood flow is restored by recompression (19, 20).

These arguments closely follow those already expounded for limb bends (21) and raise the question of how gas separated from solution in the spinal cord can compromise arterial blood flow reversibly, depending on its volume as determined by pressure in accordance with Boyle's law.

One possibility that has been introduced in speculation (22) is that the offending gas is located in extravascular sites and may cut off blood flow by pressing on a vessel in a manner not unlike that proposed for limb bends (local or Type I DCS), where bubbles probably elicit pain simply by pressing on a nerve ending (21). This introduces the "waterfall" concept of blood flow widely accepted in physiology since it was realized that the Starling resistor in the heart can be applied to many other organs such as the lung (23) or long bones (24) or in any situation where it is difficult to interpret blood flow according to the arteriovenous pressure difference. In these cases flow is determined by the difference between arterial pressure and the pressure of the fluid or gas surrounding that vessel and, hence, trying to collapse it. If extravascular gas were providing this potential closing pressure, this would offer a very simple explanation for spinal DCS consistent with both the pathological findings and the basic clinical features of spinal DCS. The vital question, however, is whether the gas separated from solution in the spinal cord has the capability of generating a local extravascular pressure equal to the perfusion pressure. In the cord this would be easiest to attain in the watershed zones (T4 and L1) and may explain why these areas are almost invariably implicated in neurological examinations of divers with spinal symptoms. Since previous mechanical studies have been primarily directed

at the effects of traumatic injury, this study is designed to assess the mechanical effects of a gaseous insult.

MATERIALS AND METHODS

Principle

From direct measurement and calculations based on the solubilities of gases it is a relatively simple matter to estimate the volume of gas separating from solution in a spinal cord upon decompression (or, at least, to estimate its order of magnitude). The vital question concerns whether this gas is sufficiently encased to generate a pressure that can indent an arteriole—which is the caliber of arterial vessels penetrating the pia mater (25)—or whether its potential pressure will be dissipated because the surrounding tissue is too compliant or allows the gas to track along structural boundaries, separating adjacent tissues in the process.

This problem can be broken down into several specific questions that can be addressed individually by experiment:

1. Does gas injected into the spinal cord dissipate or can it offer a back pressure by capillarity or by some other means? If by capillarity, then does fluid show the same back pressure resisting dissipation?
2. How compliant are the various membranes surrounding the cord to axial and circumferential stretch?
3. What are the pressure-volume characteristics of a section of cord ligated in situ when injected with air?
4. What are the mechanical factors, if any, imposed by substructures such as the myelin sheath in restraining nitrogen separated from solution in myelin from contributing to overall tissue pressure as transmitted to the arteriole wall either directly or via extracellular fluid as a hydraulic medium? In this case gas must have been formed by decompression for its location within the relevant structures to be ensured.

Materials

The source of all material was 10 mongrel dogs of both sexes used in pulmonary studies and killed by administering excessive pentobarbital. Histological examination of random cord sections showed no detectable abnormality in the material used. For the sake of comparing data from different experiments, all materials used in these studies were taken from the cord from L1 to L5 or samples thereof.

Preliminary capillarity studies

Gas deposited in the spinal cord by decompression is usually observed in sites that are both extravascular and extracellular (1). If this gas were retained in its site of formation, it would build up an appreciable local pressure as discussed for limb bends (21). In practice, however, this pressure can be dissipated by distortion or distension of adjacent tissue or by gas displacement of the extracellular fluid. If, for the moment, we consider only the latter—as though the adjacent nerve fibers were rigid—then the force resisting dissipation of the gas and its local pressure would be provided by capillarity and, hence, by surface tension.

The physics relating the pressure head (ΔP) with which fluid rises in a hollow tube of radius (r) is expressed by the well-known Laplace equation for capillary rise, viz.,

$$\Delta P = 2\gamma/r \qquad (1)$$

where γ is the surface tension. This equation also expresses the pressure (ΔP) with which fluid in the hollow tube would resist its displacement by air if the tube were now horizontal. However, no such simple relationship could be found relating ΔP to γ and r for the cases of capillarity of fluid outside of solid tubes as would seem more appropriate to the situation in the spinal cord.

A preliminary study was therefore undertaken to relate the rise of fluid within the interstices formed between closely packed glass rods to the surface tension (γ) and diameter of the rods (D). When the bundle is immersed in the fluid with all rods vertical, the fluid rises to the top of the rods in the regions where they touch. The unusually shaped meniscus rises to a minimum height above the level of fluid in the container at the center of the interstice. Analogous to the case of the hollow tube, this minimum height represents the pressure difference (ΔP) that the gas would need to exert in order to displace that fluid from the interstitial space between the rods if they were horizontal and, therefore, enables a prediction to be made of the force of capillarity resisting gas tracking between rigid cylinders. Hence ΔP has been measured for a series of 6 sets of glass rods of widely differing diameter (7, 8, 10, 12.5, 17, and 25 mm) and for two liquids of different surface tension, viz., water (72 dyn/cm) and aqueous ethyl alcohol (45 dyn/cm). Surface tensions were measured using a Wilhelmy balance (Kimray-Greenfield surfactometer).

While the above experiment has demonstrated the basic principles of interstitial capillarity, nerve fibers cannot be assumed to be rigid. Their distortion by pressure would be expected to modify the surface forces studied above, leading to the next study involving the spinal cord itself.

Pressure dissipation experiments

The sacrificed dog was laid in the supine position and deep cuts made between the vertebrae and adjacent tissue to leave an intact segment from L1 to L5 inclusive. The cord in this segment also remained intact with respect to nerve roots, vessels, ligaments, and other tissue, such that the only means of escape for fluid injected into one end of the segment and not retained by tissue distension was the other (open) end. An 18-gauge needle was inserted into the end of the segment at L5 in the cephalad direction and tied off by a suture, the other end remaining open to the atmosphere. Saline or air was then injected in known volumes and the pressure adjusted until flow stopped. This enabled the relationship between pressure and injected volume to be recorded following widely differing boluses of saline or air.

Preliminary compliance studies

The cord from L1 to L5 inclusive was excised by using a hollow cork borer closely fitting the spinal foramen to cut all nerve roots, vessels, ligaments, and other connections between the dura mater and the lining of the vertebrae. The dura and arachnoid membranes were then peeled off the excised cord together and essentially intact except for holes where nerve roots had been severed during excision.

They were tied with sutures at both ends and suspended from one end while weights were added to the other and the elongation measured using a traveling microscope (see Fig. 1*a*). This simple procedure enabled the force-elongation relationship to be determined for the membranes derived from the same cord sample that was used intact in other experiments of this study. Circumferential compliance was also measured by inserting parallel rigid wires into a 2-cm sample and pulling them apart with different forces as shown in Fig. 1*b*.

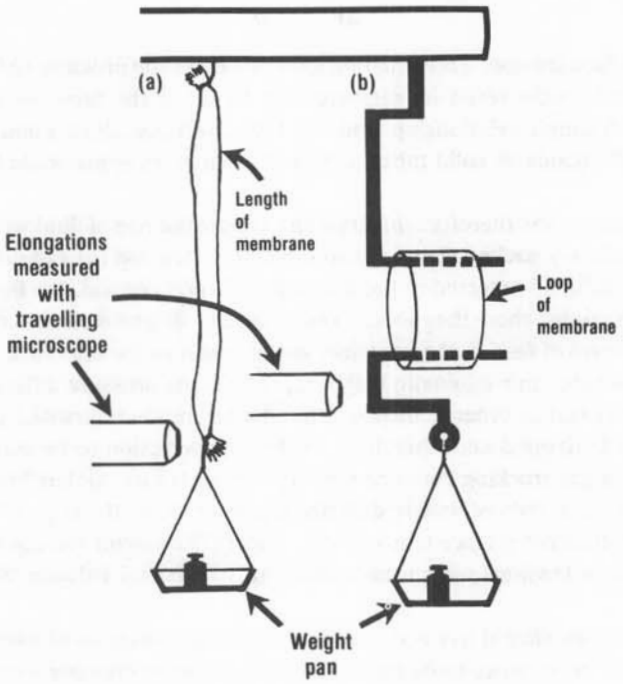

Fig. 1. Apparatus used to measure compliance of the dura and other adhering membranes excised from dog spinal cord (*a*) in the longitudinal direction and (*b*) circumferentially.

Pressure-volume relationships

Since it is debatable whether the spinal cord is effectively open- or closed-ended from a pressure-volume standpoint, the first experiment—i.e., with the intact cord between L1 and L5 lying in situ—was repeated, but with one exception. The end distal to the inserted needle was also tied off so that saline or air could not escape. The relationship between pressure and injected volume was then determined, maintaining the whole animal at 37°C.

Decompression experiments

The inner cord remaining after removing the dura and arachnoid membranes used in the second experiment was placed in a pressure chamber and compressed to 6 ATA with air and left for 48 h or 72 h—at 0°C to minimize tissue degradation and to increase the solubilities of nitrogen and oxygen during gas uptake. It was then decompressed to atmospheric pressure, warmed to 20°C ± 1°C, immersed in bubble-free and degassed saline, and transferred to a dilatometer, which was then placed in a pressure chamber (see Fig. 2). Transient pressure excursions were made to various absolute pressures (P) and the volume change (ΔV) recorded. Allowance was made for any gas starting to dissolve as a result of the pressurization by plotting the volume change against time and extrapolating the curve back to zero time in order to obtain the true value of ΔV. Since the gas itself must obey the ideal gas laws, any apparent deviation can be attributed to the pressure contributed by the restraining action of the encasing membranes and the surface tension of any fluid lining. If this contribution is p and there are n moles

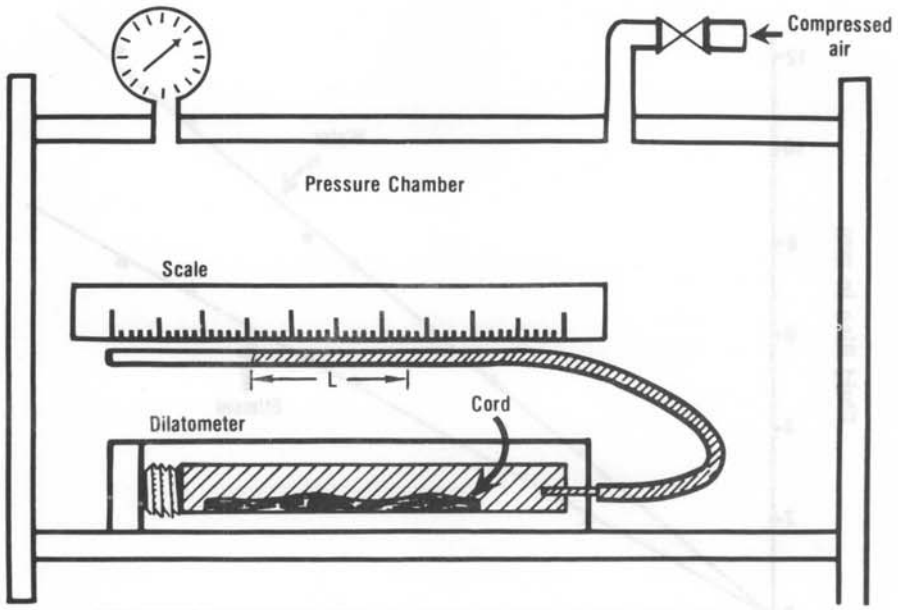

Fig. 2. Layout of dilatometer in which decompressed cord is placed surrounded by bubble-free saline. Chamber is used to pressurize the whole unit, thus transmitting, via open end of tube, pressure needed to compress the gas enclosed within the cord, whose volume is measured by position (L) of meniscus.

of dry gas separated from solution at normal pressure and an absolute temperature T, then the universal gas equation can be applied to the dry gas as:

$$(P - P_w + p)V = nRT \qquad (2)$$

where P_w is the vapor pressure of water at T and R is the universal gas constant (0.0821 liter-atm · mole $^{-1}$ · °C^{-1}). The absolute volume of gas can be determined from the dilatometer reading (ΔV) by standard algebraic procedures applied to compression dilatometry (25) to enable the tissue restraining pressure to be determined as a function of absolute gas volume (V). These are confirmed by measuring ΔV for changing absolute temperature (T) rather than pressure and, again, allowing for any gas dissolving by extrapolating ΔV back to zero time.

RESULTS

Capillarity experiments in vitro

The preliminary experiment using the different bundles of closely packed glass rods immersed in liquid showed that the fluid rise (ΔP) was greater for smaller diameter (D) of the rods and for fluid of higher surface tension (γ). The results are plotted in Fig. 3, from which it can be seen that the fluid rise ΔP is 1) directly proportional to γ; and 2) inversely proportional to D, to give the relationship:

$$\Delta P = K\gamma/D \qquad (3)$$

where K is the proportionality constant which is dimensionless and has a value calculated from the gradients in Fig. 3 of 11.3. This was confirmed in six runs.

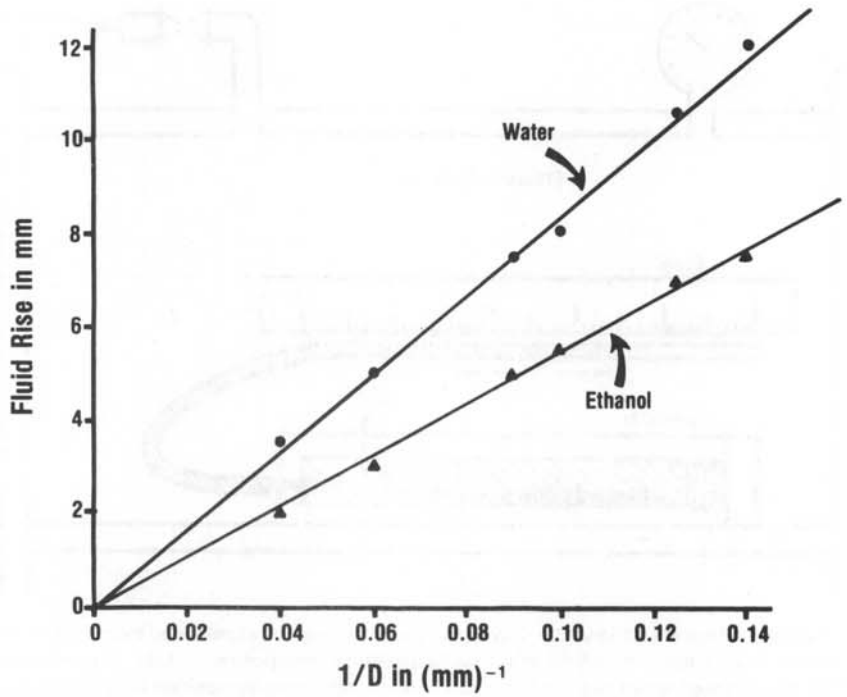

Fig. 3. Rise of lowest point of meniscus formed between closely packed solid glass rods of different diameter (D) when their ends are immersed in liquids of different surface tension, water or aqueous ethanol.

Capillarity in the spinal cord

Studies using the open-ended segment of the spinal cord in situ showed no static resistance to fluid injection. In other words, whatever volume of fluid was injected, the local pressure generated by injection would be dissipated to zero before flow would stop.

With air injection, by contrast, there were positions where flow would stop while still registering a back pressure. These back pressures showed no consistent pattern and are shown in Fig. 4 for 6 different dog spinal cords. After larger volumes had been injected, air could be seen emerging from the open end of the cord segment. There seemed to be no obvious difference between injecting in the cephalad direction at L5 (runs 1–4) or in the caudal direction at L1 (runs 5 and 6).

Preliminary compliance studies

The results of longitudinally loading the membrane complex removed from the excised spinal cord is shown in Fig. 5. Very similar curves were obtained when the experiment was repeated on 4 other samples.

The results of circumferential loading of the excised membrane are shown in Fig. 6 for samples from 5 spinal cords and, once again, show that tissue has minimal compliance that is reached after a very small extension (about 1%).

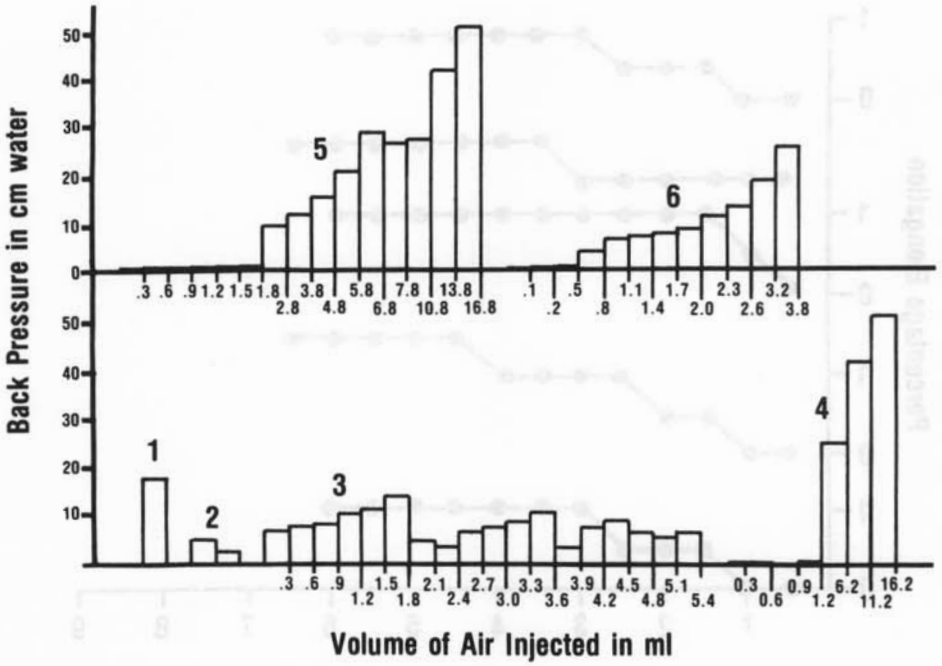

Fig. 4. Back pressure resisting gas injection into open-ended dog spinal cords (L1 to L5) left in situ when various boluses are injected either in the cephalad direction (1–4) or caudal direction (5 and 6).

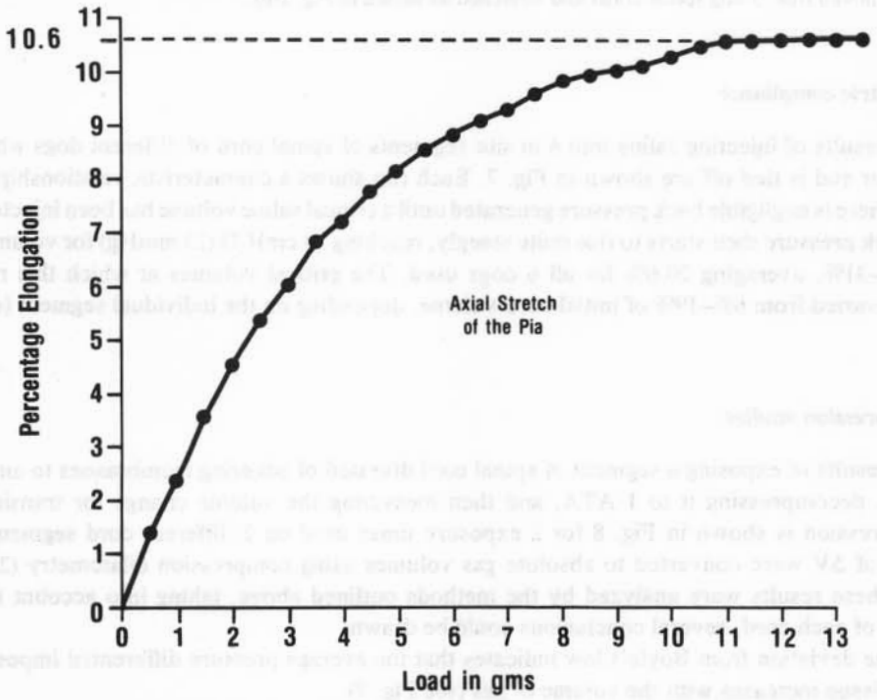

Fig. 5. Typical compliance curve for an excised membrane (dura and underlying structures) removed from a dog spinal cord and extended in longitudinal direction.

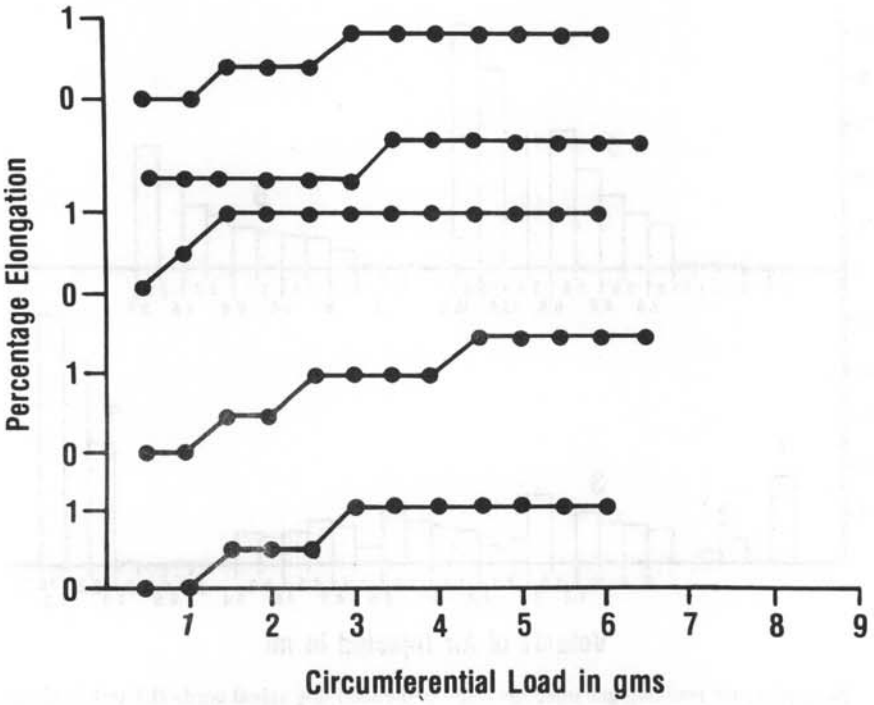

Fig. 6. Circumferential compliance curves for 2-cm lengths of membranes (dura and underlying structures) removed from 5 dog spinal cords and stretched as shown in Fig. 1(*b*).

Volumetric compliance

The results of injecting saline into 6 in situ segments of spinal cord of different dogs when the other end is tied off are shown in Fig. 7. Each run shows a characteristic relationship in which there is negligible back pressure generated until a critical saline volume has been injected. The back pressure then starts to rise quite steeply, reaching 34 cmH_2O (25 mmHg) for volumes of 14%–31%, averaging 20.6% for all 6 dogs used. The critical volumes at which this rise started varied from 6%–19% of initial cord volume, depending on the individual segment (see Fig. 7).

Decompression studies

The results of exposing a segment of spinal cord divested of adhering membraanes to air at 6 ATA, decompressing it to 1 ATA, and then measuring the volume change for transient recompression is shown in Fig. 8 for 2 exposure times used on 2 different cord segments. Values of ΔV were converted to absolute gas volumes using compression dilatometry (25). When these results were analyzed by the methods outlined above, taking into account the volume of each cord, several conclusions could be drawn:

1. The deviation from Boyle's law indicates that the average pressure differential imposed by the tissue increases with the volume of gas (see Fig. 9).

2. Even at maximum volume (21% of tissue volume) this pressure differential is no more than 25 cmH_2O (18 mmHg).

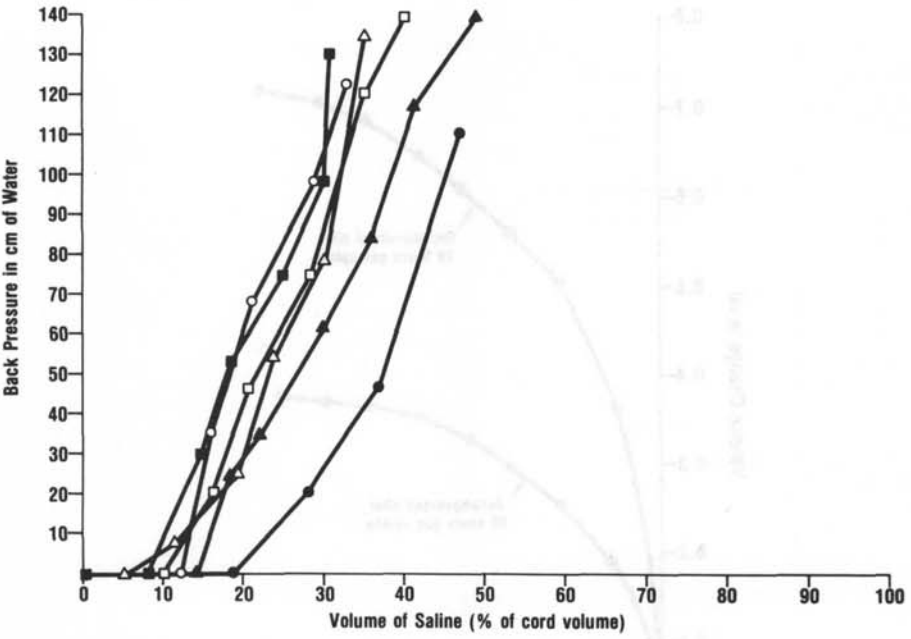

Fig. 7. Volumetric compliance curves for segments (L1–L5) of 6 dog spinal cords left in situ and injected with saline from one end while the other end was closed. Note steep rise in back pressure after injecting 6%–19% of initial volume with the pressure reaching 25 mmHg (34 cm H_2O) for 14%–31% volume increase.

3. After 72 h of gas uptake at 6 ATA and 0°C followed by decompression to 1 ATA and rewarming to 21°C, the volume of gas separated from solution amounted to about 31% of cord volume. This figure varied between 26% and 34% for 5 cord segments from as many dogs.

Although most of the experiments were conducted with the spinal cord in situ and still close to body temperature it must be remembered that excision and cooling could cause some changes. However, this applies to most studies in biomechanics, while cooling to ambient temperature has been shown to cause no more than a 5% change in mechanical stress (26).

DISCUSSION

The experiments involving air injection into the open spinal cord indicate that the resistance to dissipation of gas pockets is contributed, in part, by capillarity, since the effect is eliminated with saline—i.e., upon elimination of the air-water interface. This contribution seems to vary, as seen in Fig. 4, the variability probably arising from the tendency for tissue planes to separate suddenly and in discrete steps. While these experiments were conducted in the intact cord segment, repetition of the experiment upon excised cord viewed under the light microscope showed that separation of tissue planes did, indeed, proceed in steps, i.e., displaying an "echelon" effect consistent with the pressure steps observed in the cord in situ. The implication that mechanical distention is involved in addition to capillarity in the dissipation of gas in the cord tends to be confirmed by the preliminary capillarity studies in vitro. If the linear relationship between the pressure resisting gas dissipation and $1/D$ as seen in Fig. 3 is extrapolated to a conservative value of 200 cm^{-1} corresponding to the largest fiber likely (50 μm diam), then, according to Eq. *3*, the resistance would be 115 cmH_2O (85 mmHg). Since this value is significantly higher than the range of up to 50 cmH_2O found in the intact cord segment (Fig. 4), it is confirmed that nerve fibers are not rigid cylindrical rods, but that they deform and so

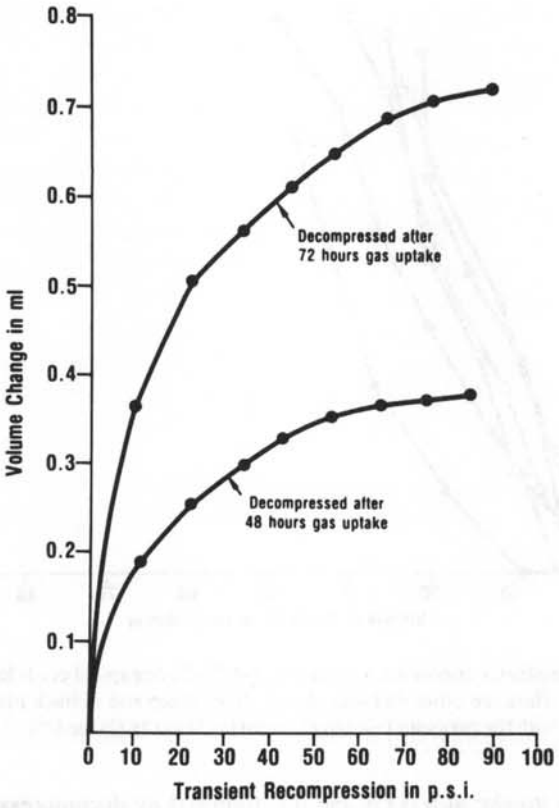

Fig. 8. Typical volume changes (extrapolated to zero time) for transient recompressions of excised segments of dog spinal cords (L1 to L5, inclusive) decompressed after exposure to 6 ATA of air at 0°C for 48 h or 72 h.

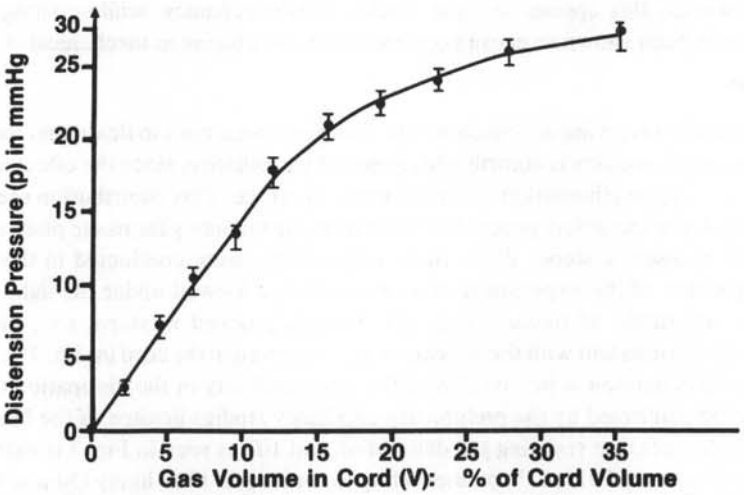

Fig. 9. Relationship between the pressure differential (p) exerted by the tissue upon the enclosed gas and its volume (V) as calculated using Eq. 2 and standard relationships for compression dilatometry (25) applied to the data in Fig. 8. Outer membranes had been removed from cord segment.

modify the resistance to gas dissipation afforded by capillarity. However, whatever the relationship between capillarity and distension, the net effect has the capability to resist the tracking of gas along tissue boundaries to the extent of allowing pressures to reach 20 or even 50 cmH_2O in some instances. The effect is variable, and this may be one factor accounting for the widely different susceptibility of the same individual to the same decompression. A practical example is the tunnel worker who, after working the same shift at the same pressure for several weeks or months, suddenly one day develops spinal symptoms upon decompression.

The results of the preliminary compliance studies (Fig. 5 and 6) show that at least one of the membranes surrounding the cord is difficult to distend in both the longitudinal and circumferential modes in the range of physiological pressures and confirms, in dog, the widely accepted view derived from human studies that the dura is particularly noncompliant (27). Thus the dura and other membranes could provide an effectively rigid outer casing against which a cord enlarged by internal gas formation can no longer expand without greatly elevating the force it exerts upon the membrane and, hence, without substantially elevating the pressure within the cord itself.

It is a moot point whether the cord has to expand to the point where the pia mater and intermediate membranes make physical contact with the dura for effective encasement, since, although cerebrospinal fluid (CSF) can be regarded as an incompressible hydraulic medium for transmitting pressure, it could filter out of the epidural space under the increased pressure and thus help accommodate distension of the cord by gas.

More significant, perhaps, are the results of the study with the closed-end cord segment left in situ. By adding fluid, we are essentially studying volumetric compliance devoid of the complications of capillarity addressed in the first two experiments. It is particularly interesting to see in Fig. 7 how there is negligible resistance to the initial expansion of the cord up to 7%–19% of its original volume, depending on the individual dog. However, the pressure for any further expansion then rises steeply and attains a value of 25 mmHg for 14%–31% volume increase. This is essentially a local pressure differential generated by the gas and, therefore, needs to be added to the normal CSF pressure of 9–14 mmHg (29) to attain a total pressure of 34–39 mmHg, which the blood would need to exceed in order to perfuse the nerve tracts of the cord by first penetrating the pia at the level of the arterioles (27)—whether these are derived from branches of the anterior spinal, posterior spinal, or radicular arteries. Hence it can be argued (see later) that if 14%–31% of gas were to separate from solution upon decompression of those dogs, then sufficient local pressure would be generated to collapse the arterioles and cut off blood flow. These figures are particularly interesting, since a 14% volume increase would be predicted for nitrogen separating from solution from an 80% fatty zone upon decompression following an exposure where the subject attained steady state breathing air at 3.7 ATA (89 fsw). This is close to the depth at which we start to observe spinal cord DCS on air diving (1–6). Repeating the calculation for 80% helium + 20% oxygen, the corresponding depth is now as great as 296 ft, which would explain why spinal symptoms are so rare on heliox diving (2, 3).

In calculating these threshold depths, the membrane and tissue barriers not only determine the back pressure of gas formed but, by slightly elevating the absolute gas pressure, set a new equilibrium level for gas coming out of solution. This results in the formation of slightly less gas phase than would occur if the encasing membranes had allowed the inside of the cord to return to true atmospheric pressure. Relative to decompression from atmospheric pressure to altitude, however, this "mechanical" differential of 34–39 mmHg now becomes appreciable relative to an absolute pressure of 226 mmHg (30,000 ft) at which neurological decompression sickness is frequently observed (28), or 179 mmHg after subtracting water vapor. The gas within the myelin sheath could be restrained by a further differential of as much as 20 mmHg

(Fig. 9) to make a total displacement of the solution equilibrium by 54–59 mmHg, which is particularly significant relative to 179 mmHg. This would require approximately 14(179 + 57)/179% = 20% of gas to separate from solution by comparison with the 14% estimated above from the mechanical studies applied to the case of decompression after hyperbaric exposure. Thus the lack of compliance of the structures surrounding the cord and gas in myelin would prevent separation of more gas from solution, and this may explain why spinal symptoms are less common than cerebral for altitude decompression—the reverse of the diving experience described earlier (2, 12).

The simple mechanical experiments undertaken in this study leave little doubt that the volume of gas that could separate from solution in the cord from potentially symptom-provoking decompressions would not be dissipated by capillarity or tissue compliance. At least, the gas would not dissipate before generating a local pressure in the region of 25 mmHg which, superimposed on a normal epidural CSF pressure of 9–14 mmHg (29), gives a pressure of about 34–39 mmHg outside of the arterioles penetrating the pia. For "encased" gas to cause actual vascular collapse with subsequent impairment of the local circulation, this value for extravascular pressure must exceed the pressure of blood within.

It is particularly difficult to measure or to find published values for the perfusion pressure of these arterioles, and this is further complicated by the known (29) sympathetic vasomotor reflex in elevating local perfusion pressure to balance the potential collapsing pressure. This compensation is also well known as the Cushing reflex in higher centers of the nervous system, but even this mechanism fails to maintain cerebral perfusion when the CSF pressure causing mechanical deformation of the medullary tegmentum reaches a head of 40–50 cmH_2O (29), i.e. 29–37 mmHg. The corresponding values in the spinal cord are likely to be appreciably lower, especially in the poorly perfused (30) watershed zones (T4 and L1), which are those most prone to decompression injury. Thus the encasing pressure has the potential to exceed the perfusion pressure with subsequent vascular collapse resulting in tissue anoxia. Moreover, we have all of the basic ingredients for applying the waterfall concept of blood flow to the decompressed spinal cord and, hence, to spinal decompression sickness (see Fig. 10). This is basically a modification of the concept of the Starling resistor widely applied in physiology to cases such as the brain (29), the lung (23), and long bones (24) where flow in collapsible vessels can be determined more by the transmural arterial pressure than by the arteriovenous pressure difference. The model depicted in Fig. 10 is drawn for a probable perfusion pressure of the order of 25 mmHg in the watershed zones.

This model is consistent with two relevant experimental observations. First, there is the finding that symptoms virtually indistinguishable from spinal decompression sickness can be induced reversibly by inflating an extradural balloon (31). Second, after the normal efflux of CSF after lumbar puncture, further fluid is displaced from the cord upon decompression of both human subjects (32) and goats (28), presumably reflecting the decreased volume of the subarachnoid space as the pia is pushed toward the dura due to inflation of the cord by bubbles (Fig. 10).

From a clinical standpoint, the model can explain the predominant involvement of the watershed zones T4 and L1 in spinal decompression sickness as the two zones with the lowest perfusion pressure and highest nitrogen content resulting from the highest lipid content. It is easy to envisage how large amounts of gas could cut off blood flow from all arterioles, resulting in the total paralysis often seen. For lesser amounts of gas, however, the closure of arterioles could be localized, depending on whether the gas had tracked along the axial planes to produce the hemiplegia often observed in divers. It is most interesting in studying neurological charts of divers with spinal DCS to see how the affected areas of the spinal cord are almost invariably adjacent—either longitudinally or laterally—but never random as would be predicted on any

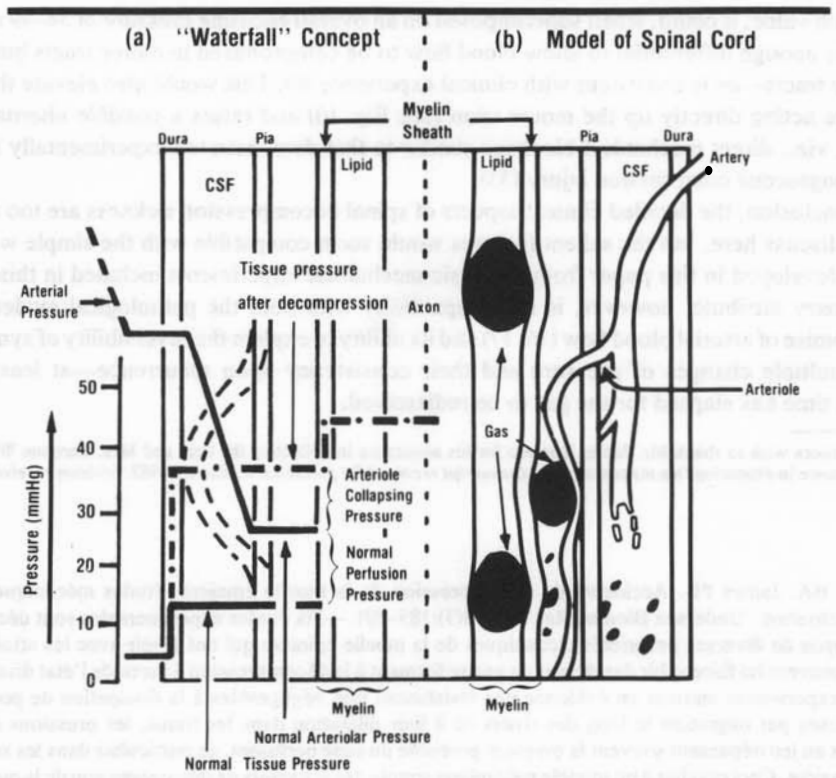

Fig. 10. Application of "waterfall" concept of blood flow (*a*) to simple mechanical model of a decompressed spinal cord (*b*). Gas in spinal cord encased by noncompliant membranes can elevate extracellular pressure above its normal CSF pressure, while gas within myelin sheath could elevate pressure around axon still further. It is also demonstrated how a sufficient volume of deposited gas could raise extravascular pressure above perfusion pressure to interrupt the circulation, causing anoxia that could be reversed by recompression.

theory of arterial occlusion by circulating bubbles. Pathological studies often reveal tracking of gas, sometimes "extending vertically for several inches" as seen in the cords of goats (1).

Tracking of gas at junctions could also account for the occasional nerve root problem observed. This is feasible because the cord and the root are encased in a common dural sheath until just beyond the spinal ganglion where the spinal nerve emerges surrounded by epineurium (27). Boycott et al. (1) have shown the clustering of gas around the anterior horn cells of the cord in the cervical and lumbar enlargements where the ratio of grey matter to white is only 2:1.

The reason why dysfunction often does not occur in all nerve tracts in the spinal cord as a total block may be because not all gas is formed in extracellular sites, but much is deposited by decompression within the myelin sheath as the fenestration (11) often described by pathologists and attributed to the high nitrogen solubility of myelin lipid. Although the mathematical analysis of the data shown in Fig. 8 indicates that pressures within the myelin sheath do not exceed 20 mmHg, they can hinder the free movement of gas between structural planes. This constraint would apply more to the lateral movement of gas, thus tending to retain gas within the same nerve tract—particularly the motor tracts with the higher lipid content associated with myelinated fibers. Whereas a pressure of up to 20 mmHg would not exceed any blood

perfusion value, it could, when superimposed on an overall encasing pressure of 34–39 mmHg, generate enough differential to allow blood flow to be compromised in motor tracts but not in sensory tracts—as is consistent with clinical experience (6). This would also elevate the local pressure acting directly up the motor axon (see Fig. 10) and raises a possible alternative to anoxia, viz., direct mechanical blockage similar to that demonstrated experimentally in dogs with nongaseous compression injury (33).

In conclusion, the detailed clinical aspects of spinal decompression sickness are too numerous to discuss here, but the salient features would seem compatible with the simple waterfall model developed in this paper from the basic mechanical experiments included in this study. Its primary attribute, however, is its compatibility with both the pathological evidence for compromise of arterial blood flow (16, 17) and its ability to explain the reversibility of symptoms upon multiple changes of pressure and their consistency upon recurrence—at least, until enough time has elapsed for the gas to be redissolved.

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Hills BA, James PB. Accidents de décompression de la moelle épinière; études mécaniques et modélisation. Undersea Biomed Res 1982; 9(3):185–201.—Six études expérimentales sont décrites à propos de diverses propriétés mécaniques de la moelle épinière qui ont à voir avec les atteintes que peuvent lui faire subir des dépots de gaz se formant à la décompression à partir de l'état dissous. Ces expériences mettent en évidence des résistances non négligeables à la dissipation de poches gazeuses par migration le long des tissus ou à leur dilatation dans les tissus, les pressions ainsi mises en jeu dépassant souvent la pression probable du sang perfusant, en particulier dans les zones lacunaires. Ceci conduit à un modèle mécanique simple des accidents de décompression de la moelle épinière, fondé sur la "cascade" vasculaire, qui s'accorde à la pathologie, aux aspects quantitatifs majeurs et à la symptomatologie, spécialement la reversibilité des troubles par recompression qu'il est si difficile d'expliquer par un mécanisme embolique. L'hypothèse est que du gaz autochtone quittant sur place l'état dissous dans la moelle épinière peut atteindre localement une pression suffisante pour dépasser celle du sang perfusant et ainsi arrêter sa circulation.

accidents de décompression de la moelle épinière
moelle épinière
gaz autochtone

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