Central nervous system decompression sickness: latency of 1070 human cases

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Francis TJR, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. Undersea Biomed Res 1988; 15(6):403–417.—Many aspects of central nervous system (CNS) decompression sickness (DCS) are poorly understood, including the temporal pattern of its presentation and the pathogenic mechanisms involved in the development of the disease. Using case histories and clinical series published in the literature and retrieved from treatment center records, this study is an attempt to define the interval between surfacing from a hyperbaric exposure and the onset of symptoms of CNS DCS. The results of 1070 cases of human CNS DCS were included in the study. The results show that the disease generally occurs rapidly: over 50% became symptomatic within 10 min of returning to 1 ATA, and in only 15% of cases was the onset of symptoms delayed for more than 1 h. Cerebral DCS had a more rapid onset than spinal cord disease: 50% of cerebral cases became apparent within about 3 min and a similar proportion of spinal cord cases within about 9 min from surfacing. The influence of these results on the diagnosis and treatment of dysbaric illness, on the safety of certain diving practices, and on possible pathogenic mechanisms is discussed.

dysbarism
spinal cord
cerebrum
embolism
diagnosis

The lesions of central nervous system (CNS) decompression sickness (DCS) may be discrete or diffusely scattered (1–5). Consequently, the clinical presentation of the disease may cover a spectrum of complexity from a relatively simple spinal cord transection to bizarre neurological or psychiatric syndromes in which the lesion(s) may be difficult to localize even when the patient is carefully and expertly examined. However, it is often possible to subdivide CNS DCS into three broad categories: cases involving the cerebrum, cases involving the spinal cord, and cases involving both. We have restricted our analysis of neurological DCS to this classification and

have not attempted to consider other presentations, such as vestibular or peripheral nerve DCS.

A considerable number of case series and reviews of the presentation, treatment, and pathology of CNS DCS have been reported in the literature, but one aspect that has been largely overlooked is the latency of the condition. Following a period of hyperbaric exposure, this may be defined as the interval between returning to 1 ATA and the onset of the first symptom of the disease. Many series have either omitted these data (6–19) or combined the latency of CNS DCS with that of more prevalent forms of DCS such as pain-only "bends" (20–34). This has led to a widely held belief that the onset of CNS DCS is usually delayed many minutes or even hours after the completion of a hyperbaric exposure, as is often the case for pain-only DCS.

There are a number of aspects of CNS DCS where latency may be important. Surface decompression (defined as when a diver omits one or more in-water stops before surfacing and is then rapidly transferred to a chamber where the decompression is completed) relies for its safety on the assumption that there is a latent period of 5 or more min between surfacing and the onset of DCS during which the transfer can safely be made. Anecdotal reports of serious DCS occurring during decompression and the relatively high incidence of type II DCS among divers undertaking surface decompression (16) indicate that this assumption may not be valid.

Latency may influence the diagnosis of cerebral DCS. This is rarely simple, and in the event of an onset within 10 min of surfacing from a dive with a decompression obligation, cerebral DCS is easily confused with pulmonary barotrauma and cerebral arterial gas embolism (CAGE). This may result in the selection of a suboptimal therapeutic table.

Finally, the latency of spinal cord DCS may have a bearing on the relevance of the various pathogenic mechanisms that have been proposed to result in dysfunction. For instance, a mechanism that is likely to require many minutes to evolve into a spinal cord-damaging insult will play only a minimal role in cases with a short latency. Equally, a mechanism for which there may be only a brief "window of opportunity," during which it is likely to operate following a return to 1 ATA, will be of little significance in the development of the injury in cases with a long latency.

We have therefore undertaken this retrospective study to determine the latency of human CNS DCS.

METHOD

We have searched the literature (principally that published in the English language) and consulted centers that currently treat hyperbaric accidents for the latency of cases of CNS DCS. We selected for inclusion in this study cases that conform to the criteria listed below.

As will be seen, the data are drawn from hyperbaric exposures of widely differing character. In particular, data from both divers and compressed-air (caisson) workers have been included.

Diagnosis of CNS DCS

We have included cases of CNS DCS where no circumstances in a case report throw doubt on the diagnosis. For hyperbaric accidents without a diagnosis, we

classified the cases into one of the groups below, providing sufficient clinical details were reported. In an attempt to avoid confusion with CAGE, we have included only cases that experienced hyperbaric conditions that carried a decompression obligation according to the U.S. Navy Diving Manual, although we recognize that CNS DCS may occasionally occur following lesser exposures (35). Only hyperbaric exposures with air as the breathing gas have been considered.

Diagnosis of cerebral DCS

Manifestations of cerebral DCS are protean and may be subtle. To include only cases in which it is reasonably certain that a lesion existed, those presenting only with nonspecific symptoms or symptoms that may be considered spurious (such as headache, fatigue, or ill-defined psychiatric symptoms) have been excluded. Cases that presented with a reduced level of consciousness have been included only if no other condition such as hypoxia, "chokes," CO or CO_2 toxicity, or head injury were reported to be concurrent or considered responsible. There is always the possibility that CNS DCS was in fact misdiagnosed CAGE, particularly where the latency was short. Consequently, cases of blow-up, emergency ascent, panic, and those in which either acute or chronic (and possibly provocative) pulmonary symptoms were reported were all excluded. The cases that have been included have suffered a grand mal seizure, experienced a reduced level of consciousness, or displayed a motor or sensory deficit with a cortical distribution. In some cases the presence of cerebral lesions were confirmed by postmortem examination.

Diagnosis of spinal cord DCS

This was defined as a patchy or total loss of sensory, motor, or autonomic function below a certain spinal level. Although it is possible for small spinal cord lesions to result in unilateral symptomatology, to avoid confusion with peripheral nerve lesions, only cases with a bilateral or Brown-Sequard syndrome distribution of symptoms or those with abnormalities of bowel, bladder, or sexual function were included in the study.

Diagnosis of both cerebral and spinal cord DCS

In a number of cases there was evidence of symptoms referable to both the brain and spinal cord. One example is case 2 reported by Brooks (36). This caisson worker, some 10 min after finishing a 3-h shift at 42 psi, became initially paraparetic and shortly thereafter lost consciousness. He died some 13.5 h after the onset of symptoms. At postmortem, pathology referable to DCS was found in both the brain and spinal cord. A more recent example is a report of electrophysiological evidence for three levels of injury in a diver with CNS DCS (37).

RESULTS

Our search of the literature resulted in 72 references in which sufficient clinical details were available to classify the cases into the preceding categories and have

reasonable confidence in the stated latency (2, 3, 21, 27, 35–102). These papers included data on 357 cases of CNS DCS. Three additional papers (103–105) included latency data on another 110 cases of type II or neurological DCS, but these were not separated into our categories and may have included cases of chokes or vestibular DCS. Furthermore, the criteria for diagnosing CNS DCS in one of these papers (105) tended to bias the results in favor of a long latency, since patients presenting with primarily cerebral symptoms, or who were symptomatic on completion of the dive, were automatically categorized as CAGE. However, we have included these data in Table 1 and Fig. 1, but not in the results of the latency of the more specific diagnoses.

A total of 603 case reports were compiled from treatment center records in Australia, Hawaii, Japan, Puerto Rico, Singapore, the United Kingdom, and the continental United States. They include diving accidents that resulted from a wide range of hyperbaric exposures such as compressed-air workers, diving fishermen, and other commercial, military, and recreational divers.

Tables 1 and 2 contain the latency data of the cases in our three categories of CNS DCS taken from the literature and from current treatment centers. It can be seen that the distribution of CNS involvement is similar in the two tables, with the classified literature cases being distributed as follows: 22.7% cerebral DCS, 66.4% spinal cord DCS, and 10.9% with both. Equivalent figures for the cases from current treatment centers are 18.4, 68.3, and 13.3%. The distribution of latency in the two tables is also broadly similar, but with the cases from the literature tending to have a shorter latency than those from current treatment centers.

The cumulative latency of all 1070 cases of CNS DCS is shown in Fig. 1. It is apparent that although there is a wide range of latency, the majority of cases became symptomatic early. The figure shows that half of all cases became symptomatic within about 8 min of returning to 1 ATA, and only about 15% presented more than 1 h after surfacing.

In Fig. 2, the cases of spinal cord and cerebral DCS have been separated, but the cases in which both cerebral and spinal symptoms occurred have been included in each group. Figure 2 shows that the latency of cerebral DCS is generally shorter than that of the spinal cord. In 50% of the cases the symptoms of cerebral DCS were manifest within approximately 3 min of returning to 1 ATA, whereas some 9 min were required for a similar proportion of spinal cord cases to become symptomatic.

TABLE 1

LATENCY OF CNS DCS FROM CASES RECORDED IN THE LITERATURE*

Category	DD	IS	5	10	15	30	60	2h	3h	4h	5h	6h	12h	24h	48+h
Cerebral	3	22	29	9	3	9	2	3	–	–	–	–	–	–	1
Spinal	17	60	25	16	20	68	16	3	7	1	–	2	–	1	1
Both	6	14	9	3	3	1	2	–	1	–	–	–	–	–	–
Unclassified	20	8	10	12	–	18	7	3	–	–	–	13	–	16	3
Total	46	104	73	40	26	96	27	9	8	1	–	15	–	17	5

*Latency in minutes or hours (h) from reaching 1 ATA; DD = during decompression, IS = immediately on surfacing.

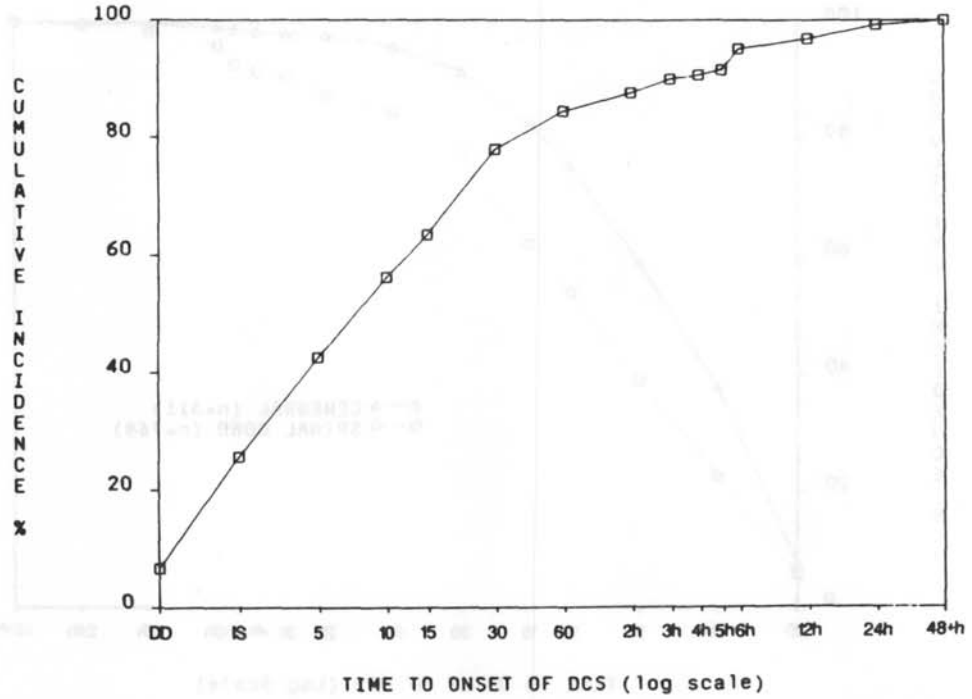

Fig. 1. Cumulative incidence of 1070 cases of human CNS DCS. The x-axis represents time; *DD* = during decompression, *IS* = immediately on surfacing; other numbers represent time intervals in minutes or hours (h) out to 48 h or more after surfacing from a hyperbaric exposure. Figure shows that 56% of cases become symptomatic within 10 min of returning to 1 ATA.

TABLE 2
LATENCY OF CNS DCS FROM CURRENT TREATMENT CENTERS*

Category	DD	IS	5	10	15	30	60	2h	3h	4h	5h	6h	12h	24h	48+h
Cerebral	5	44	16	10	10	14	5	3	–	1	1	1	1	–	–
Spinal	12	40	80	67	35	41	34	21	16	6	9	23	16	9	3
Both	9	14	13	29	6	4	4	–	–	–	–	–	1	–	–
Total	26	98	109	106	51	59	43	24	16	7	10	24	18	9	3

*Latency in minutes or hours (h) from reaching 1 ATA; DD = during decompression, IS = immediately on surfacing.

A delayed onset of cerebral DCS is rare: fewer than 4% required more than 1 h to become symptomatic.

Figure 3 presents the data from compressed-air (caisson) workers separated from divers. Although there are rather few cases in the first group, it is apparent that there are only small differences in the latency of cerebral DCS between the 2 groups. The spinal cord data are more confused. The erratic curve of the compressed-air worker DCS data is due largely to their source. By far the majority of cases were retrieved

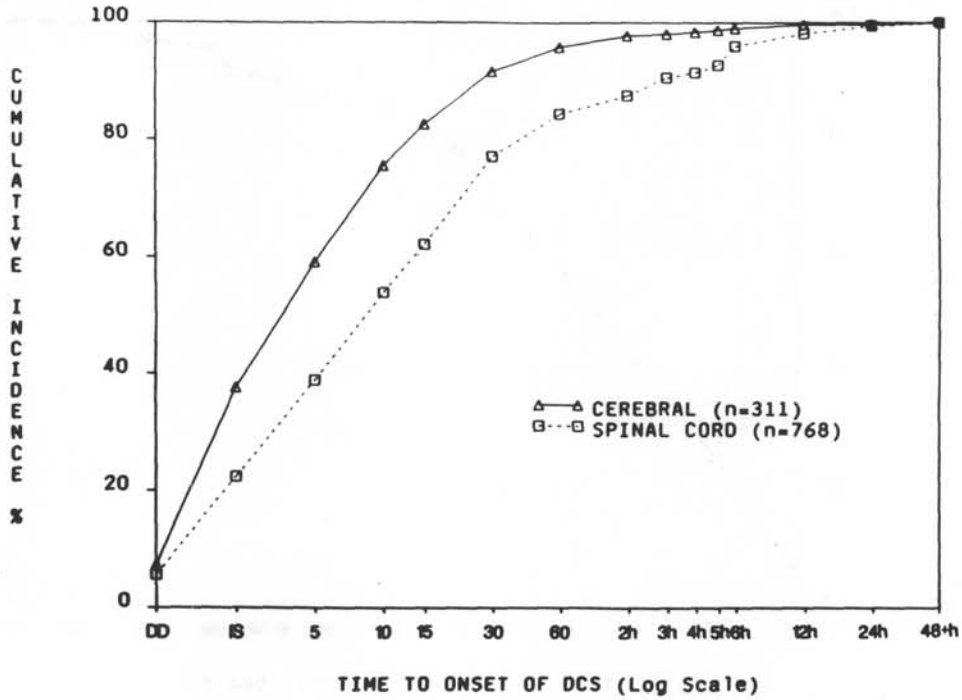

Fig. 2. Graph is presented in a similar manner to Fig. 1. It shows the latency of 311 cases of cerebral and 768 cases of spinal cord DCS. Latency of cerebral DCS is generally shorter than for spinal cord DCS.

from the early literature, and especially from Jaminet (41). His classification of latency was crude, with large groups apparently presenting immediately after leaving the caisson or at 15 or 30 min thereafter. There were also a number of cases of compressed-air workers' spinal cord DCS in the literature that had to be discarded due to vague latency statements such "on arriving home," or "on waking." These were probably longer latency cases. Thus, it is not possible to be sure that the latency of spinal cord DCS in compressed-air workers is different from that seen in divers.

DISCUSSION

Decompression sickness in man manifests itself as a number of differing syndromes that develop following a reduction of ambient atmospheric pressure. This may occur under two distinct circumstances. During the course of a flight, aviators and astronauts, for example, may experience a gradual or sudden reduction in ambient pressure from normobaric (or 1 ATA) to a hypobaric pressure. Divers and compressed-air workers, on the other hand, experience a reduction in pressure from one that is hyperbaric to normobaric before the completion of their dive or work shift. These two forms of decompression, although similar in principle, seem to result in different forms of DCS, particularly with respect to the CNS (106–109). This study has addressed CNS DCS subsequent to a hyperbaric exposure.

There are a number of sources of error in the latency data collected in this report, particularly in the cases retrieved from the literature. Many cases were reviewed in

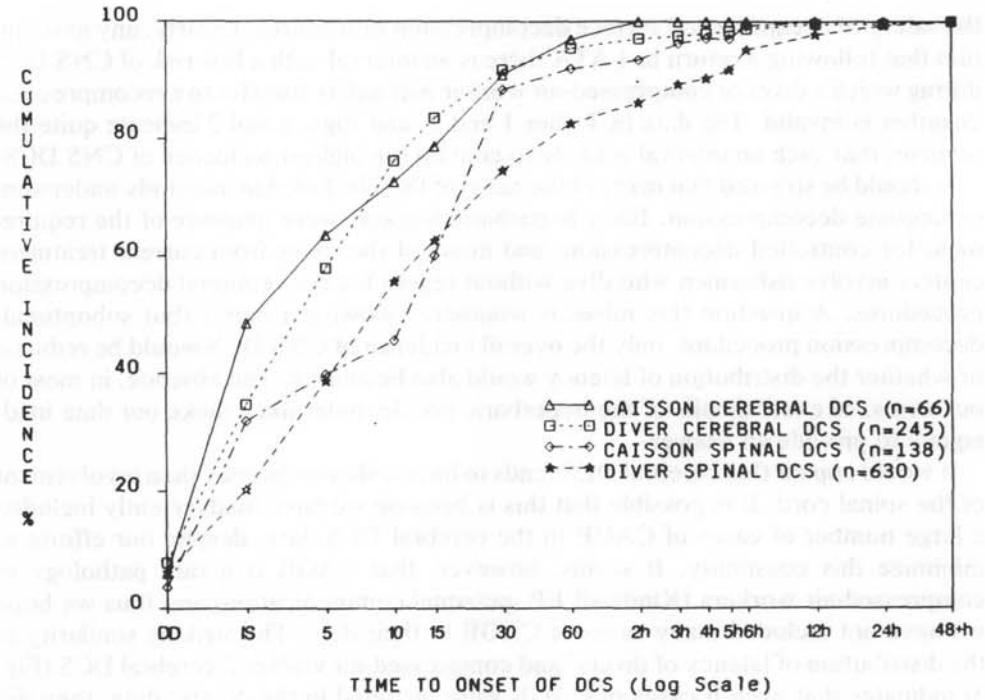

Fig. 3. Graph is presented in a similar manner to Fig. 1. It shows the latency of the cases of cerebral and spinal cord DCS divided between compressed-air (caisson) workers and divers. There is close agreement in the latency of cerebral DCS in the two exposure groups; there is less apparent agreement in the case of spinal cord DCS.

which no latency was mentioned or vaguely documented as "after a few minutes," "having walked home," or "on waking." These cases were excluded from the study. A number of cases that have been included quoted rather vague latency data such as: "after about 30 minutes." Unfortunately, even where authors have been more specific, unless someone was actually timing the events postdecompression, it is possible that quite large errors were made.

In many of the cases reviewed it is not possible to be certain of the diagnosis. Some cases recorded as "cerebral DCS" may, in fact, have been CAGE. Others may reflect high spinal, brain stem, or cranial nerve lesions; or may have been unrelated to decompression. Although the data collected from current treatment centers are likely to be more reliable than those from the literature, errors are still quite possible. For example, the prevalent inability of amateur divers to accurately time their decompression almost certainly extends into the postdecompression period.

The above sources of inaccuracy are likely to be random. Both under- and overestimates will have been made. We have attempted to avoid systematic errors in the data collection by involving 10 independent observers. We have also tried to minimize the possibility of sampling errors by making the study as large as possible and including data from widely disparate hyperbaric exposures.

Figure 1 shows that CNS DCS is usually a disease with a rapid onset, with about 56% of cases becoming symptomatic within 10 min of returning to 1 ATA. The finding that over 40% of cases have a latency of 5 min or less raises a serious question about

the safety of decanting and surface decompression procedures. Clearly, any assumption that following a return to 1 ATA there is an interval with a low risk of CNS DCS during which a diver or compressed-air worker may safely transfer to a recompression chamber is invalid. The data in Tables 1 and 2, and Figs. 1 and 2 indicate quite the reverse, that such an interval is likely to contain the highest incidence of CNS DCS.

It should be stressed that many of the cases of DCS included in this study underwent inadequate decompression. Early hyperbaric workers were unaware of the requirement for controlled decompression, and many of the cases from current treatment centers involve fishermen who dive without regard for conventional decompression procedures. A question this raises is whether, following a better (but suboptimal) decompression procedure, only the overall incidence of CNS DCS would be reduced or whether the distribution of latency would also be altered. The absence, in most of our cases, of exact details of the hyperbaric profile undertaken make our data inadequate to provide an answer.

It would appear that cerebral DCS tends to have a shorter latency than involvement of the spinal cord. It is possible that this is because we have inadvertently included a large number of cases of CAGE in the cerebral DCS data, despite our efforts to minimize this possibility. It seems, however, that CAGE is a rare pathology in compressed-air workers (Kindwall EP, personal communication) and thus we hope we have not included many cases of CAGE in their data. The striking similarity in the distribution of latency of divers' and compressed-air workers' cerebral DCS (Fig. 3) indicates that even if cases of CAGE were included in the divers' data, they did not greatly alter the distribution of latency.

Figure 2 shows that about 75% of cases of cerebral DCS present within 10 min of returning to 1 ATA. The prevalence of such short onset times emphasizes how difficult the discrimination between cerebral DCS and CAGE may be in cases of dysbaric illness. This has important implications for the rational selection of treatment tables in cases of hyperbaric accidents presenting with short-latency cerebral symptomatology. Although it remains controversial, it is standard practice, initially, to recompress cases of CAGE to 6 ATA (110). It has been shown experimentally that the recompression of cases of CNS DCS beyond 3 ATA offers no additional benefit in terms of recovery (111). In view of the difficulty in distinguishing between DCS and CAGE in dives with a decompression obligation, it would seem rational to direct efforts toward developing a common treatment regimen.

Lehner et al. (112) used sheep to show that the latency of CNS DCS following a shallow, 24-h dive was longer than following a deeper, 30-min exposure. If this finding were to be reflected in our data, one might expect to observe a longer latency in the CNS DCS of compressed-air workers than divers. Figure 3 shows that there appears to be little difference in the latency of CNS DCS in the 2 groups. This may be because much of the compressed-air data have been extracted from the reports of early projects that were undertaken before the necessity for gradual decompression was understood. Hence, many workers were exposed to decompressions more akin to those experienced by divers than modern compressed-air workers. We have gathered insufficient data to comment on the latency of CNS DCS in modern compressed-air work.

A final aspect of the latency of CNS DCS that we will consider is the possible ways in which it may influence arguments about the pathophysiological mechanisms involved in the generation of the injury. It is clear that for any proposed mechanism to account

for the majority of cases of CNS DCS, it must be capable of causing an injury to the cerebrum or spinal cord during decompression or shortly thereafter. Mechanisms that require more time to operate will only be capable of explaining a minority of cases.

For many years it has been widely accepted that the manifestations of DCS result from the nucleation of gas bubbles within tissues. These bubbles are thought to cause CNS injury through their ability to provoke ischemia. This may occur either as a result of the obstruction of the arterial supply by the impaction of bubble emboli (43, 57, 113, 114) or, in the spinal cord, as a consequence of the impairment of the venous outflow by the interaction of blood and bubbles in the epidural vertebral venous plexus (EVVP) (115–119). If, during decompression, showers of arterial bubbles were generated before the onset of DCS, the finding that cerebral DCS has a shorter latency than the spinal cord may be explained by the relatively high blood flow to the brain (and hence increased probability of embolism) and the low tolerance of cerebral tissue to ischemia compared with the spinal cord (120). Unfortunately, as Hallenbeck et al. argued (119), if this were the mechanism of CNS DCS, the incidence of cerebral DCS would greatly exceed that of the spinal cord. Our data and those from other studies show that this is not usually the case (20–29, 86, 103). Furthermore, the evidence from animal studies is that arterial gas bubbles are an uncommon early finding in nonlethal DCS (121–124) and they are unlikely to appear in the circulation sufficiently rapidly to account for cases of spinal cord DCS with a short latency (125).

An alternative mechanism whereby bubbles may generate CNS injury is by their nucleation within white matter. Autochthonous bubbles have been shown to traumatize neurons at the site of nucleation and to compress those adjacent (126). This mechanism of injury is likely to result in a more rapid onset of bubble-induced CNS dysfunction than purely ischemic, intravascular mechanisms. This is because the effects of both trauma and pressure on nerve conduction are immediate (127–129), whereas spinal cord function may continue for as much as 10 min after the onset of ischemia (120, 126).

As tissue gas clearance proceeds with time in the postdecompression period, the likelihood of bubbles nucleating within tissues becomes smaller. Thus, in the absence of circulatory embarrassment that would reduce the rate of gas clearance, the formation of autochthonous bubbles is likely to be restricted to a "window of opportunity." Cerebral perfusion is in a higher range than that of the spinal cord (130–133). The observation that cerebral DCS has a lower incidence and a shorter latency than the spinal cord is compatible with an autochthonous bubble mechanism due to the superior perfusion of the brain. This would tend to result in a more rapid tissue gas clearance than would occur in the spinal cord, thus lowering the overall probability of autochthonous bubble nucleation within a shorter "window of opportunity."

In conclusion, it is apparent that the latency of CNS DCS has relevance to a number of aspects of the disease in man and it is a subject that has been poorly addressed in the past. This study offers only an imperfect remedy to this neglect due to the problems inherent to a retrospective analysis of this sort. It is hoped that this study will result in the recognition of latency as an important element of the clinical history of a hyperbaric accident and that its recording as a matter of routine will facilitate a better study in the future.

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