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⑥ THE WORKSHOP ON LONG-TERM HEALTH
HAZARDS OF DIVING WITH REGARD TO THE
CENTRAL NERVOUS SYSTEM,

⑩ J. HALLENBECK

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Research in the 1950's called attention to the possible existence of a chronic progressive encephalomyelopathy that occurred as a result of repeated decomp- ressions from raised environmental pressures. This neurologic disorder could occur in the absence of decompression sickness, but was more common in indiv- iduals with a history of prior decompression sickness. In this presentation the general problem of chronic progressive encephalomyelopathy is addressed on three levels: (1) cases of central nervous system degeneration occurring in individuals exposed to repeated decompressions without any history of		

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preceding decompression sickness or air embolism; (2) individuals incurring central nervous system damaging decompression sickness or air embolism during the course of a decompression from raised environmental pressure, and subsequently, developing chronic progressive central nervous system degeneration despite refraining from further diving; (3) individuals afflicted by decompression sickness or air embolism who developed chronic progressive central nervous system degeneration in the setting of continued diving. Although ischemia appears to play a major role in neuraxis damage in decompression sickness and the analogous process of stroke in humans does not generally show chronic progression, it does not seem possible to confirm or deny the occurrence of this chronic progressive encephalomyelopathy at present. The author concludes that examination of the questions pertaining to chronic progressive encephalomyelopathy shows that there is a paucity of firm scientific observation in this area. He sees the need for a thorough and detailed analysis of existing medical records and a prospective study of the medical complications of divers compared with nondivers.

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THE WORKSHOP ON LONG-TERM HEALTH HAZARDS OF DIVING WITH REGARD TO THE CENTRAL
NERVOUS SYSTEM ✓

In the 1950s, Rozsahegyi and colleagues called attention to the possible existence of a chronic progressive encephalomyelopathy occurring as a result of repeated decompressions from raised environmental pressure. This progressive neurologic disorder could occur in the absence of decompression sickness but was more common in individuals with a history of prior decompression sickness. More recently, interest in the validity of this phenomenon has been intensified by cases suggesting a chronic progressive encephalomyelitic process in commercial divers. Examination of the questions in this area reveals that hard data is sparse and that there is a need for careful analysis of case histories to provide more information.

The general problem of chronic progressive encephalomyelopathy secondary to repeated decompression from raised environmental pressure can be addressed on at least three levels. One level would encompass cases of central nervous system degeneration occurring in individuals exposed to repeated decompressions without any history of preceding decompression sickness or air embolism. A second level would include individuals incurring central nervous system damaging decompression sickness or air embolism during the course of a decompression from raised environmental pressure and subsequently developing chronic progressive central nervous system degeneration despite refraining from further diving. The third level would include individuals afflicted by decompression sickness or air embolism who developed chronic progressive central nervous system degeneration in the setting of continued diving.

Several general premises which underlie the subsequent discussion should be set forth. Central nervous system damage as it occurs in decompression sickness and air embolism is regarded as primarily due to ischemia. In spinal cord damaging decompression sickness the fundamental problem is considered to be obstruction at the level of the epidural vertebral venous system with a generalized rheologic disturbance and autochthonous bubble formation in the cord contributing to the pathologic process. Cerebral symptoms when they occur are regarded as migraine-like vasomotor phenomena in some instances and in other instances may be due to arterial gas emboli. In air embolism the fundamental problem is regarded as vascular obstruction by embolic gas with consequent ischemia of tissue distal the point of obstruction. Gas bubbles in the body are considered not to be bland but instead to have an active surface capable of generating a variety of effects through surface activity at bubble-blood interfaces. Further, vessel walls and blood are not inert. Blood responds to bubble surface activity and various constituents normally present in blood are capable of interacting with acutely damaged tissue to progressively impair microvascular perfusion in the injury zone.

The possibility of a chronic progressive encephalomyelopathy related to repeated decompressions from raised environmental pressure was identified by Rozsahegyi and colleagues and was felt to resemble multiple sclerosis or arteriosclerotic myelopathy in its effect on the neuraxis. The radiologic appearance of aseptic bone necrosis in divers and compressed air workers that never experienced decompression sickness raises the distinct possibility of organ damage secondary to repeated decompressions in the absence of clinically-manifest decompression sickness. Doppler studies indicate that most decompressions are associated with the nucleation of some "silent" bubbles so that the possibility

exists during any decompression that small groups of bubbles may focally exert direct mechanical effects and indirect effects due to surface activity. Such combined effects may be responsible for observed changes in platelet count, platelet function, activity of coagulation factors and changes in the concentration of such enzymes as CPK after clinically uneventful decompressions.

On the other hand, the concept of a cumulative effect of repeated circumscribed trivial insults to the central nervous system leading to a progressive encephalomyelopathy is intuitively disturbing on several counts. The central nervous system is an eloquent tissue and even tiny zones of discrete focal damage are generally heralded by clinically apparent symptoms. Another consideration is that transient ischemic attacks in humans, a process that involves focal ischemia sufficient to cause definite neurologic symptoms, can recur many times and yet ultimately clear leaving no sequelae of neurologic dysfunction in 50 - 70% of cases. This tends to vitiate the concept of a focal ischemic process so limited in scope as to be clinically silent leading to a progressive degeneration of the central nervous system through cumulative neuronal injury. Clearly the questions in this area suggest the need for prospective analysis of case histories in divers and their non-diving cohorts.

The second level of chronic nervous system degeneration secondary to decompression from raised environmental pressures involves cases apparently resulting from one or more episodes of decompression sickness or air embolism. In these cases progression of the disorder would occur despite curtailment of further diving or compressed work. Evidence for such a process has been advanced by Rozahegyi on the basis of both clinical neurologic examinations and electroencephalographic studies. However, directly contradictory results

were obtained by Lehmann, both by clinical neurologic exam and EEG. Since the data directly bearing on this issue is insufficient and both decompression sickness and air embolism may be regarded as essentially vascular diseases, some inferences may be drawn from an analogous ischemic process effecting human brain, cerebrovascular disease. Published studies are remarkably consistent and uniform in their description of the course of recovery following cerebral thrombosis and infarction. The post-stroke period is characterized by a variable degree of improvement which tends to plateau after three to four months and deterioration is not a prominent feature of this process. Factors influencing the ultimate prognosis include the presence or absence of such complicating factors as hypertension, heart disease, and diabetes as well as the degree of residual disability. Most of the recorded cases of decompression sickness and air embolism resemble this general pattern during the convalescence period. Early return of function may be due to such factors as restoration of circulation to ischemic areas and resolution of tissue edema. Late recovery of neuronal function may well be due to the collateral sprouting of surviving axons and the assumption of new functions by surviving neurons.

One recognized chronic complication of ischemic central nervous system damage is the development of seizures. This occurs in roughly 5 to 8% of cerebral ischemic infarction cases. There is a chronic progressive ischemic process of uncertain etiology which effects spinal cord and has been found associated with chronic cor pulmonale, subacute necrotic myelitis of Foix-Alajouanine.

On the third level we consider cases in which central nervous system damage that initially occurred as a result of decompression sickness or air embolism progresses during the course of continued exposure to decompression fr

raised environmental pressure. Several considerations suggest that areas of previous central nervous damage might be at heightened risk for further damage under conditions of blood flow compromise such as might be encountered during decompression-induced bubble formation in the body. Zones of damaged central nervous system may lose autoregulation, the capacity to maintain a constant blood flow rate despite fluctuations in perfusion pressure. Under these circumstances, a drop in perfusion pressure that would not adversely effect areas in which autoregulation was preserved could render zones with impaired autoregulation ischemic. Furthermore, areas of central nervous system damaged by a previous episode of ischemia may function with persistently subnormal rates of blood flow. When such areas are detected by xenon clearance blood flow measurements in patients convalescing from a stroke they are associated with a poor long term prognosis. One would expect these zones to have a reduced capacity for collateral circulation and this could accelerate the process of neuronal infarction in focal ischemia. Focal ischemia in primates secondary to middle cerebral artery clipping does not lead to neuronal infarction for two to three hours in all animals and in some animals full neuronal recovery is possible after as long as 24 hours. Since this exceeds anyones claim for neuronal ability to withstand total glucose and oxygen deprivation and since collateral circulation has been demonstrated angiographically after middle cerebral artery occlusion, the final infarct may depend on failure of collateral circulation over time. Progressive failure of collateral circulation has been observed in middle cerebral artery occlusion. Areas with already compromised capacity for collateral circulation might be expected to require shorter periods of focal ischemia to produce nerve cell death.

In summary, several questions of practical import can not be definitively answered at the present time due to the paucity of firm scientific observation. A great deal could be learned from thorough and detailed analysis of existing medical records and prospective study of the medical complications of divers compared with non-diving cohorts.

Hallenbeck

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