# Combined arterial gas embolism and decompression sickness following no-stop dives

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Neuman TS, Bove AA. Combined arterial gas embolism and decompression sickness following no-stop diving. Undersea Biomed Res 1990; 17(5):429–436.—Decompression sickness (DCS) has been clinically classified as Type I (predominantly joint pain) or Type II (predominantly spinal cord lesions). We present 3 cases that are all characterized by severe (Type II) DCS with signs and symptoms of spinal cord injury occurring in conjunction with arterial gas embolism (AGE). We consider the AGE "minor" because only 2 of the 3 subjects initially lost consciousness, and in all cases the signs and symptoms of the AGE had essentially resolved within 1 h or by the time recompression therapy began. DCS was resistant to recompression therapy, even though treatment began promptly after the accident in 2 of the 3 cases. None of the cases had a good neurologic outcome and there has been one death. None of the divers exceeded the U.S. Navy "no-stop" limits for the depths at which they were diving. We have observed a previously unreported clinical syndrome characterized by severe Type II DCS subsequent to AGE following pressure-time exposures that would normally not be expected to produce DCS. We postulate that AGE may have precipitated or predisposed to this form of DCS.

air embolism
intravascular bubbles
hyperbaric therapy

With the development of a large recreational diving community, clinical experience has accumulated with a variety of forms of decompression accidents. Historically, in military and commercial diving, where diving practices are well supervised and the quality of training of individual divers is more comprehensive, two basic types of decompression-related illness have been reported. These are decompression sickness (DCS) and arterial gas embolism (AGE). DCS is due to inadequate decompression from depth using air or mixed gas (1). It is associated with tissue supersaturation with inert gas and a change from soluble to gaseous form in the tissues. AGE is subsequent to pulmonary overinflation on ascent with the introduction of respiratory gases into the pulmonary veins. Arterial gas embolism was first described in 1930 as a result of submarine escape training (2) and was initially confused with DCS (3).

Shortly after its initial description, AGE was recognized as a distinct clinical entity related to pulmonary overinflation, rather than DCS related to inert gas phase separation due to supersaturation (4). In submarine escape, AGE was due to either poor technique or local air trapping in seemingly healthy individuals (5).

Since the early description of AGE, the majority of world experience with AGE has come from submarine escape training facilities where, in distinction to divers, individuals are only exposed to elevated gas pressures for minutes and do not accumulate a significant amount of inert gas in body tissues (6–8). Gas embolism and its complications in this latter situation seem to be markedly different from the complications of systemic gas embolism as they are usually seen in the sport diving community (9).

Decompression sickness was originally classified in caisson workers as Type I (predominantly joint pain) or Type II (predominantly spinal cord lesions) (10). Although incomplete, this classification is still in use to describe DCS in divers. Severe DCS (Type II) is usually associated with large deviations from accepted decompression protocols or greater amounts of omitted decompression. In general, prompt treatment of any type of DCS results in a good-to-excellent outcome (11, 12). Brain injury is rare in typical DCS, a finding that has received comment in the past (13, 14).

In addition to DCS and AGE in the sport diving community there seems to be a combined clinical syndrome that we report here. The mechanism for this syndrome has been suggested in animal studies (14) and from well-established principles of gas nucleation in fluids (15).

The syndrome begins with typical events that lead to pulmonary barotrauma but also demonstrates severe and unexpected symptoms of spinal cord injury, which are not typical of AGE but are typical of DCS. The combined syndrome appears in divers who, at the completion of a moderate depth air dive that is well within prescribed safe limits [not requiring decompression stops from U.S. Navy air tables (16)], develop pulmonary barotrauma during the ascent from that dive. These individuals manifest a severe, diffuse form of decompression-related illness that would not have been expected from the dive profile.

We present here 3 cases of decompression-related illness characterized by a severe and relatively refractory neurologic syndrome involving the brain and spinal cord. In each case the illness presented with cerebral symptoms typical of AGE and was resistant to recompression therapy. All of the dives were well within prescribed safe limits (16) for the depth and time of the dive and did not require decompression stops. Additionally, 2 of the 3 cases had exposures so short as to reasonably preclude even asymptomatic venous gas emboli. Severe spinal cord neurologic deficits persisted in 2 patients, and 1 patient died. The combined syndrome suggests that presence of an antecedent AGE may initiate or augment the process of DCS in individuals who otherwise would not have sustained this latter complication.

Clinical observations from these cases suggest that an alternative to standard treatment (16) for DCS and AGE may be needed because the patients are likely to deteriorate during therapy.

## CASE REPORTS

### Case 1

A 32-yr-old female made a sport scuba dive for 7.5 min to 130 fsw. Just before beginning ascent, the patient had a spontaneous episode of severe coughing. Her

ascent, verified by her partner, was at a "standard" rate. Approximately halfway to the surface, the patient had another, more severe paroxysm of coughing but continued her ascent. On the surface she reported right pleuritic chest pain, chest tightness, dyspnea, dizziness, and disorientation.

She was brought to the local dispensary where the previous symptoms of dizziness, disorientation, chest tightness, and shortness of breath were no longer present. She was noted to have hyperflexia with clonus of the right patellar and Achilles' tendon reflexes and a positive Babinski sign. A chest radiograph revealed no pneumothorax. She was transported to a hyperbaric chamber and recompressed to 165 fsw. Fluids and steroids were administered intravenously. Total time from surfacing to the beginning of recompression was 40–45 min.

Repeat examination in the chamber revealed anesthesia bilaterally to the level of T4, with flaccid paralysis of both legs. While at depth, the sensory level improved to T10, but there was no other improvement in her neurologic status. She remained with bilateral absence of all reflexes in the lower extremities, loss of motor function in the lower extremities, and inability to appreciate pain or light touch. Treatment with USN Table VIA progressing to Table IV with multiple repeat hyperbaric treatments for 2.5 wk produced slight improvement.

One month after injury she had sensation down to L1 with dysesthesias below that level. Although able to move her lower extremities slightly, she had profound weakness in all muscle groups. Bladder catheterization remained necessary. One year after the accident, she was able to ambulate but was still markedly weak, and bladder catheterization was required.

**Case 2**

A 40-yr-old male sport scuba diver made a 70-ft maximum depth dive (average depth 65 ft) for 20 min. He remained on the surface 1 h and 45 min and made a second dive to a maximum depth of 60 fsw for 20 min in a heavy current, and ascended early from the dive to avoid it. At a depth of 6–8 fsw, he experienced difficulty and rapidly surfaced. By the report of the other divers, he became unresponsive for approximately 20 min. Upon regaining consciousness, the diver described himself as having blurred vision and being unable to move his legs or trunk. After 45 min, the diver was able to walk without assistance, and a short time afterward was able to walk normally. He noted at that time, however, that his thighs and legs were weak.

Later that evening, the diver developed weakness and paresthesias in his legs and was unable to urinate spontaneously. After 30 h with these symptoms, he was able to void with difficulty but felt he had not completely emptied his bladder.

A day later he sought medical attention and received two hyperbaric oxygen treatments. The symptoms of weakness resolved with this treatment, but severe lower extremity paresthesias persisted.

**Case 3**

A 42-yr-old male diver made a 60-ft sport dive. After 40 min at depth, he developed nausea and vomited, prompting a rapid ascent to the surface. Upon reaching the surface and attempting to enter the dive boat, he appeared disoriented and rapidly became unconscious. He was evacuated to a local chamber and placed under treat-

ment within 45 min of arriving on the surface. Treatment was to 165 ft using USN Table VIA. Upon arrival at depth, he regained consciousness but demonstrated a profound paralysis of both upper and lower extremities, slurred speech, and urinary retention. At the end of the treatment regimen, he was noted to have quadraplegia, diffuse sensory abnormalities, slurred speech, disorientation, and urinary retention. After two additional treatments with USN Table VI locally, the patient was evacuated to a larger medical facility and treated with repeated USN Table VI therapy. Adjunctive therapy consisted of i.v. fluids, steroids, and prophylactic heparinization. Minor improvement of neurologic symptoms was noted after daily recompression treatments with USN Table VI (16). On the 5th hospital day, the patient developed a cardiac arrest and could not be resuscitated. Postmortem evaluation demonstrated ischemic necrosis of the frontoparietal lobes of the brain and thoracic-lumbar and sacral spinal cord. Massive pulmonary embolism was demonstrated as well. The chambers of the heart were examined and no abnormalities were detected.

## DISCUSSION

These cases demonstrate an unusual relationship between apparent pulmonary barotrauma with associated cerebral air embolism and spinal cord DCS. This combination occurred following air dives conducted within what are conventionally considered to be safe limits for no-decompression diving (i.e., no requirement for decompression stops). This syndrome resulted in the development of severe neurologic DCS manifested as spinal cord injury, which was generally refractory to treatment with either a 60-ft oxygen treatment table or a 165-ft combined air and oxygen treatment table.

An additional case was published in a recent Undersea Medical Society Workshop (17) and is recounted by the victim who is a biomedical scientist. In that case, a similar series of events occurred after a "no-stop" air dive complicated by apparent mild pulmonary barotrauma which resulted in a brief period of unconsciousness. The initial recovery was followed by progressive and profound quadriplegia over a period of several hours. Although adequate treatment was provided, the subject sustained a severe neurologic deficit and recovered slowly. The combined syndrome resulted in a permanent spinal cord neurologic deficit.

Neurologic DCS has been described in sport divers who exceed prescribed decompression schedules (18). The usual scenario in this population is a typical lesion of the spinal cord which responds to recompression treatment (19). The cases described here do not adhere to this classic pattern and require alternative explanations to account for the severity of the illness.

The development of severe diffuse spinal cord involvement suggests that either a large amount of inert gas moved from soluble to free phase or that some other mechanism compromised blood flow in the spinal cord. The adherence to presumably safe "no-stop" limits suggests that this severe form of DCS could develop only with another initiating process.

Pulmonary barotrauma which allows even small amounts of free gas to enter the arterial circulation is likely to initiate the extensive bubble growth that produced extensive damage in these cases. We can see two mechanisms for this to occur. First, free arterial gas can cause tissue injury via a possible seeding of gas nuclei throughout

the vascular system with subsequent rapid growth of free gas bubbles in blood and tissue (20) or, second, this injury could occur through alterations in structure or function of proteins in the blood occurring at the blood-bubble interface of the AGE. We feel it is unlikely that the entire clinical syndrome we have observed can be explained by the initial entry of respiratory gases into the pulmonary venous circulation alone. Previous research (14) indicates that spinal cord damage in decompression syndromes is not caused by direct arterial embolization. In addition, AGE associated with submarine escape training where no inert gas loading of tissues is present involves the brain and not the spinal cord (7). We believe that the initial symptoms in these cases referable to cerebral dysfunction are best explained by direct arterial gas embolizations to the brain. However, another mechanism must be found to explain the associated spinal cord dysfunction.

The seeding of gas nuclei is one way to explain the secondary deterioration focused in the spinal cord. This mechanism could occur only in divers with some degree of tissue gas supersaturation resulting from uptake of inert gas while diving. The seeding of bubbles into the vascular system from air embolism in this situation would allow rapid growth of bubbles in the venous system as the arterial bubbles pass through the peripheral circulation and into the venous circulation (21). This rapid growth of bubbles in the venous circulation would occlude the drainage of the spinal cord, compromise spinal cord flow, and further augment growth of interstitial bubbles within the spinal cord. Previous studies have demonstrated that spinal cord injury from DCS is due in part to occlusion of the paravertebral veins and reduction of venous outflow from the spinal cord (14). Autopsy evidence from case 3 indicates that massive spinal cord damage occurred. This lesion is in keeping with the combined venous occlusion and interstitial bubble growth hypothesis (14). In general, cerebral air embolism that attacks the brain does not manifest symptoms of spinal cord injury (2). However, free gas in the arterial system will allow seeding of small bubbles through the peripheral circulation into the venous system (14) with the possibility of an extensive gas phase evolution because of the intravascular gas nuclei (20).

Alteration in structure and function of proteins, either alone or in conjunction with bubble nucleation, can also explain the secondary deterioration in the spinal cord found in these cases. Once alterations in the structure or function of proteins occur at the site of AGE (22–24), these altered proteins, when distributed throughout the body by the vascular system, might interact strongly at the site of gas phase separation induced by decompression. Gas phase separation has been demonstrated in asymptomatic divers (25); thus the altered proteins produced at a remote site, acting additively or synergistically with what would otherwise be asymptomatic gas bubbles forming in the epidural plexus, might produce the symptoms of spinal cord DCS. If the major factor in the venoocclusive process were activation of coagulation cascade by altered blood proteins (26), the relative resistance to standard recompression therapy would be explained.

Whatever the cause, the combination of AGE and supersaturated inert gas in tissues seems to produce a clinically severe illness not typical of Type II DCS of sport divers. Inasmuch as the final outcome in the cases presented here is often poor, combined DCS and AGE may require more aggressive treatment than the standard 60-ft oxygen (U.S. Navy Table VI) or 165-ft combined air and oxygen (Table IV or VIA) that are usually considered adequate therapy for DCS or AGE (16).

This syndrome is not rare in the diving community nor is it new. Rather it has been unreported because most previous large series of AGE have been published from the

experience at submarine escape training facilities. In such facilities, gas loading does not occur before the incident, and therefore the complication of superimposed DCS does not occur. This syndrome may also explain observation of "delayed deterioration" in AGE (27). A report by Kizer (28) of sport diving accidents described several AGE cases but did not indicate that severe DCS developed from dives considered to be within safe limits.

This syndrome is neither uncomplicated AGE nor uncomplicated DCS, but the combination of both. We suggest that AGE is the initial event, which then triggers and is followed by severe DCS. In uncomplicated DCS, the initial symptom of unconsciousness (or the other cerebral signs and symptoms) would be extremely rare, and although undoubtedly cerebral DCS is a real entity (29), our own experience and that of others (14) suggests that arterialization of inert venous gas emboli is distinctly unusual. Moon et al. (30, 31) suggested that patent foramen ovale (PFO) may be an important factor in severe DCS. Their data included 30 patients with DCS, 27 of whom had serious (Type II) symptoms. The 27 can be divided as follows:

| | Severe DCS | Moderate DCS |
|---|---|---|
| PFO | 11 | 0 |
| No PFO | 7 | 9 |

In divers with severe symptoms defined to include cerebral signs and symptoms, there was a near even distribution of subjects with and without PFO. In divers with less severe Type II DCS (sensory or sensory plus pain), none had evidence of a PFO. The work is suggestive, but does not establish a contribution of PFO to cerebral abnormalities after diving. In addition, controls were used from other studies that did not test all subjects with a Valsalva maneuver. More recent data testing normal nondivers with a Valsalva maneuver show that 20–25% of normals will demonstrate shunting from a PFO (32). This is in keeping with postmortem data showing that 30% of subjects have a PFO (33).

In the cases reported herein, the air to the brain is most likely to have arisen from the lung and not from the right atrium. Timing provides evidence for this conclusion. If DCS and PFO caused the cerebral symptoms, there should have been a delay after surfacing to the onset of symptoms, whereas a lung overpressure incident would immediately inject air into the circulation and result in embolism. We have described several other cases where this mechanism seems likely (34).

Thus, the initial cerebral symptoms noted in these cases are compatible only with AGE. Similarly, the finding of subsequent spinal cord injury in these cases is compatible only with DCS. Paraparesis in uncomplicated AGE is extremely rare. In a series of 88 cases of AGE, Elliott et al. (2) reported no cases of paraparesis. Similarly, this symptomatology cannot be explained as a reperfusion injury because the late injury should recur at the site of the initial injury. In the reported cases, brain injury was followed by spinal cord injury. A similar argument eliminates cerebral or cord edema (27) as a cause of the syndrome. Repeated embolization to the cord is also possible. For that to occur would require bilateral embolization to the same level of the spinal cord without embolization occurring to any other portion of the body. Such a combination is statistically unlikely.

In conclusion, we have observed a previously unreported clinical syndrome characterized by severe DCS subsequent to AGE after pressure-depth exposures that would not be expected to produce DCS. We postulate that AGE may precipitate or

predispose to this form of pressure-related illness involving the brain and spinal cord. Treatment of these cases may require nonconventional approaches to achieve an acceptable therapeutic outcome.

We thank Kathleen James for her help in preparing this manuscript.—*Manuscript received March 1989; accepted June 1990.*

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