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Paraplegia and decompression sickness

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Decompression sickness may include spinal cord damage, which sometimes persists. Casual divers are exposed, as well as professionals. In this study we have reviewed the clinical records of divers admitted with symptoms of spinal cord injuries in our area in the last 6 years. This study is divided into two groups: those who fully recovered (19 divers) and those with neurological sequelae (12 divers).

Keywords: tetraplegia; paraplegia; spinal cord; decompression sickness

Introduction

Scuba diving can cause neurological damage to the spinal cord whenever a decompression incident occurs.[1–4] Our rehabilitation centre in collaboration with the Sainte Anne Hospital (HIA), in charge of the hyperbaric therapy unit, has been involved in the treatment of many divers during the past 6 years. In this study we analyse 31 patients admitted for decompression sickness with symptoms of spinal cord damage. This high number is linked to the particular geographical situation of our centre: the Var Departement is a very attractive area for scuba diving. It is possible to differentiate between the two groups, according to the resolution of their illness: one group with a fast and complete recovery, and the other with a slower recovery who sometimes had significant after-effects. We studied these two groups.

Materials and methods

The first group with a good neurological recovery
This consisted of 18 men and one woman, mean age 35 years (range 27–55 years); there were 10 casual divers, eight experienced divers and one professional diver.

Dive progression
In five cases the dive progressed without any problem. In 11 the prescribed stages had not been respected (due to bad weather conditions, currents, tiredness, etc). In three successive dives took place on the same day. In three instances patients mentioned a sleepless night the preceding day.

Onset of the first symptoms
In 17 these appeared within the first minutes of surfacing; in two they were delayed by 1 and 2 h and in one they occurred at 24 h (after a flight).

First symptoms
Fourteen presented with paraparesis, one with monoplegia (upper limb), one with tetraparesis. Eight patients complained of paraesthesiae (an isolated symptom for three of them). No complete paraplegia occurred. The average depth of dive was 43 m (range 26–65 m).

The level of the lesion at the onset was very difficult to diagnose afterwards, but in most cases the thoracic spinal cord was affected. In all of the patients therapy included treatment in a decompression chamber (one to six times). Recovery was complete in all of the patients and in the majority occurred in less than 48 h. No relapse occurred in the subjects who were reassessed after several days.

The group with sequelae
This consisted of 11 men and one woman, average age 38 (range 20–56). One was a beginner, four were casual divers, four were experienced and three were professionals.

Dive progression
In almost every dive there was an incident (some were serious and are described in the case reports). Two divers had previously experienced incomplete paraplegia, caused by decompression sickness (PDS), some years before, followed by a complete recovery. In another person (with a dive within allowed limits) pulmonary tuberculosis was discovered 2 months after the accident. In all of the cases dives had been performed with normal compressed air.

Onset of the first symptoms
These occurred twice under the water, eight times at the surface and in two subjects was delayed (6 h and 48 h).

Case reports

Case 1 A 56-year-old accomplished diver; dive depth: 40 m, no incident, but complained of tiredness before diving. Aphasia occurred initially with an upper limb monoplegia, and 2 h later paraparesis appeared. Treatment consisted of recompression at 4 h (HIA). After 6 years, walking is still difficult and he requires crutches. His maximum walking distance is 200 m. Urinary voiding is difficult and incontinence sometimes occurs.

Case 2 A 37-year-old accomplished diver; dive depth: 60 m, with a shortened stage at 3 m. Initial symptoms: tetraparesis and paraesthesiae. He was treated by recompression at 3 h (HIA). There has been a good recovery after 6 years: no walking limitations, but mild spasticity remains in the lower limbs. Spontaneous micturition is present.

Case 3 A 20-year-old beginner; dive depth 38 m, no incident. Initial symptoms: tetraparesis which occurred with a delay of 48 h, after an airplane flight. She was treated by recompression after 48 h (HIA). One year later, she can walk with crutches, has a mild spasticity and sometimes urgency of micturition.

Case 4 A 34-year-old professional coral diver; resolving paraplegia (PDS) in 1985. First dive during the morning to the depth of 90 m; second dive in the afternoon at 40 m. During the following night, paraparesis occurred. He was treated by recompression after 14 h. After 4 months, only mild spasticity remained. The patient died 3 years later during a dive.

Case 5 A 48-year-old accomplished diver; dive depth: 42 m; occurrence of tremendous pain ('like a stab') compelling him to quit the last stage at 3 m. Paraparesis occurred whilst surfacing. He was treated by recompression at 4 h (HIA). After 6 years, walking is easy but he tires quickly and above all there is a neuropathic bladder with vesicoureteral reflux. The MRI showed no anomaly of the spinal cord.

Case 6 A 36-year-old accomplished diver; resolving paraplegia (PDS) in 1983; 10 years later, due to emergency surfacing from a dive of 70 m, he presented with an immediate and complete paraplegia of T4 level. The treatment was by recompression at 14 h. After 2 years, there was no recovery. The initial MRI showed evidence of spinal cord atrophy.

Case 7 A 30-year-old occasional diver; the depth and development of the dive remain unknown; onset of paraparesis when surfacing. Delay to recompression was more than 6 h. After 1 year, still has urgency of micturition; motor power in both legs is reduced with a disturbing spasticity. However the patient is able to walk without crutches. The MRI of the spinal cord was normal 1 year after the accident.

Case 8 A 48-year-old occasional diver; dive depth: 30 m; sensation of 'coldness' during the dive; onset of paraparesis 10 min after surfacing. The recompression was performed after 3 h (HIA). After 2 years, walking remains tiring. There is mild spasticity, and also urinary and sexual dysfunction. He had a normal MRI initially and also at 6 months.

Case 9 A 24-year-old occasional diver; dive depth: 37 m, no incident. First symptoms at 3 m with transient visual loss and paraparesis. Recompression after 3 h (HIA). One year later, he is able to walk without crutches, but mild spasticity persists; has spontaneous micturition. Pulmonary tuberculosis was discovered 2 months later during his rehabilitation and was treated successfully. The MRI at 6 months was normal.

Case 10 A 37-year-old professional diver. A serious unexplained problem occurred at 37 m, causing the death of his partner under the water. Paraplegia presented when surfacing. Recompression was 12 h later (due to the remote location in Tahiti). After 1 year, the patient is able to walk a couple of meters with crutches, but needs a wheelchair most of the time. Significant urinary and sexual dysfunction still persist. The MRI of the spinal cord after 6 months was normal.

Case 11 A 44-year-old professional diver; dive depth: 55 m; at 15 m he complained of a violent pain in the back ('like a stab') and paraplegia occurred while returning to the surface. There was no incident during the dive, but he had dived to 120 m, several weeks before, during a saturation dive. Immediate recompression was performed on the boat and at HIA thereafter. The initial MRI was normal. One year later walking is still limited to 1 km with a significant and disturbing spasticity and the persistance of urinary and sexual dysfunctions.

Case 12 A 44-year-old occasional diver; dive depth: 72 m but air failure at 60 m precipitated emergency surfacing with immediate paraplegia. Recompression was 2 h later (Tahiti). Normal MRI after 6 months. After 1 year, he can walk 50 m with crutches but a wheelchair is necessary throughout the day. He has a neuropathic bladder and requires self catheterisation.

Summary

Table 1 summarises the initial motor level, evolution of ASIA/IMSOP impairment scale after 2 years and the maximum depth reached.

- The average depth of dives was 55 m, range 30–90 m; all dives were done with compressed air. The lesion level is localised between T6 and T12, but the upper limit of the lesion is always difficult to pinpoint precisely.

Table 1 Clinical data

Case	*Depth*	*Initial motor level*	*Initial ASIA/IMSOP scale*	*ASIA/IMSOP outcome scale*[a]
1	40 m	C7	C	D
2	60 m	C7	D	D
3	38 m	C8	C	E
4	90 m	T7	C	E
5	42 m	T10	D	D
6	70 m	T6	A[b]	A
7	?	T4	C	D
8	30 m	T10	C	D
9	37 m	T8	C[c]	D
10	45 m	T7	B[d]	C
11	55 m	T4	B	C
12	72 m	T9	B	C

[a]2 years later; [b]+ monoplegia (upper limb); [c]+ visual loss; [d]+ loss of consciousness

- Several MRI studies were performed; in the acute situation, all but one were normal. This was a diver who had had a paraparesis during a preceding decompression incident. The MRI showed an atrophic thoracic cord, the patient had no neurological change, and the sensori-motor paraplegia remained complete. Eight others were performed over 6 months after the incident and showed no anomaly.
- The nature of the sequelae is as follows. Motor function is always affected; in seven individuals, walking is possible without orthoses but is very tiring; in two the walking distance remains less than 500 m; in two others the maximum walking distance is limited to just a few meters and the regular use of a wheelchair is necessary. In one patient the paraplegia remains complete. A degree of spasticity is always found on neurological examination, and is often important because it disturbs function. Sensory function is always impaired (but absent only in case 6), and it is very difficult to define precise levels. Subjects always complain of feelings of numbness and of neurogenic pains. Symptoms related to the conus medullaris are always present; the effects are various with changes affecting bladder, rectal or sexual control.

Discussion

The study of these two groups of divers enables us to make several statements.

Dive depth appears to be a prominent feature in the group with sequelae. In several instances the diving depth was greater than 40 m. However this depth is not the minimum one where decompression incidents occur. Others have described incidents at depths of less than 40 m[5] and even less than 20 meters.[6] The maximum depth that is normally attained with natural air is 45 m (limited by the onset of nitrogen narcosis below this level);[7] some obvious safety errors (case 12) could be explained by nitrogen narcosis.

The divers' experience is more critical in the group with sequelae. There are perhaps two reasons for this: the spinal cord could have sustained damage during previous dives without clinical symptoms. A previous accident could have affected it more seriously than may have been thought. Two observations are in favour of this hypothesis: in case 6, the initial MRI showed spinal cord atrophy. It is possible to appreciate this as a consequence of the completely resolved incident that had occurred several years earlier. In the case of diver 11 there was no incident during the dive but it involved a diver who often reached very great depths while working (and always with heliox). Whilst emerging from his dives, he noted on several occasions a feeling of numbness in his lower limbs, without paralysis. The second reason is linked to the repetition of risky technique (even though well mastered) which increases the number of incidents.

Some incidents usually occur during the dive, with a major accident. Some were the consequence of obvious mistakes.

The interest in case 9 lies in the discovery of progressive tuberculosis with apical infiltration and cavities presenting 2 months after the accident. The original chest radiograph was normal, but the patient complained of unusual tiredness which had appeared several months before the diving incident. This previous pathological state, subclinical at the time, could be held responsible for the accident occurring during decompression.

The time of onset of the symptoms is always rapid, in both groups usually when the divers emerge from the water. In the group with serious incident, we found that in three patients, the first symptoms appeared under water (5, 9, 11); and in three others (6, 10, 12) return to the surface was as an emergency with the immediate appearance of symptoms. Of these six individuals, four had major sequelae.

The spinal cord lesion was localised to the thoracic area between T4 and T12 with signs of involvement of the lumbosacral cord. The cervical (cases 2, 3) and cerebral (1, 9) symptoms were always transient.

Comparison with data from the literature

Predictions of this type of accident have been described by several authors[8–10], who also emphasised the possibility of spinal cord after-effects. We reached the same conclusions.

The delay of onset of the first symptoms is often very short in our series and appears to be linked to the magnitude of the complications shown by our patients. This is in agreement with the findings of Aharon-Peretz.[2]

Greer[8] introduces the concept of 'occult neurological accident' by noting the neurological symptoms during the systematic examination of professional divers. Aharon-Peretz also reports on the 'pathological features of the spinal cord even when the clinical manifestations during life were unimpressive'.[1,4,12] Palmer[13] documents the case of repetitive incidents that culminated in the death of a diver in unexplained circumstances. Autopsy revealed a thoracic spinal cord lesion even although the diver had completely recovered from a previous accident.

Case 6 shows the existence of an 'asymptomatic SCI lesion' on MR images. Case 11 also suggests an occult lesion. We can postulate the importance of some of the sequelae being due to previous lesions.

In two cases (4, 6), the subjects have had a decompression accident with a resolving paraparesis. After a second decompression accident, one of the subjects was permanently paraplegic and the other died later during a dive. These two patients had specific psychological profiles that contributed to non-observance of the security parameters of the dives.

The spinal cord lesions combined sensory, motor, urinary and sexual symptoms. The pathophysiological theories suggest both arterial and venous compromise with a particular vulnerability of the territory irrigated by the artery of Adamkievicz.[7,14,15] It is now admitted that the patterns of neurological decompression illness

into cerebral air embolism and type 2 decompression sickness are arbitrary. The classification of these accidents must be descriptive.[16–18]

Regarding therapy, the consensus is to treat the patient as quickly as possible with overoxygenated decompression at a moderate pressure.[18]

Conclusion

These different types of patients and all of the features that they exhibit underline the dangers of decompression accidents. The relative incidence remains small but the absolute number is increasing in our area due to the attractiveness of the local underwater sites. Professional divers appear to be exposed more often than would usually be expected. It appears to be important to underline these facts because of the seriousness of the long term neurological consequences.

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