Eight days previously, during the rehearsal for a military funeral, A. B. was acting as a member of a coffin-bearing detail, being on the left-hand side of the coffin. He therefore had his right arm abducted until parallel with the ground, with the hand round the back of his opposite number's neck, his own neck being slightly flexed to the left. Unfortunately the latter stumbled and the extra weight of the coffin was suddenly transmitted to A. B.'s shoulder at a place midway between the point of the shoulder and the junction with the neck. He continued with the rehearsal for a further two hours in some discomfort, and noticed later in the day that he had pins-and-needles in the arm. Next day there was weakness of the limb, and a small transient bruise had appeared on the shoulder.

This was the day of the actual ceremony, so he was replaced by C. D., who fared little better: the same individual on the opposite side of the coffin repeated his mistake of the previous day and lost his balance, with precisely the same effect. C. D. continued with his duty for a further 15 minutes, but after this he immediately observed weakness of his right arm.

When seen, neither had noted any return of function over the succeeding week. There was no clinical evidence of the injury to the shoulder or cervical spine in either patient, though x-ray films showed a fracture of the tip of the spinous process of C 7 in one of them (A. B.).

A. B. showed the features of a right C. 5 to 8 lesion with marked weakness and wasting of biceps and triceps and to a lesser degree of the deltoid and forearm muscles, with some weakness of grip. There was sensory blunting (and subjective paraesthesiae) of the thumb and index finger. No tendon reflex could be elicited on the affected side. There was no sympathetic involvement.

C. D. had a more extensive and severe defect. The weakness and wasting extended from C 6 to T 1 inclusive, the pectoralis major being slightly wasted. Abduction of the arm was initiated by the "trick movement" of swinging it outwards with his body, suggesting weakness of the supraspinatus, though the deltoid appeared to function normally once the arm was up. Biceps, triceps, forearm, and hand muscles showed varying degrees of weakness and wasting, and apart from a rather feeble triceps jerk the tendon reflexes were

absent. The superficial sensory deficit was more severe, and extended from the sixth cervical to the first thoracic dermatome inclusive. There was again no evidence of Horner's syndrome.

The clinical features were those of a subtotal brachial plexus injury in each case. Fortunately, recovery began some three weeks after the injury, so no actual avulsion of nerve roots had occurred. Nerve-conduction studies and electromyographic findings suggested that the type of damage was predominantly axonotmesis in A. B. and neurapraxia in C. D. Three months later recovery was almost complete in all muscle groups in both subjects.

COMMENT

Two cases of transient brachial plexus palsy are described in which the circumstances leading to the injury were unusual. The mechanism of injury was the sudden transmission of a considerable weight to the shoulder, a well-described cause of damage to the upper plexus. This part of the plexus was, in this situation, more than usually vulnerable on account of the slight flexion of the neck to the opposite side and the sudden accentuation of this position at the moment of impact. In these two cases there was evidence of involvement of the lower plexus as well, and it is suggested that this may have been due to the extended and abducted position of the arm at the time of the accident.

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Decompression Sickness : Paradoxical Response to Recompression Therapy

Brit. med. J., 1966, 2, 809-810

The essential treatment of decompression sickness is recompression, a treatment which is so effective that in doubtful cases it may be considered to be a diagnostic measure. A case is presented in which recompression to 6 atmospheres absolute¹ was associated with a serious and rapid deterioration of the patient's condition.

CASE REPORT

A member of a Royal Naval deep-sea diving team aged 26 dived on 4 June 1965 in a submersible chamber to 600 ft. (183 m.) (266.6 lb./sq. in., 18.74 kg./sq. cm. gauge) for 30 minutes, breathing a mixture of 5% oxygen and 95% helium. For 12 minutes he swam and worked outside the chamber. Initial decompression was carried out in the submerged chamber, and then he was transferred under pressure to a dry chamber on board ship, where the oxygen was enriched to 20%. At 19.09 on 5 June, after 21 hours 49 minutes' decompression on the experimental schedule, he developed a slight pain in both knees when the pressure was reduced from an equivalent depth of 70 to 50 ft. (21.3 to 15.2 m.) sea-water. At 19.35 the pain was increasing and he was therefore treated by recompression to a depth at which the pain was completely relieved. Here, at 75 ft. (22.8 m.), he remained for one hour. Subsequent decompression was by steady release of pressure

at the rate of approximately 10 ft. (3 m.) (4.4 lb./sq. in., 0.31 kg./sq. cm.) per hour. At 02.43 on 6 June at 27 ft. (8.2 m.) there was a recurrence of the aching in both knees, but relief was complete nine minutes after pressure had been increased to 30 ft. (9.1 m.). After 30 minutes at this depth decompression was continued at a slower rate than before and there was no recurrence. He arrived at the surface at 10.50, and five minutes later emerged symptom-free from the oxy-helium atmosphere of the chamber into air.

At 12.30 he complained of some difficulty in controlling his legs when getting out of a bath. He had no recurrence of pain, but there was now a feeling of "woolliness" in both legs and some numbness over the left knee. On examination there appeared to be no paralysis or weakness of the lower limbs. He had no ataxia. There was a small recent contusion on the medial aspect of the right knee which he did not remember acquiring. Power, tone, and movement of the two sides were equal. Apart from an area about 3 in. (7.5 cm.) in diameter of diminished tactile and pain sensibilities over the lower medial and anterior aspect of the left knee the sensory system was normal. Respiration was also normal.

The patient stepped into the recompression chamber at 12.40 and pressure was applied with compressed air in accordance with the Royal Naval Diving Manual (B.R. 155, 1964). During the simulated descent he complained of a sudden recurrence of pain in both knees at 60 ft. (18.3 m.) and this steadily became worse as pressure was increased to 165 ft. (50.3 m.), the maximum depth recommended for treatment. At this pressure (73.3 lb./sq. in., 5.15 kg./sq. cm.) he complained of "pins-and-needles" in both feet and of severe pain in both knees, both groins, and the left ankle. The contusion on this right knee had swollen to become a mottled red elevation about 2 in. (5 cm.) in diameter. A small bruise was noticed on the medial aspect of the left knee posterior to the area of numbness, which was unchanged, and another on the lateral aspect of the right knee. There was almost complete loss of voluntary movement of the lower limbs. Respirations had become

¹ 1 atmosphere absolute is approximately equivalent to 14.7 lb./sq. in. (1.03 kg./sq. cm.) and the pressure exerted by 33 ft. (10 m.) of sea water.

shallow and rapid, with occasional sighing gasps. The patient was pale and he vomited. He remained fully conscious and alert. After a few minutes it was obvious that there would be no immediate improvement of his condition at this pressure. It was decided that the standard therapeutic recompression schedule should be abandoned and preparations were made to take him to the maximum depth of the chamber (450 ft., 137 m.).

At 13.01 he was given a mixture of 5% oxygen and 95% helium to breathe through a mouthpiece, and the pressure was increased to 300 ft. (91.4 m.) (133.3 lb./sq. in., 9.37 kg./sq. cm. gauge). He continued to vomit and was unable to use the mouthpiece continuously. This meant that the pressure could not be increased further, since the compressed air in the chamber would be dangerously narcotic if breathed for long at greater depths. Fortunately he was soon able to retain the mouthpiece, and after a latent period of a few minutes improvement in his condition began. After 20 minutes, voluntary movement, though weak, had returned and, apart from the left ankle, all pain had gone. At about 13.40 he was fully recovered.

Decompression began at 15.07 at a steady rate of approximately 10 ft. (3 m.) per hour. The chamber atmosphere was changed from compressed air to 5% oxygen and 95% helium at the earliest opportunity and the oxygen content raised to about 15% at 120 ft. (36.6 m.) (09.15, 7 June). The rate of release of pressure was then slowed to approximately 7 ft. (2.1 m.) per hour. At 95 ft. (28.9 m.) there was a recurrence of pain in the right knee, which was relieved soon after recompressing to 110 ft. (33.5 m.). There were no other abnormal symptoms or signs. After an hour the decompression was resumed, but was halted for a further hour at 104 ft. (31.7 m.) while a dull ache at the same site subsided. The rate of decompression was slowed progressively towards the surface. The oxygen was raised to 21% at 50 ft. (15.2 m.) (05.30, 8 June) and the patient reached sea-level at 21.55, but remained breathing oxy-helium until he emerged from the chamber at 22.15. Apart from slight bruising on the medial aspect of his right knee there were no residual signs or symptoms. There was no further recurrence.

COMMENT

It would seem that the sudden worsening of the patient's condition was a result of the treatment, although there is also the possibility that the coincident timing of recompression and deterioration was due to chance. However, with the latter explanation it is necessary to accept that if he had had no recompression his condition would have become rapidly very much worse than in fact it did during these six minutes. This is because any gas present as bubbles in the tissues when he was paraplegic at 165 ft. (50.3 m.) (6 atmospheres absolute) would have occupied at sea-level six times the volume it had when compressed.

The limb-pains ("bends") of decompression sickness may become worse during recompression (Hoff and Greenbaum, 1954; Behnke, 1965; own unpublished cases), but this effect is usually transient and may be ameliorated by slowing the rate of compression until the depth of relief is reached. There is no case reported in the literature in which recompression has been associated with the onset of pain in previously unaffected sites, the swelling of bruises, or the worsening of the neurological manifestations of decompression sickness.

It is generally considered that the response of decompression sickness to treatment by recompression is evidence in favour of the illness being caused by the presence of gas bubbles. If recompression was the cause of the patient's rapid deterioration then some explanation of this apparently contradictory evidence must be offered. However, it must be remembered that the pathological mechanisms of decompression sickness are little understood, for, unless treatment is delayed, the manifestations are often even more transient than they were in this case.

It can be suggested that the increase of pressure would reduce the diameter of bubbles held up, for instance, at the pulmonary capillaries, allowing them to escape as multiple emboli throughout the arterial system. Against this is the absence of the symptoms and signs of pulmonary bubble embolism ("chokes") in the patient prior to treatment and the absence of any obvious evidence of cerebral embolism during recompression. Alternatively, the mechanism could be that of the bubbles, predominantly helium, expanding rapidly owing to the diffusion into them of nitrogen as this returns to the tissues. To support this there is a clinical impression, but no sound evidence, that the addition of nitrogen by breathing air during the last stages of helium elimination has an adverse effect on the course of decompression sickness. For instance, it has been noticed that if, after a helium dive, a diver has a minor joint pain ("niggle") on arrival at the surface, this tends to get better if he remains on oxy-helium but to get worse if he changes to air. To extend this to the worsening of helium-induced decompression sickness by rapidly increasing nitrogen tensions is hypothetical, but is a reminder that the detailed dynamics of decompression sickness are completely unknown. The mechanism suggested by Behnke (1965) for the worsening of limb-pains on recompression may be considered as a third possibility. As pressure is increased so the volume of any bubbles present must be reduced, the space vacated being reoccupied by blood or tissue fluid. If this occurs in a relatively rigid tissue such as bone a "squeeze" may occur in which the relatively low pressure is relieved at the cost of some tissue damage; but this theory is difficult to apply to bubbles in the spinal cord.

Although no single explanation is sufficient for all the manifestations of this paradoxical case, the patient's complete recovery is an important reminder that the essential treatment of decompression sickness is *adequate* recompression. Most symptoms are relieved by a pressure less than the depth of the previous dive, and most dives are made to a depth less than that at which treatment is recommended. The therapeutic depth of 165 ft. (50.3 m.) was chosen by Van Der Aue *et al.* (1945) as the optimum pressure for therapy. Their therapeutic recompression tables, though modified, are now in general use and are often adequate for decompression sickness following both oxy-nitrogen and oxy-helium dives deeper than 165 ft. (50.3 m.). However, with the increasing development of deep-sea diving for commercial use, it must be emphasized that a patient with decompression sickness should be recompressed until symptoms are completely relieved, and, if necessary, with oxy-helium to the maximum depth of the chamber available.

We are grateful to the Medical Director-General of the Royal Navy, Surgeon Vice-Admiral Sir Derek Steele-Perkins, for permission to submit this paper for publication.

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