# DIVING DOCTOR'S DIARY

## WAS IT DECOMPRESSION ILLNESS ? PROBLEMS WITH DIVING AND DOCTORS

Carl Edmonds

**Key Words**

Case history, decompression illness, medical conditions and problems.

### Case history

A 45-year-old slightly obese, experienced female diver was treated for two episodes of acute decompression illness, neurologically static, which responded to repeated hyperbaric treatments.

This enthusiastic lady has been diving for five years, mainly in warm waters and at moderate depths, rarely exceeding 18 m. She employed a dive computer, but never entered decompression. Two years ago she undertook her first diving holiday, on a live-board, undertaking multi-day and repetitive dives.

The first episode of decompression sickness was noted after 3 days on board. It occurred a few hours after the diving, and comprised paraesthesia over the first, second and third fingers and associated joints of the right hand, together with some pain and discomfort, weakness and clumsiness affecting the hand. The left hand was also affected to a slight degree, mainly involving the thumb and first metacarpophalangeal joint.

Some relief was noted during further dives, but it was not until the diving had been completed, and she had been given three oxygen recompression therapy sessions that she really improved. Even then, the improvement was gradual.

Single dives carried out over the next year were uneventful, but then a return to live-board diving caused a recurrence of almost identical symptoms and progress. The only addition to the sequence of events was a slight worsening of symptoms during the altitude exposure, flying back to Sydney.

The reason for her consultation was to determine whether or not her diving activities should be altered.

### Diagnosis

As so many of the current diving physicians are taught, any diver with symptoms following a dive should be treated as if they have acute decompression illness (the fashionable terminology). Indeed, some of the characteristics of decompression sickness are present in this case i.e. excessive diving exposure, relief with re-immersion, improvement with recompression therapy, aggravation by altitude exposure. Mild obesity and age were predisposing factors.

If, instead of employing the venerable diving principles espoused by our predecessors, including a "trial of pressure", one employs the more traditional Oslerian medical approach to the case, a different pattern emerges.

She gave a history of very mild hypertension, osteoarthritis and the development of peripheral oedema, over the previous two years, fully investigated and not shown to have any cardiac, renal or metabolic aetiology. Her oedema had previously been very well controlled on Moduretic (amiloride 5 mg and hydrochlorothiazide 50 mg).

On examination it was evident that some of the symptoms could be replicated by pressure over the carpal tunnel, and the sequence of events then became clear.

As suggested by her physician and her diving instructor, she had stopped taking the diuretic during the period of intense diving on the live-aboard. This had resulted in a gradual redevelopment of her peripheral oedema, and the production of a typical carpal tunnel syndrome, worse on the right side. Possibly immersion assisted in reduction of the peripheral oedema, also reducing the symptoms, at least for a few hours, then the oedema redeveloped. The exacerbation of her symptoms during flight might be related to the aggravation of the ischaemic affects of the carpal tunnel pathology.

Once she had finished diving (and also during the period of recompression therapies) she resumed the diuretic regime and thereby relieved her symptoms.

### Prognosis

A repeat of the investigation for the cause of the oedema was unrewarding, it now being classified as idiopathic (not that uncommon in middle-aged females). She now continues her diuretic regime during her diving activities, and has had no recurrence of the "bends". As her diving was, even previously, extremely conservative and within no-decompression limits, it was not thought necessary to modify this.

### Background

The carpal tunnel syndrome is an entrapment neuropathy of the median nerve at the wrist. Paget first

described this in 1854 in a patient who sustained a fracture of the distal radius. It has recently been reviewed by Slater.[1] The carpal tunnel is enclosed by the bones of the wrist, roofed over by the flexor retinaculum fascia. It contains nine tendons and the median nerve which lies most superficially, immediately under the ligament. The basic physiology is a reduction in the epineural blood flow which occurs with compression of 20-30 mmHg.

It may be aggravated by arthritis affecting the wrist joints, positioning of the hand and wrist, oedema of the enclosed tissues, or anything else which compromises the space available for the median nerve in the tunnel. Thus local causes, such as radius fracture, blunt trauma with haemorrhage and swelling, various tumours, systemic illnesses, metabolic diseases, overuse syndromes and aberrant anatomical structures have been incriminated.

Support for the diagnosis is based on involvement of the median nerve distribution and referral area (symptoms related to the first three fingers, thenar eminence, wrist, arm and even up to the shoulder). Pressure over the compressed nerve area, either directly or by reducing the venous return, may provoke the symptoms (Tinel's sign).

**Discussion**

This case highlighted three dubious diving medical dictums.

1 *Symptoms following diving should be classified as decompression sickness*. This may have been so during the early days of diving, when most divers were young fit males, usually in the Navy and often employing less reliable decompression regimes, and with a diving physician and recompression chamber nearby. Under those conditions, statistically it was probable that most symptoms would be diving related, encompassing decompression sickness.

Nowadays, many of the diving population more closely resembles the general practice population. This includes a wide age range, with many people having a variety of illnesses, which can develop in relationship to, or coincidentally with, the diving activities. Thus it is essential to question every diving accident regarding their past medical history.

A similarly with medication usage. The diving population now has a much wider exposure to both therapeutic and other medications, which can produce side effects that can mimic decompression sickness. Typical medications include parasympathomimetic drugs, anti-malarials, carbonic anhydrase inhibitors and many others.

2 *A trial of pressure (recompression therapy) will clarify the diagnosis*. It often does not. Often the situation in which the recompression therapy is administered will result in an improvement in a variety of conditions. This case is an example. A more typical example would be the paraesthesia associated with anxiety and hyperventilation.[2] This affects up to 6% of the Australian population. With the increased density of air in the chamber, and often the restriction to breathing from masks, the hypocapnoea produced paraesthesia may be rectified in the chamber environment. A third example is hypoxia from any cause, including salt water aspiration.

Also, especially now that multiple treatments are given, the effect of time may ameliorate many acute illnesses. Most people get better from other illnesses, as well as from decompression sickness, with time.

3 *Exposure to altitude will aggravate decompression sickness*. Although this may certainly occur, especially if there are persisting tissue bubbles, it is unlikely to be a factor if adequate oxygenation has been given and the patient rendered free of bubbles and denitrogenated. Nevertheless, exposure to altitude can aggravate any neurological manifestation[2] for many reasons (hyperventilation, hypocapnoea, alkalosis, hypoxia).[3]

**Conclusion**

Despite the oft quoted diving medical dictums it may be more relevant, now that diving physicians have to cope with a general practice type population, to give more credence to traditional approaches to medical diagnosis.

**References**

1 Slater RR. Carpal tunnel syndrome: current concepts. *J South Orthop Assoc* 1999; 8 (3) [taken from <http://www.medscape.com/SMA/JSOA/1999/v08.n03/soa0803.07.slat/soa0803.07.slat-02.html>]

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*Dr Carl Edmonds MRCP, DDHM, who was the Foundation President of SPUMS, is Director of the Diving Medical Centre, 66 Pacific Highway, St Leonards, New South Wales 2065, Australia. Phone +61-(0)2-9437-6681. Fax +61-(0)2-9906-3559.*
*E-mail <divmed@ozemail.com.au>.*