The diving doctor’s diary

Case report. Flying after diving

Carl Edmonds

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Case reports, decompression sickness, barotrauma, flying (and diving)

Prologue

I have previously used SPUMS case reports as a forum for illustrating new or poorly recognised clinical features of diving disorders. I have taken the liberty of using this report as a teaching aid/discussion case. Some details have been deliberately altered to avoid embarrassment to certain colleagues. My dictatorial attitudes have been incorporated in italics.

SB was an enthusiastic and experienced diver who had rapidly progressed through the certification system and was well qualified to undertake deep or prolonged salvage diving, using dive computers. Nevertheless, he tried to stay just within no-decompression limits, because of apprehension regarding decompression sickness (DCS), which he had experienced in the past. Unfortunately in this instance he did require decompression from an energetic multi-level salvage dive. The maximum depth was 40 metres’ sea water (msw) and it was a repetitive dive.

During the 5 msw stop he was feeling a bit tired and was developing a deep central headache. He had noticed this on a previous dive, but it had cleared up without incident. This time it progressed during the final ascent and was severe after reaching the surface.

As he was diving on a Pacific Island about three hours’ flight time from Australia, he contacted an Australian diving emergency system and explained the dive parameters and his symptoms – headache, tiredness and a “fuzzy feeling” in his head.

Transport was immediately available, so after an hour or so on oxygen (O2), which seemed to impart some relief, he was flown to Australia on a commercial flight. During the flight, on which he was not allowed oxygen, his headache became more severe.

Surface oxygen: After a decade or two’s delay, the USA and UK experts have now indisputably validated the 1970s Australian/French advocacy of this regime. However, prudence is still required. A previous diver medevac from the Cook Islands to Tahiti, resulting in the explosion of the plane with the death of all on board, was possibly attributable to a higher than normal cabin oxygen percentage.

On landing, he was taken to a recompression chamber (RCC) and given an extended US Navy Treatment Table 6 (USN 6), with good results. It was extended when the symptoms seemed to recur during the final ascent from 9 msw to the surface.

As he had no residual clinical features, he was permitted to return to New Caledonia one week later.

Diagnosis: acute decompression illness

Terminology: I think they meant DCS. I have some difficulty with the various inclusions and exclusions of the “decompression illness” appellation. I do not think they were implying cerebral arterial gas embolism, but who is to know when terminology is so ill defined.

All went well until he was being flown back, when the headache recurred in the aircraft. He arrived in Noumea with symptoms almost identical to those he had started with (perhaps a little less severe). The whole procedure was then repeated with another medevac and USN 6 recompression treatment in Australia.

The prognostic advice was then complicated by the presumed diagnoses. The clinical features were interpreted by some as indicating a serious and acute (neurological) DCS. As such, the recommended delay before further aviation exposure was up to two months, with a permanent exclusion from diving. Most of his advisers, however, suggested a couple of weeks before flying (especially considering his aviation-induced recurrence), and a month or two before diving.

Flying after DCS treatment: If RCC treatment with 100% O2 has been adequate, no further bubbles will remain, and the tissues will be effectively de-nitrogenated, so it is difficult to understand how altitude exposure can aggravate the basic pathology of DCS. This is especially so if the treatment has been instituted without air breaks (this adds more nitrogen to bubbles and tissues) and if the asymptomatic diver has breathed 100% O2 intermittently on the surface post-treatment for a number of hours.

To be ultra-conservative, and to avoid the possible aggravating factors of mild hypoxia, alkalosis and hypocapnoea associated with commercial aviation, an oxygen mask could be used during the flight, with allowance made for adequate ventilation.
If, however, the RCC treatment has been inadequate to completely remove the gas phase from the tissues, then the duration before safe flight will be proportional to the incompetence of the treatment. Some recompression facilities advise weeks’ or months’ delay before aviation exposure.

He consulted me because of the conflicting advice regarding an appropriate time delay before resumption of flying and diving; a reasonable concern considering his repeated experience and the varying advice. As I considered his oxygen recompression therapy initially to have been more than adequate, and as it was given by competent therapists (i.e., with a well-fitting oxygen mask and for a considerable duration), I decided to consider other possibilities.

A more detailed history revealed previous headaches associated with diving, some episodes of mild sinus barotrauma of descent, and the use of ‘negative pressure’ middle ear equalisation techniques. The ENT system seemed problematic, but there was no obvious non-diving ENT pathology.

After performing sinus CT scans, I advised him to return to Noumea by boat, and to change his middle ear equalisation techniques from the Toynbee/swallowing types to the conventional Valsalva, commence equalisation on the surface (pre-descent), and perform it more often (every metre or so of descent). The CT scan, performed within three days of the ‘recurrence’ and subsequent RCC ‘treatment’ revealed moderate mucosal thickening especially affecting the sphenoid sinus.

**Radiology and scanning:** This CT scan ideally could be replaced by MRI. CT and MRI have made sinus X-rays obsolete because of the explicit pathology that can now be demonstrated. Significant mucosal swelling is easily demonstrated with either scanning technique, but even with X-rays the pathology was well demonstrated in the past – if the positioning was accurate – and showed the frequency of the sphenoid pathology with diving, either alone or with other para-nasal sinus involvement. Always think ‘sphenoid or ethmoid’ when investigating diving-induced headaches.

Scans often return to normal a week or two after sinus barotrauma. That is why the consultant otolologists, who see patients ‘cold’, due to delayed referral and investigation, are at a disadvantage and may offer inappropriate reassurance. Had I been more courageous, I might have sent him home by air with advice to repeatedly perform Valsavas every minute during ascent and descent, but there was no way of assuring that the sphenoidal sinus was adequately patent, or that the advice would have been conscientiously followed.

**Para-nasal air spaces:** Some techniques of middle ear equalisation involve negative nasopharyngeal pressures – tending to induce swelling of the mucosa and narrowing of the Eastachian tube and sinus ostia. Other factors being equal, positive pressure techniques, such as the Valsalva, are less likely to be associated with barotraumas of descent, and the subsequent ascent complications. Physicians have often been deceived into assuming that middle ear equalisation affects only the middle ears. It affects the whole nasopharynx and may be very relevant to sinus equalisation when the ostia are only marginally patent.

Follow-up correspondence revealed an uneventful trip home and a successful return to diving, now employing appropriate equalising techniques.

Had the sphenoidal barotraumas not been prevented by this simple alteration in middle ear auto-inflation technique, some may have advised surgical intervention. I would not have recommended sphenoidal ostial endoscopy as a treatment (because of its potential complication rate), although I would have done so had the maxillary sinus been the one involved. This reflects a somewhat conservative attitude, as I am less concerned with operative complications in the maxillary than in the other sinuses.

**Medical literature:** The literature on sinus barotrauma in diving is less than comprehensive, but experience at a diving medical clinic at the Great Barrier Reef suggests that it is a relatively common complaint. Aviators have described some reasonable clinical series of sinus barotrauma (“aerosinusitis”) but the only two series on divers that were more than individual case reports were published in this journal. One involved 50 ‘hot’ cases as they presented post-dive at the RAN School of Underwater Medicine and the other had 50 ‘cold’ cases, a more clinically severe group, referred for treatment to the Diving Medical Centre. All these, and other relevant literature references, are to be found in the chapter on sinus barotrauma in Diving and subaquatic medicine.¹

**References**


Carl Edmonds, MB, BS, DPM, MRCP(Lond), MRCPsych, FRACP, FRANZCP, DipDHM, FAFOM

11/69-74 North Steyne

Manly, 2095 NSW, Australia

E-mail: <puddle@bigpond.net.au>

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