

## ORIGINAL PAPERS

### FLYING AFTER TREATMENT FOR DECOMPRESSION ILLNESS: WHEN IS IT SAFE?

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#### Introduction

When is it safe for a diver who has sustained an episode of decompression illness (DCI) to ascend to altitude?

In an age when many divers fly to and from their dive locations, this is a common and important question. It has been appreciated since the 1930's that altitude ascent can precipitate or exacerbate symptoms of DCI.<sup>1</sup> However, there is a wide range of opinion regarding when this altitude ascent is safe, and this variability is due to a lack of data from adequate clinical studies.

The objective of this paper is to review the literature and to suggest safe and supportable advice that can be given to divers in this situation.

#### The clinical problem

A high proportion of divers who suffer DCI have flown to their diving location. A review of patients treated for DCI at Townsville from 1977 to 1988 showed that 26% were from overseas.<sup>2</sup> A further 14% were from interstate. It is therefore common to have to advise divers on when they can fly after treatment for DCI. These divers usually feel well and are keen to travel home, so any delay of flying needs to be justified.

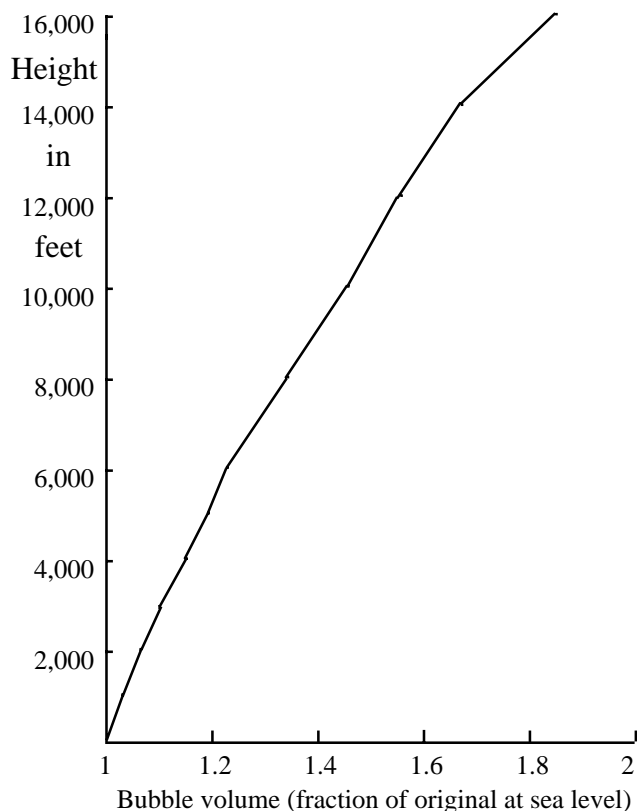
#### Bubble effects with altitude

Ascent to altitude results in a decrease in the ambient pressure. Most modern airliners, for reasons of fuel efficiency and weather conditions, fly at an altitude above 9,000 m (30,000 ft). To prevent hypoxia and for passenger comfort, the aircraft cabin is usually pressurized to give an equivalent altitude of 2,440 m (8,000 ft), at which the ambient pressure is 0.74 bar.<sup>3</sup>

Such a decrease in ambient pressure will cause any gas bubble present in a tissue to increase in volume by 35% according to Boyle's Law (figure 1). This expansion corresponds to an increase in bubble radius of 10.6%. Such changes in bubble size may appear small, but cases have been documented where such altitude exposures provoked DCI in previously asymptomatic divers.<sup>4</sup>

FIGURE 1

BUBBLE EXPANSION WITH ALTITUDE



#### Flying after diving in asymptomatic divers

Asymptomatic divers who have been exposed to reduced ambient pressure after a 3 hour surface interval have had venous bubble formation detected by Doppler at altitudes of 1,000 m to 3,000 m.<sup>5</sup> Similarly, the provocation of DCI with altitude exposure has been shown experimentally in dogs.<sup>6</sup> This risk to the asymptomatic diver decreases rapidly during the 24 hours following the dive.<sup>7</sup>

Those who go to altitude soon after diving are likely to have tissues that are supersaturated with inert gas. Ascent makes the development of bubbles, or the enlargement of previously asymptomatic bubbles, more likely. The reduction of this risk over a relatively short time span can best be explained by the elimination of much of the inert gas load via the lungs.

Conversely, if flying prior to diving is associated with the development of asymptomatic bubbles, the risk of DCI following a subsequent dive may be increased.<sup>8</sup>

This subject has been extensively reviewed recently by Sheffield,<sup>9</sup> with recommended surface intervals for flying after asymptomatic diving.

**Flying after diving followed by DCI**

The problem of flying after the development of clinical DCI differs from the situation above in two important ways. First the patient with symptomatic DCI must have developed bubbles and tissue damage before altitude exposure. Secondly, the safe time period before altitude ascent for those suffering from DCI suggested by some authors (up to 4 weeks)<sup>10</sup> is considerably longer than that for asymptomatic divers discussed above. Such an interval should allow the inert gas dissolved in the body to equilibrate with the atmospheric partial pressure of that gas. This means that it is essential to know how long bubbles can exist in tissues following their development.

Logically, it might be expected that bubbles could exist in a tissue for a relatively short time, probably for no more than several half times for that particular gas in that particular tissue. This expectation is reinforced by both mathematical and in vitro models of bubble dissolution.<sup>11</sup> Evidence exists however that this is not always the case, and that bubbles may remain in tissues for much longer.

It has long been appreciated that the presence of X-ray “streaking” in periarticular tissues can correlate with DCI.<sup>12</sup> Hills and Le Messurier (unpublished observations) followed up a diver in Adelaide using X-rays, and found that

asymptomatic tissue bubbles could still be identified 22 days after his bends-provoking dive.

Evidence also exists regarding the efficacy of delayed treatment of DCI. Divers with symptomatic DCI who delay recompression for up to 10 days can still respond with full resolution of their symptoms.<sup>13</sup> This suggests that they still had tissue bubbles. These time periods far exceed any of the theoretical half times for gas-tissue kinetics that are used for the calculation of dive tables.

**Case reports**

There are few case reports of recurrence of DCI following altitude ascent. This is probably due to these recurrences being both infrequent and under-reported. The United States Air Force (USAF) reported no cases of recurrent altitude DCI following treatment in the period 1970-1980.<sup>14</sup> This is despite a policy of allowing flying when airmen became asymptomatic, without stipulating a specific period of grounding.

However, other reports indicate that such a policy is too liberal. Recurrent episodes of altitude DCI can be considered significant and due to the presence of residual bubbles if the recurrent symptoms mimic the initial symp-

**TABLE 1**  
**SUMMARY OF OPINIONS FOR FLYING AFTER DCI**

<b>Author or Organization</b>	<b>Suggested Time interval</b>
Rayman & McNaughton (USAF) <sup>14</sup>	Once asymptomatic and treatment is completed. No specific time given.
Davis <sup>18</sup>	24 hours after treatment of Type 1. 72 hours after treatment of Type 2.
United States Navy <sup>19</sup>	24 hours after surfacing for Type 1. 48 hours after treatment of Type 2. Minimum 72 hours if symptoms persist.
Professional Association of Diving Instructors (PADI) <sup>20</sup>	72 hours following treatment of DCS.
Williamson J (Personal communication)	28 days following treatment.
Arthur and Margulies <sup>10</sup>	1 week after onset of Type 1. 30 days after onset of Type 2.
Bassett <sup>21</sup>	48 hours after treatment of symptoms resolved. At discretion of diving medical consultant if unresolved.
AS 2299 <sup>22</sup>	Not greater than 300 m for 7 days.

toms and the recurrence develops at a lower altitude than the initial episode. This is even more significant if the recurrence occurs at an altitude below 18,000 ft (5,400 m)<sup>15</sup>, which is the usually accepted lower limit for altitude DCI.

A series of cases of altitude DCI reported by Allan<sup>16</sup> showed recurrent DCI symptoms developing at up to 2 weeks after their initiation. These recurrences were considered by that author to be the result of previous injury, but bubbles had probably remained over that time to produce a recurrence of identical symptoms. Another case of recurrence at 3 days after resolution was reported by Furry.<sup>17</sup>

An unreported series of 4 divers treated at the Royal Adelaide Hospital had recurrence of their symptoms with ascent to 300 m (1,000 ft), 2 days after their last treatment. These cases indicate that some divers require a delay of at least several days after symptom resolution before ascent to altitude can be tolerated.

### Present recommendations

Current opinion about when a diver treated for DCI can fly varies greatly. Many authors and organizations recommend very different intervals. Given the lack of data, substantiation of any of these views is impossible. Table 1 gives a summary of these opinions. A review by Sheffield demonstrates a similar variability of opinion.<sup>9</sup>

### Neurological effects

It is important to consider what constitutes the clinically asymptomatic treated diver. A review of divers treated for DCI was conducted by the Royal Australian Navy.<sup>23</sup> This study involved a clinical neurological examination, a series of psychological tests, a 19 lead EEG and a CT scan of the head. Clinical resolution of symptoms occurred in 84 of the 87 treated divers. The EEG follow up demonstrated that, of 46 divers, at one week 22 and at one month 8 divers had abnormal EEGs. It is not known whether these residual abnormalities were due to the continuing presence of tissue bubbles, the haematological abnormalities subsequent to bubble formation, or the residual effects of damaged nerve tissue. Regardless of the exact pathogenesis of such changes, this data would indicate that 1 week after treatment may be too short a convalescence before altitude exposure.

### Type 1 versus Type 2 disease

Three of the opinions presented in Table 1 vary the management of DCI according to the Type 1 and Type 2 categories as originally proposed by Golding et al.<sup>24</sup> This symptomatic classification was suggested to differentiate "simple" limb bends from the "more serious" neurological and cardiopulmonary manifestations of DCI. Recent re-

views of divers suffering DCI in Australia suggest that most divers with limb bends have neurological manifestations of their disease.<sup>2,25</sup> This is further substantiated when these "pain only" sufferers are subjected to EEG examination.<sup>23</sup> This would indicate that no attempt should be made to differentiate, on the basis of presenting symptoms, when divers should ascend to altitude.

### Conclusions and recommendations

Because of the lack of systematic patient follow-up and of controlled studies, it is difficult to estimate the frequency of DCI recurrence with ascent to altitude, although the USAF review would suggest that it is uncommon.<sup>14</sup> In this context, it is not surprising that opinions about the safe time interval before altitude ascent are inconsistent. I have found unpublished evidence that asymptomatic bubbles can exist in tissues for periods of up to 3 weeks and published evidence that such stable bubbles may lead to a recurrence of DCI symptoms.<sup>16,17</sup> There is also evidence that treated asymptomatic divers have EEG abnormalities that resolve during the month after treatment.<sup>23</sup>

As it is impossible to identify which divers will have a prolonged risk of recurrence, it would seem prudent to recommend a period of 4 weeks from the end of treatment until ascent is permitted. Many divers would consider such a time interval to be too restrictive and as such would be likely to fly earlier and accept an increased risk of recurrence. However, although this interval is arbitrary, it is longer than any reported bubble survival in tissue, as well as being longer than the interval reported to be associated with recurrences. There is also good supporting evidence that this advice should not be varied on the basis of presenting symptoms.

The recommendation is based on limited information, mainly from isolated case reports. It is impossible on present information to quantify the risk of recurrence of DCI with flying after shorter periods of convalescence, or at what altitude these risks become significant.

It is important that a controlled follow-up study of divers suffering DCI is carried out. The information required is (a) the time from end of treatment to altitude ascent, (b) the altitude ascended to, and (c) the presence of any recurrent symptoms. As most treated divers in Australia cannot be reviewed by the treating Hyperbaric Unit, such a study would have to rely on patient reporting, probably by questionnaire. Such a study would allow some quantification of the risk of DCI recurrence.

With the increasing popularity of recreational diving and the greater mobility of diving populations, flying after diving will continue to occur with greater frequency. Consequently, detailed follow-up studies of treated divers are now essential.

## References

- 1 MacMillan AJF. Decompression sickness. In: Ernsting J, King P, eds. *Aviation medicine*. 2nd ed. London: Butterworth, 1988: 19-26.
- 2 Williamson J, Orton J, Callanan V et al. The Townsville diving medical and aeromedivac system. Experiences, lessons and the future. *SPUMSJ* 1988; 18(3): 82-87.
- 3 Shilling CW. In: Shilling CW, Carlston CB, Mathias RA, eds. *The physician's guide to diving medicine*. New York and London: Plenum Press, 1984: 590-1.
- 4 Edel PO, Carrol JJ and Honaker RW et al. Interval of sea-level pressure required to prevent decompression sickness in humans who fly in commercial aircraft after diving. *Aerosp Med* 1969; 40(10): 1105-10.
- 5 Balldin UI. Venous gas bubbles while flying with cabin altitudes of airliners or general aviation aircraft three hours after diving. *Aviat Space Environ Med* 1980; 51(7): 649-52.
- 6 Furry DE, Reeves E and Beckman E. Relationship of scuba diving to the development of aviators decompressions sickness. *Aerosp Med* 1967; 38: 325-8.
- 7 Balldin UI. Intracardial gas bubbles and decompression sickness while flying at 9,000 m within 12-24 hr of diving. *Aviat Space Environ Med* 1978; 49(11): 1314-8.
- 8 Lambertsen CJ. The pressure continuum: Need for rational correlation and differentiation of the flying and diving environments. *Proceedings of the 39th Undersea and Hyperbaric Medical Society Workshop*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989: 1-10.
- 9 Sheffield PJ. Flying after diving guidelines: A review. *Aviat Space Environ Med* 1990; 61: 1130-8.
- 10 Arthur DC and Margulies RA. The pathophysiology, presentation, and triage of altitude-related decompression sickness associated with hypobaric chamber operation. *Aviat Space Environ Med* 1982; 53(5): 489-94.
- 11 Kunkle TD and Beckman EL. Bubble dissolution physics and the treatment of decompression sickness. *Medical Physics* 1983; 10(2): 184-90.
- 12 Hills BA. *Decompression sickness. Vol 1*. New York: Wileys, 1977: 142-3.
- 13 Green RD and Leitch DR. Twenty years of treating decompression sickness. *Aviat Space Environ Med* 1987; 58: 362-6.
- 14 Rayman RB and McNaughton GB. Decompression sickness: USAF experience 1970-1980. *Aviat Space Environ Med* 1983; 54(3): 258-60.
- 15 Fryer DI. Decompression sickness at 18,500 feet, a case history with comment. *Aerosp Med* 1964; 35: 479-81.
- 16 Allan JH. Traumatic calcifications: A precipitating factor in "bends" pain. *Aviat Med* 1945; 16: 235-41.
- 17 Furry DE. Incidence and severity of decompression sickness in navy hospital corpsmen. *Aerosp Med* 1973; 44 (4): 450-452.
- 18 Davis JC. Treatment of decompression sickness and arterial gas embolism. In: Bove AA and Davis JC, eds. *Diving Medicine*. 2nd ed. Philadelphia: Saunders, 1990: 258.
- 19 *US Navy Diving Manual*. Vol 1, revision 1. Navy Department, Washington D.C., June 1985: 8-47.
- 20 *PADI Divemaster Manual*. Santa Ana, California: Professional Association of Diving Instructors 1985: 143.
- 21 Bassett BE. Diving and altitude. Recommendations for divers. *SPUMS J* 1983; 0: 6-9.
- 22 *Underwater Air Breathing Operations AS2299, 1990*. Standards Association of Australia.
- 23 Gorman DF, Edmonds CW, Parsons DW et al. Neurologic sequelae of decompression sickness; a clinical report. *Ninth International Symposium on Underwater and Hyperbaric Physiology*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987: 993-8.
- 24 Golding FC, Griffiths P and Hempleman HV et al. Decompression sickness during construction of the Dartford tunnel. *Brit J Industr Med* 1960; 17: 167-80.
- 25 Gorman DF, Pearce A and Webb RK. Dysbaric Illness at the Royal Adelaide Hospital 1987: A factorial analysis. *SPUMS J* 1988; 18(3): 95-102.

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## ON NO-STOP TIME LIMITS, SAFETY STOPS AND ASCENT RATES

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### Introduction

The past ten years, or so, have witnessed a number of important changes in diving protocols and table procedures, such as shorter no-stop time limits, slower ascent rates, discretionary safety stops, repetitive dive profiles requiring all dives to be shallower than the one before, multi-level techniques, both faster and slower tissue half-times controlling repetitive dives, lower critical tensions (M-values) and longer flying-after-diving surface intervals. Stimulated by