Flying after recompression treatment for decompression illness: why wait four weeks?

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Flying (and diving), decompression illness, treatment, treatment sequelae, review article

Abstract

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The risk of a relapse associated with flying following recompression treatment for decompression illness (DCI) is unknown. Recommendations regarding the safety of flying following treatment for DCI are varied and personal, and few are published. The author's view that a diver should wait four weeks is based on:

- a review of the current literature
- personal clinical observations
- case reports
- postulated pathophysiological mechanisms for recorded relapses such as reactivation of 'primed' leucocytes and endothelium by new bubble formation and growth of 'stabilised' bubbles
- the physics and physiology of airflight including hypobaric hypoxia, hypobaric hypovolaemia, gas cavity expansion in accordance with Boyle's law and a decrease in gravity.

This evidence is presented in the following review.

Introduction

Flying and altitude exposure following compressed-air diving (CAD) are associated with a risk of decompression illness (DCI) due to a lower barometric pressure. Various recommendations have been published concerning when it would be safe to fly following diving; however, few have been tested.[1–3] These recommendations have also been used as a guide concerning the minimal stay at 'ground level' before flying following recompression treatment for DCI. Similarly, these recommendations are varied and based on unsubstantiated opinion and bias; few are published. Some of these recommendations are listed in Table 1.

The objects of this paper are to review current literature, postulate relapse mechanisms and provide supportable advice that can be given to divers following treatment for DCI.

Environmental and physiological factors associated with airflight

CABIN PRESSURE: HYPOBARIC HYPOXIA

Modern commercial aircraft fly at altitudes between 9,000 and 12,000 metres above sea level (mASL) to keep fuel costs low and to avoid unfavourable air conditions. With ascent the atmospheric pressure drops so that at 12,000 mASL the pressure is 176 mmHg (23.38 kPa).[4] However, the pressure inside the cabin is maintained by drawing in external air and limiting its outflow. Most aircraft cabin structures can withstand a pressure differential of 430 mmHg (57.14 kPa), thus the pressure inside the cabin at 12,000 mASL would be 606 mmHg (80.53 kPa). This is the air pressure found at an altitude of 1,600 m, therefore, the 'cabin altitude' is said to be 1,600 m.[4] With the decrease in atmospheric and cabin pressure there is a corresponding decrease in the inspired partial pressure of oxygen (P_IO_2) in the air breathed, causing a hypobaric-induced hypoxia. Depressurisation to 'cabin altitude' takes about 20 minutes.[5,6] 'Unpressurised' aircraft flights in Australia are limited to 3,000 mASL (approximately 68 kPa).[5,6]

Table 1
Recommendations for flying after treatment for DCI (TT USN 6A – US Navy treatment table 6A)

Organisation	Suggested time interval
Australian Standard AS2299	7 days
Royal Adelaide Hospital	4–6 weeks
Townsville General Hospital	3 weeks
Royal Hobart Hospital	2 weeks
Prince of Wales Hospital	1–2 weeks
Fremantle Hospital	2 weeks
PADI	3 days
NAUI	1 day
DAN (unconfirmed)	3 days
US Navy	24 hours for Type 1
	48 hours for Type 2
	72 hours if symptoms persist
	72 hours after TT USN 6A

To prevent hypobaric hypoxia the aircraft cabin is usually pressurised to give an equivalent altitude of 2,440 mASL, at which the ambient pressure is 562.4 mmHg (74 kPa).[3] From the alveolar gas equation[7] the alveolar partial pressure of oxygen (P_AO_2) at a 'cabin altitude' between 2,000–2,500 mASL, excluding the saturated vapour pressure of water, would be between 63 and 68 mmHg (approximately 9.03 kPa) and because of the alveolar/arterial difference the arterial partial pressure of oxygen (P_aO_2) would be between 35 and 58 mmHg (4.78–7.70 kPa).

Hypoxia causes pulmonary vasoconstriction and mild hyperventilation, due to hypoxic stimulation of peripheral chemoreceptors, shifting the oxygen dissociation curve to the left and marginally increasing the P_IO_2 and hence P_AO_2 (the alveolar gas equation).[7]

The drop in cabin pressure will cause a 4% reduction in the amount of oxygen carried by the blood and has a minimal effect on healthy passengers. Even with a P_aO_2 of 56 mmHg the passenger lies on the flat part of the oxygen-haemoglobin dissociation curve.[7] However, this reduction may be critical for tissues with a marginal blood supply, particularly when the dissociation curve is shifted to the left.[6] A reduction in P_aO_2 will reduce the flux of nitrogen from air bubbles present due to a reduction in the 'oxygen window' effect, thus slowing bubble resolution.[6,8]

Acute hypoxia induced by reducing the P_IO_2 to 71 mmHg (9.43 kPa) promotes a rapid microvascular response characterised by increased leucocyte rolling and adherence to the venular endothelium, leucocyte emigration to the perivascular space, increased vascular permeability and proinflammatory features in the endovascular compartment. This appears to be a generalised response. After three weeks of acclimatisation to hypoxia this microvascular response resolves.[9,10] Although the induced hypoxia during flight is not as severe as this it is not known what degree of hypoxia (if any) in combination with other factors may activate a previously 'primed' or 'activated' endothelium.

CABIN PRESSURE: DECREASE IN GRAVITY

Gravity decreases with altitude. This decrease initially causes:

1 a decrease in venous and arterial pressures in the dependent capillary beds promoting intravascular fluid retention, hence decreasing blood pooling in the legs causing an increase in venous return, and hence thoracic and pulmonary blood volume;[7] and
2 a linear decrease in gas density with the decrease in atmospheric (cabin) pressure promoting, perhaps, better alveolar gas mixing; and
3 these combining to produce a more uniform distribution of ventilation and blood flow.

CABIN PRESSURE: HYPOBARIC HYPOVOLAEMIA AND OTHER CARDIOVASCULAR ALTERATIONS

Cabin air is exchanged every 3–4 minutes and because it is drawn in at altitude it has a very low water content. Engineering constraints do not allow for humidification systems in aircraft; hence, cabin atmosphere has a low relative humidity (15–25%) which will lead to water loss from the respiratory tract.[4] In addition, the increase in preload due to the increase in thoracic blood volume results in a diuresis. This induced diuresis and loss of respiratory fluid promotes hypovolaemia (called hypobaric hypovolaemia).

The combination of hypoxic pulmonary vasoconstriction and increase in pulmonary capillary blood volume results in pulmonary hypertension. This increases the work performed by the right heart and promotes peripheral, particularly ankle, oedema. The increased hypoxic vascular permeability may also contribute to this.[9]

Early in hypovolaemic shock the lumen of capillaries become narrower as a result of swelling of hypoxic endothelial cells and adhesion of activated polymorphonuclear leucocytes to the endothelium of the post-capillary venules. The interaction of leucocytes with the endothelium is followed by the release of vasoactive mediators and toxic oxygen species promoting macromolecular leakage, interstitial oedema and redistribution of tissue perfusion, hence compromising oxygen delivery.[11] Although the altitude-induced hypobaric hypovolaemia is not severe enough to initiate these mechanisms it may have an additive effect to endothelial reactions occurring because of hypobaric hypoxia or because the endothelium has been 'primed' due to bubbles.

CABIN PRESSURE: BOYLE'S LAW

Gas spaces will expand with a decrease in ambient pressure in accordance with Boyle's law and Pascal's Principle;[12] hence, any tissue bubbles present during altitude exposure will grow. At an ambient pressure of 74 kPa bubble volume will increase by about 35% (assuming a spherical bubble), which corresponds to an increase in radius of nearly 11%. If bubbles have been present for some time they will have become 'stabilised' with a coating of surfactant, haematological and immunological active substances.[6] These bubbles will be in equilibrium with the surrounding tissues and will consist of nitrogen, carbon dioxide, oxygen and water vapour. As the ambient pressure drops, these bubbles will enlarge and quantities of these gases will diffuse into the bubbles until equilibrium is again achieved. So the actual prediction of bubble/gas expansion by Boyle's law may initially be an underestimation.

A summary of the pressure changes with altitude and airflight and other relevant clinical information are listed in Table 2.

Table 2
Changes in pressure, temperature, gas space volume expansion and alveolar oxygen pressure (P_AO_2) breathing air, 30% oxygen and 100% oxygen (saturated water vapour pressure included) with airflight.[3–6] Table modified from Everest et al[13]

Altitude (mASL)	Pressure (kPa)	°C	Expansion %	P_AO_2 breathing Air (mmHg)	30% (mmHg)	100% (mmHg)	General comments
Sea level	101 (760 mmHg,)	15		100	178	710	Average temperature
304.9	97.4 (733 mmHg)	13	+3.6	96	169	680	Minimum altitude for helicopters
609.8	93.8 (706 mmHg)	11	+8.0	91	162	657	Altitude for helicopters above sea terrain
914.7	90.5 (681 mmHg)		+12.0	85	153	626	
1219.6	87.2 (656 mmHg)	7	+16.0	81	146	604	Cabin altitude for ambulance aircraft
2134.3	77.9 (586 mmHg)	1	+29.0	67	125	535	Cabin altitude for commercial aircraft
3049.0	69.5 (523 mmHg)	-5	+45.0	55	106	474	Hypoxic threshold, ceiling for helicopters
6098.0	46.4 (349 mmHg)	-25	+117.0	20	55	299	Upper cruise altitude for turbo prop aircraft
8200.0	37.5 (282 mmHg)		+170.0	<10	35	231	
12196.0	18.7 (141 mmHg)	-56	+439.0	-	-	94	Upper cruise altitude for commercial aircraft

Pathophysiology of decompression illness (DCI) and postulated mechanisms of a relapse following treatment

The pathophysiology of DCI involves bubble formation and either intra- or extra-vascular growth. These bubbles distort tissues, obstruct perfusion and interact with formed blood elements or proteins (stimulating platelets, denaturing lipoproteins, activating and aggregating leucocytes, activating complement and coagulation pathways, releasing cytokines, and causing capillary leakiness). Bubbles may also damage both the luminal surfactant layers and endothelial cells of blood vessels.[14]

Relapse following initial treatment is multifactorial:[15]
- persistence of gas bubbles
- new bubble growth
- tissue ischaemia and oedema
- reactivation of blood proteins, leucocytes and endothelium
- barotrauma on ascent
- a combination of some or all of these mechanisms.

These mechanisms may also be responsible for relapse with airflight. However, barotrauma on ascent is unlikely.

Divers Alert Network (DAN) data reported that 40% of divers were asymptomatic following one recompression treatment, with diminishing clinical benefit with successive treatments.[16] Only 67.7% of divers had complete relief of symptoms following six treatments (six days, assuming one treatment per day). This would indicate that following an initial US Navy treatment table 6 (Royal Navy table 62), 'stabilised' bubbles and their pathophysiological effects may still be present in some cases, but are responsive to recompression and hyperbaric oxygen therapy (HBOT).

Case reports: altitude exposure

There is a paucity of published case reports regarding a relapse with airflight following treatment for DCI. A series of four cases at the Royal Adelaide Hospital relapsed with symptoms on ascent to 300 mASL two days following treatment.[3] Allan showed recurrence of DCI symptoms two weeks post development in aviators.[17] Fury also reported recurrence after three days.[18] Millar reported nine cases of relapse following treatment with an altitude exposure (range 100 mASL to 'cabin' pressure). Six of these relapsed within seven days, while three did so 10 to 21 days post treatment (altitude exposure 400–1,000 mASL).[6]

Uguccioni et al reported on 126 divers some of whom had a return of or worsening of their symptoms during commercial flight.[19] Of the 74 who 'flew' within 72 hours of treatment, 20 had a return of symptoms. Seventeen of these did not have a full resolution of symptoms prior to flight. Of the 52 who 'flew' after 72 hours, 11 relapsed of whom five did not have full symptom resolution prior to the flight. These data show that, in divers still symptomatic prior to a flight, the risk of a worsening of symptoms decreases from 27% to 21% if they wait 72 hours.

In an unpublished series of 46 divers treated at the Townsville General Hospital, Australia (personal communication, Bishop E, 2004), 28 flew after treatment, of whom eight relapsed. No relapses occurred if divers waited 35 days before flying.

Recently a diver was treated at the Royal Adelaide Hospital who had relapsed twice following airflight. Each relapse was worse than the previous one. His initial symptoms were considered 'mild': paraesthesia in one arm and the face,

which was treated with surface oxygen. He waited three days before flying and relapsed following this brief flight. He was treated with standard recompression protocols and then waited five days on medical advice before his next flights, which included a flight from Canada to Australia. His final presentation was with paraplegia. The time frame over which his relapses occurred was three weeks.

Vann et al reported a DAN online survey that involved 121 divers, comparing relapse rates in divers who 'flew' and those who did not.[20] Online surveys have limitations, but the data showed that:

- those who relapsed 'flew' within 13 days
- those who 'flew' but did not relapse had a range of 1–28 days.

Case reports: persistence of bubbles

The effect of recompression treatment with HBOT on inert gas elimination and bubble dynamics remains poorly understood and understudied. There are data, however, which show that asymptomatic divers following treatment do still have circulating bubbles.[21]

Acott and Gorman published a case history of a diver who developed postoperative symptoms of decompression illness following a general anaesthetic involving the use of nitrous oxide.[22] The diver had his operation one week following a group of dives in which two dives were followed by symptoms of decompression illness that were 'treated successfully' with surface oxygen. He presented two weeks after his operation with symptoms suggestive of DCI. These had been present for the two weeks. His symptoms resolved with HBOT.

Hills and Le Messurier (reported by Butler[3]) followed up an asymptomatic abalone diver using X-rays that showed bubbles still present 22 days following a 'bends'-provoking dive. Gorman et al reported a series of asymptomatic divers following treatment for DCI who had persistent abnormalities detected by EEG, psychological tests and/or CT scan at four weeks following treatment.[23] The cause of these abnormalities was unknown but they may have been due to continuing presence of bubbles, and their continuing haematological activity, or residual nerve damage.

Personal clinical observations during follow up of treated divers over a decade have shown that divers do not 'feel 100%' until at least four weeks following treatment. However, it is not known whether this is due to persistent bubbles or their haematological and immunological effects.

Historically, there are data to show that bubbles exist for prolonged periods in excess of those predicted by mathematical models. Boycott et al showed the presence of bubbles in their experimental goats' spinal cords 15 and 27 days following decompression, and in blood six days after; the goats were asymptomatic prior to euthanasia.[24]

New bubble growth

There are data to suggest that bubble formation may occur during flight. Eckenhoff showed that venous bubbles were detected in 50% of subjects following decompression from saturation at only 3.5 msw depth.[25] This pressure difference is similar to that in exposure to an altitude of 74 kPa. Conkin et al demonstrated venous gas emboli in subjects subjected to 70 kPa or less.[26]

Discussion

Vann et al and others argue that altitude exposure relapses within five days following treatment may be an example of the natural history of DCI seen in those divers; that is, they would have relapsed regardless.[20] However, it is difficult to maintain this argument for times longer than this. Anecdotal data from case histories indicate that bubbles may remain in tissues much longer than predicted by mathematical and *in vitro* models of bubble dissolution.[3] The persistence of these bubbles is due to a surrounding 'semi rigid' haematological barrier, which provides a diffusion barrier. These 'stabilised' bubbles will change shape and size during airflight, distorting tissues with local pressure effects.[6]

Airflight may give rise to new bubble formation (see earlier) and animal data suggest that even a small number of 'silent bubbles' will cause endothelial damage and impairment.[27,28] The additive effect of the expansion of 'stabilised' bubbles with new bubble formation may further cause activation of haematological and immunological events during airflight resulting in an increase in inflammation and oedema. These haematological and immunological events may be 'primed' by earlier pathophysiological processes. It is unknown how long the vascular endothelium and leucocytes stay 'primed' following initial bubble stimulation (with DCI), but in the acute hypoxic model it is three weeks.[10]

In addition, the 'primed' endothelium may be reactivated by the physiological effects of 'hypobaric hypoxia and hypovolaemia' with airflight.

Proposed management to decrease risk with flying

ROLE OF INFLIGHT OXYGEN ADMINISTRATION

The postulated mechanism that hypoxia is mainly responsible for relapse during airflight is too simplistic. Oxygen via nasal prongs at 3 $l.min^{-1}$ will elevate the fraction of inspired oxygen (F_IO_2) to approximately 30%; therefore, at cabin altitude the P_IO_2 will be 154 mmHg (20.5 kPa). Although oxygen may be used to negate any hypoxic effect

during airflight, it will not diminish other physiological adjustments to the hypobaric environment, such as the decrease in gravity and the expansion of air-containing spaces, particularly bubbles. In Australia the aviation industry considers oxygen to be a hazardous material and it can be used on a commercial aircraft only on a prescription basis, which requires 48 hours' notice.[4]

PRE-FLIGHT DENITROGENATION WITH NORMOBARIC OR HYPERBARIC OXYGEN (HBO)

Pre-flight denitrogenation has been shown to decrease the incidence of altitude DCI in astronauts. Rice et al showed that 30 minutes of pre-flight normobaric oxygen breathing was effective.[29] However, earlier studies by Behnke and others show that denitrogenation may take up to 240 minutes.[30] In man, the initial elimination was measured at 50 $ml.min^{-1}$, decreasing to 0.1 $ml.min^{-1}$ at nine hours. Bornstein reported that 25–33% (200–300ml) of the total nitrogen was eliminated from a lean man in nine minutes.[31] Nitrogen elimination in animals was an exponential function plateauing at 240 minutes.[30] Any pre-flight denitrogenation, as practised by NASA and various military airforces, would require the diver to breathe oxygen while boarding the aircraft, which would prove to be impracticable in most circumstances.

Pre-flight HBO therapy would denitrogenate and oxygenate tissues but its effect on the endothelium and leucocytes is unknown even if they are 'primed' but not activated. Practicalities of the application of HBO, which treatment table one should use, and at what stage it should be applied before boarding the aircraft, would limit any usefulness. Currently, there are no data to suggest it would be useful.

Recommendations

The physiological changes associated with airflight following treatment may create an unpredictable DCI risk. However, if flight is unavoidable what can be done to reduce the risks of relapse?

- Explain to the patient (diver) that the risks associated with flight are unknown.
- Ensure the airline is aware that there may be a problem and has an adequate supply of 100% oxygen available.
- Maintain hydration before and during the flight.
- Consider the role of a prior hypobaric exposure before flight; at least the patient could be treated if any problems did occur.
- Check the flight path and note if there are any recompression facilities available en route should problems arise.
- Check if there is a hyperbaric facility at the patient's destination or where the nearest facility is to be found.
- Advise a review of the diver by a diving physician as soon as possible following the flight.
- Send a detailed clinical summary with the patient.

My recommendation is to wait four weeks before airflight following decompression illness because:

- there are no published case histories where a relapse has occurred after this time period;
- the pathophysiology associated with DCI may take three to four weeks to abate.

Conclusions

Case histories that show early relapse with or without airflight within five to seven days of treatment would indicate that airflight is not advisable for at least seven days post treatment. No relapses have been reported in divers who waited 35 days. Flight between seven and 35 days has a diminishing risk. However, this risk cannot, at present, be quantified.

References

1 Sheffield PJ. Flying after diving guidelines: A review. *Aviat Space Environ Med.* 1990; 61: 130-8.

2 MacMillan AJF. Decompression sickness. In: Ernsting J, King P, editors. *Aviation medicine*. London: Butterworth; 1988. p. 19-26.

3 Butler C. Flying after treatment for decompression illness: When is it safe? *SPUMS J.* 1992; 22: 189-92.

4 Lovell M. In-flight medical emergencies. A difficult medical and legal environment. In: Keneally J, Jones M, editors. *Australian anaesthesia.* Melbourne: ANZCA; 2003. p. 63-72.

5 Aeronautical Information Publications, Civil Aviation Authority, Australia as cited in 6.

6 Millar I. Post-diving altitude exposure. *SPUMS J*. 1996; 26: 135-40.

7 West J. *Respiratory physiology: the essentials*. 6th ed. Baltimore: Lippincott Williams & Wilkins; 2000.

8 Vann RD, Thalmann ED. Decompression physiology and practice. In: Bennett PB, Elliott DH, editors. *Physiology and medicine of diving*. 4th ed. London: Saunders; 1993. p. 376-442.

9 Gonzalez NC, Wood JG. Leucocyte-endothelial interactions in environmental hypoxia. *Adv Exp Med Biol*. 2001; 502: 39-60.

10 Ten VS, Pinsky DJ. Endothelial response to hypoxia: physiological adaptation and pathological dysfunction. *Curr Opin Crit Care*. 2002; 8: 242-50.

11 Kreimeier U, Frey L, Messmer K. Small volume resuscitation. In: Prys-Roberts C, editor. *Current opinion in anaesthesiology.* 1993; 6: 400-8.

12 Edmonds C, Lowry C, Pennefather J, Walker R. *Diving and subaquatic medicine.* 4th ed. London: Arnold; 2002. p. 12-4.

13 Everest E, Munford B. Transport of the critically ill. In: Bersten A, Soni N, editors. *Oh's intensive care manual*. 5th ed. Sydney: Butterworth Heinemann; 2003. p. 21-32.

14 Francis TJ, Gorman DF. Pathogenesis of the decompression disorders In: Bennett PB, Elliott DH,

editors. *Physiology and medicine of diving.* 4th ed. London: Saunders; 1993. p. 454-80.
15 Moon RE, Gorman DF. Treatment of the decompression disorders. In: Bennett PB, Elliott DH, editors. *Physiology and medicine of diving.* 4th ed. London: Saunders; 1993. p. 506-41.
16 Divers Alert Network. *Report on decompression illness and diving fatalities.* Durham: DAN International Annual Review; 2000. p. 51.
17 Allan JH. Traumatic calcifications: A precipitating factor in 'bends' pain. *Aviat Med.* 1945; 16: 479-81.
18 Fury DE. Incidence and severity of decompression sickness in navy hospital corpsman. *Aerosp Med.* 1973; 44: 450-2.
19 Uguccioni DM, Dovenbarger JA, HobgoodJA, Moon RE. Commercial airflight after recompression therapy for decompression illness. *SPUMS J.* 1998; 28: 178.
20 Vann RD, Freiberger JJ, Denoble PJ, Dovenbarger J, Nord D, et al. The risk of relapse from flying after recompression therapy for decompression illness: preliminary results. Proceedings UHMS Annual Scientific Meeting, Sydney; May 2004.
21 Eckenhoff R, Osborne SF, Parker JW, Bondi KR. Direct ascent from shallow air saturation exposures. *Undersea Biomed Res.* 1986; 13: 305-15.
22 Acott CJ, Gorman DF. Decompression illness and nitrous oxide anaesthesia in a sports diver. *Anaesth Intensive Care*. 1992; 20: 249-50.
23 Gorman DF, Edmonds CW, Parsons DW. Neurological sequelae of decompression sickness; a clinical report. In: *9th International Symposium on Underwater and Hyperbaric Physiology.* Bethseda, Maryland: Undersea and Hyperbaric Medical Society; 1987. p. 993-8.
24 Boycott AE, Damant GC, Haldane JS. Prevention of compressed air illness. *J Hyg Lond*. 1908; 8: 342-443.
25 Eckenhoff R. Human dose response relationship for decompression and edogenous bubble formation. *J Appl Physiol*. 1990; 69: 914-8.
26 Conkin J, Foster PP, Powell MR, Waligora JM. Relationship of the time course of venous gas bubbles to altitude decompression illness. *Undersea Hyperb Med.* 1996; 23: 141-9.
27 Nossum V, Hjelde A, Bergh K, Brubakk AO. Lack of effect of anti-C5a monoclonal antibody on endothelial injury by gas bubbles in the rabbit after decompression. *Undersea Hyperb Med.* 2000; 27: 27-35.
28 Nossum V, Hjelde A, Brubakk AO. Small amounts of venous gas embolism cause delayed impairment of endothelial function and increase polymorphonuclear neutrophil infiltration. *Eur J Appl Physiol.* 2002; 86: 209-14.
29 Rice GM, Vacchiano CA, Moore JL, Anderson DW. Incidence of decompression sicknes in hypoxia training with and without 30 minute prebreathe. *Aviat Space Environ Med.* 2003; 7: 6-61.
30 Behnke AR. Some early studies of decompression. In: Bennett PB, Elliott DH, editors. *Physiology and medicine of diving.* 1st ed. London: Bailliere Tindall Cassell; 1969. p. 226-51.
31 Bornstein A. Weitere untersuchugen uber Herzschlagvolumen. *Z Exp Path Ther.* 1913; 14: 135-50.

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