

33 m (60-110 fsw). This risk has been applied to the new no-decompression limits, and the same level of risk has been used for decompression dives up to a total decompression time of 20 minutes. Longer dives have been allowed to ramp up to 5% when the total decompression time reaches 60 minutes. The old 12 hour clean rule has gone, and on some dives it will take up to 33 hours to be clean.

These new tables are no longer considered to be safe or unsafe by the US Navy, rather, they simply have a greater or lesser chance of causing DCI. Additionally, they give information about the level of risk at various times during a dive. This is an exciting approach to the way we conduct our diving, and reinforces the point that diving is merely a risk acceptance activity.

In conclusion, I believe that our thinking regarding how we dive needs to change if we are to reduce further the incidence of DCI in recreational diving. The holy grail of no-stop diving may not be such a laudable goal after all, and the data suggests that staged decompression after every dive will substantially reduce a divers risk of DCI. Further, studies suggest that these stops need to be made before significant bubbling has occurred if a benefit is to be realised. Spending, say, 1 minute at 18 msw, 2 to 3 minutes at 10 msw and 5 to 10 minutes at 5 msw after each dive should significantly reduce risk. An increase in risk is seen as dives get deeper, but this effect is not nearly as great as with time. DCI can be expected to occur occasionally, even in relatively unprovocative exposures. Thus it should not be regarded as an accident. It is expected to happen occasionally, and it does not always represent a loss of control as is implied by the use of the term "accident".

But can we trust recreational divers to discipline their diving to this extent so that we will see a decrease in those presenting for treatment of DCI? Realistically I think not, as current studies show an alarming number of divers who are unable to manage even their air supply, with those that make the statistics probably representing only the tip of the iceberg. However, I do hold out hope for those who have a genuine interest in reducing the risk of DCI in their dive practice, mainly us older, once bolder types.

I believe that the evidence is overwhelming for staged decompression even following a dive profile that, according to some algorithm, incurs no decompression debt. The objective of our procedures after all is to REDUCE the probability of DCI to an ACCEPTABLE minimum, and I believe we have the tools at our disposal to enable us to do this within a predicted probability of risk.

Key Words

DCI, risk, tables.

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A list of references consulted is available from Dr Gordon.

POST DIVING ALTITUDE EXPOSURE

Ian Millar

The Alfred Hospital experience

During the 68 month period 1 November 1987 to 30 June 1993, the Alfred Hospital, Melbourne, Hyperbaric Unit treated a total of 401 cases diagnosed as suffering decompression illness (DCI). Of these, 44 had involved post diving altitude exposure. Only one of these was associated with medical retrieval; cases where air ambulance transport occurred but did not aggravate DCI were not included in the 44. This series provides illustrations for many of the dilemmas associated with determining safe limits for altitude exposure after diving and after treatment for DCI.

Review of available case records revealed the following:

All but 5 were recreational divers.

Note that several patients appear twice. DCI was initially provoked by altitude exposure after diving and subsequently relapsed with further altitude exposure post treatment

ASYMPTOMATIC BEFORE EXPOSURE

Twenty one cases were asymptomatic prior to their altitude exposure ranging from 300 to 800 m above sea level (ASL).

Twelve of these developed symptoms during surface based travel. In this group 8 involved altitude ascent several hours after dives which had been preceded by multiday repetitive diving. Five also involved post dive exercise and alcohol as risk factors.

3 late onset cases involved altitude ascent one to three days after dives involving risk factors of multiple ascents or vigorous underwater exercise.

1 case involved returning to place of residence at 800 m altitude on the Sunday evening of a routine entry level training weekend.

Nine developed symptoms after flying. Six of these involved flying at between 12 and 24 hours after multi-day, repetitive, deep diving. One involved flying at 24 hours after extreme multi-day diving. One involved flying 2 days after a single extreme air dive in emergency circumstances.

One involved multi-day diving with detail regarding the interval not recorded.

SYMPTOMS BEFORE EXPOSURE TO ALTITUDE

16 cases had some abnormal symptoms present prior to altitude exposure.

In 3 of these, symptoms had resolved without treatment prior to altitude, but returned and increased with an altitude exposure some days after the symptoms.

In several of the remainder, who flew with symptoms, these symptoms were initially mild and had not been recognised as DCI prior to altitude exposure.

One patient suffered deterioration during a 300 m altitude exposure whilst driving home several hours after symptom onset

The remainder were associated with flying after delays ranging from 17 hours to several days and in one case, two and a half weeks after the diving which caused symptoms to arise.

Four cases were notable for minimal exacerbation resulting from altitude despite moderate symptoms and preceding deep repetitive multi-day diving

POST-TREATMENT RELAPSES

Nine cases involved post treatment relapse associated with altitude exposure (Table 1)

DETERIORATION DURING RETRIEVAL

One case involved neurological deterioration during a 90 minute helicopter retrieval at 900 metres above sea level (ASL).

Sources of altitude exposure

Cabins of commercial aircraft are usually pressurised to an altitude equivalent not exceeding 2,500 m above sea level (ASL) or 0.75 atmospheres absolute (ATA) approximately. Cabin altitudes in the order of 1,500- 2,000 metres ASL are more common during domestic flight sectors.¹ Depressurisation to cruising cabin altitude

TABLE 1
RELAPSES WITH ALTITUDE AFTER
SUCCESSFUL TREATMENT

Travel method	Effective altitude experienced	Time after treatment
Car	100 m	Several hours
Car	300 m	1 day
Car	700 m	2 days
Car	800 m	5 days
Commercial Aircraft	? 2,000 m	5 days (Against advice)
Light Aircraft	100 m	7 days
Train	400 m	10 days
Walk	1,500 m	10 days
Car	1,000 m	3 weeks

usually occurs over 15 to 20 minutes. In Australia, unpressurised flight is limited to 3,000 m ASL (10,000 feet or 0.68 ATA approximately).² Light aircraft flight commonly involves climb rates of 300 to 600 m per minute. Many diving venues allow the possibility of significant altitude exposure after diving as a result of travel over hills or mountains. Of the Alfred Hospital cases, 24 involved flying after diving while 21 cases involved car, bus, train or horse travel to altitudes. The lowest altitudes associated with initial onset of DCI were approximately 300 m ASL although these cases involved other risk factors including alcohol, exercise and prior multi-day deep diving.

After treatment for decompression illness, most relapses were associated with surface based rather than air travel. The case involving apparent exacerbations of symptoms with altitudes of approximately 100 m only involved travel between the patient's home and the Alfred Hospital each day for hyperbaric treatment. In addition to the relapses listed above, several patients who did not present for retreatment reported temporary return of symptoms during altitude excursions by road and air and, in one case, following travel by elevator to the top of a 200 m building.

Although this series includes many cases associated with altitudes and intervals usually considered safe, several cases are notable for an apparent lack of significant altitude related deterioration when obvious symptoms were present before a flight back to Australia after intensive diving activity during Pacific island holidays.

Theoretical Considerations

Many flying after diving tables and theoretical models have been based upon prediction of nitrogen wash out using tissue half time models. Most models do not take into account the effect of established bubbles which have

been demonstrated following most diving activity.^{3,4} Further, it has been demonstrated that extrapolation of diving decompression models into the hypobaric realm would result in an excessive risk of altitude DCI.⁵ Nitrogen diffuses out of established bubbles at a far slower rate than it does from solution in tissues and the "oxygen window" effect that is largely responsible is much reduced at altitude.⁶ In the past, it was generally accepted that the threshold for safe decompression from a saturated state to a lesser pressure was a relative pressure reduction of approximately 50%. Hence dives to 10 msw could be of unlimited duration and the threshold for altitude decompression sickness was generally accepted to be 5,500-6,000 m. Venous gas bubbles have been detected, however, in 50% of subjects following decompression from saturation at only 3.5 msw.⁷ In the case of decompression from surface to altitude with no prior diving, NASA research has estimated a threshold for DCI at 3,400 m in the case of 6 hour altitude exposures.⁸

Flying usually occurs at least some hours after diving and it is likely that most blood borne bubbles will have cleared before flight. The pathology of DCI associated with altitude is therefore likely to result from tissue bubbles which were present before the altitude exposure and from newly generated blood born bubbles. These presumably arise as a result of a lowered threshold for altitude DCI resulting from persisting supersaturation of nitrogen in "slow" tissues after extensive diving. Exercise has been shown to produce a surge of bubbles from moving limbs and this is likely to be a significant factor during walking or horse riding to altitude. Dehydration is common during flying and is a further risk factor for DCI.

Twenty six of the Alfred cases had symptoms of incipient or actual DCI or had recently been treated for DCI prior to altitude exposure. Many of the remainder had undertaken diving likely to be associated with persistent "silent bubbles". The presence or absence of such asymptomatic but potentially critical bubbles before altitude exposure may help explain the extreme variability of experience with regard to the consequences of travel to altitude following diving.

It is clear therefore that the pathophysiological consequences of post diving altitude exposure cannot readily be predicted by extrapolation from normal decompression theory. Even if individual factors are considered for any one situation, predictability will remain low and conservatism will be necessary to minimise risk.

Bubble expansion with altitude

When bubbles are present before an ascent, they will expand during time at altitude and may remain enlarged after return to ground level. Boyle's law related

bubble diameter increase can be calculated and is often used to illustrate altitude effects. This, however, significantly under estimates the *in vivo* effects of altitude. The pressure reduction to an average commercial aircraft cruising altitude of 2,000 m is approximately 20%, from 1 ATA to 0.8 ATA. This correlates to a bubble volume increase of 25%. The actual volume of bubbles in the body will, however, increase somewhat more than this as a result of the effect of gases other than nitrogen. Although a decompression generated bubble will initially principally consist of nitrogen, subsequent equilibration with surrounding tissues result in water vapour, carbon dioxide and oxygen entering the bubble. As the total pressure drops with altitude, additional quantities of these gases will diffuse into the bubble, enlarging it further until these gases are again equilibrated. As a result of this, the predicted volume increase from a 20% decrease in pressure is approximately 30% rather than the 25% that would apply if the bubble were in an inert environment. Further nitrogen may also diffuse from tissues into the bubble, resulting in further bubble growth.

A 30% increase in volume results in a diameter increase of only approximately 9% (for a spherical bubble). At lower altitudes, altitude related pressure change can be approximated as 1,000 m ASL = 1 msw ascent and an ascent of 300 m represents a pressure decrease of only 3% approximately. The diameter increase of a spherical bubble would thus be a mere 1% in the case of DCI exacerbated at an altitude of 300 m.

Bubble diameter is, however, unlikely to be the critical determinant of DCI severity in the case of altitude exposure many hours after diving when the bulk of venous gas bubbles will have dissipated. Bubbles in brain surface arteries have been shown to become trapped at pre-capillary bifurcations where they form round ended cylinders which fill the vessel. Trapped bubbles will thus expand in length in almost direct proportion to their volume increase, increasing friction and increasing the likelihood of the bubble remaining fixed in its position.⁹

Bubbles which have remained stable for some time will have a "shell" of haematologically and immunologically active substances surrounding the gas bubble and triggering inflammation and oedema in adjacent tissues. This shell is likely to be semi-rigid and may provide a gas diffusion barrier which is partly responsible for the long term persistence of bubbles despite the forces of partial pressure gradient and surface tension that normally work to decrease bubble size with time. The components of this shell are thought to be activated by the physical and chemical forces acting at the blood-bubble or tissue-bubble interface.¹⁰ It follows, therefore, that further activation could result from changes in molecular shape forced by changes in bubble wall tension, size and curvature as a result of depressurisation with altitude. This mechanism could underlie exacerbation of pre-existing DCI as a result

of an increased inflammation and oedema rather than merely in response to local pressure effects or increased blood vessel obstruction by the offending bubbles.

Hypoxia

Altitude exposure also results in significant hypoxia which may aggravate DCI pathology by several mechanisms. Commercial aircraft flight may involve a decrease in arterial oxygen partial pressure in the order of 30%. While the functions of haemoglobin and of the respiratory system in normal persons serves to maintain oxygen delivery at sufficient levels for normal, healthy tissues, this reduction in oxygenation may be critical for tissues which have marginal blood supply as a result of bubbles and adjacent inflammation associated with incipient or actual DCI.

New venous gas bubbles may be a factor in some cases, especially when a rapid ascent, higher altitude flight closely follows deeper multi-day repetitive diving activity. Excess venous bubbles are presumed to be the cause of pulmonary DCI ("the chokes") as a result of pulmonary vascular overload. They also can result in "paradoxical embolisation" of the arterial system if bubbles pass through the pulmonary capillaries or via right to left shunting through a patent foramen ovale when this abnormality is present and when pulmonary obstruction by bubbles is sufficient to result in right heart pressures exceeding those on the left.¹¹ Hypoxia is known to trigger pulmonary vascular hypertension to a variable degree.¹² This mechanism may be partly responsible for the comparatively high incidence of "chokes" which is seen with non-diving related altitude DCI as well as causing an increased risk of bubble arterialisation during flying after diving.

Altitude Exposure After Treatment For DCI

After treatment for DCI, there should not be any significantly increased levels of dissolved nitrogen left in any body tissues. The frequency of post treatment relapse would seem to indicate, however, that persistent bubbles, residual tissue inflammation and a sensitivity to re-activation of inflammation and oedema can remain for many days or even weeks even when complete resolution initially seems, clinically, to have been obtained.

Most Australian hyperbaric units currently seem to use one month post treatment as a guideline with regard to the appropriate post treatment interval before flying. The Alfred Hospital utilises this guideline although the author's clinical practice has involved proposing variations to this advice in certain circumstances. Factors in favour of early altitude exposure being considered acceptable include:

Mild DCI

Early Treatment

Rapid response to treatment

Early and complete resolution

Apparently "single site" DCI with a low likelihood of significant disability if recurrence occurs

Single ascent, low altitude, short duration of proposed altitude exposure

Treatment facilities available at destination if required

Patient with good understanding of problems, acceptance of risk and willingness to seek help if necessary

Intervals longer than one month seem appropriate in those patients who do not achieve clinical resolution and who experience symptom exacerbation with or after non-specific stimuli such as physical exertion, viral disease or lack of sleep. Local circumstances may allow such patients to undertake a voluntary trial of altitude exposure in the form of trips to hill locations of increasing altitude and duration. It seems preferable for such "at risk" patients who believe that they must fly for business or personal reasons to experience short haul domestic flight before embarking upon a long duration overseas trip to a destination without hyperbaric treatment facilities.

Current Recommendations

The range of recommendations with regard to flying after diving have received several reviews in recent years. A gulf appears to exist between decompression theory based tables and dive computer function on the one hand and individual or consensus based blanket guidelines on the other.^{13,14}

The recommendations in published guidelines for flying after non-saturation diving range from a delay of a few hours only to 24 hours or more. Decompression diving is often treated differently from no-decompression diving and a few publications provide for reduced intervals in the case of lower altitudes or emergency circumstances. After reviewing many of these, the 1979 Undersea and Hyperbaric Medical Society workshop on Flying After Diving issued consensus guidelines (Table 2).¹⁴

It is noteworthy that the late Dr A. Bühlmann indicated that he saw a necessity for guidelines for road travel. For dives involving less than 60 minutes dive time in the previous 12 hours he proposed a delay of 4 hours only before travel to altitudes up to 3,000 m ASL. This was apparently based upon his decompression research and experience gained by Swiss divers who have followed tables allowing altitude exposure only a few hours after diving. Such divers usually gain their altitude exposure during road or rail travel rather than flight, however, and a comparison of typical ascent rates is of interest (Table 3).

TABLE 2

FLYING AFTER DIVING RECOMMENDATIONS

No-decompression diving

- a Less than 2 hrs total dive time in previous 48 hrs
12 hrs delay
- b Multi-day, unlimited air diving
24 hrs delay

Decompression diving

24 hrs minimum, preferably 48 hrs delay

Recommendations

With the number of Alfred Hospital cases requiring treatment following surface based travel nearly equalling those following flying, there is clearly a need for guidelines to cover more than simply commercial airline flight. Flying after diving guidelines sometimes do assume minimum altitudes. If air transport is necessary for DCI, retrieval is usually arranged at 300 m ASL or less. This seems a reasonable limit for immediate land based travel and helicopter transport also. The practice of some emergency retrieval aircraft flying at less than 30 m ASL seems unnecessarily and dangerously low.

It is apparent that a significant number of divers fly with symptoms of DCI or having suffered symptoms of DCI in the period preceding flying. It is unknown, of course, as to how many divers do this without suffering sufficient adverse consequences to result in them seeking help. Nevertheless, there is clearly a need for more awareness of the risks associated with DCI prodromal symptoms such as lethargy, malaise, and transient “pins and needles” or “niggles”. Caution also is required if dive limits have been exceeded in any way or if adverse factors such as rapid ascent, undue exertion or thermal stress have occurred. If obvious symptoms are apparent, consultation with an experienced diving and hyperbaric medicine physician is, of course, appropriate.

All divers planning flying after diving should make their bookings with contingency plans in place to allow for rescheduling of flights if required. Where hyperbaric facilities are not present, post diving oxygen breathing is likely to greatly reduce risk if flying cannot be delayed and an “at risk” situation has occurred or potential DCI prodrome symptoms have been noted.

It is inevitable that some cases of altitude related DCI will continue to occur, given the unpredictable nature of the condition. It would appear that the physics and pathophysiology of altitude exposure may create a far greater

TABLE 3

ASCENT RATES OF VARIOUS ACTIVITIES

Ascent type	Rate of Ascent	Pressure change per min
Recreational diving	9-18 m per minute	30-65%
Air travel	1,000 m altitude in 2-10 minutes	1-5%
Driving over mountains	1,000 m altitude in 15-60 minutes	0.15-0.6%

unpredictability in DCI risk than is the case following diving activity alone. Divers should make their travel plans with an awareness of this. Although even a 12 hour delay prior to flight is probably unnecessarily cautious following most diving involving one dive per day well within conservative no-decompression limits, the current common practice of waiting 24 hours seems more appropriate as a general standard. Multi-day, deep repetitive dives should be followed by at least 36 hours or more before flying (two night’s rest). Divers should remember that travel to hill locations during a diving holiday increases risk and any abnormal symptoms, injuries or intercurrent illnesses suffered during a trip should be an indication to cancel or at least severely restrict remaining diving activity or alternatively, to delay flying. The scheduling of shallower dives towards the end of a diving holiday seems desirable.

If DCI symptoms do arise during commercial air travel, 100% inspired oxygen will not be available. Nevertheless, use of low concentration therapy oxygen which is carried by some operators may be of value given that the effect of additional oxygen at altitude is far higher than at the surface or in hyperbaric situations.⁶

Following DCI treatment, hyperbaric physicians should assess the clinical progress of the patient before allowing even minor altitude exposure in the first few days post treatment. The current one month guideline appears generally reasonable provided that patients who have responded poorly are individually assessed if flying is planned.

References

- 1 Emmerman M. *Flying and diving, a new look.* Lifeguard Systems Inc. 1987
- 2 Aeronautical Information Publications, Civil Aviation Authority, Australia
- 3 Spencer M. Decompression limits for compressed air

- determined by ultrasonically detected blood bubbles. *J Appl Physiol* 1976; 40 (2):229-235
- 4 Dunford R. et al. Ultrasonic doppler bubble incidence following sports dives *Undersea Biomed Res* 1988; 15 (Suppl); 45-46
 - 5 Conkin J and Van Liew H. Failure of the straight line DCS boundary when extrapolated to the hypobaric realm. *Aviation Space & Environmental Med* 1992; 63 (11); 965-970
 - 6 Van Liew H. et al. The oxygen window and decompression bubbles: estimates and significance *Aviation, Space & Environmental Med* 1993; 64 (9 pt 1): 859-865
 - 7 Eckenhoff R. et al. Human dose-response relationship for decompress and endogenous bubble formation *J Appl Physiol* 1990; 69 (3): 914-918
 - 8 Kumar K. et al. Threshold altitude resulting in decompression sickness. *Aviation, Space & Environmental Med* 1990 61 (8): 685-689
 - 9 Gorman D. et al. The distribution of arterial gas emboli in the pial circulation. *SPUMS J* 1987; 17 (3): 101-116
 - 10 Hallenbeck J. and Anderson J. Pathogenesis of the decompression disorders. (Chapter 17) in Bennett PB and Elliott DH Eds. *The Physiology and Medicine of Diving 3rd Ed.* London: Balliere Tindall, 1982
 - 11 Moon R. et al. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989; 1: 513-514
 - 12 Schoene R. Pulmonary edema at high altitude. Review, pathophysiology and update. *Clinics in Chest Medicine* 1985; 6: 491-507
 - 13 Lippmann J. Dive computers and flying after a dive to 39 m for 10 minutes. *SPUMS J* 1990; 20 (1): 6-7
 - 14 Sheffield P. *Flying after diving; Proceedings of the 39th Undersea and Hyperbaric Medical Society Workshop*. Bethesda, Maryland: UHMS, 1989

Key Words

Altitude, decompression illness, flying, treatment sequelae.

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