

minutes before, 30 minutes before and just before the dive) and another immediately after the dive.

A blood glucose level over 160 mg/dl (8.96 mmol/l) before every dive.

Depth limited to 30 m.

Use the diabetic dive log to accumulate "reference" data for similar dives.

Sufficient hydration (a minimum of 2 l before the first dive but not more than 1 l/hour).

Late testing (12-15 hours) after the dive to detect and prevent hypoglycaemia due to muscle-storage depletion.

Oxygen and emergency equipment at the dive site.

We have demonstrated that diving in tropical waters, while on insulin, can be safe for a healthy diabetic with a stable blood glucose situation when training and experience under non-diabetic "buddy-control" is given.

References

- 1 Schnabel A. Diabetes und körperliche Belastung. *Schwerpunkt Medizin* 1987; 10 (3): 37
- 2 Goldgewicht C, Slama G, Patpoz and Tchobroutsky G. Hypoglycaemic reaction in 172 type I diabetic patients. *Diabetologica* 1983; 24: 95-99
- 3 Parker J. *The Sports Diving Medical*. Melbourne; J L Publications, 1994
- 4 Edge C, Lindsay D and Wilmshurst P. Diabetes and diving. *Diver* 1992; 37 (2): 35-36

Key Words

Buddy, diabetes, drugs, environment, fitness to dive, research, safety, training.

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HYPERBARIC RETRIEVALS IN TOWNSVILLE: IS A PORTABLE CHAMBER USEFUL?

Christopher Butler

Abstract

A review of the retrievals of divers with decompression illness (DCI) to the recompression chamber at the Townsville General Hospital (TGH) was conducted for a 2 year period. For the second half of the study a portable recompression chamber was not available for retrievals.

Assuming that portable recompression chamber retrievals were justified in divers with unstable moderate and severe disease, only 1 diver in 108 cases of DCI treated at TGH may have potentially had a better outcome had the facility been available over the second year of the study.

Using patient outcome at discharge as the end point, we cannot demonstrate any superiority of the portable chamber over expeditious sea-level air transport to the hospital based chamber.

Introduction

Recreational diving, by its very nature, tends to be conducted in remote locations. The Great Barrier Reef stretches for some 2,000 km along the Queensland coast and is one of the world's premier dive locations. Currently (November 1995) there is only one hospital based hyperbaric unit in Queensland, situated at the Townsville General Hospital (TGH).

All cases of decompression illness (DCI) treated at the TGH over a 2 year period have been reviewed. During the second year of this study, divers were transported without the availability of a portable recompression chamber (PRCC). This has provided an opportunity to assess the usefulness of such a unit in support of a hospital based multiplace chamber.

Methods

A retrospective review of patient records from 25/4/93 to 30/6/95 was conducted. Due to the loss of the chamber life support technician employed by the Queensland Emergency Services, the PRCC (a Dräger DuoCom) became unavailable for diver retrievals on the 30th June 1994. Subsequently divers who would have previously been retrieved and treated during transport were transferred by air at sea-level cabin pressure and then definitively treated in the TGH multiplace chamber.

During the first year of the study the TGH unit's operation was interrupted for a 66 day period while the main chamber was moved to its current location within the hospital. The PRCC during this time was used to transfer patients interstate to other hospital based facilities. This period represents an abnormal use pattern, in that the portable chamber was not being used in support of the hospital based facility. Because of this, all patients managed during the 66 day period were excluded from the study.

Patients

The patients were divided into two groups.

Year 1: 25/4/93 to 30/6/94 (excluding 66 days, 365 days available for treatment) PRCC available.

Year 2: 1/7/94 to 30/6/95 (365 days available for treatment) PRCC unavailable.

All patients who received treatment at TGH for new episodes of DCI were included. Patients were excluded from the study if:

- 1 They were being treated for recurrence of DCI. These patients were only considered for their initial presentation.
- 2 They had undergone a "trial of pressure" to establish the diagnosis of DCI, and this proved negative.
- 3 For whatever reason they did not receive recompression.

Each patient file was reviewed and the following information recorded.

Severity of disease.

This was stratified into 3 groups (Table 1) for the purposes of the review.

- 1 "Mild Disease" patients who were symptomatic for DCI but no objective signs could be elicited.
- 2 "Moderate Disease" patients who had symptomatic DCI and signs of a subtle nature. These included positive Sharpened Romberg test, impaired higher function on simple testing, objective sensory changes, mild weakness or changes in deep tendon reflexes.
- 3 "Severe Disease" patients with life or mobility threatening DCI. These included pulmonary and cardiovascular manifestations, loss of consciousness, bladder or bowel impairment or severe weakness.

TABLE 1

DCI SEVERITY

DCI	Year 1 (PRCC)		Year 2 (No PRCC)	
	Divers	%	Divers	%
Mild	44	50	63	58
Moderate	41	47	41	38
Severe	3	3	4	4
Total	88	100	108	100

TABLE 2

DCI STABILITY

DCI	Year 1 (PRCC)		Year 2 (No PRCC)	
	Divers	%	Divers	%
Stable	75	85	95	88
Unstable	11	15	13	12
Total	88	100	108	100

Stability of disease

Patients were classified as stable or unstable (Table 2). Unstable patients had deteriorating symptoms or signs at the time of referral.

Location

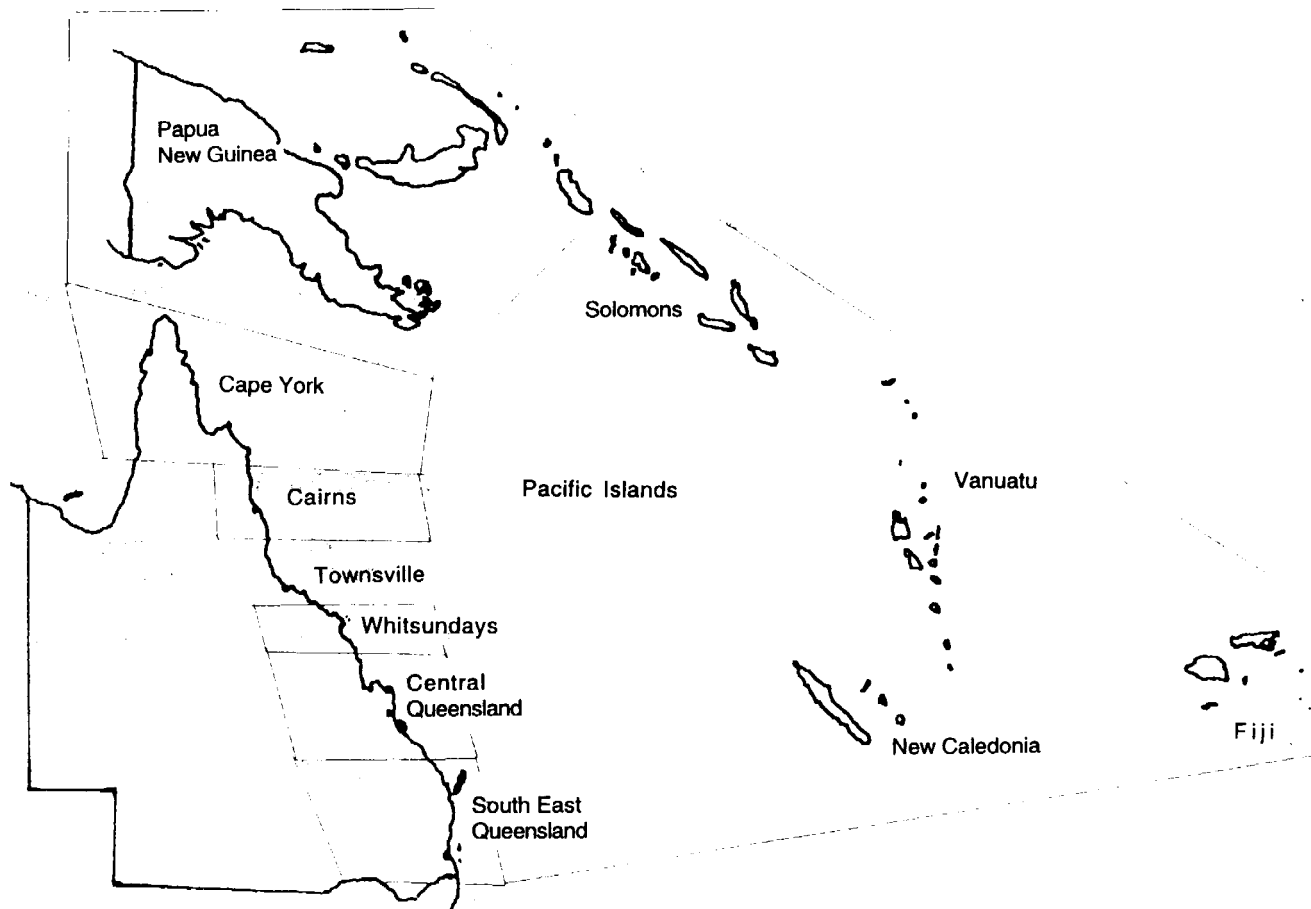
The locations where the diver first sought medical attention were grouped into 8 zones (Table 3 and Figure 1).

- 1 Townsville.
- 2 Whitsundays, including Bowen, Airlie Beach, Whitsunday islands, Proserpine and Mackay.
- 3 Cairns and district.
- 4 Central Queensland.
- 5 Cape York and surrounding islands.
- 6 South East Queensland.
- 7 Papua New Guinea.
- 8 Pacific islands (Vanuatu, New Caledonia, Fiji).

Method of transportation

This was either by sea level air transport (helicopter or pressurised fixed wing aircraft), PRCC or road transport.

FIGURE 1
AREAS FROM WHICH PATIENTS WERE RETRIEVED



Results

The cases of DCI that were treated at the TGH hyperbaric unit in the 2 years under review have been classified by location, disease severity, disease stability and method of transport.

In year 2, following the decommissioning of the portable recompression unit, 108 DCI patients were treated at Townsville. Only 24 patients (22%) presented directly to TGH or doctors in the Townsville area.

The majority of the patients transported by air at sea level pressure were managed in that way because it represented the most practical method of transporting them to Townsville. Most of the patients with mild or stable moderate disease presenting to Cairns or the Whitsunday region were transported by road.

If one accepts that use of the PRCC was justified in cases of moderate unstable or severe disease presenting outside the Townsville area, the facility would have been the method of choice for transporting 9 patients in the year following its withdrawal, had it been available.

The outcome at the time of discharge for this group was asymptomatic seven patients, mild residual symptoms one patient and severe residual disease (paraplegia) one patient.

The patient with mild residual symptoms was retrieved from Noumea following initial recompression and so a portable unit would not have allowed for earlier treatment.

It can be seen that if a selective use pattern had been applied to the PRCC (had it been available) during year 2, only 1 patient in the 108 cases of DCI would have potentially benefited from the facility being available. This takes no account of the number of treatments required, and only considers the outcome at discharge.

In year 1, before the loss of the PRCC, 88 divers were treated at TGH for DCI. If the same selection criteria were retrospectively applied for the use of the PRCC (unstable moderate or severe disease), 7 patients would have ideally been transferred in the PRCC.

TABLE 3

PATIENT LOCATION

	Year 1 (PRCC)		Year 2 (No PRCC)	
	Divers	%	Divers	%
Cairns	32	36	40	37
Townsville	25	28	24	22
Whitsunday	8	9	17	16
Papua New Guinea	2	2	12	11
South East QLD	9	10	4	4
Cape York	7	8	2	2
Central QLD	3	3	4	4
Pacific Islands	2	2	5	5
Total	88	100	108	100

The outcomes for this group were 1 patient (transferred with the PRCC) discharged with moderate residual weakness, one suffered a relapse after being discharged symptom free, 1 had non-specific fatigue at discharge and 2 were asymptomatic at discharge. Two of the 7 patients considered likely to benefit from transfer in the PRCC were actually transferred at sea level and were asymptomatic at discharge.

Discussion

The pathophysiology of DCI involves tissue damage as a result of bubble formation causing direct tissue injury or ischaemia secondary to impaired perfusion.¹ Intuitively, the outcome from an episode of DCI should be related to the degree of bubble formation and the length of time that the bubbles were in a position to cause tissue damage.

This philosophy has led to the common practice of employing portable recompression chambers to allow the rapid treatment of divers. This has been the standard of practice for the treatment of seriously affected divers in our institution.² Although the West Australian experience placed doubt on the benefit of this approach, the use of the PRCC has been recommended by other Australian units.^{3,4}

Leitch and Green in a large review of cases of cerebral arterial gas embolism indicated that delay in recompression resulted in a poorer outcome.⁵ A study of 49 cases of spinal cord DCI reported by Bull also gives some support to this contention.⁶ With severely affected divers, increasing the time from onset of symptoms to recompression correlated with a poorer outcome. However, less severely affected divers tended to have a good outcome regardless of the time to recompression.

TABLE 4

METHOD OF TRANSPORT

	Year 1 (PRCC)		Year 2 (No PRCC)	
	Divers	%	Divers	%
Road	54	61	73	68
Sea level pressure	19	22	35	32
PRCC	15	17	-	-
Total	88	100	108	100

Kizer came to a similar conclusion in a review of delayed treatment of mainly civilian divers treated in Hawaii.⁷ This lends support to the policy of emergency retrieval and treatment of severely affected divers.

The loss of the portable recompression unit for diver retrieval has forced a re-evaluation of this practice in our institution. From the data presented above, the use of the PRCC would not appear to greatly effect patient outcome from DCI in the diving population that was treated in Townsville, provided that expeditious sea level transfer and treatment was available. No attempt has been made to relate the use of the PRCC to the number of recompressions that a diver requires.

It was important to consider the disadvantages of the PRCC in patient management. These chambers take time to load into an aircraft (approximately 40 minutes) and provide poor access to a potentially sick patient. The PRCC was also heavy, bulky and expensive in both maintenance and manpower. Their utility in the helicopter available to us (Bell 412) was limited and we have only routinely deployed them in a fixed-wing aircraft.

Our experience indicates that the PRCC adds little to the management of divers with the pattern of DCI that we treat. Provided that an efficient sea level retrieval system was available, we could not endorse the widespread use of portable units.

References

- 1 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine*. 3rd Ed. Oxford: Butterworth Heinemann, 1992; 159
- 2 Williamson J, Orton J, Callanan V, et al. The Townsville diving medical and aeromedivac system. Experiences, lessons, and the future. *SPUMS J* 1988;18 (3): 82-87
- 3 Oxer HF. Is transport of diving casualties under pressure worth it? *Proceedings IX International*

- Congress on Hyperbaric Medicine.* Sydney, Australia 1987; 137-138
- 4 Gorman D. Decompression sickness and arterial gas embolism in sports scuba divers. *Sports Medicine* 1989; 8 (1): 32-42
- 5 Leitch D and Green R. Pulmonary barotrauma in divers and the treatment of cerebral arterial gas embolism. *Aviat Space Environ Med* 1986; 57: 931-938
- 6 Bull R. Effect of severity, time to recompression with oxygen, and re-treatment on outcome in forty-nine cases of spinal cord decompression sickness. *Undersea Hyperbaric Med* 1993; 20 (2): 133-145
- 7 Kizer K. Delayed treatment of dysbarism. A retrospective review of 50 cases. *JAMA* 1982; 247 (18): 2555-2558

Key Words

Decompression illness, transport, treatment sequelae.

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THE WORLD AS IT IS

SOME DIABETICS ARE FIT TO DIVE, BUT WHICH ONES?

THE AUSTRALIAN EXPERIENCE AND SPUMS POLICIES

John Williamson

SPUMS, the South Pacific Underwater Medicine Society, has at times been accused of advocating that "only perfect physical specimens should dive"! Regarding diabetes and diving, the original SPUMS statement published in 1992,¹ opposed diving for all but the diet-controlled diabetic. It contained no hard supporting data. The paper was followed by some vigorous opposing views² and criticism³, but little more than anecdotal opposing data.

Bryson, Edge and colleagues^{4,5} and Dear and colleagues⁶ gave notice of data collection in 1994. This and recent early data from Stephen Prosterman, of the Diabetic Association of the Virgin Islands ("Camp DAVI"),⁷ lends support to the premise that certain insulin dependent diabetics (IDDM) can dive safely under the right control conditions. Opposing opinions continue.^{8,9} On-going data collection from DAVI is promised.⁷

What "decent" Australian data exists, relevant to diabetes and diving? Medline contains none. There are none in the data base of the "DES Australia" phone. This contains detailed records of 1,950 calls since 1987, both Australian and international. Data from 1987-1990 are published¹⁰ and 1991-1995 are in preparation for publication. There appear to be none contained within the "Project Stickybeak" data base, a continuous series of detailed mortality and morbidity events in Australian and New Zealand diving from about 1969 to the present.¹¹

That there are Australian (and thus likely New Zealand) divers with Type I diabetes mellitus is certain.^{2,3} There is an interesting new study by Lerch, Thurm and Lutrop from North Queensland which supports diving for selected insulin-dependant diabetics. It is being prepared for publication in the SPUMS Journal (see pages 62-66).

There are extensive worldwide data on both Type I and Type II diabetes mellitus unrelated to diving. In addition consultation with experienced, but non-diving, diabetologist colleagues has occurred. Present evidence is that diving produces a fall in blood glucose levels in diabetics^{6,7} and that measurement of peri-diving blood glucose levels is necessary for safety.^{4,5} More "hard in-water data" are necessary.

The case against

Diving diabetics, even experienced ones, cannot always be relied upon to measure their peri-diving blood glucose levels.⁷

Irregular food absorption (eg. sea sickness), or even meal timing (eg. alcohol), may occur during diving activities. Conditions may also predispose to insulin or drug administration errors.^{8,12}

Hypoglycaemia symptoms usually begin at a blood glucose level of less than 2.5 mM/l (45 mg/decilitre). The onset can be rapid (minutes), will affect central neurological function (judgement, vision, consciousness) and any warning autonomic symptoms which normally precede those of CNS dysfunction (sweating, shaking, palpitations) may be hidden underwater.

Undetected autonomic neuropathy⁹ in a diving diabetic may result in masking of warning symptoms of