ORIGINAL ARTICLES

INITIAL TREATMENT OF DECOMPRESSION ILLNESS: A SURVEY OF AUSTRALIAN AND NEW ZEALAND HYPERBARIC UNITS

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Summary

Current treatment for decompression illness (DCI) is usually based on experience, current research data and ongoing clinical assessment. In order to ascertain the current Australasian practice in the treatment of DCI, a four page survey was forwarded to doctors associated with hyperbaric units in Australia and New Zealand. This comprised five clinical scenarios with questions relating to initial therapies, need for hyperbaric oxygen, treatment tables and follow up practice. Thirteen completed forms and protocols from two hyperbaric units were returned (63% response). Results indicated a wide variation in initial assessment policies, treatment protocols and follow up for patients presenting with various categories of DCI. Current opinion is that some recompression profiles may not prevent long term sequelae of DCI, however interpretation of outcome data is difficult due to the variability in treatment policies. It would be of value to have standardised protocols for assessment, treatment and follow up of DCI in order to formulate a rational and effective treatment plan for this group of patients.

Introduction

Anecdotal experience and application of current research data, combined with widely differing backgrounds in training, ensure that there is no uniform approach to the treatment of patients requiring hyperbaric treatment for DCI. This variable approach means that it is difficult to compare the results from various units and to indicate which are the most appropriate treatment protocols. As a result of discussions about the treatment tables used in the management of a group of divers with delayed presentation of DCI, it became obvious that there was a wide variation in treatment options and therefore an attempt was made to ascertain the treatment protocols of the different units around Australia and New Zealand.

Methods

A four page questionnaire was sent to medical staff involved in the clinical decision-making in all the hyperbaric units in Australia and New Zealand. This involved the completion of a structured series of questions relating to specific clinical scenarios that could be encountered in the daily running of a hyperbaric unit. The questionnaire was confidential and anonymous.

The recipients were asked to answer the following five questions for three scenarios.

1 What investigations would you perform, if any, before recompression?
2 What recompression tables would you use initially? What recompression schedule would you use if the symptoms failed to respond to the initial therapy?
3 What other measures would you use in conjunction with recompression therapy?
4 What follow up treatments would you use?
5 How long would you continue to treat the patient?

CASE 1

A 27 year old Japanese diver made a rapid uncontrolled ascent from 15m. Immediately on surfacing, he lost consciousness and started to convulse. He was immediately dragged into the boat where he started to breathe normally and stopped fitting. He remained unconscious. 100% oxygen was administered. Within 90 minutes of surfacing he was transported 100 km by helicopter, flying at 25 m altitude, to the local recompression chamber. (Acute neurological DCI, probable CAGE)

CASE 2

A 39 year old tuna farm diver, who had been treated for DCI 3 weeks previously, presented with increasing malaise, excessive lethargy, inability to sleep, recurrence of his shoulder pain and slight paraesthesia in his left hand. These symptoms were similar to his initial presentation, but less intense. (Relapsing neurological and musculoskeletal DCI)

CASE 3

An experienced cave diver presented to your unit following a series of dives to 55 m on air. His last dive was 6 hours previously (with in water decompression using DCIEM tables). He phoned you complaining of difficulty with walking, weakness in his legs with paraesthesia and patchy loss of sensation in his legs. He had one episode of urinary incontinence. He was transported to your unit by ambulance on 100% O_2, with no significant relief of symptoms. On examination he had flaccid paralysis of both lower limbs and a palpable bladder. This was associated with loss of sensation to light touch and pin prick to a sensory level of T 8. There was nothing else significant in his dive profile or past history. (Progressive neurological DCI)

For the other two scenarios the questions were different.

CASE 4

You were contacted from a Pacific island 1,000 km from the nearest recompression facility. A 29 year old accountant, on holiday, presented to the resort doctor with severe pain in both shoulders and slight weakness in both legs following a wall dive to 30 m for 40 minutes. The diver felt the weakness was progressing. He had no travel insurance. (Acute progressive DCI without a chamber)

Q1 What would be your advice?
Q2 Would you consider in-water oxygen therapy?

CASE 5

A 45 year old phoned you for advice 2 days after a weekend of cray fishing. He undertook a series of 5 dives in 2 days at depths of no greater than 7 m; in-water time was approximately 70 minutes each dive. He complained of slight paraesthesiae in his hand, but felt otherwise well. His GP found no objective signs of DCI on examination, however a trial of 100% O_2 produced a slight decrease in his feeling of pins and needles in his hand. He was 6 hours from the nearest recompression facility. (Possible mild DCI)

Q1 Would you recommend HBO treatment?
Q2 What would be your initial treatment?

Results

A total of 24 questionnaires were sent out. Thirteen (54%) were returned completed. A further two responses were given as flow charts of departmental protocols, giving a total response rate of 63%. In some cases some respondents failed to answer all questions, while in other questions more than one response was indicated on the survey form.

Two units did not respond directly to the questions set, but forwarded their set protocols. One unit commences all patients on RN 62 table and either completed the RN 62 or went to a Comex 30 ($He:O_2$) + 50 m extension. Treatments were followed up by RN 61 or RN 62 tables.

The other unit categorised their patients into *Critically ill* (requiring CPR) treated with RN 63, *Severely ill* (unconscious or with severe CNS problems) given Comex 30 ($He:O_2$) and *Mild to moderately ill* treated with RN 62 or Comex 30. Follow up treatments would be either Comex 30, RN 62 or RN 61

Initial investigations

Investigations performed before recompression for cases 1-3 are shown in table 1. The initial investigations for DCI ranged from none to CT and MRI scanning. It is interesting to note that whereas in other branches of acute medicine, blood screening tests, e.g. complete blood count, electrolytes, liver function tests, cardiac enzymes and a coagulation screen, are often routinely performed, this is not the norm in this group of patients. While the cost-benefit analysis of such "routine" tests must be taken into account, objective evidence of disease severity and treatment progression needs to be identified. Recently it has been suggested that serum creatine kinase may define severity and possible prognosis in divers presenting with gas embolism.[1] Other markers of DCI, such as complement activation, platelet count, white cell count and activity, need further study in man for diagnostic, therapeutic and prognostic purposes. Divers need to be assessed in a similar fashion to other acutely ill patients. Full radiological and blood test investigations should be considered before they are subjected to potentially long periods of recompression. However as in other aspects of acute care, delays incurred by these investigations must be balanced against the urgency of recompression and the patient's condition.

Adjuvant therapy

Most respondents used intravenous fluid therapy, with other modalities such as lignocaine, non-steroidal analgesics, steroids, dextran and inotropes used less commonly (Table 2). Lignocaine as adjuvant therapy was considered by a large number of respondents both for use within the hyperbaric chamber and also as first line therapy if HBO was not immediately available. The benefits of lignocaine are well documented in animal studies and include reduction in neutrophil-endothelial adhesion, reduction in free oxygen radical release along with reducing intracranial hypertension.[2,3] Human data is small and confined to anecdotal case histories where it has been used in cases refractory to recompression therapy.[4] Prospective controlled studies are needed to assess the potential role of lignocaine in the treatment of DCI. Important questions to answer include dose response data, length of therapy and efficacy. Consideration also needs to be given to the possible role of lignocaine in association with hyperbaric oxygen and any effect it may have on CNS convulsive thresholds. The same comments can be aimed at the other forms of adjunctive therapy. Well constructed, randomised clinical studies have not been done to validate most of the adjunctive treatments in man.

Treatments

The initial recompression schedules used to treat cases 1-3 are shown in table 3. Table 4 defines the treatment tables used by the respondents. The reason for using the stated table was not specifically asked for, however it is likely that the choice is determined by several factors which include past experience along with current

TABLE 1

PRE-TREATMENT INVESTIGATIONS*

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Chest X-ray	7	Chest X-ray	1	Chest X-ray	5
CT scan	2	Cervical spine X-ray	2	CT scan	3
Full blood count	6	MRI scan	1	MRI scan	3
Arterial blood gas	2	Shoulder X-ray	2	Electrolytes	5
Electrocardiogram	2	Full blood count	5	Full blood count	5
		Electrolytes	3	Electrocardiogram	3
		Somatosensory evoked potentials	1	Spirometry	1

* Each reply may have had more than one response

TABLE 2

ADJUNCTIVE TREATMENT FOR DCI

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Intravenous fluids	13	Intravenous fluids	5	Intravenous fluids	11
Lignocaine	9	Lignocaine	5	Lignocaine	7
Steroids	1	NSAIDS	1	NSAIDS	2
Inotropes	1				
Dextran	1				

Each reply may have had more than one response
NSAIDS; non-steroidal anti-inflammatory agents

TABLE 3

TREATMENT SCHEDULES FOR DCI*

	Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Initial	RN 62	9	RN 62	11	RN 62	12
	RN 63	2	18: 60: 30	1	30 m[a]	3
	Comex 30	2	14m[a]	1	50 m[a]	1
Fail	RN 62 (extended)	4	RN 62 (extended)	2	RN 62	4
	RN 63	4	Comex 30	1	30 m[a]	6
	30 m[a]	8	Continue with current table[b]	9	50 m[a]	1

* details of recompression schedules are described in Table 4
a Depth but not table stated
b In 9 cases, if the symptoms had not settled, the original table would be continued without alteration.

TABLE 4

DEFINITION OF TREATMENT TABLES.

Some respondents only gave depths rather than specific tables, therefore details of exact treatment schedules cannot be given.

RN 61

Royal Navy table 61 or United States Navy (USN) table 5. Maximum depth 18 m. 18 m for 45 minutes then decompress at 0.3 msw/minute to 9 m; 9 m for 25 minutes then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 2 hours 15 minutes.

RN 62

Royal Navy table 62 or USN table 6. Maximum depth 18 m. 18 m for 75 minutes, then ascend to 9 m at 0.3 msw/minute; 9 m for 135 minutes then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 4 hours 45 minutes.

RN 63

Royal Navy table 63 or USN table 6A. Maximum depth 50 m. 50 m for 30 minutes then decompress to 18 m at 8 msw/minute; 18 m for 75 mins then decompress at 0.3 msw/minute to 9 m; 9 m for 150 minutes then ascent to surface over 30 minutes. Breathing medium oxygen and air, however modifications using helium oxygen mixtures are used. Duration 5 hours 19 minutes.

18: 60: 30.

Maximum depth 18 m. 18 m for 60 minutes, then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 1 hour 30 minutes.

10: 90: 30.

Maximum depth 10 m. 10 m for 90 minutes, then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 2 hours.

Comex 30

Maximum treatment depth 30 m. There are several variations on this table. An example of one is as follows: 30 m for 120 minutes, then decompress to 24 m over 35 minutes; 24 m for 35 minutes then decompress to 18 m over 35 minutes; 18 m for 95 minutes then decompress to 12 m over 35 minutes; 12 m for 185 minutes then ascent to surface over 25 minutes. Breathing medium 50-50 helium-oxygen or nitrogen-oxygen and oxygen with air breaks. Duration 7 hours 15 minutes.

There are many ways of measuring (expressing) chamber pressure. All involve a gauge which measures the pressure above atmospheric. These may be expressed as depth, feet of sea water (fsw), metres of sea water (msw)(which have been used in this paper) or as pressures, pounds per square inch (psi) (seldom used in Australasia), bar (equal to 10 msw), Pascals (Pa = Newton x m^2) or multiples of the Pascal such as kilopascal (kPa) and megapascal (MPa). KPa are convenient as they are used in measuring medical gas supplies. Msw and fsw should not be used for expressing partial pressures of gases.

Users of any measuring system must remember to add atmospheric pressure to the chamber pressure before trying to calculate actual gas pressures. This is difficult using msw so the conversion table below is provided for readers to work things out for themselves in more familiar units.

Depth to pressure conversions.

Depth	Pressures	
msw	bar	kPa
At surface	1.0	100
9	1.9	190
10	2.0	200
12	2.2	220
18	2.8	280
24	3.4	340
30	4.0	400
50	6.0	600

research data. There is at present debate as to the effectiveness of the US Navy schedule, with long term problems being reported despite initial treatment success.[5] Similarly the use of helium as part of the recompression schedule may have a place in clinical practice based upon data from Hyldegaard[6] along with clinical data in man with spinal cord DCI.[7] Interim data from the Royal New Zealand Navy heliox study, whilst showing a reduction of initial recompression treatments and cost saving, failed to show significant clinical benefit of heliox over oxygen.[8] Finally, saturation treatments have been used for the initial treatment and for those refractory cases of DCI, however again there are no controlled trials of efficacy.

Recompression schedules used for follow up treatments were even more variable than those used for initial therapy (Table 5). Nine replies indicated that recompression therapy would continue until the symptoms has failed to improve or resolved plus one additional treatment i.e. plateau + 1. Two units continued for a plateau + 2 while one reply indicated a set protocol was used, but did not state the details.

TABLE 5

FOLLOW-UP TREATMENTS FOR DCI

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
RN 62	3	RN 61	1	RN 62	2
RN 61	3	18: 60: 30	2	RN 61	3
18: 60: 30	2	14 m[a]	1	18: 60: 30	3
14 m[a]	1	10 m[a]	6	14 m[a]	1
10 m[a]	4	RN 61 + 10 m x 3	1	10 m[a]	4

[a] Depth but not table stated

There appears to be no consensus on the appropriate oxygen dose (partial pressure, duration or frequency) of follow up treatment tables, although most units would continue treatment in the acute phase up to either resolution or plateauing of symptoms. In one of the few studies looking at follow up treatments in cases of DCI, Wilson suggested in a retrospective study that there were fewer recurrences of DCI in divers treated at 2.8 ATA compared to 2.4 ATA.[9] This needs to be repeated in a controlled prospective study.

Patients are increasingly demanding to know the rationale for treatment in all branches of medicine. Hyperbaric medicine needs to be able to provide hard data on which to base treatment plans. This uniformity of treatment would also help reduce the possibility of insurance and other funding debating management. Moreover in these days of Casemix, DRGs and fiscal constraints, there is a real need to justify and support rationales for different therapeutic modalities.

In-water oxygen therapy

The advice given to case 4 included 100% surface oxygen (10), intravenous fluids (10) and the need for retrieval (9). Lignocaine was advocated for use by 2 respondents. Specific problems of in-water recompression were mentioned in some replies. These included, "symptoms of DCI may worsen in water", "in-water oxygen convulsions" "divers have died using in-water oxygen" and "unable to evaluate a diver in the water". The major problem seen by the respondents to this survey was the danger of in-water convulsions leading to barotrauma, gas embolism and death. Other problems included the inability to adequately clinically evaluate divers underwater. Perhaps the view that, for the trained specialist with technical and resuscitative skills who has performed the exercise before, it may be a possible temporising measure until definitive treatment is available, is too restrictive.

While in-water decompression on oxygen has been shown to be safe in over 18,000 dives when breathed at depths between 3 and 6 m, its use at deeper depths for in-water recompression is not supported.[10-12] Indeed at the 1994 UHMS meeting in Denver, a panel of four diving medical specialist all agreed that they would seriously consider the use of in-water oxygen if the clinical condition warranted it. Experience in navies and off shore commercial diving has shown that immediate treatment gets better results than treatment delayed by an hour or two. In water oxygen therapy has a simple protocol, which can be carried out wherever there is 6 m of protected water. It is an emergency treatment approved for use when appropriate by the Royal Australian Navy (RAN) and the United States Navy (USN).[12] The patient is conscious and accompanied by an attendant diver at all times. He or she can assess changes in their own condition. Case 4 had progressive neurological DCI and many hours to wait for evacuation, just the case that naval diving manuals suggest should be treated by underwater oxygen at 6 m. However respondents in the survey preferred 100% surface oxygen and intravenous fluid hydration.

Overseas retrieval

Diving incidents overseas can be financially disastrous for those uninsured. As an example, from a Diving Emergency Service (DES) call in 1994, the cost of a retrieval from Fiji to Melbourne was costed in the vicinity of A$40,000. It is also necessary that divers are adequately insured so that retrieval can be an easily carried out possibility. Severe DCI requires rapid assessment by a doctor knowledgeable in diving related problems and

subsequent retrieval, if necessary to a recompression facility. This type of retrieval however is not without risk as was shown in 1995 over the South Pacific when a plane retrieving two divers crashed, killing all aboard.

Late vague symptoms

The problem of a diver who has vague neurological symptoms a few days after a dive is not uncommon. 10 respondents elected to treat the diver with a RN 62, with one considering taking him to 50 m if the symptoms failed to resolve. The question to treat is often tempered by the distance factor; i.e should one suggest that a diver with a mild ache and paraesthesiae in his wrist which is not worrying, and possibly getting better, travel hundreds of kilometres to the nearest recompression facility. Does the present data support such advice? That question is still unanswered.

Discussion

The survey was intended to obtain a snapshot of how the various hyperbaric units in Australasia treat various types of decompression illness. Although the response rate was only 63%, there was at least one reply from each unit in Australia and New Zealand, therefore the survey does represent current practice in this area. There is wide variation in the assessment, preliminary investigations, adjunctive therapy and treatment tables used for DCI.

While the results from conventional recompression therapies may be variable, there is great difficulty in comparing the results from different units due to the difference in treatment regimens. Australia and New Zealand are in a unique position, both geographically and because of the small number of doctors involved in hyperbaric medicine, to foster the collaboration of the various hyperbaric units in the quest for more rational and uniform treatment profiles. Groups such as the South Pacific Underwater Medicine Society (SPUMS) or the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) could provide a platform on which to base consensus statements on policies regarding assessment, investigation, treatment profiles and follow up management.

There is already established a national database for decompression illness in the United Kingdom. A larger prospective study looking at the incidence and treatment of DCI is being planned in Europe under the guidance of Dr Henrik Stanstrup. The aim is to collect multi-centre data from all hyperbaric units in Europe, with the goal of providing a valid database for future research studies. This European study hopes to submit data on 1,000 incidents of DCI annually. The numbers from Australia are approximately 300-400 per year. If such data could be collated and interpreted in a uniform and consistent way, perhaps some of the uncertainties may become clearer.

Acknowledgments

I would like to thank all of those members of the Hyperbaric Units in Australia and New Zealand who completed and returned the survey forms.

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Key words

DCI, hyperbaric facilities, investigations, tables, treatment.

Data from this survey was presented at the XXIst Annual Meeting of EUBS. The abstract is published in Proceedings of the XXIst Annual Meeting of European Underwater and Biomedical Society held in Helsinki, Finland, June 28th-July 1st 1995. Editors Seppo A Sipinen and Matti Leinio. Helsinki, Finland: Finnish Society of Diving and Hyperbaric Medicine, 1995; 197.

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THE VALSALVA MANOEUVRE A CRITICAL REVIEW

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Abstract

The Valsalva manoeuvre is commonly used in diving to equalise middle ear pressures during descent. A forceful expiration with the nostrils and mouth held shut results in an increased nasopharyngeal pressure and opening of the Eustachian tubes. The correctly performed manoeuvre is easily taught, effective and usually without complications.

When performed incorrectly, prolonged periods of raised intrathoracic pressure may lead to decreased venous return, decreased arterial pressure and increased pressures within the superior and inferior vena cavae. An intact autonomic nervous system will initiate compensatory cardiovascular reflexes. The manoeuvre has clinical and research uses which rely on induced physiological changes and the initiation of reflex responses. The physiology and clinical uses of the manoeuvre are discussed.

The inappropriate use of the manoeuvre has been associated with significant morbidity. This includes pulmonary and aural barotrauma, hyper- and hypotension, cardiac arrhythmias, arterial and venous haemorrhage, gastric reflux and stress incontinence. The complications of the manoeuvre are discussed.

Introduction

The Valsalva manoeuvre was first described in 1704 by the Italian physician Anton Maria Valsalva (1666-1723) as forced expiration against a closed glottis. For divers it is the process of making a forceful attempt at expiration while holding the nostrils closed and keeping the mouth shut for the purpose of adjusting middle ear pressure.

For many divers the manoeuvre is their only means of equalising middle ear pressures upon descent. However, it is not without its dangers and the inappropriate use of the manoeuvre has been associated with significant morbidity and even mortality. The purpose of this review is to summarise the physiological changes which occur during a forceful Valsalva manoeuvre and to describe some of the documented dangers and complications.

To put this it into its proper perspective, the vast majority of manoeuvres are performed correctly and without the generation of intrathoracic or intravascular pressures likely to lead to complications.

Physiology

A standardised Valsalva manoeuvre has been described and is divided into four phases.[1] The patient is requested to exhale against a resistance of 40 mmHg for 20 seconds while heart rate and blood pressure are monitored. During Phase 1 of the manoeuvre, the increase in intrathoracic and intra-abdominal pressure will cause aortic compression and an increase in peripheral resistance resulting in a transient increase in blood pressure.

The increase in intrathoracic pressure during Phase 2 (maintenance or strain phase) hinders venous return to the heart and pressures in the superior and inferior vena cavae are increased.[1-3] The decrease in venous return leads to a decrease in ventricular end-diastolic volume, cardiac output and consequently systolic arterial pressure.[1,3-7] This fall in arterial pressure is detected by baroreceptors in the carotid artery sinuses and results in a decrease in afferent nervous discharge from the sinuses to the brain stem via Herring's and then the glossopharyngeal nerves. The glossopharyngeal nerves relay in the nucleus tractus solitarius and the decrease in their rate of discharge has an inhibitory effect upon the vagus nerve centre (parasympathetic) and an excitatory effect upon the vasomotor centre (sympathetic). This results in a reflex tachycardia and peripheral vasoconstriction after about seven seconds of strain.[1,3] The increased pressures within the vena cavae are transmitted in a retrograde fashion along