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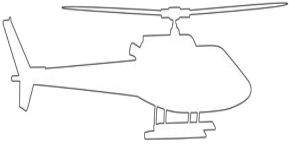
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Prehospital Care of Injured Divers:

Oxygen Delivery and Retrieval Considerations



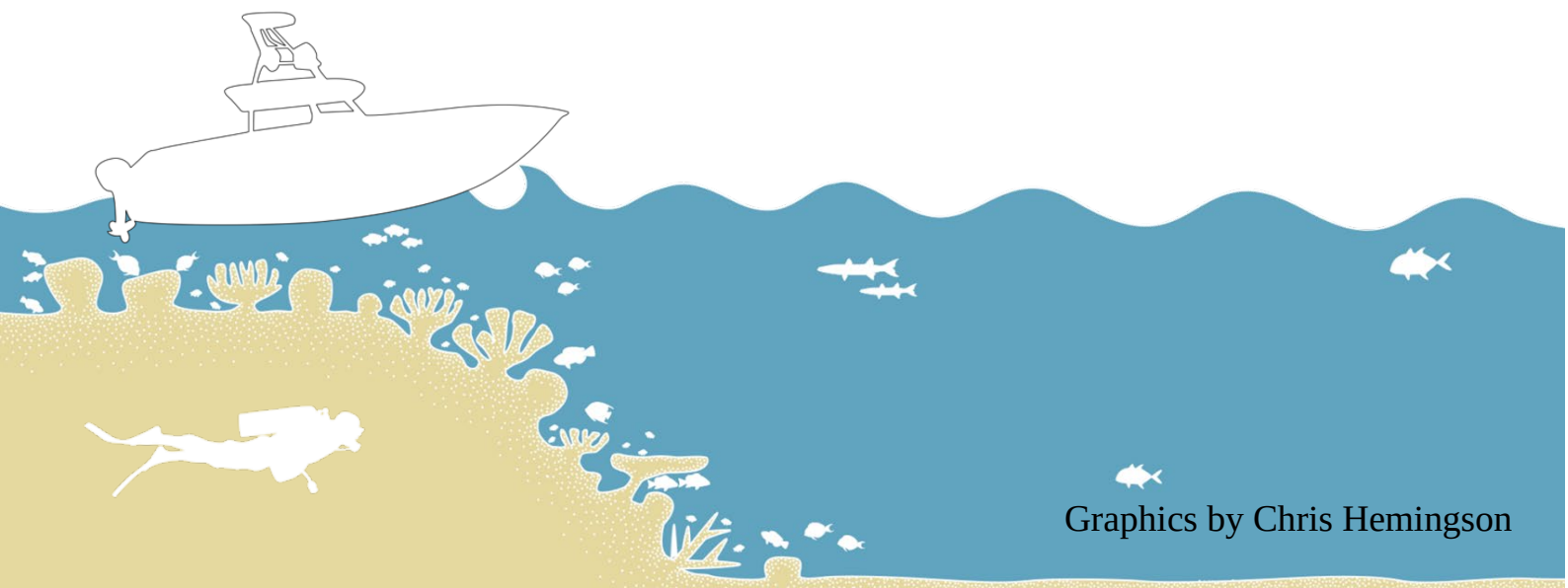
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A thesis submitted in fulfillment of the requirements
for the degree of Doctor of Philosophy

College of Medicine and Dentistry
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Graphics by Chris Hemingson

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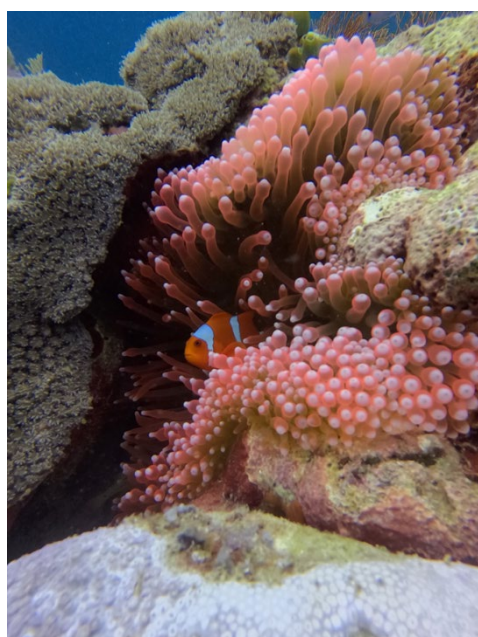
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Raja Ampat, Indonesia

Statement on the Contribution of Others

Nature of assistance	Contribution	Name and affiliation
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Transcutaneous oximetry	Conducting research	Ms Annie Brouff (TUH) was employed as a part-time research nurse using the EMF grant
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	Treatment tables	Michael Hammond (TUH)
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French translations	Reviewed the French translation of dive articles	Dr Margaux Hein (JCU) Dr Pauline Narvaez (JCU) Dr Hugo Harrison (JCU)
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The Contribution of Authors to Publications

Thesis chapter	Publication	List of authors: DB - Denise F Blake MC - Melissa Crowe DL - Daniel Lindsay SJ – Simon J Mitchell NP – Neal W Pollock RT – Richard Turk AB – Annie Brouff PL – Peter Leggat
Chapter 2	Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. Diving Hyperb Med. 2024;54(4):308-15.	DB conceived and designed the study with input from all authors. DB and RT collected the data. DB and DL analysed, and all authors interpreted the data. DB drafted the manuscript, and all authors revised and approved the final version.
Chapter 3	Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review. Diving Hyperb Med. 2025;55(2):79-90.	DB conceived and designed the study with input from all authors. DB and RT collected the data. DB and DL analysed, and all authors interpreted the data. DB drafted the manuscript, and all authors revised and approved the final version.
Chapter 5	Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Pollock NW. A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations. Diving Hyperb Med. 2018;48(4):209-217.	DB conceived and designed the study with assistance from all authors. DB and AB collected the data. DB and DL analysed, and all authors interpreted the data. DB drafted the paper, and all others revised and approved the final version.
Chapter 6	Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Leggat PA, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates. Diving Hyperb Med. 2020;50(1):34-42.	DB conceived and designed the study with assistance from all authors. DB and AB collected the data. DB and DL analysed, and all authors interpreted the data. DB drafted the paper, and all others revised and approved the final version.

Declaration

I, *Denise Faye Blake*, declare that this thesis is my own original work and has not been submitted in any form for another degree or diploma at any university or institution of tertiary education. Information derived from published and unpublished work of other scholars has been duly acknowledged in the text, and a list of references is provided.

I declare that the research included in this thesis was ethically conducted with approval received for each component of the research from the Townsville Health Service District and James Cook University Human Research Committees. The following ethics approval codes relate to the research relevant to this thesis: HREC/16/QTHS/196, H6859, LNR/2019/QTHS/51229, H7767.

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Publications Arising from Thesis

Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review. *Diving Hyperb Med.* 2025;55(2):79-90. doi: 10.28920/dhm55.2.79-90. PMID: 40544136.

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Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Pollock NW. A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations. *Diving Hyperb Med.* 2018;48(4):209-217. doi: 10.28920/dhm48.4.209-217. PMID: 30517952.

Presentations and Electronic Posters

2025 October “Retrieval pathways of divers with diving-related illnesses in North Queensland: a 15-year retrospective review”. Hyperbaric Technicians and Nurses Association, Annual Scientific Meeting, Townsville, Queensland – oral presentation.

2025 October **Invited Speaker** – Wenoll oxygen first aid rebreather system. Boating and diving, James Cook University. Marine scientists, diving officers, community oxygen instructor.

2025 May **Invited Speaker** “Retrieval pathways of divers with diving-related illnesses in North Queensland: a 15-year retrospective review”. Senior emergency department registrar education day.

2025 April “Retrieval pathways of divers with diving-related illnesses in North Queensland: a 15-year retrospective review”. Retrieval Services Queensland education day - Queensland clinical co-ordinators.

2025 March **Invited Speaker** – “Efficiency and effectiveness of different oxygen delivery devices”. Canadian Underwater Conference and Exhibition, Vancouver, Canada.

2025 March **Invited speaker** Cozumel and Playa del Carmen, Mexico “Efficiency and effectiveness of different oxygen delivery devices” – Hyperbaric staff and dive community.

2024 September European Underwater and Baromedical Society Annual Scientific Meeting, Brest, France “Oxygen treatment and retrieval pathways of divers with diving-related illnesses in North Queensland: a 15-year retrospective review” – oral presentation.

2024 June Dive medic technician course instructor, Commercial Diving Institute of Canada, Kelowna, BC. Teaching use of medical oxygen rebreather system.

2023 June Undersea and Hyperbaric Medicine Society Annual Scientific Meeting, San Diego, California, USA “Divers treated in Townsville, Australia: severe symptoms lead to poorer outcomes” – electronic poster.

2023 June **Invited Speaker** - Dive medic technician course, Commercial Diving Institute of Canada, Kelowna, BC “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates.”

2023 May **Invited Speaker** Mayo Clinic Diving and Hyperbaric Medicine conference, Cozumel, Mexico “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” - Diving physicians and dive community.

2023 February **Invited Speaker** - Dive medic technician course, Commercial Diving Institute of Canada, Kelowna, BC “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates”.

2021 October My Research Rules, The Townsville Hospital Research Symposium “North Queensland retrieval of divers: a retrospective review” – Hospital staff and researchers.

2020 October Emergency Department, The Townsville Hospital “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – Junior emergency department doctors.

2020 February Great Barrier Reef Marine Park Authority Science seminar series, Townsville “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – Marine scientists and dive safety officer.

2019 Remote Area Dive, Townsville “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – dive club members.

2019 October My Research Rules, The Townsville Hospital Research Symposium “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – **Winner early candidature student \$2,500.**

2019 September Emergency Department, The Townsville Hospital “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – Emergency department medical trainees.

2019 July James Cook University cohort doctoral conference “Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates” – Graduate students and supervisors.

2019 April Remote Area Dive, Townsville “A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve using four different mask configurations” - dive club members.

2018 October James Cook University Science with sushi “A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve using four different mask configurations” - Marine scientists, graduate students, oxygen instructor, boating and diving staff.

2018 July James Cook University cohort doctoral conference “A comparison of the tissue oxygenation achieved using four different oxygen masks” – Graduates students and supervisors.

2018 June Medical Grand Rounds, The Townsville Hospital “A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations” – Medical and nursing staff.

2018 February **Invited speaker** Cozumel and Playa del Carmen, Mexico “A comparison of the tissue oxygenation achieved using different delivery devices and flow rates” - Diving physicians and dive community.

Related Presentations and Publications during Candidature

Blake DF, Crowe M, Mitchell SJ, Aitken P, Pollock NW. Vibration and bubbles: a systematic review of the effects of helicopter retrieval on injured divers. *Diving Hyperb Med.* 2018;48(4):235-40. doi: 10.28920/dhm48.4.241-251. PMID: 30517957.

2018 July James Cook University Cohort Doctoral conference “Helicopter vibration and simulator development” electronic poster. **Awarded best poster \$500.**

2017 June Undersea and Hyperbaric Medicine Society Annual Scientific Meeting, Naples, Florida, USA “Helicopter vibration and simulator development” – electronic poster.

Abstract

Introduction: Arterial gas embolism (AGE) and decompression sickness (DCS), collectively termed decompression illness (DCI), are risks for scuba divers. AGE results from pulmonary barotrauma introducing gas directly into the vascular system. DCS is caused by the formation of bubbles primarily from dissolved inert gas. Arresting bubble growth and encouraging their involution is the primary treatment for DCI. Breathing a high concentration of oxygen creates a diffusion gradient, leading to the transport of inert gas to the lungs for exhalation. Current prehospital care recommendations for divers with signs and symptoms of DCI is for oxygen delivery at the highest concentration possible while awaiting transport to definitive care. Dive site remoteness and limited access to facilities may delay specialist assessment and/or hyperbaric oxygen treatment (HBOT). Townsville has the only hyperbaric facility along the Great Barrier Reef, treating divers from the reef and neighbouring islands. The aim of this thesis was to examine the prehospital care of injured divers in north Queensland and determine the optimum oxygen delivery system. This thesis contains two sections: (1) a retrospective review analysing oxygen therapy and retrieval pathways of injured divers presenting to the Townsville Hyperbaric Medicine Unit (THMU) and (2) a comparison of oxygen delivery devices and flow rates (2a and 2b) using a novel approach to measure oxygenation and assessing the efficacy of the optimal oxygen delivery device (2c) in reducing the number of venous gas emboli (VGE) after a dry hyperbaric chamber dive.

Methods: (1) A 15-year retrospective review of diver presentations to the THMU was conducted. Charts were reviewed collecting data on time of symptom onset, initial disease severity, time to oxygen commencement post-symptom onset, time to HBOT and clinical outcome. Non-parametric tests were used to compare initial disease grade with time to event data (symptom onset, oxygen commencement, HBOT). Retrieval details were collected including platform, retrieval origin and destination and retrieval type (primary, secondary, tertiary). Retrieval destination appropriateness for divers with severe initial disease grade and primary retrievals was examined. Retrieval pathways of divers with three retrieval legs were evaluated for appropriateness. Initial disease severity, diagnosis, need for HBOT, and distance from Townsville were assessed for appropriateness of primary destination and staging.

(2a) Sixteen participants took part in four interventions breathing oxygen from a demand valve using an intraoral mask with and without a nose clip, a pocket face mask and an oronasal mask. (2b) Twelve participants breathed oxygen from five interventions: the optimized demand valve (from 2a), a non-rebreather (NRB) mask at 10 and 15 L·min⁻¹ and an oxygen rebreather system with an oronasal mask and intraoral mask. Nasopharyngeal inspired oxygen and transcutaneous oximetry were measured in parts 2a and 2b. Non-parametric tests were used to compare nasopharyngeal and tissue oxygen levels.

Participants rated mask comfort, ease of breathing and overall use of each device. (2c) In a randomized, double-blind cross over study, 12 participants performed two no-decompression dives in a dry hyperbaric chamber riding a cycle ergometer, breathing air or oxygen at $15 \text{ L} \cdot \text{min}^{-1}$ commencing 30 min after dive completion. Transthoracic echocardiography (TTE) commenced after the participants exited the chamber and continued every 15 min for 120 min. VGE were graded by two blinded reviewers using a TTE expanded grading scale. Time to onset of non-zero VGE grades, time to maximum VGE grade and VGE resolution were determined.

Results: (1) 306 injured divers were included with retrieval pathways as long as 3,200 km. Median time to symptom onset was 60 (IQR 10, 360) min with most divers having mild symptoms (71%). Median time to oxygen therapy was 4:00 (IQR 00:30, 24:27) h:min with the NRB mask most frequently used (44%, 28/63). Median time to start of HBOT was 38:51 (22:11, 69:15) h:min, with the most common reason for delay being delayed presentation (35%, 100/283) followed by extreme (500 to > 1,700 km) retrieval distance (32%, 91/283). Divers with a higher initial disease severity had significantly shorter times to symptoms onset, oxygen commencement and HBOT. A high proportion of injured divers received oxygen treatment (87%, 267/305) while awaiting transport. Of the severe cases, one with immersion pulmonary oedema did not require further transfer to Townsville, and one with a final diagnosis of cerebral AGE should have primarily gone to Townsville. There were 15 cases with three retrieval legs, four international retrievals. Of the 11 Australian cases, six were identified as inappropriately staged during the retrieval process (three with cerebral AGE, one spinal DCS and two doing decompression diving with moderate DCS symptoms). Most divers had good clinical outcomes, and no treated diver died. Higher initial disease severity accelerated the speed of care and was the only factor associated with poorer outcome.

(2) Similar inspired and transcutaneous oxygen values were obtained with all three oxygen delivery systems: demand valve, NRB mask and oxygen rebreather system. (2a) The demand valve provided the highest oxygenation when used with an intraoral mask. (2b) The NRB mask performed best with the higher oxygen flow rate of $15 \text{ L} \cdot \text{min}^{-1}$. Oxygenation was significantly lower with the NRB at $10 \text{ L} \cdot \text{min}^{-1}$. The oxygen rebreather system with either an oronasal mask or intraoral mask is a good choice when oxygen supplies are limited. The NRB mask was rated the most comfortable to wear, easiest to breathe with, and easiest to use. (2c) Maximum VGE grades were higher during the air breathing session for all time periods from 60 to 120 min post dive. VGE were evident at 120 min with air but not with oxygen.

Conclusions: Most injured divers received oxygen first aid and were transported to Townsville for definitive care with a variable number of stages. Many different retrieval assets and legs were

necessary when dive sites were remote and distances vast. These findings have provided valuable insight into the retrieval pathways of divers transported to Townsville and identified areas of education for retrieval physicians. All three available oxygen delivery systems promoted for use with injured divers can provide high oxygenation levels when used with the appropriate mask and oxygen flow rate: demand valve with an intraoral mask, NRB at an oxygen flow rate of $15\text{ L}\cdot\text{min}^{-1}$, and oxygen rebreather system with either an air cushion or intraoral mask. These results have provided an understanding of the varied systems available and will provide valuable information for divers and medical personnel to assist with device selection in diving emergencies.

Keywords: Decompression illness, Echocardiography, Hyperbaric oxygen treatment, Oxygen therapy, Retrieval platform, Scuba diving, Transcutaneous oximetry, Venous gas emboli (VGE)

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List of Abbreviations

AGE	Arterial gas emboli
AIMS	Australian Institute of Marine Science
ATA	Atmospheres absolute
BCD	Buoyancy compensator device
BMI	Body mass index
CAGE	Cerebral arterial gas emboli
CCR	Closed circuit rebreather
CEBM	Oxford Centre for Evidence-Based Medicine
CNS	Central nervous system
COVID	Corona virus disease
CPAP	Continuous positive airway pressure
DAN	Divers Alert Network
DCI	Decompression illness
DCIEM	Defence and Civil Institute of Environmental Medicine
DCS	Decompression sickness
DMT	Dive medical technician
EMF	Emergency Medicine Foundation
ETCO ₂	End-tidal carbon dioxide
FAO ₂	First aid oxygen
FIO ₂	Fraction of inspired oxygen
GBR	Great barrier reef
GCS	Glasgow coma scale
GRADE	Grading of recommendations, assessment, development and evaluation
HBOT	Hyperbaric oxygen treatment
HMU	Hyperbaric medicine unit

IMCA	International Marine Contractors Association
IOM	Intraoral mask
IV	intravenous
IWR	In-water recompression
KISS	Kisman integrated severity scale
kp	kilopond
kPa	kilopascals
LA	Left arm
LOE	Level of evidence
msw	Metres of sea water
MORS	Medical oxygen rebreathing system
N ₂	Nitrogen
NBOT	Normobaric oxygen therapy
NC	Nose clip
NRB	Non-rebreather mask
O ₂	Oxygen
PAP	Positive airway pressure
PEEP	Positive end-expiratory pressure
PFO	Patent foramen ovale
PhD	Doctor of Philosophy
PRISMA	Preferred reporting items for systematic reviews and meta-analysis
P _{tc} O ₂	Tissue oxygen partial pressure
QAS	Queensland ambulance service
QATSDD	Quality assessment tool for studies of diverse designs
RA	Right arm
RN	Royal Navy

RPE	Rating of perceived exertion
rpm	Revolutions per minute
RQ	Research question
SANDHOG	San Diego Diving and Hyperbaric Organizations
SPUMS	South Pacific Underwater Medicine Society
TCOM	Transcutaneous oximetry measurement
TTE	Transthoracic echocardiography
VO _{2max}	Maximal oxygen consumption
VGE	Venous gas emboli
W	Watts

The Researcher's perspective

I am a Canadian trained emergency physician who moved to Townsville, Australia 20 years ago to pursue a career in diving medicine. I am blessed to be able to combine my love for the ocean and diving with a career in medicine.

My love for the underwater world started when, as a child, I first snorkelled in Tobago, West Indies. I became an open water diver at the end of high school and a scuba instructor during university. My teaching focused on the rescue course as my previous work as a lifeguard and paramedic allowed me to provide extra training to my students. I have been a scuba diver for over 40 years.

I completed a bachelor's degree in nursing and a post graduate certificate in intensive care nursing. I completed my first DAN introductory dive medicine course in Bonaire, Netherland Antilles during nursing school and was hooked. I started medical school at the University of Manitoba and during a summer break completed a 2-week course in diving and hyperbaric medicine course in San Antonio, Texas. During medical school and my emergency medicine residency I travelled to Townsville, Australia to do electives in dive medicine. I moved to Townsville at the completion of my emergency medicine training to pursue a career in diving medicine. I started working at the hyperbaric medicine unit and completed a post graduate diploma in diving and hyperbaric medicine from the University of Auckland. This started my love for research.

My university appointment at James Cook University (JCU) is in Marine Biology where I taught diving physiology. I started performing annual dive medicals for JCU students, forming relationships that have continued throughout the years. I have published many peer-reviewed research papers and supervised other doctors doing their SPUMS Diploma of Diving and Hyperbaric Medicine. My other roles include medical advisor for a commercial dive company in Townsville (Pacific Marine Group), SPUMS representative on the Australian Standards 2299 committee for scientific diving, dive medical technician instructor (IMCA) and teaching at a commercial dive school in Kelowna, BC, Canada. I have a dive medicine clinic where I perform recreational and occupational dive medicals and review divers with dive related problems.

I am an emergency physician in Townsville and have worked as a clinical coordinator, assessing the need for aeromedical transfer for unwell patients in North Queensland. This background has led me to pursue a PhD with a focus on prehospital care for injured divers in North Queensland. I have identified areas for further education for retrieval and emergency physicians and defined oxygen mask selection and flow rate to improve treatment of injured divers. I hope you enjoy reading my thesis.

CHAPTER 1 – INTRODUCTION

1 Introduction

Scuba diving allows air-breathing humans to enter and experience the wonderment of the underwater world. The Great Barrier Reef (GBR) is the world's most extensive and best-known coral reef ecosystem and a popular dive site. The GBR attracts both domestic and international tourists, many of whom participate in a variety of water-related activities, including scuba diving and snorkelling. Although scuba diving is a relatively safe sport, 483 diving-related fatalities were reported in Australia from 1970 to 2018, 116 in Queensland.¹ Even though death is relatively rare, many divers are injured each year requiring treatment in a hyperbaric facility. In 2018, 112 divers were treated for decompression illness (DCI) in Australia, 34 in Queensland (Hyperbaric Technicians and Nurses Association, unpublished data).

1.1 Defining Decompression Illness

DCI is a term encompassing both arterial gas embolism (AGE) and decompression sickness (DCS). Arterial gas embolism results from pulmonary barotrauma or arterialisation of bubbles passing through a right to left shunt (e.g., a patent foramen ovale [PFO]). DCS, on the other hand, is caused by the formation of bubbles primarily from dissolved inert gas.² DCS was originally called caissons disease due to its occurrence during mining and the building of bridges in the 1800s and the 'bends' due to the hunched over position of those afflicted, prior to it being identified during in-water pressure exposures. There is potential difficulty in clinically distinguishing between AGE and DCS, however, the modern trend in therapy is to treat both in the same manner.² These considerations provided the motivation for referring to them collectively as DCI.³

1.2 Pathophysiology of Decompression Sickness

Human beings are not 'fit' to dive underwater. Divers require a mask to provide an air space in front of their eyes to see, fins for propulsion, an exposure suit to protect from marine life and maintain body temperature, a buoyancy compensator device (BCD) to adjust buoyancy at depth and the surface, a weight belt to assist with commencing descent, and a dive computer to monitor depth and record dive bottom time. The most important item required by a diver is a gas supply for the diver to breathe at depth. Immersion in water comes at a price with DCS a risk for divers. During a dive, an increase in the ambient pressure resulting from the weight of the surrounding water leads to a higher breathing gas partial pressure being delivered to the lungs, and more inert gas (nitrogen in air diving) dissolving in the blood and body tissues. The deeper the dive the higher the partial pressure of nitrogen in the inhaled gas and the longer the dive, the more time for the nitrogen to diffuse into the tissues of the

body. This is called on-gassing. The problem is not the dive itself, but at some point, the diver must ascend back to the surface. As the diver ascends, both the ambient pressure and the partial pressure of the inspired inert gas decrease. The on-gassing process is reversed as tissue inert gas diffuses into the blood for carriage back to the lungs and exhalation. However, if the pressure of dissolved gas in tissue significantly exceeds the ambient pressure (a condition referred to as supersaturation) the gas may form bubbles in body tissues, lymphatics or within blood vessels. When bubbles are formed within the vascular system, they are called venous gas emboli (VGE). These bubbles are considered to be the primary vector of injury leading to the symptoms of DCS.² The exact mechanism through which these bubbles produce the symptoms of DCS is unknown but thought to be related to mechanical damage, ischemia and inflammatory responses.²

1.3 Venous Gas Emboli

Inert gas bubbles do not always lead to symptoms of DCS as the pulmonary capillaries in the lungs act as filters for these bubbles. Bubbles can be detected using acoustic Doppler and more recently trans thoracically using portable ultrasound machines to visualize the four chambers of the heart. Venous gas emboli (VGE) loads are graded using a semi-quantitative ordinal scale with grade 0 being no bubbles and grade V a whiteout, where individual bubbles cannot be discerned.⁴ Bubble formation is generally accepted as an indicator of decompression stress and DCS risk in research where the generation of clinical DCS would be an unacceptable endpoint.⁵ There is some evidence that there is a relationship with the VGE grade and incidence of DCS⁶ although the variation among and within individuals is large.⁷

1.4 Clinical Diagnosis of Decompression Sickness

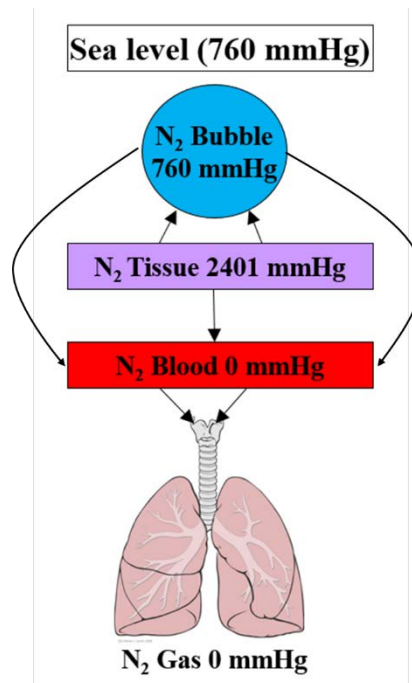
DCS symptoms can range from mild joint aches to severe neurological symptoms such as leg paralysis with urinary retention. There are no tests to diagnose DCS. The history and physical exam of the injured diver lead to the clinical diagnosis. The history should include information on the dive profiles, breathing gas, risk factors and dive computer use. Characterization of the symptoms and time of onset (before, during or after the dive) can assist with the likelihood of a diagnosis of DCS. The physical exam should be guided by the symptoms and include a detailed neurologic and otic exam.

1.5 First Aid Oxygen Treatment of Decompression Sickness

Early treatments for DCS included alcohol massages and ingestion, elevation of the legs and wearing of bimetallic or galvanic bands.⁸ Repressurization in the caissons was found to alleviate symptoms

with the use of normobaric oxygen suggested for treatment in the late 1800s⁹ with some uptake of its use documented in the 1960s.¹⁰ However, it was only in 1991 that the Divers Alert Network (DAN) created an oxygen first aid training program focusing on the delivery of oxygen in the event of a diving emergency.¹¹ Normobaric oxygen therapy is thought to assist with the removal of inert gas located in the body tissues and bubbles. Breathing a high concentration of oxygen significantly reduces nitrogen in the breathing gas, creating an increased gradient such that nitrogen diffuses out of the tissues and VGE into the blood then to the lungs to be exhaled (Figure 1.1).² In addition to helping to displace dissolved inert gas and gas in bubbles, breathing a high concentration of oxygen also supplies oxygen to hypoxic tissues¹² and reduces inflammation.^{13,14}

Figure 1.1 Diffusion gradient for nitrogen off-gassing while breathing near 100% oxygen after a dive



Portable oxygen delivery units are available to provide injured divers with immediate dive site first aid oxygen. The regulator designed specifically for injured diver use has two components: a demand valve and a variable litre flow device. The demand valve is similar to a scuba regulator and delivers oxygen only when the diver inspires, allowing for conservation of finite oxygen supplies in remote locations. The constant flow device can be used with a non-rebreather (NRB) mask or other constant flow delivery device. The current recommendation is that oxygen be delivered to divers with DCS symptoms and signs at the highest concentration possible to provide near 100% normobaric oxygen.² The oxygen flow rate used with a NRB mask has historically been 15 L·min⁻¹ to provide a high concentration of oxygen. The guidelines have changed suggesting the reduction of the NRB flow rate to 10 to 15 L·min⁻¹ allowing the oxygen supply to last longer.¹⁵ However, it has been shown that

decreasing the flow rate from 15 to 10 L·min⁻¹ significantly decreased peripheral tissue oxygenation.¹⁶ The same study also showed an unexpected lower peripheral tissue oxygenation when participants were breathing oxygen using the demand valve with a Tru-Fit silicone oronasal mask compared to a NRB mask at 15 L·min⁻¹. Discussion has surrounded these findings as it is believed that the demand valve should be able to administer near 100% oxygen.² The optimal mask configuration for use with the demand valve and the ideal oxygen flow rate for use with a NRB mask to provide high levels of oxygenation is still unknown.

Normobaric oxygen therapy is recommended to be started early in divers with signs and symptoms of DCS.² Many dive sites may be remote and therefore the transport to medical care delayed. Oxygen should be available at dive sites for treatment of symptomatic divers while arrangement for retrieval and transport to definitive care is in progress. Many dive sites on the GBR may be 50 to 100 km offshore and over 500 km away from a hyperbaric facility. Therefore, delivery devices that can provide oxygen therapy for extended periods should be investigated.

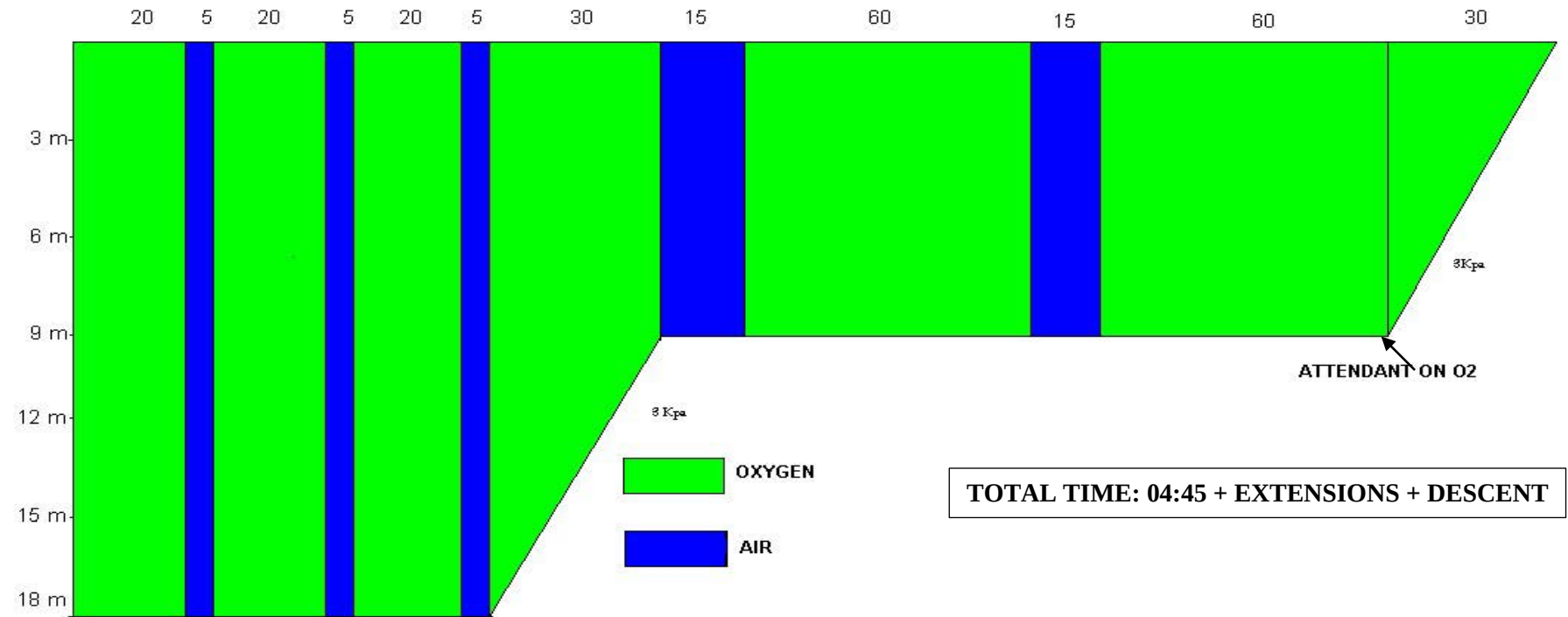
Pulmonary oxygen toxicity is a risk for divers when receiving normobaric oxygen for longer than 12 hours.¹⁷ Symptoms are cough and pleuritic chest pain. Pulmonary oxygen toxicity is a concern from a patient comfort perspective, reversible in its early stages and extremely unlikely to cause harm in this group of patients. If symptoms of pulmonary oxygen toxicity occur, discussing the benefits and risks of ongoing oxygen therapy with an experienced diving physician and the diver is warranted. Little is known about the incidence and outcomes of pulmonary oxygen toxicity in injured divers treated with normobaric oxygen.

1.6 Hyperbaric Oxygen Treatment

Recompression breathing oxygen is currently considered the definitive treatment for DCS.² Hyperbaric oxygen treatment (HBOT) involves returning the diver to a pressured environment breathing 100% oxygen. HBOT provides a high ambient pressure reducing bubble volume, enhancing diffusion of inert gas out of the bubbles, and providing oxygenation to hypoxic tissues.² HBOT most commonly occurs in hyperbaric chambers, but in rare cases, particularly in commercial dive operations, it may be provided by in-water recompression (IWR). Commonly used hyperbaric chamber treatment tables for HBOT include the Royal Navy (RN) 62 and the Comex 30 (Figures 1.2 and 1.3 respectively). The RN62 table involves compression to 284 kPa (2.8 ATA, 18 msw) with three oxygen breathing sessions separated by air breaks. The diver is then assessed and if symptoms are improving or resolved the chamber is then decompressed to 192 kPa (1.9 ATA, 9 msw) to

complete a further 2 oxygen breathing periods. The treatment time is 4 h and 45 min. The table can be extended at 284 kPa for two further oxygen breathing sessions if there is inadequate resolution of symptoms. The Comex 30 treatment table compresses the diver to 405 kPa (4 ATA, 30 msw) initially breathing heliox (50% helium/50% oxygen) until decompression to 284 kPa when the diver is switched to oxygen. The treatment time is 7 h and 30 min. This table has historically been used for severe DCS and cerebral arterial gas emboli (CAGE). If residual symptoms remain, follow-up recompression is usually once daily using a shorter treatment table until complete resolution of symptoms or no sustained improvement is seen over two consecutive days.² Although HBOT is considered the definitive treatment for DCS there is consensus among experts that some transfers can be avoided and divers with mild symptoms can be treated adequately on-site with high concentration normobaric oxygen.^{18,19}

Figure 1.2 Royal Navy (RN) 62 Treatment Table



EXTENDING TABLE 62

If relief from signs and symptoms is incomplete after 75 min on Table 62, a further one or two periods of oxygen breathing may be introduced at 18m. These periods would be of 20-min duration interspersed with 5-min air breathing. Decompression from 18m – 9m should be preceded by 5 min air breathing. Similarly, another 60min oxygen breathing period may be introduced at 9m following an intervening period of 15-min air breathing. Final decompression to the surface will still be on oxygen. Should any of these extra oxygen-breathing periods be employed, the attendant will breathe oxygen during the final 60 min at 9m and during the final decompression to the surface.

Depth	Duration	Breathing Mix	Elapsed Time
18 m	75 min	Oxygen: 3 BIBS sessions 20 min ON + 5 min OFF	01:15
18 – 9 m	30 min	Oxygen: 1 BIBS session 30 min ON	01:45
9 m	150 min	Oxygen: 2 BIBS sessions 15 min OFF + 60 min ON	04:15
9 – 0 m	30 min	Oxygen: 1 BIBS session 30 min ON	04:45

TIME: 07:30 + DESCENT

Depth	Duration	Breathing Mix	
30 m	60 min	Heliox 50/50 : 1 BIBS session 60 min ON	Air
30–24 m	30 min	Heliox 50/50 : 1 BIBS session 25 min ON + 5 min OFF	Air
24 m	30 min	Heliox 50/50 : 1 BIBS session 25 min ON + 5 min OFF	Air
24–18 m	30 min	Heliox 50/50 : 1 BIBS session 25 min ON + 5 min OFF	Air
18 m	60 min	Oxygen : 2 BIBS sessions 25 min ON + 5 min OFF	Air

TIME: 07:30 + DESCENT

1.7 In-Water Recompression

IWR breathing oxygen is an established, albeit not universally accepted, strategy for treating DCI,²⁰ especially at remote dive sites when access to a recompression chamber is not readily available.²¹ This procedure is not without risks and requires special equipment and training, with a clear protocol in place prior to use.¹⁹ A major risk of IWR is central nervous system oxygen toxicity leading to a seizure and subsequent drowning.²¹ IWR may be performed on commercial dive sites where surface supplied oxygen and diver support systems are available. With an increase in technical diving, this procedure may become more commonly used.²²

1.8 Retrieval of Injured Divers

Access to recompression may be delayed due to the remoteness of the dive site and limited access to hyperbaric facilities. First aid treatment for divers includes surface oxygen while preparing for transport to a recompression chamber. Oxygen may reduce or resolve symptoms, and hasten recovery, and therefore should be initiated at the dive site, especially in remote locations where long delays to recompression may be required.¹⁸

The GBR is a chain of 2900 reefs, 600 islands and 300 coral reefs, extending 2,300 km from the northern tip of Queensland, Australia to Bundaberg with over 2 million visits made to the reef each year.²³ The vast majority of dive sites are concentrated in the Cairns and Whitsunday (Mackay) areas. Diving excursions on the GBR include day trips on large vessels, and live aboard holidays ranging from two to seven days duration. Midway along the coastline parallel to the GBR is located the city of Townsville. The Townsville University Hospital houses the only hyperbaric facility along the GBR, providing specialist advice and HBOT for injured divers. The next nearest hyperbaric facility is located 1,400 km south of Townsville in Brisbane.

Townsville is a centre of excellence in marine science. The Australian Institute of Marine Science (AIMS) is located 50 min drive time south of Townsville and James Cook University, located in the city, is well known for its Marine Biology program. In 1968, the federal government proposed the plan to open an AIMS facility and Townsville became the chosen site.²⁴ Since AIMS was to house the largest group of professional divers in north Queensland, a twin lock Comex deck recompression chamber was installed at AIMS and in 1977 commenced clinical use.²⁵ With stringent safe diving practices, few scientific divers required the chamber, and it was increasingly used by injured recreational divers. Logistical strains on staff (both AIMS and Townsville medical staff) and extensive

time travelling to AIMS led to the relocation of the chamber to the Townsville General Hospital in 1989 (personal communication J Hardman, March 2018).

The Townsville Hospital relocated to its current site in October 2001, with the installation of a new multi-place rectangular hyperbaric chamber (Fink Engineering Pty Inc., Warana, Queensland, Australia). Data from the first two years of the new hyperbaric chamber service (2001-2003) were presented at a diving medicine workshop.²⁶ The focus of this publication was the road transfer of divers with mild DCS showing no poorer outcomes than those divers who were retrieved by an aeromedical asset.²⁶ There has been no data since this earlier publication on divers treated in Townsville and no published data on the retrieval pathways taken to transport divers to Townsville or reasons for delays.

A centralised Queensland retrieval service assists with the transport of injured divers to definitive treatment, with the northern zone co-ordination centre located in Townsville. Rotary and fixed wing assets are located along the Queensland coast. Rotary wing assets are located in Thursday Island, Cairns, Townsville and Mackay. Fixed wing assets are located in Cairns and Townsville, including a jet in Townsville for international and long-haul retrievals (Figure 1.4). Referrals to the coordination centre may come directly from dive boats (skippers), the Queensland Ambulance Service (supervisors), health care facilities (nurses or doctors) or diving physicians (emergency hotlines or hyperbaric facilities). It is the job of the retrieval service clinical coordinator to provide medical advice and determine the urgency, retrieval platform, and appropriate destination for injured divers.

Figure 1.4 Coastal fixed and rotary wing bases in North Queensland; number of symbols represents number of assets



Townsville is centrally located on the Queensland coast and is the location of the only hyperbaric facility along the GBR and home to the northern zone retrieval service. Ongoing analysis of the incidence, care and outcome of injured divers referred to Townsville University Hospital Hyperbaric Medicine Unit (HMU) is essential for improving healthcare quality and identifying areas for improvement.

1.9 Aims, Research Questions and Objectives

The aims of this thesis were to examine the current prehospital care of injured divers in North Queensland and determine the optimum first aid oxygen delivery system. This thesis consists of two sections: (1) a retrospective review analysing oxygen therapy and retrieval pathways of injured divers presenting to the Townsville HMU and (2) a comparison of oxygen delivery devices and flow rates (Parts 2a and 2b) using a novel approach to measure oxygenation and then (Part 2c) the assessment of the efficacy of the optimal oxygen delivery device in reducing the number of venous gas emboli (VGE) after a simulated dive in a hyperbaric chamber measured using transthoracic echocardiography (TTE).

Mapping of the retrieval pathways will provide insight into the appropriateness of destination and staging decisions. Analysis of pre-hospital care and retrieval pathways of these injured divers could assist with establishing education strategies for retrieval physicians and guide resource allocation. Determining optimal oxygen delivery options will provide information to assist with appropriate device selection by dive site coordinators and providers of prehospital care.

Research Questions (RQ)

RQ 1. How many injured divers were treated at the Townsville University Hospital in the last 15 years and what were the demographics, medical and dive history, symptoms and clinical outcomes of these divers?

Objective 1: Outline the demographics, diving history, qualifications, medical and surgical history of the injured divers.

Objective 2: Describe the incident dive, place of incident, nature of dive, dive team, breathing gas and circuit use, and possible contributing factors.

Objective 3: Determine time of symptom onset, symptom profile and initial disease grade.

Objective 4: Determine time to HBOT and explore reasons for delay.

Objective 5: Determine clinical outcome and factors contributing to outcome.

RQ 2. What was the retrieval method and pathway used to transfer the injured divers to Townsville?

Objective 1: Determine retrieval platform, type of retrieval and pathway.

Objective 2: Determine initial disease severity, primary retrieval destination, and destination appropriateness.

Objective 3: Analyse cases requiring three retrieval legs for appropriateness of staging.

RQ 3. Did the injured diver receive normobaric oxygen prior to HBOT and if so, what oxygen delivery device was used?

Objective 1: Collect treatment details including time to commencement of pre-HBOT oxygen therapy, type of oxygen delivery device and change of symptoms with oxygen.

Objective 2: Collect data on oxygen therapy duration and any symptoms of oxygen toxicity.

RQ 4. What is the published literature surrounding VGE reduction or clinical outcome in injured scuba divers or animal models after treatment with normobaric oxygen prior to hyperbaric oxygen therapy?

RQ 5. In healthy divers, when delivering oxygen with a demand valve which oxygen mask configuration provides the best peripheral tissue oxygen partial pressure determined using transcutaneous oximetry?

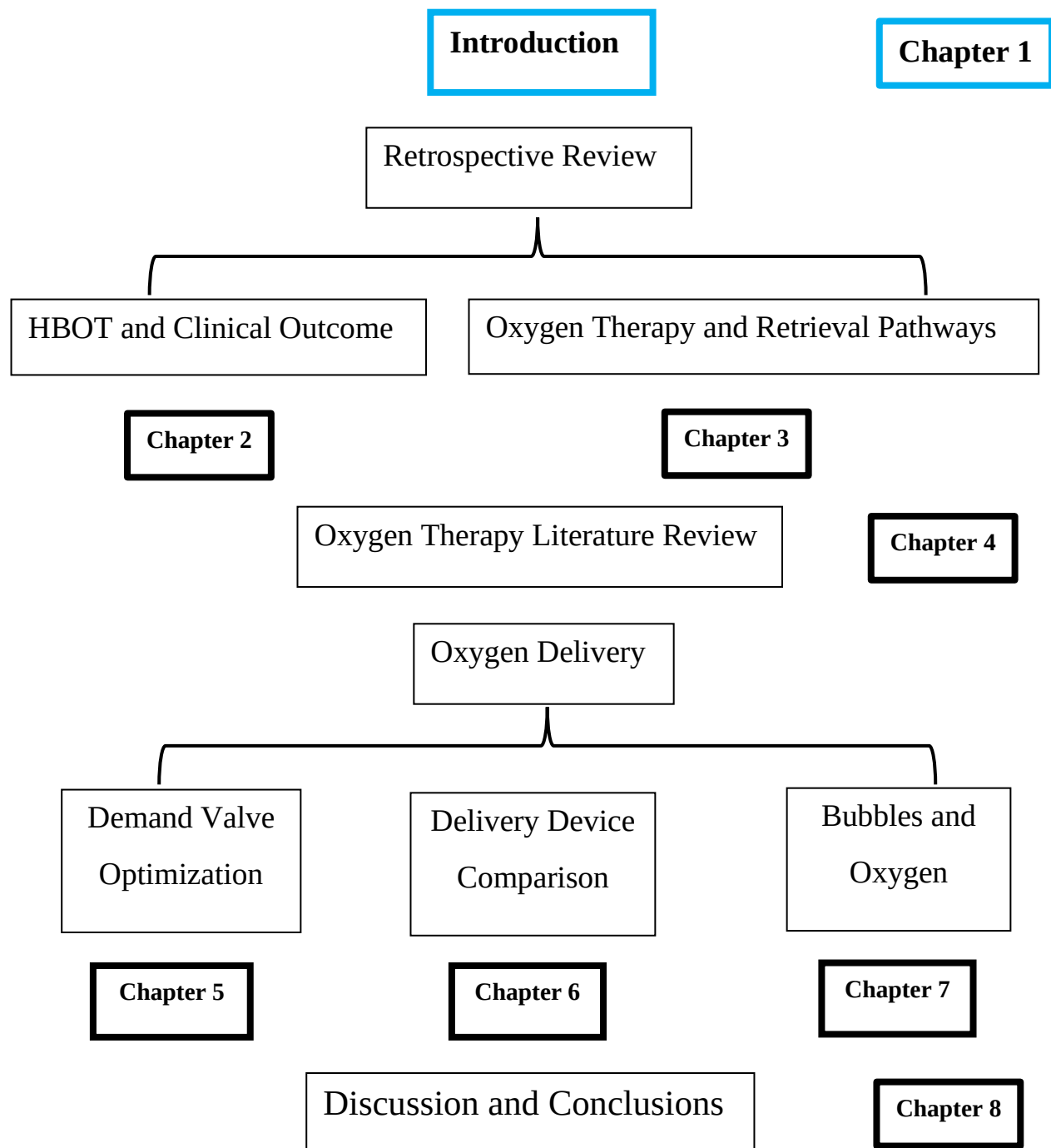
RQ 6. In healthy divers, when delivering oxygen with commonly used devices and flow rates is there a difference in peripheral tissue oxygen partial pressure determined using transcutaneous oximetry?

RQ 7. In healthy divers, does administration of normobaric oxygen after a simulated dive change venous gas emboli grade determined by transthoracic echocardiography compared to breathing air?

1.10 Thesis format

This thesis is presented as a series of published papers and manuscripts submitted or in preparation for publication preceded by an introductory chapter and concluded with a discussion chapter. All papers have been published in the same journal and therefore the style of that journal will be used throughout the thesis. A short introduction will precede each paper. Reference lists will be included at the end of each chapter. The thesis contains two main sections, one a retrospective review of diving injuries referred to Townsville over a 15-year period. Two retrospective studies are presented in this section (Chapters 2 and 3). These chapters set the scene for the thesis by presenting the type of injured divers treated in Townsville, current prehospital care of injured divers and the pathways and duration of

retrievals to Townsville for specialist diving medicine assessment and care. The second section is focused on normobaric oxygen delivery for treatment of injured divers. This section includes a scoping literature review of normobaric oxygen therapy (Chapter 4) and three data studies investigating the efficacy of oxygen delivery devices (Chapters 5-7). Chapters 5 and 6 will evaluate the best device and oxygen flow rate for effective oxygen delivery and the final data chapter (Chapter 7) will present preliminary evaluation of VGE grades breathing oxygen with the optimal delivery device compared to air following a simulated scuba dive. Evaluating commonly available oxygen delivery devices will provide data to assist with appropriate choice of delivery device to those caring for injured divers. The following flow chart will guide the reader through the thesis.



References

- 1 Lippmann J. A review of snorkelling and scuba diving fatalities in Queensland, Australia, 2000 to 2019. *Diving Hyperb Med.* 2022;52(2):108–18. doi: 10.28920/dhm52.2.108-118. PMID: 35732283.
- 2 Mitchell SJ. Decompression illness: a comprehensive review. *Diving Hyperb Med.* 2024;54(1 Supp):1-53. doi: 10.28920/dhm53.4.supp.1-53. PMID: 38537300.
- 3 Francis TJR. The classification of dysbarism. In: Francis TJR, Smith DJ. Eds. *Describing decompression illness: Proceedings of the 42nd workshop of the Undersea and Hyperbaric Medical Society*. Bethesda, MD: Undersea and Hyperbaric Medical Society. 1991.
- 4 Møllerløkken A, Blogg SL, Doolette DJ, Nishi RY, Pollock NW. Consensus guidelines for the use of ultrasound for diving research. *Diving Hyperb Med.* 2016;46(1):26-32. PMID: 27044459. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/46March/Mollerlokken_dhm.46.1.26-32.pdf
- 5 Pollock NW. Use of ultrasound in decompression research. *Diving Hyperb Med.* 2007;37:68–72. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/37June/Pollock_dhm.37.2.68-72.pdf
- 6 Eftedal OS, Lydersen S, Brubakk AO. The relationship between venous gas bubbles and adverse effects of decompression after air dives. *Undersea Hyperb Med.* 2007;34(2):99-105.
- 7 Doolette DJ, Murphy FG. Within-diver variability in venous gas emboli (VGE) following repeated dives. *Diving Hyperb Med.* 2023;53(4):333-339. doi: 10.28920/dhm53.4.333-339. PMID: 38091593 PMCID: PMC10944664.
- 8 Acott C. A brief history of diving and decompression illness. *SPUMS J.* 1999;29(2):98-109. [cited 29 June 2025]. Available from:
https://www.dhmjournal.com/images/IndividArticles/29June/Acott_SPUMSJ.29.2.98-109.pdf
- 9 Bert P. *Barometric pressure [La pression barometrique]*. Translated by M A Hitchcock and F A Hitchcock College Book Company, Colombus, Ohio, 1943.
- 10 Edmonds C. Underwater oxygen treatment of decompression sickness a review, *SPUMS J.* 1995;25(3):122-8. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/25Sept/Edmonds_SPUMSJ.25.3.122-128.pdf
- 11 Gunderson J. Appendix B. Training and education; pp. 81-4. In: Tillmans F, editor. *DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents*. Durham, NC: Divers Alert Network; 2020. [cited 2025 June 3]. Available from:
<https://www.ncbi.nlm.nih.gov/books/NBK582510/>

- 12 Lippmann J. First aid oxygen administration for divers. SPUMS J. 2003;33(4):192-8. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/33Dec/Lippmann_dhm.33.4.192-198.pdf.
- 13 Wang Q, Zhang X, Suo Y, et al. Normobaric hyperoxia therapy in acute ischemic stroke: A literature review. Heliyon. 2024;10(1):e23744. Epub 20231220. doi:10.1016/j.heliyon.2023.e23744. PMID: 38223732; PMCID: PMC10787244.
- 14 Li C, Jiang M, Chen Z, et al. The neuroprotective effects of normobaric oxygen therapy after stroke. CNS Neurosci Ther. 2024;30(7):e14858. doi: 10.1111/cns.14858. PMID: 39009510; PMCID: PMC11250159.
- 15 Bird N, Nochetto M. Emergency oxygen for scuba diving injuries: student handbook. 9th ed. Durham: Divers Alert Network; 2021. [cited 11 March 2025]. Available from: <https://dan.org/wp-content/uploads/2021/08/dan-emergency-oxygen-handbook-v3.pdf>
- 16 Blake DF, Naidoo P, Brown LH, Young D, Lippmann J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates. Diving Hyperb Med. 2015;45(2):79-83. (Partial retraction March 2016)
- 17 Comroe JH, Dripps RD, Dumke PR, Deming M. Oxygen toxicity: The effect of inhalation of high concentrations of oxygen for twenty-four hours on normal men at sea level and at a simulated altitude of 18,000 feet. JAMA. 1945;128(10):710–17. doi:10.1001/jama.1945.02860270012004.
- 18 Mitchell SJ, Doolette DJ, Wachholz C, Vann RD, editors. Management of mild or marginal decompression illness in remote locations. 2004 Workshop Proceedings. Washington, DC: Undersea and Hyperbaric Medical Society; 2005.
- 19 Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. Diving Hyperb Med. 2018;48(1):45–55. doi:10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.
- 20 Toklu AS, Cimsit M, Yildiz S, Uzun G, Korpinar S, Sezer H, Aktas S. Decompression sickness cases treated with recompression therapy between 1963 and 1998 in Turkey: review of 179 cases. Undersea Hyperb Med. 2014;41(3):217–21. PMID: 24984316. [cited 2025 June 5]. Available from: <https://www.uhms.org/uhm-search/uhm-journal-volume-41/number-3/decompression-sickness-cases-treated-with-recompression-therapy-between-1963-and-1998-in-turkey-review-of-179-cases.html>
- 21 Doolette DJ, Mitchell SJ. In water recompression. Diving Hyperb Med. 2018;48(2):84–95. doi: 10.28920/dhm48.2.84-95. PMID: 29888380.

22 Mitchell SJ, Doolette DJ. Recreational technical diving part 1: an introduction to technical diving methods and activities. *Diving Hyperb Med.* 2013;43(2):86–93. PMID: 23813462. [cited 29 June 2025]. Available from:

https://www.dhmjournal.com/images/IndividArticles/43June/Mitchell_dhm.43.3.86-93.pdf

23 Australian government: Great Barrier Reef Marine Park Authority. Tourism visitation data. Townsville (AU): GBRMPA; 2023 [updated 2024 Sep 22; cited 2025 Feb 20]. Available from: <https://www2.gbrmpa.gov.au/help/tourism-visitation-data>.

24 Vernon C, editor. AIMS: the first twenty-five years. Townsville: Australian Institute of Marine Science; 1998.

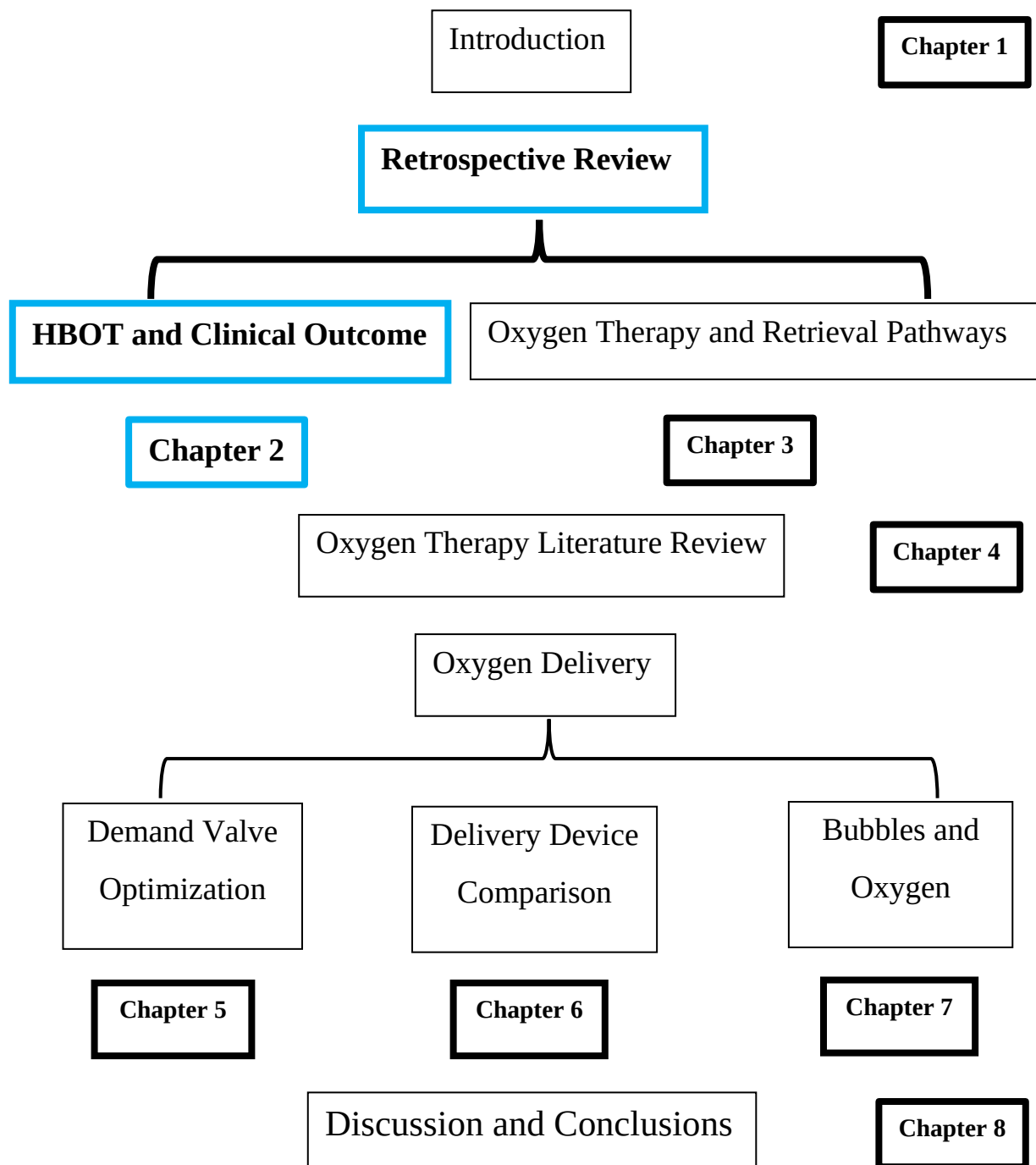
25 Williamson J, Orton J, Callanan V, Ellery W, Hardman J, Palmer R. The Townsville diving medical and aeromedivac system experiences, lessons, and the future. *SPUMS J.* 1988;18(3):82-7. [cited 29 June 2025]. Available from:

https://www.dhmjournal.com/images/IndividArticles/18Sept/Williamson_SPUMSJ.18.3.82-87.pdf

26 Griffiths D, Webb R. Ground transportation of diving injuries to Townsville. In: Mitchell SJ, Doolette DJ, Wachholz CJ, Vann RD, editors. Management of mild or marginal decompression illness in remote locations workshop proceedings; May 24-5, 2004; Sydney, Australia. Durham, NC: Divers Alert Network; 2005. p. 100–10. [cited 2025 June 5]. Available from: <https://world.dan.org/wp-content/uploads/2021/06/remotewrkshpfinal05-1.pdf>

CHAPTER 2 - DIVERS TREATED IN TOWNSVILLE, AUSTRALIA: WORSE SYMPTOMS LEAD TO POORER OUTCOMES

This chapter is an exact copy of the published journal article. This is the first of two published retrospective review papers. This chapter sets the scene for understanding the number and extent of diving injuries along the GBR in North Queensland. The focus of this chapter is the characterization of the injured divers presenting to Townsville, including initial disease severity, time to HBOT, reasons for delay and clinical outcome.



Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes

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Keywords

Decompression illness; Decompression sickness; Hyperbaric oxygen treatment; Oxygen; Recompression; Scuba diving

Abstract

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Introduction: Hyperbaric oxygen treatment (HBOT) is considered definitive treatment for decompression illness. Delay to HBOT may be due to dive site remoteness and limited facility availability. Review of cases may help identify factors contributing to clinical outcomes.

Methods: Injured divers treated in Townsville from November 2003 through December 2018 were identified. Information on demographics, initial disease severity, time to symptom onset post-dive, time to pre-HBOT oxygen therapy (in-water recompression or normobaric), time to HBOT, and clinical outcome was reviewed. Data were reported as median (interquartile range [IQR]) with Kruskal-Wallis and chi-square tests used to evaluate group differences. Significance was accepted at $P < 0.05$.

Results: A total of 306 divers (184 males, 122 females) were included with a median age of 29 (IQR 24, 35) years. Most divers had mild initial disease severity ($n = 216$, 70%). Time to symptom onset was 60 (10, 360) min, time to pre-HBOT oxygen therapy was 4:00 (00:30, 24:27) h:min, and time to start of HBOT was 38:51 (22:11, 69:15) h:min. Most divers (93%) had a good (no residual or minor residual symptoms) outcome and no treated diver died. Higher initial disease severity was significantly associated with shorter times to symptom onset, oxygen therapy, and HBOT, and with worse outcomes. The paucity of cases receiving HBOT with minimal delay precluded meaningful evaluation of the effect of delay to HBOT.

Conclusions: Most divers had mild initial disease severity and a good outcome. Higher initial disease severity accelerated the speed of care obtained and was the only factor associated with poorer outcome.

Introduction

The Great Barrier Reef (GBR) is one of the most popular places to dive in Australia. It is the largest living structure on the planet and extends for 2,300 kilometres along the Queensland coastline. With 2.4 million visitor days per year, the GBR provides 64,000 jobs and contributes \$6.4 billion to the Australian economy in annual revenue.¹ Diving is a relatively safe sport, but 483 fatalities were reported with the

activity in Australia from 1970 to 2018, 116 in Queensland.² Although death is relatively rare from diving, many divers are injured each year requiring treatment in a hyperbaric facility. In 2018, 112 divers were treated for decompression illness (DCI) in Australia, 34 in Queensland (Hyperbaric Technicians and Nurses Association, unpublished data). Decompression illness is a collective term embracing decompression sickness (DCS) caused by bubble formation from dissolved gas, and arterial gas embolism (AGE) caused

by pulmonary barotrauma.³ In this paper 'DCS' is used when the goal is to specifically refer to the consequences of bubble formation from dissolved gas, and the collective term 'DCI' is used to refer to both DCS and AGE.

Hyperbaric oxygen treatment (HBOT) is currently considered the definitive treatment for DCI.^{3,4} Access to HBOT may be delayed due to the remoteness of a dive site and limited access to hyperbaric chambers. Delay to HBOT greater than three hours has been associated with poorer outcomes in severely injured divers.⁵ The Townsville University Hospital operates the only hyperbaric chamber in north Queensland, providing physician advice and HBOT for injured divers from the Whitsunday Islands north to the Torres Strait as well as for divers from some of the surrounding Pacific Islands. However, no recent literature has been published on injured divers in north Queensland.

The aim of this retrospective review was to outline the incidence, care and outcome of injured divers referred to Townsville University Hospital hyperbaric medicine unit.

Methods

Ethics approval was granted from the Townsville Hospital and Health Service (LNR/2019/QTHS/51229) and James Cook University (H7767). The Townsville Hospital relocated to its current site in October 2001, with the installation of a new multi-place rectangular chamber (Fink Engineering Pty Inc., Warana, Queensland, Australia). Data from the first two years of service at the new site were presented at a diving medicine workshop.⁶ This retrospective review includes all injured divers treated at the Townsville hyperbaric unit after the previous report, from 4 November 2003 through 31 December 2018. Yearly patient logs and electronic discharged summaries were reviewed to identify cases for inclusion.

Retrieval Services Queensland databases (Queensland neonatal emergency transport service, clinical coordination retrieval information system, and Brolga) were searched using key words and relevant diagnoses (cerebral arterial gas embolism, decompression [including illness and sickness], drown*, snorkel*, and scuba), hyperbaric med*, and offshore retrievals by rotary wing asset to identify cases. Identifying data (name, date of birth, and date of incident) were collected so that cases could be linked with hyperbaric unit data to ensure that no cases were missed or duplicated.

Individual charts were reviewed, and data extracted to pre-formatted forms. Where available, the Queensland state-wide diver injury assessment form provided valuable information ([Appendix A](#)[#]). Diver age, sex, region of origin, body habitus, medical history and known medication use was collected. Body habitus was classified using body mass index

if height and weight data were available otherwise from clinical descriptions in the medical charts, passport photos, or staff memory. Diving history including qualification, reported number of previous dives, years of diving, previous DCI as well as a description of the incident dive (day of week, month, and place of incident, nature of the dive, dive team, breathing gas and circuit type, dive computer use, potential contributing factors, maximum depth, and total dive time) and symptom profile were obtained from medical records and dive logs. Due to the complexity of dive profiles and the lack of dive computer downloads, only maximum depth, and total dive time (also known as surface-to-surface or run time) were documented. If a dive log had depths recorded as fractions of a metre (e.g., 24.3 m) it was recorded that a dive computer had been used. Time of symptom onset was defined in two ways. First, using a binary definition: during the dive (symptom onset underwater during the dive) or post-dive (after arriving at the surface). Second, calculating an actual time duration from the time the injured diver arrived at the surface after the incident dive to the time of symptom onset. Due to the unavailability of details on time to symptom onset underwater, the time of arrival at the surface was used as the starting time point for calculating time to treatment for all divers.

Initial disease grade was classified as mild, moderate, or severe using a system developed in Townsville (Table 1).⁶ This grading system is subtly different to the widely accepted paradigm that arose from the 2005 remote DCI workshop,³ but it was adopted here for having been applied to the Townsville patients in 'real time' over the study period. Treatment details were collected including time to commencement of pre-HBOT oxygen therapy (if administered) after symptom onset.

Retrieval details were collected including platform (boat, rotary wing, fixed wing, or road) and type of retrieval (primary, secondary, or tertiary). Primary retrievals were classified as retrievals from a pre-hospital location. If a dive boat called for medical advice and was directed to return to shore, this was classified as a primary retrieval by boat. If the dive boat returned to shore without any urgency after completing their trip, this was not considered a retrieval. Secondary retrievals were defined as retrievals from a place of medical care to a second facility providing higher care. This may be a second retrieval leg after a primary retrieval or the transfer between two health care facilities after diver self-presentation. Tertiary retrievals were defined as transfers from a secondary site to a third facility. Road retrievals included ambulance, bus, or car. Time to start of HBOT following symptom onset, final diagnosis, and clinical outcome at completion of HBOT (characterised as in Table 2)⁷ were determined.

[#] Appendix A can be found on the DHM Journal website: <https://www.dhmjournal.com/index.php/journals?id=344>

Table 1

Initial disease severity grade using the established Townsville Hospital categories;⁶ mild and moderate symptoms are invariably decompression sickness (DCS) while arterial gas embolism events would be classified as severe

Severity	Definition
Mild	Symptomatic DCS with no objective signs except: Minor skin rash Lymphatic DCS Sharpened Romberg test less than 30 seconds
Moderate	Symptomatic DCS with subtle signs: Impaired higher function Impaired Romberg test Subjective sensory changes Minor weakness due to pain Cutis marmorata
Severe	Symptoms threatening life or mobility: Loss of consciousness Cardiopulmonary DCS Spinal DCS

Table 2

Clinical outcome classification at the end of hyperbaric oxygen treatment⁷

Well, no residual signs or symptoms
Minor symptoms, no functional significance
Residual symptoms, moderate impairment
Major incapacity
Dead

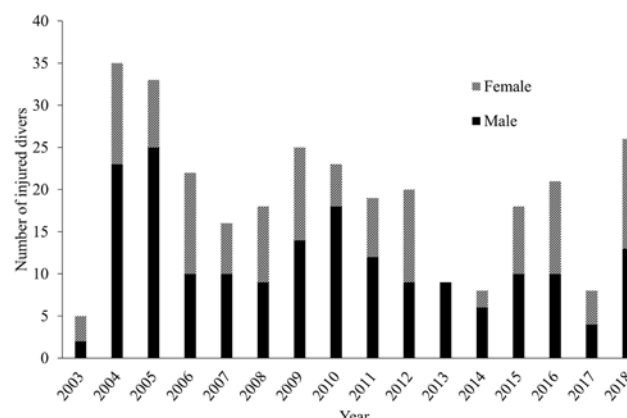
Two researchers (DB and RT) performed the data extraction. Forms were compared and consensus reached. Individual Retrieval Services Queensland records were accessed to clarify retrieval information not apparent in the hospital medical records. All collected data were de-identified and entered into an Excel worksheet, and subsequently exported into Statistical Package for the Social Sciences version 28.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) for analysis.

ANALYSIS

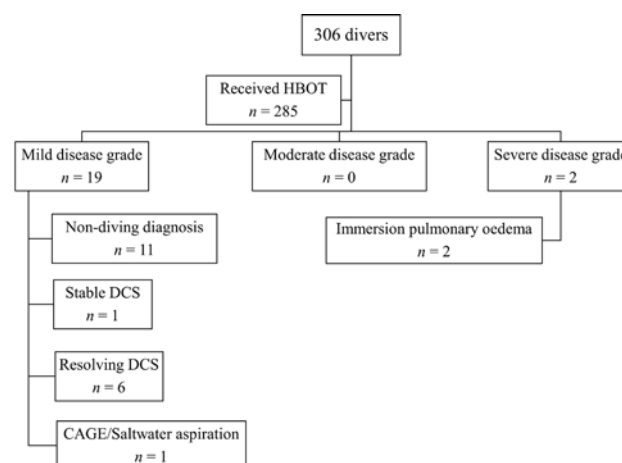
Data are presented using frequencies and percentages for categorical variables, and median and interquartile range (IQR) for continuous variables as all data were not normally distributed as assessed using the Shapiro-Wilk test. The Mann-Whitney U test was used for analysis comparing time to symptom onset, oxygen commencement, and HBOT between divers with onset of symptoms at depth versus onset of symptoms after the dive. Comparison of initial disease

Figure 1

Number of injured divers by sex presenting for hyperbaric oxygen therapy at the Townsville hyperbaric medicine unit per calendar year during the study period 4 November 2003 through 31 December 2018

**Figure 2**

Breakdown of cases by initial disease grade of divers who did not receive hyperbaric oxygen treatment (HBOT); CAGE – cerebral arterial gas embolism; DCS – decompression sickness



grade with time to event data (symptom onset, oxygen delivery, and HBOT) and oxygen duration was completed using the Kruskal-Wallis test for analysis. Dunn's test was used for *post hoc* analysis with Bonferroni correction. Statistical significance was accepted at $P < 0.05$ for all tests. As data were missing from some medical records, the *n* presented throughout the results denotes the number of records for which the information was documented.

Results

DIVER DEMOGRAPHICS

A total of 310 injured divers were identified during the study period. Four divers were excluded as their paper medical records had been destroyed following national medical record guidelines, one in 2003 and three in 2004, leaving

Table 3

Injured diver characteristics; data are *n* (%) unless otherwise specified; ^aWorld Health Organization regions; *n* = number of divers for which the data was documented in each category; DCI – decompression illness; IQR – interquartile range

Characteristic (<i>n</i> = 306)	
Male	184 (60)
Female	122 (40)
Median age (years)	29 (IQR 24, 35) (Range 14–74)
Region of origin^a (<i>n</i> = 306)	
Western Pacific	159 (52)
Australia	143 (47)
Europe	103 (34)
Americas	43 (14)
Africa	1 (< 1)
Eastern Mediterranean	0
South-east Asia	0
Body habitus (<i>n</i> = 165)	
Underweight	5 (3)
Normal	135 (82)
Overweight	14 (8)
Obese	11 (7)
Relevant medical history	
Medical/Surgical history = yes (<i>n</i> = 292)	148 (48)
Medication use = yes (<i>n</i> = 248)	107 (43)
Diving qualification (<i>n</i> = 216)	
Uncertified	19 (9)
Student	25 (12)
Open water	57 (26)
Advanced	19 (9)
Rescue	13 (6)
Divemaster/Assistant instructor	16 (7)
Instructor	47 (22)
Commercial/Military	20 (9)
Relevant diving history	
Median number of previous dives (<i>n</i> = 198)	55 (IQR 9, 325) (Range 0–17,000)
Median years of diving (<i>n</i> = 75)	8 (IQR 2, 13) (Range 0–53)
Previous DCI = yes (<i>n</i> = 207)	41 (20)

306 divers for the analysis, displayed by year and sex in Figure 1. The term ‘injured divers’ is used intentionally to reflect the fact that some divers did not receive HBOT and had diagnoses other than DCI including cases considered to be suffering severe symptoms arising from immersion pulmonary oedema. (Figure 2). Most of the divers were from overseas, young, and certified with a wide range of reported previous diving experience (Table 3). Over half were male and the majority were of normal body habitus. A small number of divers reported having a previous incident of DCI (Table 3). Just under half of the divers had a history of a medical or surgical condition and many used a medication

(acute or chronic) in the 48 hours before or after the incident dive (Table 3).

INCIDENT DIVE AND CONTRIBUTING FACTORS

Most incidents occurred during recreational dives (Table 4), during the summer (Southern Hemisphere) months, and on weekends (Friday through Sunday). Less than one-third of the incidents were in occupational divers and incidents were rare in scientific divers (Table 4). Just over half of the divers were diving with a buddy and the vast majority were breathing compressed air with open-circuit equipment. Only

Table 4

Incident dive characteristics; *n* = number of divers for which the data was documented

Characteristic	<i>n</i> (%)
Nature of dive (<i>n</i> = 260)	
Introductory	16 (6)
Certification course	55 (21)
Recreational	116 (45)
Occupational	69 (27)
Scientific	4 (1)
Technical	0
Dive team (<i>n</i> = 130)	
Solo	5 (4)
Buddy	68 (52)
Threesome	2 (1)
Group > 3	41 (32)
Surface support	14 (11)
Breathing gas (<i>n</i> = 177)	
Air	164 (93)
Nitrox 32%	6 (3)
Nitrox other %	6 (3)
Oxygen	1 (< 1)
Breathing circuit (<i>n</i> = 181)	
Open	158 (88)
Surface supply	22 (12)
Freediving	1 (< 1)
Dive computer (<i>n</i> = 160)	
Computer used	133 (83)

half of the medical records had documentation of whether a dive computer was used. In those with documentation, the majority used a computer (Table 4). The maximum dive depth recorded in 301 cases ranged from 1.8 to 50 metres of seawater (msw) (median 18, IQR 14, 25 msw). The total dive time recorded in 279 cases ranged from one to 210 minutes (min) (median 37, IQR 29, 45 min). Medical record documentation was poor for pre/post-dive contributing factors. However, dehydration and seasickness were commonly noted in those charts with documentation (Table 5). Possible contributing factors were varied, with repetitive dives and inadequate surface interval being the most frequently reported (Table 5).

SYMPTOMS AND TREATMENT AT SCENE

Most symptoms commenced after the incident dive (*n* = 275, 90%) rather than during the dive. The diagnoses of the divers with symptom onset during the dive were: 17 DCS, 10 cerebral AGE, one inner ear barotrauma and three immersion pulmonary oedema. All divers diagnosed with DCS had performed multiple dives often over several days. The median time to symptom onset post-dive (*n* = 269) was 1 hour (IQR 0:10, 6:00 h:min). One extreme outlier was identified. This diver had a time to symptom onset of 384 h. This diver was exposed to altitude by flying after diving, so the diagnosis of DCS was considered plausible, and the diver was recompressed, but the final diagnosis was

Table 5

Possible contributing factors for decompression illness pre-, during and post-dive; *n* = number of divers for which the data was documented; *factors listed on the Queensland diver assessment form; ^bcold or overheated combined as a single factor on the Queensland diver assessment form

Possible contributing factor	<i>n</i> (%)
Dehydration (<i>n</i> = 153)	88 (58)
Seasickness (<i>n</i> = 64)	37 (58)
Rough seas (<i>n</i> = 61)	32 (53)
Alcohol/Drug use (<i>n</i> = 133)	61 (46)
Possible contributing dive factor^a (<i>n</i> = 306)	
Multiple repetitive dives (> 3 / day)	138 (45)
Surface interval < 120 min	136 (44)
Multi-day diving (> 3 consecutive days)	97 (32)
Rapid ascent	95 (31)
Excessive exertion	93 (30)
No safety stop	75 (25)
Reverse profile	72 (24)
Ear problems	67 (22)
Equipment problems	44 (14)
Violated computer/table guidance	35 (11)
Altitude exposure	32 (11)
Buoyancy problems	20 (7)
Thermal stress ^b	16 (5)

non-diving related. Most divers were classified as having mild initial disease grade (*n* = 216, 70%). Paraesthesia was the most common presenting symptom followed by arthralgia/myalgia and poor balance/ataxia (Figure 3). Half of the divers received treatment at the scene (*n* = 155/304), most commonly oxygen (143/155, 92%). Twenty-four divers (*n* = 24/155, 15%) had analgesia, 32 (*n* = 32/155, 21%) had fluids (30 oral, one intravenous and oral, one intravenous only) and two (*n* = 2/155, 1%) had antiemetics at the scene. Time to symptom onset was shorter for divers treated at the scene (*n* = 130) (median 20 min, IQR 00:05, 1:30 h:min) compared to the group of injured divers not treated at the scene (*n* = 139) (median 4 h, IQR 1, 16 h).

RETRIEVAL

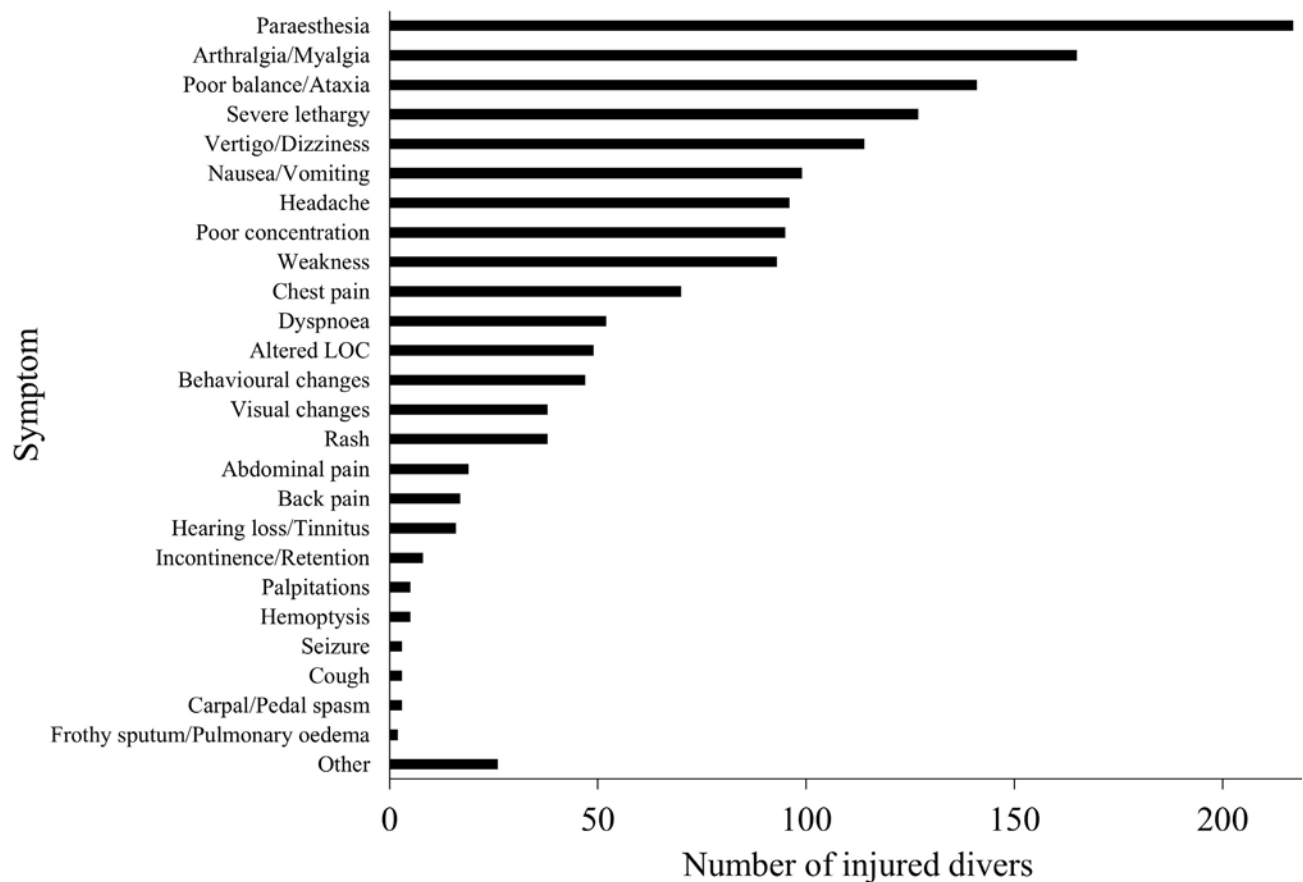
One-third of the injured divers (*n* = 104) were primarily retrieved, half by boat (*n* = 52/104). More than three quarters of the injured divers required a secondary retrieval (*n* = 236), half by road. Only 24 injured divers had a tertiary retrieval.

RECOMPRESSION

A total of 285 (93%) of the injured divers received HBOT. Figure 2 depicts, by initial disease grade, the divers that did not receive HBOT. Nineteen of 216 injured divers initially classified as having a mild disease grade were not

Figure 3

Frequency of presenting symptoms of the injured divers who may have exhibited more than one symptom; the 'other' category consists of 26 discrete symptoms. LOC – level of consciousness

**Table 6**

Time to hyperbaric oxygen treatment (HBOT) post-symptom onset for all divers and subgroups; * $P = 0.001$ vs divers with symptom onset during the dive; IQR – interquartile range

Parameter	Median (IQR) (h:min)
Time to HBOT all injured divers, $n = 283$	38:51 (22:11, 69:15)
Time to HBOT for divers with symptom onset post-dive, $n = 256$	41:30 (22:26, 70:37)*
Time to HBOT for divers with symptom onset during the dive, $n = 27$	23:48 (9:45, 31:06)
Time to HBOT for divers treated at scene, $n = 145$	26:12 (17:20, 49:48)
Time to HBOT for divers primarily retrieved, $n = 97$	21:40 (10:30, 38:10)

recompressed and for two the time to HBOT could not be calculated. Of these 19 non-recompressed divers, 11 had a non-diving related final diagnosis, seven had a final diagnosis of decompression sickness with one a disease evolution of stable and the other six resolving. One injured diver had a final diagnosis of possible cerebral AGE and saltwater aspiration. Due to a previous medical condition and resolution of symptoms, it was decided not to recompress this diver. All injured divers with initial disease grade of moderate ($n = 57$) were recompressed. Two injured divers classified as having initial severe disease grade ($n = 33$)

were not recompressed. The final diagnosis in both cases was immersion pulmonary oedema. Divers with symptom onset during the dive had shorter times to HBOT compared to those with post-dive symptom onset (Table 6). Time to HBOT decreased as initial disease grade severity increased.

Of the 283 divers that underwent HBOT, none had HBOT commenced under three hours and only eight had HBOT commenced under six hours. Only 35 divers (12%) commenced HBOT under 12 hours and only 93 divers (33%) commenced HBOT under 24 hours. Three extreme

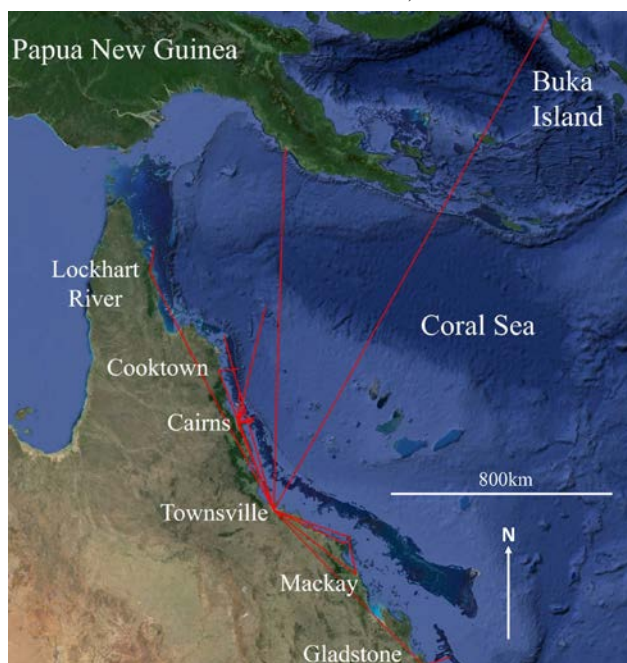
Table 7

Reasons for delays to hyperbaric oxygen treatment (HBOT); IQR – interquartile range; NBOT – normobaric oxygen treatment; * retrieval pathways for these cases are shown in Figure 4

Reason for delay	<i>n</i> (%)	Median (IQR) time to HBOT (h:min)	Initial disease severity		
			mild	moderate	severe
Delayed presentation for medical review	100 (35)	48:30 (29:58, 91:53)	83	15	2
Extreme retrieval distance (500 to > 1,700 km)	91 (32)	22:03 (11:48, 41:15)	42	27	22*
NBOT overnight then transferred	24 (8)	31:18 (23:11, 45:23)	20	4	0
Kept diving	22 (8)	84:45 (56:36, 244:11)	18	2	2
Initial misdiagnosis	15 (5)	73:32 (49:50, 134:56)	12	2	1
No delay	10 (4)	5:39 (3:32, 6:54)	3	3	4
NBOT with symptom reoccurrence	10 (4)	48:16 (30:08, 78:03)	9	1	0
NBOT overnight with morning HBOT	9 (3)	22:22 (19:34, 24:39)	7	2	0
Refused initial transfer	2 (< 1)	68:03 (24:31)	1	1	0

Figure 4

Retrieval pathways for divers with severe initial disease grade and extreme retrieval distance; *n* = 22



outliers were identified with a time to HBOT of greater than 373 h from symptom onset. All three divers were occupational divers who presented late for initial medical review. Reasons for delay to HBOT are listed in Table 7. Initial misdiagnosis and the lack of knowledge of the need for HBOT led to a delay in the referral and transfer of some divers to Townsville. Extreme retrieval distance was the most common reason for delay to HBOT for divers with severe initial disease severity. Retrieval pathways for these divers (*n* = 22) are shown in Figure 4. Only one of these divers was directly transferred to Townsville with the remaining 21 requiring more than one retrieval leg.

The initial recompression treatment table used was most often a modified Royal Navy (RN) 62 (US Navy treatment table 6), with only a small number of table extensions required (Table 8). Most divers required only a few treatments and had a good outcome (Table 8). Sixteen divers required more than 10 treatments, only one of these had mild initial symptoms (symptoms initially resolved on normobaric oxygen therapy) and only three had complete resolution of symptoms. Seven of these divers had a modified RN 62 as their first follow-up treatment table, four of whom had a Comex 30 as their initial treatment table. The other follow-up treatments were a combination of 180 kPa (100 min with 2 x 5 min air breaks) and 140 kPa (120 min with 2 x 5 min air breaks) treatment tables.

There were statistically significant differences between initial disease grade and time to symptom onset, time to oxygen commencement, and time to HBOT (Table 9). Divers with more severe initial disease grade had a shorter time to symptom onset, oxygen commencement, and HBOT. There was no statistically significant difference for duration of pre-HBOT oxygen therapy between the three initial disease grade groups ($P = 0.408$).

POSSIBLE CONTRIBUTING FACTORS FOR CLINICAL OUTCOMES

The small group of injured divers with major incapacity at the completion of hyperbaric treatment (*n* = 2) had severe initial disease grade, short times to symptom onset post-surfacing (1 min) and to oxygen commencement (5 min), and HBOT (6 h 49 min and 7 h 3 min). Due to small numbers, the clinical outcome groups 'moderate impairment' (*n* = 19) and 'major incapacity' (*n* = 2) were combined into one group for further analysis. There were no statistical differences between the clinical outcome groups for time to symptom onset, time to oxygen commencement or time to HBOT in the group of divers with severe initial disease grade (Table 10). There was

Table 8

Initial hyperbaric treatment table, number of treatments and clinical outcome; IQR – interquartile range; *n* = number of divers for whom the data was documented

Treatment parameter	<i>n</i> (%) or median (IQR), range
Initial treatment table (<i>n</i> = 285)	
Royal Navy 62	262 (92)
Comex 30	12 (4)
Other	11 (4)
Extensions and treatment numbers	
Table extension (<i>n</i> = 283)	30 (11)
Median (IQR) treatments (<i>n</i> = 285)	3 (2, 4), range 1–37
Clinical outcome (<i>n</i> = 306)	
Well, no residual signs or symptoms	147 (48)
Minor symptoms, no functional significance	138 (45)
Residual symptoms, moderate impairment	19 (6)
Major incapacity	2 (1)
Death	0

Table 9

Comparison of initial disease grade with timelines as specified (median IQR hours:minutes [h:min]); * Kruskal-Wallis test; # does not include injured divers with symptom onset during the dive; ^asignificant difference between mild and moderate; ^bsignificant difference between mild and severe; ^csignificant difference between moderate and severe; IQR – interquartile range; HBOT – hyperbaric oxygen treatment

Timeline	Mild	Moderate	Severe	<i>P</i> -value*
Time of symptom onset post-dive [#] (h:min)	2:00 (0:15, 8:00) ^{a,b} <i>n</i> = 199	0:15 (0:02, 2:00) ^a <i>n</i> = 51	00:10 (0:01, 1:00) ^b <i>n</i> = 19	< 0.001
Time to pre-HBOT oxygen start post-symptom onset (h:min)	9:00 (0:39, 31:57) ^{a,b} <i>n</i> = 169	1:27 (0:15, 11:48) ^a <i>n</i> = 54	00:15 (0:06, 3:40) ^b <i>n</i> = 31	< 0.001
Time to HBOT (h:min)	46:55 (26:10, 79:15) ^{a,b} <i>n</i> = 195	24:31 (12:10, 43:16) ^{a,c} <i>n</i> = 57	11:28 (7:57, 23:48) ^{b,c} <i>n</i> = 31	< 0.001

Table 10

Comparison of clinical outcome after completion of hyperbaric oxygen treatment (HBOT) with timelines as specified (median IQR hours:minutes [h:min]) for divers with initial severe disease grade; *Kruskal-Wallis test; ^adoes not include injured divers with symptom onset during the dive; ^bone extreme outlier excluded due to a non-DCI final diagnosis

Timeline	No residual symptoms	Minor residual symptoms	Moderate / major residual symptoms	<i>P</i> -value*
Time to symptom onset post-dive ^a (h:min)	0:01 (< 0:01, 0:20) <i>n</i> = 6	0:01 (0:01, 3:00) <i>n</i> = 5	00:45 (0:01, 2:00) ^b <i>n</i> = 7	0.322
Time to oxygen start post-symptom onset (h:min)	0:10 (0:10, 3:00) <i>n</i> = 11	2:22 (0:07, 6:22) <i>n</i> = 12	00:11 (0:05, 5:16) <i>n</i> = 8	0.462
Time to start HBOT (h:min)	8:08 (6:57, 66:59) <i>n</i> = 9	16:53 (8:16, 23:33) <i>n</i> = 12	13:42 (7:40, 41:18) <i>n</i> = 10	0.347

a statistically significant association between initial disease grade and combined clinical outcome ($P < 0.001$, df 4). *Post hoc* analysis showed that divers with moderate or severe initial disease grades had poorer outcomes.

Discussion

Divers with higher initial disease grade had earlier time to symptom onset, oxygen commencement, shorter time to HBOT and poorer outcomes. These findings appear to be consistent with other studies where initial disease severity is

related to outcome, but other contributing factors are difficult to determine.^{5,8,9} However, almost all the divers in this study had a substantial delay to HBOT, precluding meaningful evaluation of the effect of time to HBOT on clinical outcome.

Many factors (Table 5) have been proposed as possibly contributing to the risk of DCS and outcomes.^{3,10,11} Numerous retrospective reviews^{5,8,9,12–14} have reported the incidence of these factors, with one study reporting that 76% of injured divers had one or more contributing factors.¹⁵ Despite ongoing attempts, there appears to be no consistent association between these proposed contributing factors and DCS risk or outcome. The retrospective nature of these studies probably greatly contributes to the difficulty in delineating pertinent risk factors. Incomplete documentation often leads to exclusion of cases^{16,17} or possibly missing pertinent negatives in data sets as only positive responses are often recorded. Self-reporting would only include the items a diver believed to be a possible risk factor.^{13,18} A prospective study collecting information on possible risk factors would greatly improve our understanding of risk and help focus educational opportunities for divers, dive operators, and dive medical personnel.

The divers in our study were largely young (Table 3), possibly reflecting the Australian backpacker (younger people travelling overseas, often on a working visa, staying in hostels) commonly taking scuba lessons and diving on the GBR. Injured divers are often young^{12,14,16,19–21} especially compared to deceased divers. Divers Alert Network (DAN) fatality data from 2018 found a median age of 56 years of age²² in deceased divers and Queensland data for 2000–2019 found a median age of 48 (IQR 32, 57) years.² Older divers are more likely to have medical conditions and poor physical fitness. Previous medical conditions are frequently listed in fatality reviews² but infrequently documented in retrospective reviews for divers treated for DCI. Health surveillance of recreational divers has been an issue discussed in the diving medicine fraternity;²³ however, any recommendations would be difficult to enforce. Divers are encouraged to be reviewed by a medical practitioner after a change in health.²³ Despite this recommendation, an online survey completed by DAN found divers with diabetes, cardiovascular, or respiratory disease rarely modified their diving practices or sought specialist advice.²⁴ Identifying medical or surgical conditions when divers are treated for DCI could provide an opportunity for discussion with a diving physician, potentially decreasing the risk of death in later years.

Many of the injured divers treated in Townsville were from overseas, possibly due to a regional phenomenon reflecting the high load of visitors who often participate in scuba courses to dive on the GBR. The percentage of overseas divers seems to be even higher than described in other tourist areas.^{14,20} This is also reflected in the seasonality of presentations with more cases in the Australian summer

months when the ocean water is warmer. In the northern hemisphere, higher call volume for advice is also found in the summer months.^{22,25} More injured divers presented over the weekend days. This is unsurprising as dive trips are often planned around other commitments as weekend getaways.

The median time to symptom onset of an hour post-dive in our study was similar to that described in previous reports.^{17,19} Other studies reported time to symptom onset of: 30 min,¹³ 41 min,¹⁶ and 90 min.¹² Divers in our study with a severe initial disease grade had shorter times to symptom onset. This was consistent with other studies focusing on divers with spinal cord DCS, the time to symptom onset from surfacing being considerably shorter: 5 min,⁸ 10 min,⁵ and 15 min.⁹ Longer times to symptom onset have been associated with better outcomes,⁵ while severe initial symptoms are associated with poorer recovery.²⁶ Together, short delays to symptom onset and severe symptoms should lead to prompt initiation of first aid treatment and arrangement for transport to a recompression facility.³

The most common presenting symptoms of paraesthesia and arthralgia/myalgia in our study are in keeping with previously published data.^{3,16,21} These symptoms may be mild and vague, often making DCS difficult to diagnose by an inexperienced practitioner. The DAN America ‘hotline’ was established in 1980 to help injured divers by providing advice for both pre-hospital and hospital care.²² An Australian hotline, called the Diver Emergency Service (DES), started operation in 1983 providing similar advice in the Asia-Pacific region. DAN World assumed responsibility for the Australian hotline in 2019. Phone advice can be obtained from DAN as well as directly from diving physicians around the world assisting with the diagnosis of diving related injuries, and guidance on treatment and disposition. This is a valuable service especially for centres that may not frequently care for injured divers.

The median time to HBOT in our study was considerable. Other studies have reported median times to HBOT of 6 h (Switzerland),¹³ 24 h (Turkey),¹⁶ 32.5 h (Poland),¹⁹ and 2 days (New Zealand).¹² Consistent with our study, two studies in France found that divers with severe initial disease had shorter times to HBOT, 3 h⁸ and 2 h 44 min.⁵ Delay to recompression seems to increase the risk of incomplete recovery, but only in severely injured divers.^{5,26} Previous research found an improvement in outcomes when divers with severe disease received HBOT within six hours.²⁶ A more recent study has found that divers with spinal DCS treated with HBOT within three hours of symptom onset had less sequelae at time of discharge.⁵ None of the divers in our study had HBOT starting within three hours and only eight divers had HBOT commenced within six hours. Most divers in our study presented late for HBOT. Delayed HBOT, greater than 48 hours, has still been found to alleviate symptoms,^{27,28} therefore delayed presentation should not preclude HBOT.

Many factors contributed to the delay to HBOT in our study (Table 7). Time to HBOT not only varies with initial disease severity but also by geographical location and distribution of hyperbaric facilities.^{20,28} The Townsville hyperbaric unit covers a large geographical area with divers often in remote locations requiring long and complex retrievals. Further in-depth analysis of retrieval pathways will provide information on the factors leading to long retrieval times, identifying areas for improvement.

During our study period no formal follow-up occurred. Follow-up of divers with incomplete recovery is infrequently documented in retrospective reviews and often commented on in the limitations.^{15,20} One study presented clinical outcome at one month post-injury in divers with spinal cord DCS, but the details of how this was done were not included.⁸ Another study contacted divers treated over a two-year period, 1.5 to 3.5 years later.²¹ In this study, 13 divers “*had reduced but lingering symptoms at discharge from hospital*”.²¹ Out of the 30 divers treated over the two-year period, 24 were contacted, one having died in a subsequent diving accident.²¹ Six divers had residual symptoms at the time of contact, but interestingly three of these did not report having symptoms upon completion of their HBOT.²¹ One diver suffered a concussion in the intervening years and it could not be determined if the reported symptoms were from DCS or the concussion. No other information was provided on possible reasons for recurrence of symptoms in the divers who had been free of symptoms on discharge.²¹ In the current era of electronic communication, it would seem easier to contact previously treated divers whether they were local or tourists, though securing responses is likely to remain challenging. Historically, the Townsville HMU sent out follow-up letters to divers requesting information on clinical outcome and recurrence of symptoms during air travel. This information led to the changing of the advice on flying after hyperbaric treatment for DCI, decreasing the time to three weeks post completion of treatment from previous advice to wait for 4–6 weeks. Despite the reduction, this remains a very conservative recommendation. Follow-up letters are no longer sent to divers, and follow-up information was not documented in any of the charts in this review. Follow-up questionnaires could provide valuable data on recovery of divers especially those discharged with residual symptoms. At the time of discharge, divers are presented with a treatment summary and discussion ensues around returning to diving and flying. This would be an ideal time to verify electronic contact details and discuss the sending of a follow-up questionnaire. This would provide continuity of care for the divers and help with organising clinical review if necessary. Active follow-up of all treated patients would improve the knowledge of the incidence of ongoing permanent sequelae and allow for better prognostication and advice to patients on discharge.

RECOMMENDATIONS

Queensland has a state-wide diver injury assessment form ([Appendix A](#)[#]). The form contains information on assessing potential diving related injuries and a fillable section to enter information on dive profiles, risk factors, symptoms, physical assessment, and treatment provided. This form was designed to provide guidance for facilities infrequently encountering injured divers, providing a template of pertinent factors to be collected and discussed when referring to the hyperbaric facilities. Recommended changes to this form have been identified from this study. Thermal stress should be divided into cold and overheated and include the phase of dive at which this occurred.⁴ There was poor documentation of thermal stress in the current study perhaps indicating a lack of knowledge of the role it may play in DCS risk. Current diving practice would indicate that a 60 min surface interval between dives is now considered standard, therefore, this item should be changed from 120 to 60 min. Lastly, documenting the incident dive location would assist in identifying high risk dive sites and allow for improved analysis of retrieval pathways and time to HBOT.

LIMITATIONS

This study was retrospective and limited by incomplete records and missing data. Missing data may have contributed to the difficulty to detect correlations between initial disease severity, contributing factors, timelines, and clinical outcomes. Time to treatment for divers with symptom onset during the dive may have been longer than reported as arrival at the surface was used as the starting point for timeline calculations. Divers are encouraged to return to the hyperbaric unit for review should symptoms reoccur, however, there was no attempt at follow up of divers after completion of their hyperbaric treatment, therefore final outcome is unknown. It is unknown if any injured divers were treated with normobaric oxygen therapy either on dive boats or at other health care facilities and not transferred to Townsville for treatment. Therefore, the true incidence of DCI in the Townsville catchment area is unknown.

Conclusions

This review describes 15 years of activity at the Townsville hyperbaric medicine unit. Most divers had mild initial disease severity, required few hyperbaric treatments and had a good outcome. Higher initial disease severity accelerated the speed of care obtained and was the only factor associated with poorer outcome. Improved documentation may enhance the ability to understand the impact of contributing factors on clinical outcomes.

[#] Appendix A can be found on the DHM Journal website: <https://www.dhmjournal.com/index.php/journals?id=344>

References

- 1 Australian Government: Great Barrier Reef Marine Park Authority. Reef facts. August 22, 2022. Updated September 11, 2023. [cited 2023 Dec 17]. Available from: <https://www2.gbrmpa.gov.au/learn/reef-facts>.
- 2 Lippmann J. A review of snorkelling and scuba diving fatalities in Queensland, Australia, 2000 to 2019. *Diving Hyperb Med*. 2022;52:108–18. doi: 10.28920/dhm52.2.108-118. PMID: 35732283. PMCID: PMC9522589.
- 3 Mitchell SJ. Decompression illness: A comprehensive overview. *Diving Hyperb Med*. 2024;54(1Suppl):1–53. doi: 10.28920/dhm54.1.suppl.1-53. PMID: 38537300. PMCID: PMC11168797.
- 4 Mitchell SJ, Bennett M, Moon RE. Decompression sickness and arterial gas embolism. *N Engl J Med*. 2022;386:1254–64. doi: 10.1056/NEJMr2116554. PMID: 35353963.
- 5 Andre S, Lehot H, Morin J, Louge P, de Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. *Emerg Med J*. 2022;0:1–6. doi: 10.1136/emered-2021-211227. PMID: 35135892.
- 6 Griffiths D, Webb R. Ground transportation of diving injuries to Townsville. In: Mitchell SJ, Doolette DJ, Wachholz CJ, Vann RD, editors. Management of mild or marginal decompression illness in remote locations workshop proceedings; May 24–5, 2004; Sydney, Australia. Durham, NC: Divers Alert Network; 2005. p. 100–10. [cited 2023 Dec 17]. Available from: <https://world.dan.org/wp-content/uploads/2021/06/remotewrkshpfinal05-1.pdf>.
- 7 Bennett M. The retrieval of diving injuries in New South Wales a retrospective review of two years of practice. *SPUMS Journal*. 1995;25:142–7. [cited 2023 Dec 17]. Available from: https://www.dhmjournal.com/images/IndividArticles/25Sept/Bennett_SPUMSJ.25.3.142-147.pdf.
- 8 Gempp E, Blatteau J-E. Risk factors and treatment outcomes in scuba divers with spinal cord decompression sickness. *J Crit Care*. 2010;25:236–42. doi: 10.1016/j.jcrc.2009.05.011. PMID: 19682840.
- 9 Blatteau, J-E, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: influence of time to recompression. *Diving Hyperb Med*. 2011;41:129–34. PMID: 21948497. [cited 2023 Dec 17]. Available from: https://www.dhmjournal.com/images/IndividArticles/41Sept/Blatteau_dhm.41.3.129-134.pdf.
- 10 Pollock NW, Buteau D. Updates in decompression illness. *Emerg Med Clin N Am*. 2017;35:301–19. doi: 10.1016/j.emc.2016.12.002. PMID: 28411929.
- 11 Cialoni D, Pieri M, Balestra C, Marroni A. Dive risk factors, gas bubble formation, and decompression illness in recreational SCUBA diving: analysis of DAN Europe DSL data base. *Front Psychol*. 2017;8:1587. doi: 10.3389/fpsyg.2017.01587. PMID: 28974936. PMCID: PMC5610843.
- 12 Haas RM, Hannam JA, Sames C, Schmidt R, Tyson A, Francombe M, et al. Decompression illness in divers treated in Auckland, New Zealand, 1996–2012. *Diving Hyperb Med*. 2014;44:20–5. PMID: 24687481. [cited 2023 Dec 17]. Available from: https://www.dhmjournal.com/images/IndividArticles/44March/Haas_dhm.44.1.20-25.pdf.
- 13 Thaler J, Pignel R, Magnan M-A, Pellegrini M, Louge P. Decompression illness treated at the Geneva hyperbaric facility 2010–2016: a retrospective analysis of local cases. *Diving Hyperb Med*. 2020;50:370–6. doi: 10.28920/dhm50.4.370-376. PMID: 33325018. PMCID: PMC8038901.
- 14 Danker R, Gall N, Freidman G, Arad J. Recompression treatment of Red Sea diving accidents: a 23-year summary. *Clin J Sport Med*. 2005;15:253–6. doi: 10.1097/01.jsm.0000168074.89744.4f. PMID: 16003040.
- 15 Lundell RV, Arola O, Suvilehto J, Kuokkanen J, Valtonen M, Räisänen-Sokolowski AK. Decompression illness (DCI) in Finland 1999–2018: special emphasis on technical diving. *Diving Hyperb Med*. 2019;49:259–65. doi: 10.28920/dhm49.4.259-265. PMID: 31828744. PMCID: PMC7039777.
- 16 Toklu AS, Cimsit M, Yildiz S, Uzun G, Korpınar S, Sezer H, Aktas S. Decompression sickness cases treated with recompression therapy between 1963 and 1998 in Turkey: review of 179 cases. *Undersea Hyperb Med*. 2014;41:217–21. PMID: 24984316.
- 17 Mutzbauer TS, Staps E. How delay to recompression influences treatment and outcome in recreational divers with mild to moderate neurological decompression sickness in a remote setting. *Diving Hyperb Med*. 2013;43:42–5. PMID: 23508662. [cited 2023 Dec 17]. Available from: https://www.dhmjournal.com/images/IndividArticles/43March/Mutzbauer_dhm.43.1.42-45.pdf.
- 18 Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment. *Diving Hyperb Med*. 2022;52:78–84. doi: 10.28920/dhm52.2.74-84. PMID: 35732278. PMCID: PMC9527095.
- 19 Kot J, Sićko Z, Michałkiewicz M, Lizak E, Góralczyk P. Recompression treatment for decompression illness: 5-year report (2003–2007) from national centre for hyperbaric medicine in Poland. *Int Marit Health*. 2008;59:69–80. PMID: 19227740.
- 20 Guillén-Pino F, Morera-Fumero A, Henry-Benítez M, Alonso-Lasheras E, Abreu-González P, Medina-Arana V. Descriptive study of diving injuries in the Canary Islands from 2008 to 2017. *Diving Hyperb Med*. 2019;49:204–8. doi: 10.28920/dhm49.3.204-208. PMID: 31523795. PMCID: PMC6884094.
- 21 Juhl CS, Hedetoft M, Bidstrup D, Jansen EC, Hyldegaard O. Decompression illness treated in Denmark 1999–2013. *Diving Hyperb Med*. 2016;46:87–91. PMID: 27334996. [cited 2023 Dec 17]. Available from: https://www.dhmjournal.com/images/IndividArticles/46June/Juhl_dhm46.2.87-91.pdf.
- 22 Denoble P, Caruso J, Nelson C, Chimiak J, Moore J, Tillmans F. Diving fatalities. In: Tillmans F, editor. DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents. Durham, NC: Divers Alert Network; 2020. p. 7–24. [cited 2023 Dec 17]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK582510/>.
- 23 Mitchell SJ, Bove AA. Medical screening of recreational divers for cardiovascular disease: consensus discussion at the Divers Alert Network fatality workshop. *Undersea Hyperb Med*. 2001;38:289–96. PMID: 21877558.
- 24 Lippmann J, Taylor D McD, Stevenson C, Williams J, Mitchell SJ. Diving with pre-existing medical conditions. *Diving Hyperb Med*. 2017;47:180–90. doi: 10.28920/dhm47.3.180-190. PMID: 28868599. PMCID: PMC6159622.
- 25 Monnot DPM, Boisvert J, Buteau D, Pollock NW. Retrospective review of enquiries to the Québec diving medicine call centre: 2004 through 2018. *Diving Hyperb Med*. 2021;51:152–60. doi: 10.28920/dhm51.2.152-160. PMID: 34157730. PMCID: PMC8426130.
- 26 Blatteau J-E, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, Cochard G et al. Prognostic factors of spinal cord decompression sickness in recreational diving: retrospective

- and multicentric analysis of 279 cases. *Neurocrit Care*. 2011;15:120–7. doi: [10.1007/s12028-010-9370-1](https://doi.org/10.1007/s12028-010-9370-1). PMID: [20734244](https://pubmed.ncbi.nlm.nih.gov/20734244/).
- 27 Inman AL, Sorrell LP, Lagina AT. Decompression sickness responsive to delayed treatment with hyperbaric oxygen: a case report of two divers. *Undersea Hyperb Med*. 2020;47:551–4. doi: [10.22462/10.12.2020.3](https://doi.org/10.22462/10.12.2020.3). PMID: [33227830](https://pubmed.ncbi.nlm.nih.gov/33227830/).
- 28 Sokolowski SA, Räisänen-Sokolowski AK, Tuominen LJ, Lundell RV. Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcomes. *Diving Hyperb Med*. 2022;52:271–6. doi: [10.28920/dhm52.4.271-276](https://doi.org/10.28920/dhm52.4.271-276). PMID: [36525684](https://pubmed.ncbi.nlm.nih.gov/36525684/). PMCID: [PMC10026386](https://pubmed.ncbi.nlm.nih.gov/PMC10026386/).

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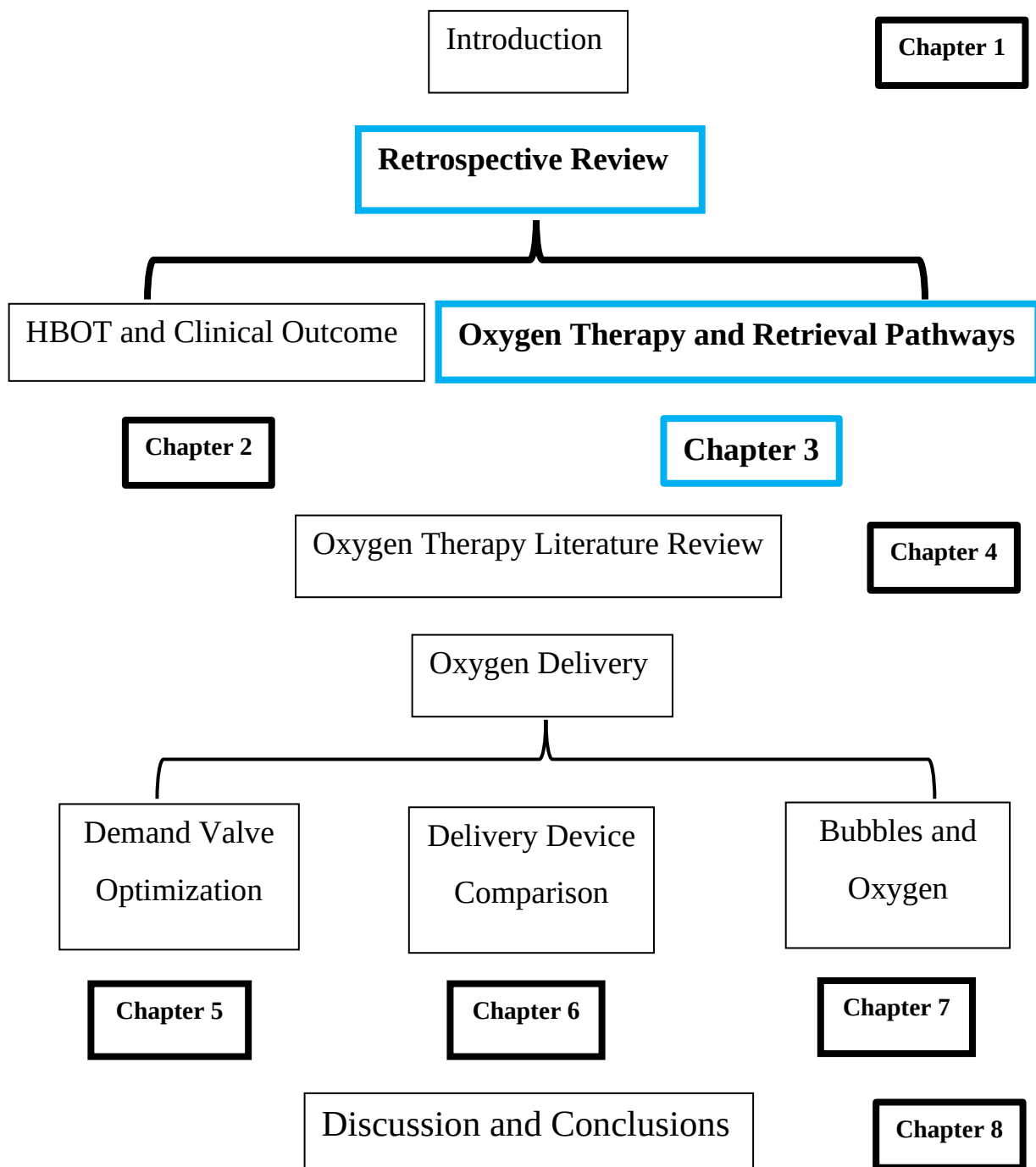
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CHAPTER 3 - OXYGEN TREATMENT AND RETRIEVAL PATHWAYS OF DIVERS WITH DIVING-RELATED CONDITIONS IN TOWNSVILLE, AUSTRALIA: A 15-YEAR RETROSPECTIVE REVIEW

This chapter is an exact copy of the published manuscript. This chapter is a second retrospective review paper. Additional important information is included in this retrospective study that enabled analysis of the retrieval pathways and pre-HBOT oxygen treatment of the injured divers identified in the previous chapter.



Original articles

Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review

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Keywords

Arterial gas embolism; Decompression sickness; First aid; Oxygen toxicity; Retrieval platform; Scuba diving; Treatment

Abstract

(Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review. *Diving and Hyperbaric Medicine*. 2025 30 June;55(2):79–90. doi: [10.28920/dhm55.2.79-90](https://doi.org/10.28920/dhm55.2.79-90). PMID: [40544136](https://pubmed.ncbi.nlm.nih.gov/40544136/).)

Introduction: First aid for injured divers includes oxygen delivery prior to definitive care. Delay to specialist assessment and/or hyperbaric oxygen treatment (HBOT) may be due to dive site remoteness and limited access to facilities. Townsville has the only hyperbaric facility along the Great Barrier Reef. Analysis of oxygen therapy and retrieval pathways of divers treated in Townsville may assist with establishing future education strategies and resource allocation.

Methods: Data were retrospectively collected on divers assessed at the Townsville hyperbaric medicine unit from November 2003 through December 2018. Demographics, dive incident location, oxygen treatment, retrieval platform and pathway, and initial disease grade were reviewed. Data are presented as frequencies and percentages.

Results: A total of 306 cases were included (184 males). Divers typically received oxygen therapy (87%, 267/305 known) prior to specialist review. The non-rebreather mask was the most frequently used (44%, 28/63) followed by in-water recompression (24%, 15/63). While 34% of the divers were retrieved from the scene ($n = 104$), only 11 (11%, 11/104) were retrieved directly to Townsville. Most divers initially classified as severe were retrieved from the scene (82%, 27/33), only two directly to Townsville. Fifteen cases had three retrieval legs (5%, 15/306).

Conclusions: Most injured divers received oxygen first aid and were transported to Townsville for definitive care with a variable number of retrieval stages. Continuing education of retrieval physicians should address knowledge of diving related injuries and highlight cases that may benefit from expedited transfer.

Introduction

The Great Barrier Reef (GBR) is the world's most extensive coral reef ecosystem, extending from the northern tip of Queensland, Australia to just north of Bundaberg. As one of the best-known reef systems in the world, over two million visits are made to the reef each year.¹ Midway along the coastline parallel to the reef system is located the city of Townsville.

The Townsville University Hospital houses the only hyperbaric facility along the GBR, providing specialist advice and recompression/hyperbaric oxygen treatment (HBOT) for divers with decompression sickness (DCS) or arterial gas embolism (AGE) (collectively referred to as decompression illness [DCI]) on the GBR and neighbouring Pacific Islands. The next nearest hyperbaric facility is located 1,400 km south of Townsville in Brisbane. For the

purpose of this study, the term ‘injured divers’ refers to those with suspected DCI or another malady after a dive such as immersion pulmonary oedema thought to require review by a diving specialist. A centralised Queensland retrieval service assists with the transport of injured divers to definitive treatment, with the northern zone co-ordination centre located in Townsville. Rotary and fixed wing assets are located along the Queensland coast, including a jet in Townsville for international and long-haul retrievals (Figure 1). Referrals to the coordination centre may come directly from dive boats (skippers), the Queensland Ambulance Service (supervisors), health care facilities (nurses or doctors) or diving physicians (emergency hotlines or hyperbaric facilities). It is the job of the clinical coordinator to provide medical advice and determine the urgency, retrieval platform, and appropriate destination for injured divers.

First aid treatment for divers may include oxygen delivery while obtaining specialist advice and preparing for evacuation.² In relevant scenarios, surface oxygen may reduce or resolve symptoms, and hasten recovery,³ and therefore should be initiated as soon as possible when symptoms develop, and maintained until definitive treatment can be delivered. Many dive sites are remote, requiring a variety of retrieval platforms and stages to be used in transferring injured divers to a facility capable of providing HBOT. Organising transport to a hyperbaric facility can be challenging and early advice from a diving physician can assist in appropriate patient selection, treatment options, level of urgency and destination decisions.

The aim of this retrospective review was to analyse oxygen therapy and the retrieval platform and pathways of injured divers presenting to the Townsville hyperbaric medicine unit. Mapping of the retrieval pathway will provide insight into the appropriateness of aircraft base locations and destination decisions. Analysis of pre-hospital care and retrieval pathways of these injured divers could assist with establishing future education strategies and resource allocation.

Methods

Ethics approval was granted from the Townsville Hospital and Health Service (LNR/2019/QTHS/51229) and James Cook University (H7767). This retrospective review includes all injured divers assessed and treated at the Townsville hyperbaric medicine unit (THMU) after a previous report,⁴ from 4 November 2003 through 31 December 2018. Cases were identified for inclusion by reviewing yearly HMU patient logs and electronic discharge summaries.

Retrieval Services Queensland databases (Queensland neonatal emergency transport service, clinical coordination retrieval information system, and Brolga [retrieval information system]) were searched using keywords and relevant diagnoses (cerebral arterial gas embolism,

Figure 1

Map of Queensland with northern coastal aeromedical retrieval bases and assets. The vehicle symbol (helicopter or plane) indicates the number of aeromedical assets at each base; Thursday Island to Townsville 1,000 km; Cairns to Townsville 350 km, approximately 60 min rotary flying time; Mackay to Townsville 389 km, approximately 60 min rotary flying time



decompression [including illness and sickness], drown*, snorkel*, and scuba), hyperbaric med, and offshore retrievals by rotary wing asset to identify cases. Identifying data (name, date of birth, and date of incident) were recorded so that cases could be correlated with THMU data, looking for any missed cases and ensuring no cases were duplicated.

Individual charts were reviewed, and data extracted to pre-formatted templates. The divers' ages in years and sex were recorded. Time of symptom onset was defined in two ways. Firstly, whether symptom onset occurred underwater during the dive or post-dive after arriving at the surface. Secondly, an actual time duration from arrival at the surface to symptom onset. Due to the unavailability of details on time to symptom onset underwater, the time of arrival at the surface was used as the starting time point for these cases. Initial disease grade was classified using a previously published scale (Table 1).⁴ Treatment details were recorded including time to oxygen commencement post-symptom onset, oxygen delivery method, and change of symptoms with oxygen. Oxygen therapy duration was calculated and documentation of any symptom(s) of oxygen toxicity collected.

Retrieval details were collected including platform (boat, rotary wing, fixed wing, or road), oxygen delivery, retrieval origin and destination, and type of retrieval (primary, secondary, or tertiary). Primary retrievals were classified as retrievals from the scene of the dive incident or other prehospital location.⁵ If a dive boat called for medical advice and was directed to return to shore, this was classified as a primary retrieval by boat. If a dive boat returned to shore without any urgency after completing their trip, this was not considered a retrieval. Secondary retrieval was defined as a transfer between health care facilities. This may be a second

Table 1

Initial disease severity grade using the established Townsville Hospital categories;⁴ mild and moderate symptoms are invariably decompression sickness (DCS) while arterial gas embolism events would be classified as severe. It is acknowledged that these classifications do not correspond exactly with the final definition of mild DCS arising from the workshop where Reference 4 was presented, but they were historically applied to the Townsville data and so are used here

Severity	Definition
Mild	Symptomatic with no objective signs except: Minor skin rash Lymphatic DCS Sharpened Romberg test < 30 seconds
Moderate	Symptomatic with subtle signs: Impaired higher function Impaired Romberg test Subjective sensory changes Minor weakness due to pain Cutis marmorata
Severe	Symptoms threatening life or mobility: Loss of consciousness Cardiopulmonary DCS Spinal DCS

retrieval leg after a primary retrieval or the transfer between two health care facilities after self-presentation. Tertiary retrieval was defined as a third retrieval leg transfer between health care facilities. Road retrievals included ambulance, bus, or car. Assessment by a health care professional and oxygen use prior to review in the THMU were collected. In relevant cases, time to start of HBOT following symptom onset was determined. All treatments were completed in a multi-place rectangular hyperbaric chamber (Fink Engineering Pty Inc., Warana, Queensland, Australia). Clinical outcome at the end of HBOT was classified as no residual symptoms, minor residual symptoms, or moderate/major residual symptoms.^{6,7}

Two researchers (DB and RT) performed the data extraction. Forms were compared and consensus reached. Individual Retrieval Services Queensland records were accessed to clarify retrieval information not apparent in the hospital medical records. All collected data were de-identified and entered into a pre-formatted Excel (Microsoft Office 365, Redmond, Washington, USA) worksheet.

ANALYSIS

Data are presented using frequencies and percentages for categorical variables, and medians and interquartile range [IQR] for continuous variables as all data were non-normally distributed as assessed using Shapiro-Wilk tests. The Mann-Whitney U test was used for analysis comparing

times for oxygen commencement between divers with symptom onset during versus symptom onset after the dive and for oxygen duration in divers with and without symptoms of oxygen toxicity. The Kruskal-Wallis test was used for analysis comparing times for oxygen commencement for divers treated at the scene by initial disease grade. Spearman rank-order correlations were computed to examine the relationship between time to symptom onset and time to oxygen commencement. Pairwise two-tailed z tests were used for *post hoc* analysis with Bonferroni correction. Significance was accepted as $P < 0.05$. The Statistical Package for the Social Sciences version 28.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) was used for analysis.

Retrieval data including mode of retrieval, and primary, secondary, or tertiary retrieval destinations were collected. Geospatial mapping of the retrieval pathway was completed by entering latitudes and longitudes or names of dive sites and retrieval destinations into Google Earth Pro (NOAA, 2015). Sites and destination placemarks were visually verified (zooming in) on the map and adjustments made to coordinates to ensure appropriate positioning. Retrieval destination appropriateness for divers with severe initial disease grade and primary retrievals was examined. Retrieval pathways of divers with three retrieval legs were evaluated for appropriateness. Initial disease severity, diagnosis, need for HBOT, and distance from Townsville were used to assess appropriateness of primary destination and staging. Identified cases were reviewed by two diving medicine and retrieval experts with extensive knowledge of the geographic area and capabilities of retrieval assets and facilities. As data were missing from some medical records, the *n* presented throughout the results denotes the number of records for which the information was documented.

Results

A total of 310 injured divers were identified during the study period with retrieval pathways as long as 3,200 km. Four divers were excluded as their paper medical records had been destroyed following national medical record guidelines, leaving 306 divers for the analysis. The median age of the divers was 29 [interquartile range 24, 35] years and 60% were male. Most were recreational divers (72%, 187/260) performing no-decompression diving. Other diver demographics and dive incident details for this cohort have been published elsewhere.⁷

Most symptoms started after the incident dive (90%, 275/306). Median time to symptom onset was 60 [10, 360] min in the divers whose symptoms commenced after surfacing and shorter for divers receiving oxygen at the scene (20 [5, 90] min), and for those primarily retrieved (15 [2, 90] min). Time to symptom onset was shorter in divers with severe initial disease grade (details previously published).⁷

Most of the injured divers received oxygen therapy (87%, 267/305) prior to assessment by a diving physician. Of the divers with a final diagnosis of DCI, 89% (245/274) received oxygen therapy. A high percentage of divers treated at the scene were given oxygen (Table 2). Other treatments included analgesia, antiemetics, and fluid administration. There was poor documentation of the type of oxygen delivery system used, but of those with documentation the non-rebreather mask was most often used with demand valve systems being less common. Most divers had partial or full relief of their symptoms with oxygen treatment (Table 2). In cases where the oxygen delivery method and change in symptoms were documented, symptom improvement was seen in 81% (35/43) of divers breathing surface oxygen and in 78% (7/9) divers receiving in-water recompression (IWR) on oxygen. Further evaluation comparing oxygen delivery device and change of symptoms could not be completed due to incomplete data.

The vast majority (14/15) of divers who received IWR were conducting occupational dives. The majority conducted their dives using a surface-supplied breathing system (9/15). Two were using open-circuit scuba and four had no documentation of the breathing system used. All IWR was done using surface-supplied systems (Table 2). Most divers undertaking IWR (9/15) were in remote locations 500 to 1,000 km from Townsville, three were diving 350 km from Townsville, one was diving 200 km from Townsville and two dive sites were unknown.

Of the divers who had symptom onset during the dive, most (71%, 22/31) received oxygen at the scene. Divers with symptom onset during the incident dive had significantly shorter times to oxygen commencement than divers with symptom onset post-dive (Table 3). Those who had severe initial disease grade and received oxygen at the scene had shorter times to oxygen onset than those with mild initial disease grade (Table 3). The Spearman rho showed a significant positive correlation between time of symptom onset and time of oxygen start ($r_s = 0.4$, $n = 225$, $P < 0.001$). Duration of oxygen delivery for the group of injured divers is presented in Table 3. Divers with symptom onset during dives did not have significantly longer oxygen treatment durations than those with symptom onset post-dive ($P = 0.85$). Divers with oxygen treatment started at the scene had significantly longer oxygen duration than those who did not have oxygen started at the scene (Table 3). Only 35% (46/132) of the injured divers received continuous oxygen.

The presence or absence of oxygen toxicity was infrequently documented. Of those cases with documentation ($n = 41$), 88% described oxygen toxicity. Most of the cases were mild pulmonary oxygen toxicity (92%) with only three cases of possible central nervous system (CNS) toxicity (one nausea, one lip tingling, and one metallic taste). Pulmonary oxygen toxicity was documented in seven cases while breathing normobaric oxygen (two during fixed wing retrievals), in 15 cases while during HBOT and in 11 cases the timing was

Table 2

Pre-hospital treatment of injured divers; ^atreatment included fluids, medications, and oxygen; ^b n = number of divers for which the information was documented; CPR – cardiopulmonary resuscitation; ROSC – return of spontaneous circulation

Parameter	<i>n</i> (%)
Treatment ^a at scene $n = 304^b$	155 (51)
Oxygen at scene $n = 155^b$	143 (92)
Other (CPR, ROSC then oxygen)	1 (< 1)
Initial disease grade mild $n = 216^b$	82 (38)
Initial disease grade moderate $n = 57^b$	37 (65)
Initial disease grade severe $n = 33^b$	24 (73)
Oxygen delivery method $n = 63^b$	
Non-rebreather mask	28 (44)
In-water recompression (IWR)	15 (24)
IWR oxygen	12 (19)
IWR air	3 (5)
Demand valve	13 (21)
Bag valve mask	4 (6)
Simple face mask	3 (5)
Change of symptoms with oxygen $n = 124^b$	
Partial relief	86 (69)
Full relief	15 (12)
No relief	17 (14)
Worse	3 (2)
Relapsing	2 (2)

undetermined. The three cases of possible central nervous system oxygen toxicity were all during HBOT. The cases of oxygen toxicity during HBOT were all given air breaks and treatments continued. Pre-HBOT oxygen delivery duration was significantly longer in the oxygen toxicity group (Table 3). Three divers with oxygen toxicity were international retrievals, one had received HBOT on a Pacific Island prior to the transfer to Townsville. The removal of the oxygen mask by the diver was not recognised as oxygen toxicity by the retrieval team and the diver was sedated to enforce the wearing of the mask. Only one diver who had received IWR on oxygen had possible CNS oxygen toxicity (nausea). Of the divers who received continuous oxygen ($n = 46$), six had documented symptoms of pulmonary oxygen toxicity and two had documentation of no symptoms of oxygen toxicity. The median duration of oxygen delivery in the group who had continuous oxygen and symptoms of oxygen toxicity was 7:20 [3:56, 10:29] h:min.

Of the injured divers primarily retrieved, the majority had oxygen delivered during the retrieval (Table 4). Of the divers who had a secondary retrieval, most had oxygen delivered during the aeromedical retrieval (rotary wing = 93%, 14/15, fixed wing = 91%, 84/92 [eight cases missing data]) (Table 5). The rotary wing secondary retrieval where the

Table 3

Time to and total duration of oxygen delivery pre-hyperbaric oxygen therapy post incident dive; * $P < 0.001$ Mann-Whitney U test; [†] $P = 0.006$ Mann-Whitney U test; [‡] $P = 0.017$ Kruskal-Wallis test

Parameter	Median [IQR] h:min
Time to oxygen start post-symptom onset, $n = 254$	4:00 [0:30, 24:27]
Time to oxygen start where symptom onset was post-dive, $n = 227$	5:00 [0:30, 26:30]*
Time to oxygen start where symptom onset was during dive, $n = 27$	00:15 [0:10, 2:30]*
Time to oxygen start post-symptom onset where treated at scene, $n = 132/143$	00:30 [0:10, 2:00]
Mild initial disease grade, $n = 74/82$	00:30 [0:15, 2:30] [‡]
Moderate initial disease grade, $n = 35/37$	00:20 [0:10, 1:58]
Severe initial disease grade, $n = 23/24$	00:10 [0:05, 00:30] [‡]
Duration of oxygen delivery, $n = 256$	10:00 [6:00, 16:14]
Duration of oxygen delivery where symptom onset was during dive $n = 27$	9:05 [7:30, 12:05]
Duration of oxygen delivery where oxygen was started at scene $n = 145$	11:25 [6:55, 18:11]*
Duration of oxygen delivery where oxygen was NOT started at scene $n = 109$	8:00 [4:07, 13:17]*
Duration of oxygen delivery in divers with no oxygen toxicity or no documentation of oxygen toxicity $n = 221$	9:03 [5:07, 15:35] [†]
Duration of oxygen delivery in divers with documentation of oxygen toxicity $n = 35$	14:16 [9:06, 18:05] [†]

Table 4

Primary retrieval details; QAS – Queensland ambulance service;
^a n = number of divers for which the information was documented;
^bWorld Health Organization definition

Parameter	n (%)
Primary retrieval, $n = 306$	104 (34)
Oxygen during primary retrieval $n = 81^a$	77 (95)
Primary retrieval initial disease grade $n = 104$	
Mild	42 (40)
Moderate	35 (34)
Severe	27 (26)
Primary retrieval platform $n = 104$	
Boat	52 (50)
Rotary wing	26 (25)
Road (QAS = 19, self = 2)	21 (20)
Fixed wing	5 (5)
Primary retrieval destination $n = 104$	
Cairns	37 (35)
Townsville	11 (10)
Torres Strait and Pacific Is ^b	10 (9)
Lizard Island	9 (9)
Cooktown	8 (8)
Alva Beach/Ayr	7 (7)
Whitsunday islands/Proserpine	7 (7)
Lockhart River	4 (4)
Mossman/Port Douglas	4 (4)
Gladstone	2 (2)
Mackay	1 (1)
Other	4 (4)

Table 5

Secondary retrieval details; QAS-Queensland ambulance service;
^a n = number of divers for which the information was documented;
^bWorld Health Organization definition

Parameter	n (%)
Secondary retrieval $n = 306$	236 (77)
Oxygen during secondary retrieval $n = 115^a$	111 (97)
Secondary retrieval platform $n = 235$	
Road (QAS = 20, bus = 38, self = 53)	127 (54)
Fixed wing	92 (39)
Rotary wing	15 (6)
Boat	1 (< 1)
Secondary retrieval destination $n = 236$	
Townsville	211 (89)
Cairns	19 (4)
Torres strait and Pacific islands ^b	8 (3)
Whitsunday islands/Proserpine	2 (1)
Alva beach/Ayr	1 (< 1)
Mackay	1 (< 1)
Mossman/Port Douglas	1 (< 1)
Other	3 (1)

Table 6

Tertiary retrieval details; ^a*n* = number of divers for which the information was documented

Parameter	<i>n</i> (%)
Tertiary retrieval <i>n</i> = 306	24 (8)
Oxygen during tertiary retrieval <i>n</i> = 20 ^a	18 (90)
Tertiary retrieval platform <i>n</i> = 24	
Fixed wing	18 (75)
Rotary wing	4 (17)
Road (bus = 2)	2 (8)
Tertiary retrieval destination <i>n</i> = 24	
Townsville	24 (100)

diver did not receive oxygen was completed using an Australian military helicopter. The pattern was similar for tertiary retrievals with 90% of the divers retrieved aeromedically receiving oxygen (Table 6).

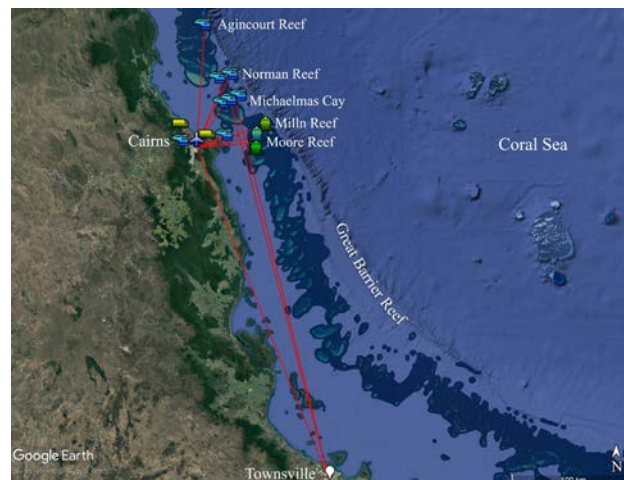
Most of the injured divers were assessed by a health care professional prior to arrival at the THMU (95%, 290/306). This included physicians diving on the dive boat, primary retrieval physicians, nurses in primary health care centres, general practitioners, and emergency physicians. Just under half (49%, 151/306) of the injured divers had at least two medical assessments prior to review by a hyperbaric physician, 14% (42/306) had three medical assessments and 2% (7/306) had four medical assessments. A high percentage of the divers received oxygen at these visits (78% [199/256], 86% [123/143], 80% [32/40] and 50% [3/6] respectively).

The most common primary retrieval destination was Cairns (Figure 1), and the most common retrieval platform was a boat (Table 4). The 'other' destination category consisted of different ports along the Queensland coast (Magnetic Island, Bowen and Gympie) as well as an international site (Indonesia) (Table 4). More than three-quarters of the injured divers required a secondary retrieval. Road transfer was the most common modality with Townsville the most frequent destination (Table 5). The 'other' destination category included Brisbane, Magnetic Island, and Julia Creek (Table 5). Only 24 injured divers had a tertiary retrieval, all to Townsville (Table 6).

The majority of the injured divers initially classified as severe (82%, 27/33) were retrieved from the scene (Table 4). The six divers not retrieved from the scene had a variety of reasons for not being primarily retrieved. Two had symptom onset during flights post diving, one went back to their accommodation and symptoms worsened, one presented to a general practitioner after returning from the dive trip, one self-transported to a hospital in Papua New Guinea, and for one diver there were two doctors on the dive boat who provided care while the boat steamed back to shore. Eighteen (18/27) of these severe cases were within a radius where they could have been primarily retrieved directly to Townsville. Only two were retrieved directly to

Figure 2

Retrieval pathways, assets, and destinations of injured divers with severe initial disease grade in the Cairns area. The vehicle symbol describes the asset used (helicopter, boat, ambulance, or plane), and the number of symbols represents the number of divers transported using that asset

**Figure 3**

Retrieval pathways, assets, and destinations of injured divers with severe initial disease grade in the Townsville area. The vehicle symbol describes the asset used (helicopter, boat, or ambulance), and the number of symbols represents the number of divers transported using that asset

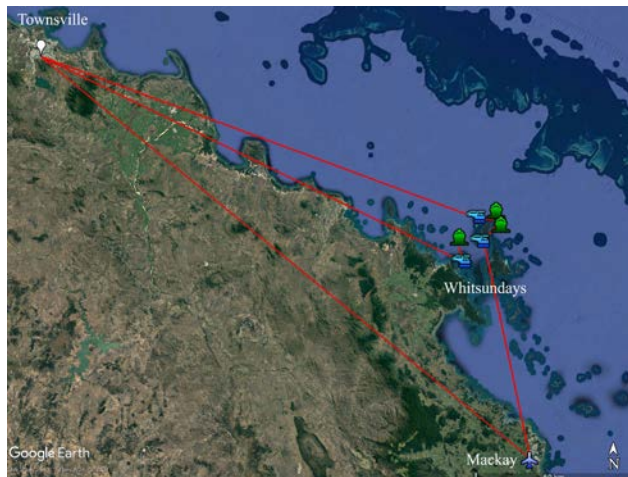


Townsville (Figure 2). Three went to Ayr hospital (nearest facility) initially and then were transferred to Townsville (Figure 3). Three in the Whitsunday area went by boat to a nearby island, one was then transferred to Mackay hospital (nearest facility) and then onto Townsville, and two were transferred directly to Townsville (Figure 4). Ten cases were primarily retrieved to Cairns and then transferred to Townsville, eight by fixed wing and two by rotary wing (Figure 2). Of these 18 severe cases, 14 had a final diagnosis of cerebral AGE, three immersion pulmonary oedema, and one central neurological DCS.

There were 11 primary retrievals direct to Townsville (Figure 5), three were international retrievals direct from

Figure 4

Retrieval pathways, assets, and destinations of injured divers with severe initial disease grade in the Whitsunday area. The vehicle symbol describes the asset used (helicopter, boat, or plane), and the number of symbols represents the number of divers transported using that asset

**Figure 5**

Retrieval pathways and assets of injured divers primarily retrieved to Townsville. The vehicle symbol describes the asset used (helicopter, boat, ambulance, or plane), and the number of symbols represents the number of divers transported using that asset



Papua New Guinea. The other local cases included seven divers with severe neurological symptoms and one diver with severe respiratory distress. Three of the divers did not receive HBOT. Their final diagnoses were cerebrovascular accident, migraine, and immersion pulmonary oedema. Three divers had a final diagnosis of cerebral AGE, one peripheral neurological DCS, and one vestibular DCS. Of the 93 additional primary retrievals, four of the divers had moderate to severe initial disease grades, were transported during daylight, and were within a radius where they could have been transferred directly to Townsville. All the other cases required an initial destination other than Townsville due to severity or distance, needing staging, stabilisation, or treatment at the closest medical facility. Time to start of HBOT was shorter for divers that were primarily retrieved (Table 7).

There were 15 cases with three retrieval legs (Figure 6). Four cases were international retrievals. Two of these cases were treated on a Pacific Island and then transferred to Townsville for further treatment. The 11 Australian cases were retrieved from the dive site by boat, then onward travel for medical review and then onto Townsville for HBOT. Six of these 11 cases were identified as inappropriately staged during the retrieval process. Three of these cases were cerebral AGE, one spinal DCS and two divers were doing decompression diving and had moderate symptoms of DCS.

Most divers had a good clinical outcome and no treated diver died.⁷ A higher percentage of divers with moderate (98%, 56/57) or severe (97%, 32/33) initial disease grade received oxygen therapy prior to HBOT than those with mild initial disease grade (83%, 179/216). Divers with moderate or severe initial disease more often had oxygen treatment commenced at the scene of the incident (Table 2). All divers with severe initial disease were retrieved ($n = 33$)

and more frequently primarily retrieved (82%, 27/33) than those with mild (19%, 42/216) or moderate (61%, 35/57) initial disease (Table 4).

Discussion

Townsville is strategically located on the east coast of Queensland, providing hyperbaric services to divers on the GBR and neighbouring Pacific Islands. Covering such a large area, there is often a necessity to transport injured divers a great distance to receive HBOT. Most divers in this study received oxygen treatment while awaiting transport and were appropriately staged or primarily transported to Townsville for definitive care.

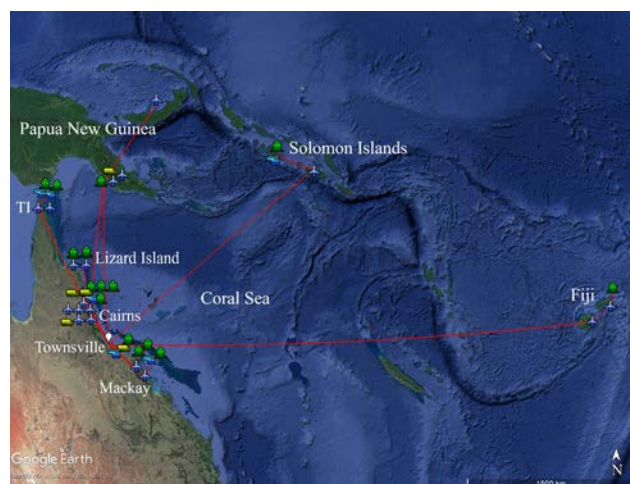
A large percentage of the injured divers treated in Townsville received oxygen either at the scene, during transport, or at a health care professional visit. Oxygen delivery has long been recommended as a first aid measure for injured divers.⁸ Divers Alert Network (DAN) created an oxygen first aid training program in 1991 focusing on the delivery of oxygen in the event of a diving emergency.⁹ This program was modified to meet Australian guidelines and introduced in Australia in 1994.¹⁰ Unfortunately, data from calls to DAN Asia-Pacific in 2018 showed only 24% of injured divers received oxygen first aid.¹¹ This low level of oxygen administration reported by DAN is consistent with other published international studies,^{12–17} but unexpected since our incidence of oxygen treatment in North Queensland was much higher. The higher incidence of oxygen delivery in our study may be due to Queensland workplace health and safety code of practice for oxygen use in recreational diving originally published in 1992 (Workplace health and safety Queensland, personal communication, 2023) requiring diving first aid qualifications for all dive masters and instructors and the availability of oxygen at all dive sites.

Table 7
Time to hyperbaric oxygen therapy (HBOT) from time of symptom onset

Parameter	Median [IQR] h:min
Time to HBOT for all injured divers <i>n</i> = 283	38:51 [22:11, 69:15]
Time to HBOT for all divers primarily retrieved <i>n</i> = 97	21:40 [10:30, 38:10]
Time to HBOT for divers NOT primarily retrieved to Townsville <i>n</i> = 89	21:11 [10:43, 35:24]
Time to HBOT for divers primarily retrieved to Townsville <i>n</i> = 8	27:05 [5:43, 48:45]

Figure 6

Retrieval pathways and assets of injured divers having three retrieval legs. The vehicle symbol describes the asset used (helicopter, boat, ambulance, or plane), and the number of symbols represents the number of divers transported using that asset; TI – Thursday Island



Although the incidence of oxygen treatment in our study was high, documentation of oxygen delivery device and flow rate was often missing. This appears to be consistent with other studies. One study examining the self-treatment of technical divers experiencing mild symptoms of DCI found a high percentage of divers hydrated orally (74%) and rested (70%) but only a small percentage breathed oxygen (21%) with no documentation of the delivery device used.¹⁸ One paper stated that no divers received oxygen prior to HBOT¹⁹ and one did not mention oxygen in their description of pre-hospital treatment, though they commented on fluids and acetylsalicylic acid administration.²⁰ One study stated that oxygen was “*routinely administered*”²¹ and two others that oxygen was administered during transport^{22,23} but details on delivery device and flow rate was not provided.²² One small study only mentioned in the limitations that most divers received oxygen, stating that there were no data available on the inspired fraction or duration of therapy.²⁴ In fact, only one of these studies commented on the type of oxygen delivery device used.²³ The current recommendation is to provide the highest possible concentration of oxygen.^{2,8}

Similar to previous reports,^{3,23} the non-rebreather mask was the most frequently used device in our cohort although the oxygen flow rate was infrequently documented. To provide a high level of oxygen to the tissues, a non-rebreather mask may require a flow rate of 15 L·min⁻¹.²⁵ However, a high flow rate will compromise the duration of available oxygen which is of relevance when treatment occurs at remote dive sites.

After the non-rebreather mask, IWR (breathing air or oxygen) was the second most frequently documented form of oxygen treatment at the scene. IWR breathing oxygen is an established, albeit not universally accepted, strategy for treating DCI,²⁶ especially at remote dive sites when access to a recompression chamber is not readily available.²⁷ This procedure is not without risks and requires special equipment and training, with a clear protocol in place prior to use.²⁸ Indeed, all the divers in our study who performed IWR used surface-supplied breathing systems and all but one had been performing occupational dives. The use of a surface-supplied breathing system suggests that the expertise, equipment, and processes were in place to perform IWR, however, some of the IWR procedures were conducted using air which is not recommended.²⁷ Despite this, the indication for performing IWR or the protocol used was not documented in the medical chart. Most of the dives were conducted in remote areas but four were conducted in areas reasonably close to Townsville (< 400 km). Given the limited details recorded, further comment on the use of IWR in these cases cannot be made.

Our cohort of injured divers received oxygen for a considerably longer time than previously reported data,³ but the administration was not necessarily continuous. Oxygen delivery duration is often not stipulated in recommendations.² When reported, the recommended durations are often vaguely written and vary from several hours,²⁹ even if relief of symptoms, to until arrival at a recompression facility.^{30,31} Other recommendations include giving oxygen for four to five hours without breaks depending on the time to the chamber,³² or giving five min air breaks every 30 min if time to recompression is likely to be greater than four hours.³³ A more concrete recommendation is the administration of as close to 100% oxygen as possible for 12 hours with 15 min breaks every four hours.³⁴ This recommendation is based

on the risk of pulmonary oxygen toxicity as an estimate of the maximum amount of time oxygen can be safely breathed rather than the dose of oxygen an injured diver requires.

Oxygen toxicity is a risk for injured divers breathing oxygen. Though poorly documented, 36 divers in our study appeared to demonstrate some level of oxygen toxicity, all but three being pulmonary toxicity, and all with long exposures. Divers with oxygen toxicity had a median oxygen duration (Table 3) greater than the recommended 12 hours.^{34,35} Only a small number of the divers in this work received continuous oxygen delivery, with the majority suffering from oxygen toxicity not having had continuous oxygen. Pulmonary oxygen toxicity is a concern from a patient comfort perspective, reversible in its early stages and extremely unlikely to cause harm in this group of patients. If symptoms of pulmonary oxygen toxicity occur, discussing the benefits and risks of ongoing oxygen therapy with an experienced diving physician is warranted. Future guidelines on pre-hospital management of DCI should provide recommendations on the duration of oxygen delivery and address the possible occurrence of pulmonary oxygen toxicity especially during long retrievals or onward transfer after receiving HBOT at another facility.

Air breaks in oxygen breathing during treatment at the scene or during retrievals were for logistical reasons rather than for scheduled air breaks or limited oxygen supplies. Air breaks (15 min off oxygen every four hours) are commonly recommended for injured divers receiving oxygen overnight awaiting HBOT, though this was infrequently documented. Air breaks have been recommended for divers receiving oxygen for longer than 12 hours.³⁴ This recommendation is thought to reduce the risk of pulmonary oxygen toxicity as well as giving the diver a break from wearing a mask, allowing them to eat and drink. It is interesting that the research supporting the recommendation of only providing oxygen for 12 hours also showed that a 15 min air break every four hours did not decrease the risk of pulmonary oxygen toxicity even though it did decrease its severity.³⁵ In our experience of long evacuations, clinically significant pulmonary oxygen toxicity seems extremely rare and it is not clear whether potential compromise of DCI treatment by imposing air breaks is justified, or at what time point in a long evacuation this should occur. Further research would be necessary to establish answers to these questions which seem relevant to only a small minority of cases.

Aeromedical assets are frequently used to transfer injured divers as dive sites can be remote and hyperbaric centres few and far between.²⁰ In our study, rotary wing assets were more often used for the primary versus other retrieval legs and for at least one retrieval leg in 55% (18/33) of divers classified as having severe initial disease. Other studies reported 16%³⁶ and 39%⁶ of divers retrieved by helicopter. Comparable with our results, asset choice can be for geographical considerations¹⁴ or for severity.⁶ Helicopters often provide the most timely and efficient means of transfer¹⁴ and may be

chosen independent of illness severity.³⁷ Routes chosen for helicopter retrievals were at the lowest altitude possible. It has been suggested that the vibration generated from rotary aircraft may worsen symptoms of DCS, but there is no published research to support this premise.³⁸ Even though in our study rotary wing assets were more often used for the primary retrieval leg than for other retrieval legs, due to the location of the dive sites, boat retrieval predominated as the choice for primary retrieval platform.

Logistic difficulties often result in a variety of assets being tasked to transport injured divers to definitive care.^{14,39} Previous examination of divers with mild or moderate DCI treated at our facility found that divers transported by surface transport, without oxygen or fluids, had a similar outcome following HBOT to those retrieved using an aeromedical asset.⁴ Road retrieval predominated for the secondary retrieval leg in the current study, as it has been the continued practice to have divers with mild symptoms travel to Townsville either by bus or private vehicle. This leaves aeromedical assets available for the transfer of acutely injured patients. Most of these divers had been assessed by a healthcare practitioner ($n = 88/91$, 97%) and discussed with the hyperbaric medicine physician on-call prior to the decision of suitability for road transfer.

The THMU provides care and support to injured divers along the Queensland coast from Rockhampton north to the Torres Strait Islands as well for some Pacific Islands. Providing coverage for this large area often leads to the need for staging of injured divers at various sites during their retrieval to Townsville. Retrieval destination and asset choice are determined at the time of referral based on the diver's clinical condition, location, asset and staff availability, time of day, weather conditions, and competing tasks. Perhaps not surprisingly, some of our divers required three retrieval legs using different assets. The majority of the current cases that required three retrieval legs were due to these geographical constraints. However, four divers likely should have come to Townsville on their second retrieval leg and two remained at their secondary destination overnight and likely should have been transferred earlier. Similar studies described one asset per retrieval^{6,14,36} or only focused on one type of retrieval asset.²³

Despite the multiple factors playing a role in retrieval decisions, few cases were identified as being inappropriately transported. Of the severe cases, one diver with immersion pulmonary oedema did not require further transfer to Townsville, and one diver with a final diagnosis of cerebral AGE should have primarily gone to Townsville. Four primarily retrieved cases were identified that could have been directly retrieved to Townsville. As described above, six cases with three retrieval legs were inappropriately staged. Decisions on the timeliness of retrieval to Townsville for assessment and possible HBOT is often jointly made by the hyperbaric physician and the clinical coordinator. Documentation of this decision-making process could allow

for future analysis and the development of a clinical decision support tool for determining the urgency of the retrieval and the appropriate destination.³⁷

LIMITATIONS

This study was retrospective and limited by incomplete records and missing data. Poor documentation of type of oxygen mask used, gas flow rates, starting and stopping times, air breaks, and change in symptoms limited the ability to assess the efficacy of these devices. Documentation of the referral to the THMU and the decision-making process about the retrieval was poor. There was no documentation on tasking deliberations or resource availability. With this decision-making information unavailable, determination of the appropriateness of destination and retrieval timeliness was decided based on the available medical and retrieval notes.

RECOMMENDATIONS

Improved documentation by all persons involved in the care of injured divers, from those providing first aid to diving physicians, in the area of oxygen therapy would allow for a more comprehensive assessment of the effectiveness of the treatment, problems with delivery, and side effects. A prospective study would improve acquisition of this data. Information to be collected could include: type of oxygen delivery system used, gas flow rate, time oxygen started and stopped, IWR protocols used, reasons for stopping, air breaks, change in symptoms of DCI, or any symptoms of oxygen toxicity. Oxygen therapy data should be collected for the whole patient journey, starting from on scene care through completion of treatment.

Despite this need for further research, pre-hospital care and transport of injured divers is well coordinated in Queensland. However, it is important for first responders to obtain early expert advice from an experienced diving physician to assist with decisions regarding on site treatment, retrieval urgency and platform selection as well as appropriate destination. Not all injured divers require recompression, such as divers with immersion pulmonary oedema or divers with mild DCS as recently defined,²⁸ and can be managed at local facilities with advice from diving experts. Early consultation with experts in the field will ensure that injured divers receive timely treatment at the most appropriate facility reserving aeromedical assets for those needing urgent transport.

Conclusions

A high proportion of injured divers received oxygen treatment while awaiting transport and were appropriately staged or primarily transported to Townsville for definitive care. In-water recompression was used in several cases of occupational diving. Pulmonary oxygen toxicity is a concern when providing extended multi-hour durations of oxygen therapy. The use of many different retrieval assets and legs

may be necessary when dive sites are remote and distances to a hyperbaric facility are vast. Continuing education for retrieval physicians should address knowledge of diving related injuries and highlight cases that may benefit from expedited transfer. Improved documentation by all carers of injured divers may enhance the ability to understand the impact of oxygen therapy on divers' outcomes.

References

- 1 Australian government: Great Barrier Reef Marine Park Authority. Tourism visitation data. Townsville (AU): GBRMPA; 2023 [updated 2024 Sep 22; cited 2025 Feb 20]. Available from: <https://www2.gbrmpa.gov.au/help/tourism-visitation-data>.
- 2 Mitchell SJ. Decompression illness: a comprehensive overview. *Diving Hyperb Med*. 2024;54(1 Supp):1–53. doi: [10.28920/dhm54.1.supp.1-53](https://doi.org/10.28920/dhm54.1.supp.1-53). PMID: [38537300](https://pubmed.ncbi.nlm.nih.gov/38537300/). PMCID: [PMC11168797](https://pubmed.ncbi.nlm.nih.gov/PMC11168797/).
- 3 Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiburger JJ. First aid normobaric oxygen for the treatment of recreational divers. *Undersea Hyperb Med*. 2007;34:43–9. PMID: [17393938](https://pubmed.ncbi.nlm.nih.gov/17393938/). [cited 2024 November 21]. Available from: https://www.researchgate.net/publication/51377358_First_aid_normobaric_oxygen_for_the_treatment_of_recreational_diving_injuries.
- 4 Griffiths D, Webb R. Ground transportation of diving injuries to Townsville. In: Mitchell SJ, Doolette DJ, Wachholz CJ, Vann RD, editors. Management of mild or marginal decompression illness in remote locations workshop proceedings; May 24–5, 2004; Sydney, Australia. Durham, NC: Divers Alert Network; 2005. p. 100–10. [cited 2024 Nov 21]. Available from: <https://world.dan.org/wp-content/uploads/2021/06/remotewrkshpfinal05-1.pdf>.
- 5 Muyambi K, Gardiner F, Sollid S, Hyldmo PK, Yisma E, Spring B, et al. Aeromedical retrieval services characteristics globally: a scoping review. *Scand J Trauma Resusc Emerg Med*. 2022;30:71. doi: [10.1186/s13049-022-01053-x](https://doi.org/10.1186/s13049-022-01053-x). PMID: [36510297](https://pubmed.ncbi.nlm.nih.gov/36510297/). PMCID: [PMC9743498](https://pubmed.ncbi.nlm.nih.gov/PMC9743498/).
- 6 Bennett M. The retrieval of diving injuries in New South Wales a retrospective review of two years of practice. *SPUMS Journal*. 1995;25:142–7. [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/25Sept/Bennett_SPUMSJ.25.3.142-147.pdf.
- 7 Blake DB, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. *Diving Hyperb Med*. 2024;54:308–19. doi: [10.28920/dhm54.4.308-319](https://doi.org/10.28920/dhm54.4.308-319). PMID: [39675739](https://pubmed.ncbi.nlm.nih.gov/39675739/). PMCID: [PMC12018695](https://pubmed.ncbi.nlm.nih.gov/PMC12018695/).
- 8 Lippmann J. First aid oxygen administration for divers. *SPUMS Journal*. 2003;33:192–8. [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/33Dec/Lippmann_dhm.33.4.192-198.pdf.
- 9 Gunderson J. Appendix B. Training and education. In: Tillmans F, editor. DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents. Durham, NC: Divers Alert Network; 2020. p. 81–4. [cited 2024 Nov 21]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK582510/>.
- 10 Lippmann J. DAN S.E. Asia-Pacific oxygen provider programs. *SPUMS Journal*. 1998;28:107–10. [cited 2024 Nov 21]. Available from: <https://www.dhmjournal.com/images/>

- [IndividArticles/28June/Lippmann_SPUMSJ.28.2.107-110.pdf](#).
- 11 Lippmann J. Diving accidents in Australia. In: Tillmans F, editor. DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents. Durham, NC: Divers Alert Network; 2020. p. 49–50. [cited 2024 Nov 21]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK582510/>.
 - 12 Sokolowski SA, Räisänen-Sokolowski AK, Tuominen LJ, Lundell RV. Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcomes. *Diving Hyperb Med*. 2022;52:271–6. doi: [10.28920/dhm52.4.271-276](#). PMID: [36525684](#). PMCID: [PMC10026386](#).
 - 13 Lundell RV, Arola O, Suvilehto J, Kuokkanen J, Valtonen M, Räisänen-Sokolowski AK. Decompression illness (DCI) in Finland 1999–2018: Special emphasis on technical diving. *Diving Hyperb Med*. 2019;49:259–65. doi: [10.28920/dhm49.4.259-265](#). PMID: [31828744](#). PMCID: [PMC7039777](#).
 - 14 Guillén-Pino F, Morera-Fumero A, Henry-Benítez M, Alonso-Lasheras E, Abreu-González P, Medina-Arana V. Descriptive study of diving injuries in the Canary Islands from 2008 to 2017. *Diving Hyperb Med*. 2019;49:204–8. doi: [10.28920/dhm49.3.204-208](#). PMID: [31523795](#).
 - 15 Bessereau J, Genotelle N, Brun P-M, Aboab J, Antona M, Chenaitia H, et al. Decompression sickness in urban divers in France. *Int Marit Health*. 2012;63:170–3. PMID: [23129100](#). [cited 2024 Nov 21]. Available from: https://journals.viamedica.pl/international_maritime_health/article/view/26137/20931.
 - 16 Haas RM, Hannam JA, Sames C, Schmidt R, Tyson A, Francombe M, et al. Decompression illness in divers treated in Auckland, New Zealand, 1996–2012. *Diving Hyperb Med*. 2014;44:20–5. PMID: [24687481](#). [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/44March/Haas_dhm.44.1.20-25.pdf.
 - 17 Chevasutho P, Premmaneesakul H, Sujiratanana A. Descriptive study of decompression illness in a hyperbaric medicine centre in Bangkok, Thailand from 2015 to 2021. *Diving Hyperb Med*. 2022;52:277–80. doi: [10.28920/dhm52.4.277-280](#). PMID: [36525685](#). PMCID: [PMC10017199](#).
 - 18 Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment. *Diving Hyperb Med*. 2022;52:78–84. doi: [10.28920/dhm52.2.74-84](#). PMID: [35732278](#). PMCID: [PMC9527095](#).
 - 19 Hadanny A, Fishlev G, Bechor Y, Bergan J, Friedman M, Maliar A, et al. Delayed recompression for decompression sickness: retrospective analysis. *PLoS One*. 2015;10(4):e0124919. doi: [10.1371/journal.pone.0124919](#). PMID: [25906396](#). PMCID: [PMC4408070](#).
 - 20 Thaler J, Pignel R, Magnan M-A, Pellegrini M, Louge P. Decompression illness treated at the Geneva hyperbaric facility 2010–2016: a retrospective analysis of local cases. *Diving Hyperb Med*. 2020;50:370–6. doi: [10.28920/dhm50.4.370-376](#). PMID: [33325018](#). PMCID: [PMC8038901](#).
 - 21 Blatteau, J-E, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: Influence of time to recompression. *Diving Hyperb Med*. 2011;41:129–34. PMID: [21948497](#). [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/41Sept/Blatteau_dhm.41.3.129-134.pdf.
 - 22 Gempp E, Blatteau J-E. Risk factors and treatment outcomes in scuba divers with spinal cord decompression sickness. *J Crit Care*. 2010;25:236–42. doi: [10.1016/j.jcrc.2009.05.011](#). PMID: [19682840](#).
 - 23 Oode Y, Yanagawa Y, Omori K, Osaka H, Ishikawa K, Tanaka H. Analysis of patients with decompression illness transported via physician-staffed emergency helicopters. *J Emerg Trauma Shock*. 2015;8:26–9. doi: [10.4103/0974-2700.145396](#). PMID: [25709249](#). PMCID: [PMC4335152](#).
 - 24 Mutzbauer TS, Staps E. How delay to recompression influences treatment and outcome in recreational divers with mild to moderate neurological decompression sickness in a remote setting. *Diving Hyperb Med*. 2013;43:42–5. PMID: [23508662](#). [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/43March/Mutzbauer_dhm.43.1.42-45.pdf.
 - 25 Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Leggat PA, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates. *Diving Hyperb Med*. 2020;50:34–42. doi: [10.28920/dhm50.1.34-42](#). PMID: [32187616](#). PMCID: [PMC7276271](#).
 - 26 Toklu AS, Cimsit M, Yildiz S, Uzun G, Korpınar S, Sezer H, Aktas S. Decompression sickness cases treated with recompression therapy between 1963 and 1998 in Turkey: review of 179 cases. *Undersea Hyperb Med*. 2014;41(3):217–21. PMID: [24984316](#). [cited 2024 November 21]. Available from: <https://www.uhms.org/uhm-search/uhm-journal-volume-41/number-3/decompression-sickness-cases-treated-with-recompression-therapy-between-1963-and-1998-in-turkey-review-of-179-cases.html>.
 - 27 Doolette DJ, Mitchell SJ. In water recompression. *Diving Hyperb Med*. 2018;48:84–95. doi: [10.28920/dhm48.2.84-95](#). PMID: [29888380](#). PMCID: [PMC6156824](#).
 - 28 Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med*. 2018;48:45–55. doi: [10.28920/dhm48.1.45-55](#). PMID: [29557102](#). PMCID: [PMC6467826](#).
 - 29 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377:153–64. doi: [10.1016/S0140-6739\(10\)61085-9](#). PMID: [21215883](#).
 - 30 Moon RE, Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Med*. 1997;68:234–43. PMID: [9056035](#).
 - 31 Müller PHJ, Beuster W, Hühn W, Knessl P, Roggenback HJ, Warninghoff V, et al. Diving accident guidelines of the German society for diving and hyperbaric medicine: summary version. *Diving Hyperb Med*. 2008;38:212–17. [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/38Dec/Muller_dhm.38.4.212-217.pdf.
 - 32 Acott C. Ten years of treating divers. *SPUMS Journal*. 2000;30:27–31. [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/IndividArticles/30March/Acott_SPUMSJ.30.1.27-31.pdf.
 - 33 Mitchell S, Gorman D. Diving accidents. *General Practitioner*. 1993, p. 8–9.
 - 34 Moon R. Could normobaric oxygen be accepted as a definitive treatment? In: Denoble PJ, Bird N, Chomiak J, editors. Treatment of decompression illness in recreational diving: Differences in current treatment practices and possible reconciliation workshop proceedings; June 26, 2019; Rio Grande, Puerto Rico. Durham, NC: Divers Alert Network; 2020. p. 34–44. [cited 2024 Nov 21]. Available from: <https://world.dan.org/wp-content/uploads/2021/08/2019-pre-course-proceedings-web.pdf>.

- 35 Comroe JH, Dripps RD, Dumke PR, Deming M. Oxygen toxicity: The effect of inhalation of high concentrations of oxygen for twenty-four hours on normal men at sea level and at a simulated altitude of 18,000 feet. *JAMA*. 1945;128:710–17. doi: [10.1001/jama.1945.02860270012004](https://doi.org/10.1001/jama.1945.02860270012004).
- 36 Zeindler PR, Freiburger JJ. Triage and emergency evacuation of recreational divers: a case series analysis. *Undersea Hyperb Med*. 2010;37:133–9. PMID: [20462146](https://pubmed.ncbi.nlm.nih.gov/20462146/). [cited 2024 Nov 21]. Available from: <https://www.uhms.org/uhm-search/uhm-journal-volume-37/number-2-mar-apr-2010/triage-and-emergency-evacuation-of-recreational-divers-a-case-series-analysis.html>.
- 37 Andre S, Lehot H, Morin J, Louge P, de Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. *Emerg Med J*. 2022;Feb 7:emermed-2021-211227. doi: [10.1136/emermed-2021-211227](https://doi.org/10.1136/emermed-2021-211227). PMID: [35135892](https://pubmed.ncbi.nlm.nih.gov/35135892/).
- 38 Blake DF, Crowe M, Mitchell SJ, Aitken P, Pollock NW. Vibration and bubbles: a systematic review of the effects of helicopter retrieval on injured divers. *Diving Hyperb Med*. 2018;48:235–40. doi: [10.28920/dhm48.4.241-251](https://doi.org/10.28920/dhm48.4.241-251). PMID: [30517957](https://pubmed.ncbi.nlm.nih.gov/30517957/). PMCID: [PMC6355309](https://pubmed.ncbi.nlm.nih.gov/PMC6355309/).
- 39 Bennett M. Evacuation methods in diving injuries. *SPUMS Journal*. 1998;28(3Suppl):10–14. [cited 2024 Nov 21]. Available from: https://www.dhmjournal.com/images/28Suppl/Bennett_SPUMSJ_28_3Suppl.10-14.pdf.

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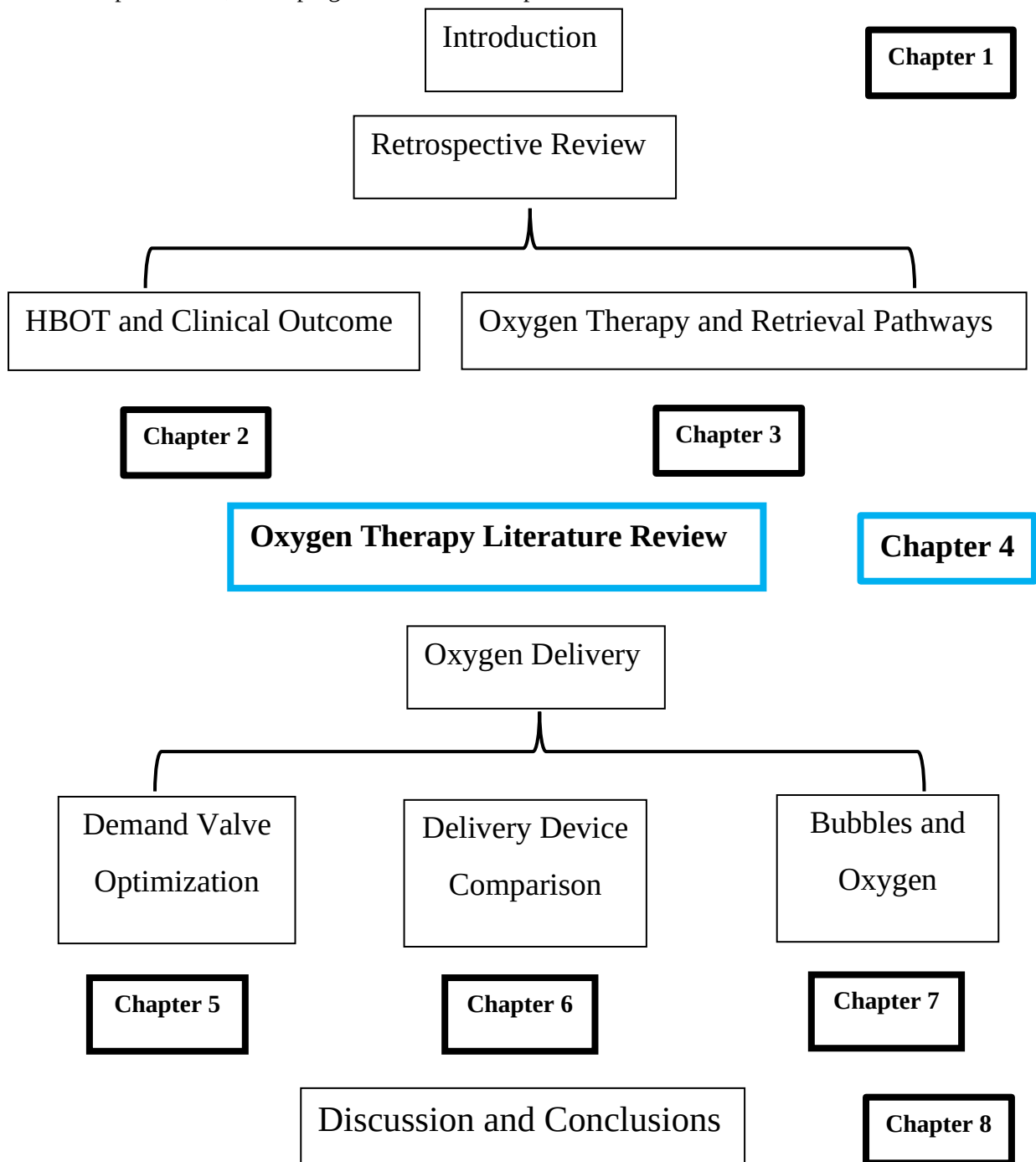
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CHAPTER 4 - OXYGEN THERAPY: A SCOPING REVIEW OF THE EFFICACY OF NORMOBARIC OXYGEN USE IN SCUBA DIVERS WITH DECOMPRESSION ILLNESS

The data presented in the previous two chapters showed a high percentage of pre-hyperbaric oxygen treatment, however, there is a lack of research evaluating the efficacy of pre-hyperbaric oxygen for injured divers. Therefore, this chapter is a scoping review exploring the peer-reviewed published literature surrounding normobaric oxygen treatment for injured scuba divers. The focus was on venous gas emboli reduction, evidence of symptom resolution and improved clinical outcome to inform the following data chapters investigating oxygen delivery device performance. It is written in a format for publication, in keeping with the other chapters.



Oxygen therapy: a scoping review of the efficacy of normobaric oxygen use in scuba divers with decompression illness

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Abstract

Introduction: High concentration normobaric oxygen is recommended as first line treatment for injured scuba divers with suspected decompression illness (DCI). The purpose of this scoping review was to examine the literature surrounding normobaric oxygen treatment of injured scuba divers to find any evidence that oxygen contributes to venous gas emboli reduction, symptom resolution or improved clinical outcome prior to hyperbaric oxygen treatment (HBOT).

Method: A systematic literature search of key databases following preferred reporting items for systematic reviews and meta-analysis extension for scoping reviews (PRISMA-ScR) guidelines was conducted to identify articles investigating normobaric oxygen use in injured scuba divers. Titles, abstracts and full articles were screened independently by two researchers for inclusion. Relevant study characteristics and data were extracted and summarized.

Results: Eight studies were identified for inclusion in the review. Three animal studies provided evidence of clinical improvement and bubble reduction breathing oxygen versus air. One prospective study found decreased bubbles in divers breathing normobaric oxygen after a decompression dive compared to breathing air. One retrospective study found a decrease in HBOTs for divers who received first aid oxygen. One retrospective study found a low level of normobaric oxygen use and no significant effect on final outcome. Two other retrospective studies found a high use of normobaric oxygen with little or no change in symptoms during retrieval and no significant effect on final outcome in severely injured divers.

Conclusion: There is a paucity of well conducted peer-reviewed articles examining normobaric oxygen treatment and clinical outcome in scuba divers with suspected DCI. Animal and human studies provide some evidence supporting a reduction in bubbles with oxygen breathing. Retrospective reviews found that breathing oxygen led to a decrease in the number HBOTs required but no significant improvement in final outcome for severely injured divers. This limited evidence does point to benefits for delivering high concentrations of normobaric oxygen prior to HBOT but more well controlled research is needed to confirm these initial findings.

Keywords: Bubbles; Decompression sickness; First aid; Review article; Venous gas embolism (VGE)

Introduction

Scuba diving allows air-breathing humans to enter and appreciate the underwater world. Although diving is a relatively safe sport, 483 diving-related fatalities were reported in Australia from 1970 to 2018, 116 in Queensland.¹ Although death is relatively rare from diving, many divers are injured each year requiring treatment in a hyperbaric facility. In 2018, 112 divers were treated for decompression illness (DCI) in Australia, 34 in Queensland (Hyperbaric Technicians and Nurses Association, unpublished data).

DCI is a term encompassing the clinical consequences of two different pathophysiological processes: introduction of bubbles to the arterial circulation by pulmonary barotrauma (referred to as arterial gas embolism [AGE]); and formation of bubbles from inert gas (referred to as decompression sickness [DCS]). Both involve bubbles as presumed primary vectors of injury. There is potential difficulty in clinical distinction between them, and the modern trend in therapy is to treat both in the same manner.² These considerations provided the motivation for referring to them collectively as DCI.³

DCS is a pressure related problem caused by previously dissolved inert gas coming out of solution as ambient pressure is reduced leading to bubble formation. DCS has also been called caissons disease due to its preponderance during mining and the building of bridges. Historical treatments for DCS have included alcohol massages and ingestion, elevation of the legs and wearing of bimetallic or galvanic bands.⁴ Repressurization in the caissons was found to alleviate symptoms with the use of normobaric oxygen suggested for treatment in the late 1800s.⁵ However, it was only in 1991 that the Divers Alert Network (DAN) created an oxygen first aid training program focusing on the delivery of oxygen in the event of a diving emergency.⁶ Recompression is currently considered the definitive treatment for DCS and hyperbaric chamber treatment the gold standard.² Access to recompression may be delayed due to the remoteness of the dive site and limited access to hyperbaric chambers. First aid treatment for divers includes surface oxygen while preparing for transport to a recompression chamber. Oxygen may reduce or resolve symptoms, and therefore should be initiated at the dive site, especially in remote locations where long delays to recompression may occur.

Normobaric oxygen therapy is thought to assist with the removal of inert gas located in the body tissues and bubbles and reduce inflammation caused by these bubbles. It has been hypothesized that oxygen may decrease bubble generation, evolution or distribution in an injured diver potentially improving their condition.^{2,7} Current guidelines⁸ have been written based on expert opinion and to our knowledge there is no systematic review or other strong evidence that could guide the clinician regarding the use of normobaric oxygen therapy in injured scuba divers. Therefore, the purpose of this scoping review was to systematically examine the literature surrounding normobaric oxygen treatment

of injured divers or in animal models to find any evidence that oxygen contributes to venous gas emboli (VGE) reduction, symptom resolution or improved clinical outcome and identify knowledge gaps to aid in the planning of future research.

Methods

A scoping review was deemed appropriate because of the diverse body of literature on this topic and the need to identify the types of research available. A five-step framework was used to guide the scoping review⁹ which included identifying the research question, identifying relevant studies, selecting studies, charting the data and collating, summarizing and reporting the data.

1. IDENTIFYING THE RESEARCH QUESTION

This scoping review aimed to answer the following question formulated using the PCC (participant, concept, context) framework⁹:

“What is the published literature surrounding VGE reduction or clinical outcome in injured scuba divers or animal models after treatment with normobaric oxygen prior to hyperbaric oxygen therapy?”

2. IDENTIFYING RELEVANT STUDIES

A systematic search of the literature was conducted on October 10, 2024 to identify articles investigating normobaric oxygen therapy use in injured scuba divers. The review was conducted following the guidelines of the preferred reporting items for systematic reviews and meta-analyses extension for scoping reviews (PRISMA-ScR).¹⁰ Databases searched included MEDLINE, CINAHL, Informit, Embase, Scopus, Web of Science, and Cochrane Central with no date limits (i.e., from database inception to October 2024). No language restrictions were applied. The search strategy was developed with the assistance of a senior medical research librarian. Medical subject headings (MeSH) and key words were used as search terms in three groups: diving* OR scuba OR "self-contained underwater breathing apparatus" AND bends OR "caisson disease" OR "decompression sickness" OR "decompression illness"; bends OR "caisson disease" OR "decompression sickness" OR "decompression illness" AND oxygen* OR "oxygen inhalational therapy"; "caisson disease" OR "decompression sickness" OR "decompression illness" AND "emergency therapy" OR "emergency treatment" OR "first aid". Results of the search were exported to a reference managing database (Endnote™ 21.4, Clarivate, Philadelphia, PA, USA) and then into Covidence (Melbourne, Australia) for the screening process. PRISMA-ScR guidelines were followed for the study selection process (Figure 4.1).¹⁰ The protocol for this review was not registered.

3. STUDY SELECTION

Titles and abstracts were screened, and full text of potentially relevant articles were obtained for review. To be included in the review, articles needed to report on normobaric oxygen treatment of injured divers or animal models to find any evidence that oxygen contributes to VGE reduction, symptom resolution or improved clinical outcome prior to hyperbaric oxygen treatment (HBOT). Studies reporting intravascular bubble counts were included since such measurements are commonly used for DCS risk assessment.¹¹ Exclusion criteria for each stage of the review are listed in Figure 4.1. Exclusion in the initial screening included articles pertaining to animals that dive, hyperbaric oxygen, pre-dive oxygen, oxygen decompression, and in water recompression on oxygen. Full text review exclusions included articles with no data and only expert opinion, consensus statements, historical review of oxygen therapy, conference abstracts and non-peer reviewed conference proceedings, non-peer reviewed reports and no normobaric oxygen data or analysis. Backward citation searching was performed by reviewing the reference lists of identified articles for additional relevant articles. Forward citation searching was also performed using PubMed. Available non-English language (German, Italian, Japanese, Korean, and Spanish) abstracts and relevant articles were translated. Articles were reviewed at each PRISMA-ScR stage by two reviewers (DFB and NWP) with consensus reached via face-to-face discussions.

4. CHARTING THE DATA

Data from eligible studies were extracted including author, year, country, methods, research design, sample size, results and authors conclusions. Charting was done independently and confirmed by a second investigator.

5. COLLATING, SUMMARIZING AND REPORTING

Study results are summarized in table form with relevant information described including a section on limitations of the studies. (Table 4.1). A narrative synthesis of the results was undertaken to address the research question.

Results

The combined searches initially identified 2309 records with 1467 records remaining after duplicates were removed (Figure 4.1). After initial screening, 184 full text articles were assessed for eligibility. Limited scientific literature was available on this topic, and only eight articles met the broadened inclusion criteria. Table 4.1 contains a summary of the eight relevant articles. Study characteristics including participants, study design, methods, study results, author conclusions and study limitations are presented. No prospective studies examining the use of normobaric oxygen and clinical outcome were identified. Three animal articles provided some evidence of bubble resolution with oxygen versus air breathing. One study examined VGE and oxygen use in healthy divers. Four articles presented clinical outcome data. The articles included in this review have been categorized into three groups for comparison: animal studies, VGE and oxygen delivery, and oxygen (and fluids) use and clinical outcome.

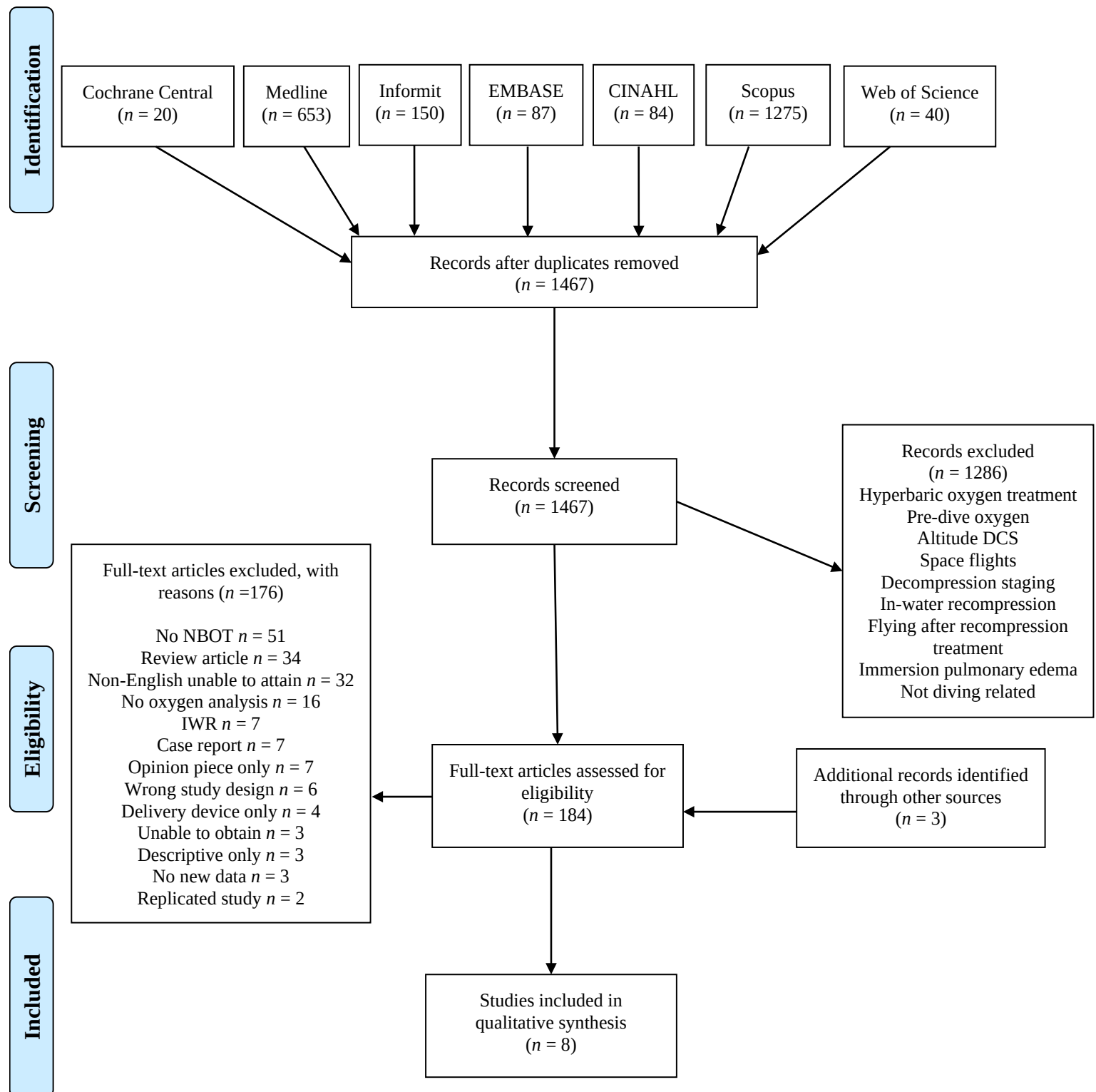
ANIMAL STUDIES

Three experimental studies on animals provided evidence of clinical improvement and bubble reduction breathing normobaric oxygen versus air.

The first animal study¹² compressed anesthetized rats to 3.27 bar (327 kPa) for 4 h with a three-stage decompression over 20 minutes (min). The animals were placed under a microscope and had their abdomens opened to expose adipose tissue. Once a bubble was identified, the animals breathed air, heliox (80:20) or oxygen. The field was photographed at intervals and bubble volume was calculated. During air breathing bubbles continued to grow over the 2-to-3-h observation period. During heliox breathing, bubbles diminished in size. During oxygen breathing, bubbles initially grew or remained stable then diminished at a considerable rate. The authors concluded that heliox and oxygen ultimately caused bubbles to shrink in size, with the rate of bubble size change (shrink or grow) probably dependent on the local tissue vascularization.

The focus of this study¹² was the use of heliox as a breathing gas to decrease the size of nitrogen bubbles, though both an air and oxygen breathing arm were included. The group allocation process or the photography time intervals were not described. Ten animals died but their allocated group details were not reported. Heliox is not used as a first aid breathing gas for injured divers but rather as a breathing gas during recompression.

Figure 4.1 Prisma flow diagram; NBOT-normobaric oxygen; IWR-in-water recompression; HBOT-hyperbaric oxygen therapy; DCS-decompression sickness



The second animal study¹³ compressed anesthetized golden hamsters to 7 ATA (709 kPa) for 30 min and then returned them to 1 ATA (101 kPa) over 3 min. Animals were removed from the chamber and placed on a microscope stage to directly view bubbles in the femoral vessels. The animals blood pressure and heart rate were recorded every 5 min for 35 min. Animals were randomly assigned to 3 groups: no treatment, 100% oxygen via a tracheostomy and 1.5 ml saline intravenously (IV) or 100% oxygen and 1.5 mL perfluorocarbon IV. Survival was defined as breathing on their own at 30 min. A postmortem was performed on all animals. The control group animals had unstable vital signs, massive levels of venous then arterial bubbles and 15/16 died before 30 min. The oxygen and saline group had venous emboli and some arterial bubbles. The surviving animals (10/16) in this group had no arterial bubbles seen at 35 min post decompression. The oxygen and perfluorocarbon group animals had venous emboli, some arterial bubbles which resolved by 30 min. Only one animal in this group died. Postmortem exam showed that the control animals had massive levels of bubbles in the body compared to other two groups. Animals that died before 35 min had more bubbles than the surviving animals. The authors concluded that oxygen and perfluorocarbon therapy improves hemodynamic status and decreases bubbles in acute DCS.

The focus of this study¹³ was the use of perfluorocarbon; therefore, there was not an oxygen alone arm but rather oxygen and saline IV. Even though this was animal research, the study provided some evidence that oxygen (and fluids) decrease bubble generation after a decompression dive. The bubble grading technique was not described.

This third animal study¹⁴ assessed bubble generation in healthy goats after compression in a hyperbaric chamber. Phase one compressed 40 animals to 2.2 bar (220 kPa) for 24 h and then returned them to the surface at a rate of 1 bar·min⁻¹ (100 kPa·min⁻¹). Animals were monitored for 8 h and if presented signs of DCI, they were randomly allocated to oxygen or air breathing. Breathing of the assigned gas continued until DCI sign resolution or 3 h plus 1 h to a maximum of 4 h with a 10 min air break every hour. Audio Doppler was performed at 2-, 5- and 15-min post surfacing then every 15 min until 2 h, then hourly until 8 h post surfacing or until bubbles ceased. Twenty-three animals showed signs of DCI, 12 allocated to the oxygen group and 11 allocated to the air group. The time to symptom onset was significantly shorter in the oxygen group (38 vs 88 min). Just under half (5/12) of the goats in the oxygen group had complete persistent resolution of their signs of DCI. The time to sign resolution was 157 (SD 53) min. No animal had complete persistent resolution of signs in the air group (0/11). There was no significant difference in VGE grades between the DCI and no DCI groups. VGE decreased to zero by 8 h in the animals with no DCI and those treated with oxygen. The VGE grades fell earlier in the oxygen versus air group with a statistically significant difference at 90 min post-exposure.

Table 4.1 Characteristics of studies included in the scoping review using the PCC (participant, concept, context) framework⁹; IV-intravenous; DCI-decompression illness; VGE-venous gas emboli; NRB-non-rebreather mask; IWR-in-water recompression; DCS-decompression sickness; AGE-arterial gas embolism; DAN-Divers Alert Network; FAO₂-first aid oxygen; HBOT-hyperbaric oxygen treatment; CAGE-cerebral arterial gas embolism

Authors (year of publication)	Participants	Study design	Methods	Results
Animal studies				
Hyldegaard et al (1989) ¹²	56 female rats 250-359 g	Non-random allocation to breathing air, heliox (80:20) or oxygen	Animal anaesthetized and compressed to 327 kPa breathing air for 4 h, 3 stage decompression over 20 min. Abdominal adipose exposed under a microscope to view bubbles. During gas breathing, fields were photographed and bubble volume calculated.	All animals developed bubbles. 10 animals died. With air breathing the bubbles continued to grow during the observation period. Bubbles continually diminished in size when heliox was breathed. During oxygen breathing, the bubbles initially grew or remained stable for a period then decreased at a considerable rate.
Lynch et al (1989) ¹³	48 female hamsters 80 to 100 g	Randomized allocation to no treatment, 100% oxygen and 1.5 ml saline IV or 100% oxygen and 1.5 ml perfluorocarbon IV	Animal anaesthetized and compressed to 7 ATA for 30 min then returned to 1 ATA over 3 min. Vital signs were monitored, and bubbles were viewed in the femoral vein. Surviving animals (after 35 min) were killed for a detailed postmortem examination.	Control group-unstable vital signs, massive levels of venous and then arterial bubbles, high rate of death (15/16). Oxygen and saline-bubbles in femoral vein and eventually in artery; only 6/16 died. Oxygen and perfluorocarbon-bubbles in femoral vein but only in artery of 10 animals; only 1/16 death. Postmortem-control animals had massive levels of bubbles in the body compared to other groups; animals that died before 35 min had more bubbles than surviving animals.
Loveman et al (2015) ¹⁴	Healthy female or male castrated goats 57 ± 10 kg (range 41-81 kg) Phase 1 - 40 animals Phase 2 – 39 animals	Randomized allocation to air or oxygen breathing if signs of DCI. Phase 1 saturation dive. Phase 2 simulated submarine escape.	Phase 1 - Animal compressed to 2.2 bar for 24 h then returned to surface at rate of 1 bar·min ⁻¹ . Animals were monitored for 8 h, those with signs of DCI were allocated to air or oxygen breathing until resolution of signs or 3 hours via a plastic hood with latex neck seal; 10 min air break every h. Breathed oxygen for 1 h post sign resolution (max 4 h). Audio Doppler was performed before compression and initially at 2-, 5- and 15-min post compression and then every 15 min for 2, then hourly for 8 h or until bubbles ceased. Phase 2 – Above compression profile followed by a pressurization to 21 bar in 28 s with a 4 s hold, followed by a linear decompression to the surface at 0.275 bar·s ⁻¹ . Group allocations,	Phase 1 - 23 animals showed signs of DCI; 12 allocated to oxygen and 11 allocated to air. Time to sign onset was significantly shorter in the oxygen group (38 vs 88 min). 5/12 in the oxygen group had complete persistent resolution of signs. Time to resolution was 157 (53) min. No animal had complete persistent resolution of signs in the air group (0/11). There was no significant difference in VGE grades between the DCI and no DCI groups. VGE decreased to zero by 8 h in the animals with no DCI and those treated with oxygen. The VGE grades fell earlier in the oxygen versus air group with a statistically significant difference at 90 min post-exposure. Phase 2 – 19 animals showed signs of DCI with one case of pulmonary barotrauma. Three animals were euthanized and 16 were randomly allocated to the oxygen and air breathing groups. 6/8 in the oxygen group had complete resolution of

			breathing gas and Doppler assessment followed the above protocol.	their symptoms. 5/8 animals in the air group had resolution of their symptoms with one recurring overnight. Animals in the oxygen group had more severe initial symptoms. No significant difference in time to resolution between the 2 groups. There was no significant difference in VGE grades between the DCI and no DCI groups. VGE decreased to zero by 8 h in the animals with no DCI and those treated with oxygen. The VGE grades fell earlier in the oxygen versus air group with a statistically significant difference at 300 min post-exposure.
Bubbles and oxygen				
Blatteau et al (2009) ¹⁵	19 healthy military divers 34 (SD 7) y (range 23-48 y)	Randomized breathing order	Initial dive 30 msw for 30 min, 9-min stop at 3 msw, open circuit air, fin swimming. 10 min post surfacing randomly assigned to 30 min IWR, normobaric oxygen 15 L·min ⁻¹ , control. Supine position for normobaric oxygen and control. Bubbles measured at precordial site by blinded Doppler operator at 40-, 60- and 80-min post surfacing; at rest and after 2 leg movements. Graded bubbles using Spencer scale and converted into Kisman integrated severity score (KISS).	Raw data presented. Peak bubble grade at 60-min post surfacing for control dive and 40-min for IWR and normobaric oxygen dives. KISS significantly lower for IWR vs normobaric oxygen and control. KISS significantly less for normobaric oxygen vs control.
Oxygen treatment and outcome				
Bond et al (1990) ¹⁶	447 recreational injured divers; included DCS and AGE; 77% male, 35 ± 9.8 y	Retrospective review	2 years of DAN America injury database reviewed for HBOT treatment table use and oxygen administration prior to recompression.	302 divers with DCS only; 68 (23%) divers with DCS received FAO ₂ ; 51% complete relief if breathed oxygen, 56% complete relief no oxygen (OR 0.8 [0.48 – 1.41]). Significant improved outcomes in divers treated with regular tables.
Longphre et al (2007) ¹⁷	2231 recreational divers; included DCS and AGE; no diver demographics reported	Retrospective review	6 years of DAN America injury database reviewed for oxygen administration prior to recompression.	1045 (47%) divers received FAO ₂ ; 14% complete relief and 51% improved with FAO ₂ (<i>n</i> = 330/1045 with documentation). 157/417 (38%) used NRB. 33% who received FAO ₂ within 4 hours after surfacing required only 1 HBOT. No outcome difference at end of treatment.
Oode et al (2015) ¹⁸	28 patients with DCI (SANDHOG criteria ¹⁸) 71% male, 45 ± 2 y	Retrospective consecutive case series	4 years of transportation in physician-staffed helicopters. 34 identified; 6 excluded due to cardiopulmonary arrest at scene.	0 deterioration, 20 no change, 8 improved during flight. All received oxygen; all but 1 received IV fluids. All flights < 300 meters.
Andre et al (2022) ²⁰	195 divers; 42 female, 48 ± 12 y	Retrospective review	9-year review of divers with spinal cord DCS and time to HBOT < 24 h.	Median time to symptom onset 10 min; median time to recompression was 2 h and 44 min; prehospital oxygen, hydration, aspirin or evacuation platform were not associated with outcome. 88% of the divers received normobaric oxygen; 57% (13/23) recovery in the no oxygen group and 67% (115/172) recovery in the oxygen group (<i>P</i> = 0.45).

				Divers with higher initial disease severity had worse outcomes; time to HBOT of > 3 h increase in sequelae in this group.
Authors	Conclusions			Limitations
Animal studies				
Hyldegaard ¹²	Most bubbles eliminated in the same time period during oxygen and heliox breathing. Rate of change in bubble size (shrink or grow) probably dependent on surrounding tissue vascularity.			Animal study; no documentation of which group the deceased animals were in; no description of allocation of animals to intervention groups; imaging interval not defined. Heliox is not used as a first aid breathing gas.
Lynch ¹³	Oxygen and perfluorocarbon therapy improves hemodynamic status and decreases bubbles in acute DCS.			Animal study; decompression dive with no deco stop. Used oxygen and saline as treatment. No description of bubble grading.
Loveman ¹⁴	Oxygen is an effective first aid measure in the treatment of DCI. Still a high level of signs that remained after oxygen therapy or recurred. HBOT still gold standard in the treatment of DCI.			Animal study; saturation dive; no description of concealment or blinding of Doppler technician; no blinded independent review of Doppler signals, unsure if signal recorded; no description on duration of listening for bubbles; no movements used.
Bubbles and oxygen				
Blatteau ¹⁵	IWR breathing oxygen reduced venous bubbles and is more effective than normobaric oxygen.			All military divers; no data on sex of participants; no details on IWR procedure; no description of normobaric oxygen delivery device; no sample size calculation or power analysis; state all data presented as means (SD) and then use non-parametric tests; incorrect post hoc test used; minimal statistical analysis with table and figures not referred to in the text; older technique of bubbles detection only performed until 80 min post surfacing; no description of saving of the scan recordings or independent review; state peak bubbles at 40 min in 2 treatment arms but only started scanning at 40 min.
Oxygen treatment and clinical outcome				
Bond ¹⁶	No significant improvement in treatment outcome in divers who received FAO ₂ .			Retrospective review, initial data not designed for FAO ₂ analysis; no description of time to NBO, duration of oxygen or device used.
Longphre ¹⁷	FAO ₂ administration decreases the odds of more than 1 HBOT if oxygen commenced less than 4 h after surfacing.			Retrospective review, initial data base not designed for FAO ₂ analysis; used time of oxygen delivery from time of surfacing rather than symptom onset; only percentages presented in many cases so unable to reproduce analysis; no comparison group, those who did not get HBOT; no follow-up.
Oode ¹⁸	Subjective improvement via helicopter if <300 meters and administration of oxygen and fluids.			Unsure if any patients had CAGE; No details on patients requiring intubation (<i>n</i> = 4); oxygen saturation improvement clinically insignificant; no analysis between groups with change in symptoms; no data on HBOT or follow up.
Andre ²⁰	Initial disease severity and time to HBOT associated with presence of sequelae at time of discharge. Prehospital management items not associated with improvement therefore do not delay transport for recompression.			Title of article misleading, focus of the article is not prehospital management; no description of oxygen delivery devices, time to onset of oxygen treatment from time of symptom onset or duration of oxygen treatment; no assessment of change in symptoms “under oxygen” prior to HBOT; data on oxygen use and outcomes presented in a table but not discussed in the manuscript.

The second phase of the study compressed 39 animals using the compression profile stated above, followed by a simulated submarine escape. The simulated escape consisted of pressurization to 21 bar (2100 kPa) in 28 s with a 4 s hold, followed by a linear decompression to the surface at a rate of $0.275 \text{ bar}\cdot\text{s}^{-1}$ ($27.5 \text{ kPa}\cdot\text{s}^{-1}$). Group allocations, breathing gas and Doppler assessment followed the above protocol. Nineteen animals showed signs of DCI with one case of pulmonary barotrauma. Three animals had early severe symptoms requiring humane euthanasia. The remaining 16 animals were randomly assigned to the oxygen and air breathing groups. In the oxygen breathing group, 6/8 animals had complete resolution of their symptoms. In the air breathing group, 5/8 animals had resolution of symptoms with one recurring overnight. Animals in the oxygen group had more severe initial symptoms with animals in the air group having only limb symptoms. There was no significant difference in the time to resolution of symptoms between the two groups. There was no significant difference in VGE grades between the DCI and no DCI groups. VGE decreased to zero by 8 h in the animals with no DCI and those treated with oxygen. The mean VGE grade fell earlier in the oxygen breathing group with a statistically significant difference at 300 min post exposure.

The authors concluded that oxygen is an effective first aid measure in the treatment of DCI but since a high level of signs remained or recurred after oxygen therapy, recompression therapy with oxygen should still be the good standard for the treatment of DCI.

This was an animal study with a saturation dive.¹⁴ The main focus was to assess first aid treatment of submariners requiring rapid escape during an incident. Concealment or blinding of the Doppler technician or the duration of listening for bubbles was not described. There was no description of blinded independent review of Doppler signals nor indication if the Doppler signals were recorded. The term DCI was used throughout the paper to include possible AGE and DCS arising from the rapid ascents.

HUMAN STUDIES

Four human studies provided some evidence of bubble reduction breathing normobaric oxygen versus air, a decrease in the number of hyperbaric oxygen treatments (HBOT) required if normobaric oxygen was administered but no change in clinical outcome in injured divers who breathed normobaric oxygen prior to HBOT.

BUBBLES AND OXYGEN

This study¹⁵ examined cardiac bubbles using acoustic Doppler in military divers after a decompression dive. Nineteen divers performed three dives. Ten min post surfacing, divers were randomly assigned

to: IWR breathing oxygen, lying supine breathing normobaric oxygen or no oxygen breathing. Doppler was performed at 40-, 60- and 80-min post surfacing at rest and after two leg movements. Bubbles were graded using the Spencer scale and then converted into Kisman integrated severity score (KISS). Peak bubble grade was at 60-min post surfacing for the control dive and at 40-min for IWR and normobaric oxygen dives. KISS was significantly lower after IWR breathing oxygen versus breathing normobaric oxygen or after the control dive. KISS was significantly less after breathing normobaric oxygen versus after the control dive.

The participants in this study were all military divers, however, no data on sex was provided nor sample size calculation presented. The IWR procedure including oxygen delivery system or diver support system was not described. The oxygen mask used for the normobaric treatment arm was not described. The incorrect post hoc test was used; however, raw data was presented allowing recalculation. There was minimal statistical analysis with a table and figures not referenced in the text. This article was published in 2009 and therefore an older technique of bubble detection was performed.¹¹ Bubble assessment was continued until 80 min post surfacing rather than the recommended 120 min.¹¹ The saving of the scan recordings or independent review was not described. The stated peak bubbles were at 40 min in two treatment arms but scanning only started at 40 min and therefore the peak may have been earlier. There were no limitations mentioned.

OXYGEN TREATMENT AND CLINICAL OUTCOME

This third group of articles examined clinical outcome in divers treated with normobaric oxygen. They were all retrospective chart reviews. Normobaric oxygen treatment was not the focus of these studies, but they were still included in the review as data was presented that allowed for the evaluation of oxygen treatment and one article¹⁷ is frequently referenced in other articles. One study had a low level of normobaric oxygen use and no significant improvement in outcome. One study found a decrease in HBOTs for divers who received first aid oxygen. The other two studies found a high use of normobaric oxygen with little or no change in symptoms during retrieval and no significant effect on final outcome in severely injured divers.

One study retrospectively reviewed a two-year period of the DAN America injury database for initial HBOT treatment table, oxygen delivery prior to recompression and outcome.¹⁶ The authors examined all cases (DCS and AGE) for initial HBOT treatment table (regular vs enhanced) and possible interactions with other variables. Pre-recompression oxygen therapy and treatment outcome was analysed. A total 327 cases were included in the study. 302 divers had a diagnosis of DCS with 68 (23%) receiving FAO₂. Of those who breathed oxygen, 51% were symptom free at the end of

treatment and 56% had ongoing signs/symptoms after the last HBOT (OR 0.8 [0.48 – 1.41]). There were significant improved outcomes in divers treated with regular tables.

This article used data from the DAN database with 72 cases excluded due to incomplete records. There was no data on time to normobaric oxygen delivery, duration of treatment or delivery device used. There was a low level of normobaric oxygen use. The focus of this study was clinical outcome based on initial treatment table use. Tables were defined as regular tables (USN Table 5 or 6) or enhanced (USN Table 6A or Table 6 with extensions). There was no data on number of HBOT treatments each diver received.

One study retrospectively reviewed a six-year period of the DAN America injury database for data on oxygen delivery prior to recompression.¹⁷ The authors examined all cases (DCS and AGE) for use of first aid oxygen, timing and mode of administration, symptom resolution and number of hyperbaric oxygen treatments. A total of 2,231 cases were included with a total of 1,045 (47%) receiving first aid oxygen. The NRB mask was most commonly used (157/417, 38%) but flow rate was unknown. The median time to oxygen commencement was 2.2 h after symptom onset and 4 h after the end of the dive. Duration of oxygen delivery was poorly documented (229/1,045, 33%) but for those with documentation the mean and median duration was 132 and 93 min respectively. Sixty-five percent of those with documentation (330/1,045) had complete relief or improvement post oxygen treatment and prior to HBOT. More divers treated with oxygen (67%) had complete relief after the first HBOT than those that did not receive oxygen (58%), but this difference was not seen at discharge. Of the divers who required more than one HBOT, 45% had received oxygen and 50% had not (OR = 0.83, 0.70-0.98). Divers who received oxygen within 4 h of surfacing required less HBOT sessions (OR = 0.49, 0.36-0.69). Time from symptom onset to oxygen commencement was not predictive of number of HBOT sessions. First aid oxygen did not influence the number of HBOTs for more severe cases. Divers with higher severity more frequently received timely oxygen.

This article¹⁷ used data from the DAN database which was not designed to analyse the efficacy of oxygen therapy and therefore many data items were missing. Many items were not presented in the article but used in analysis (age, sex, time to symptom onset, case severity [PSI], number of HBOT sessions). The odds ratio of 0.83 for more than one HBOT session is rated as weak with the confidence interval almost reaching 1. Only 365 (35%) divers had documentation of time to oxygen delivery post surfacing. Percentages were only presented for time to oxygen greater than or less than four h of surfacing. Assuming this percentage was for those with documentation, this would be 120 divers receiving oxygen less than or equal to 4 h from surfacing and requiring more than one HBOT. Those receiving oxygen greater than 4 h from surfacing would be 135 divers requiring more than one HBOT. It would have been of interest to compare the group that received oxygen 4 h after surfacing

and the no oxygen group. However, no analysis was done comparing these two groups. Time to symptom onset was “most often within the first hour after the dive”, with times only presented by symptom severity. Time to oxygen delivery from time of surfacing was analysed without examining the severity and time to symptom onset as well. The authors state that NRB mask does not deliver 100% oxygen and recommend the use of the demand valve.

OXYGEN (AND FLUIDS) TREATMENT AND CLINICAL OUTCOME

The charts of 28 consecutive DCI patients retrieved via helicopter over a four-year period from Izu Peninsula, Japan were retrospectively reviewed.¹⁸ Six divers who had cardiopulmonary arrest at the scene were excluded. Diagnosis of DCI was made using the San Diego Diving and Hyperbaric Organizations (SANDHOG) criteria.¹⁹ Patient demographics and dive characteristics were collected. Patients were classified by type of DCS: central nervous system (CNS), chokes, choke/bends and choke/CNS. All patients underwent oxygen therapy with delivery device and flow reported. All participants were transported in the supine position and all, but one subject received IV fluids. All flights were below an altitude of 300 metres above sea level. Changes in vital signs, Glasgow coma scale (GCS) and change of subjective symptoms before and after the flights were compared. A statistically significant improvement in oxygen saturation pre- and post-flight was found. Four patients required intubation. No patient deteriorated enroute, 20 did not change status and eight improved. Time from request for transfer and arrival at the medical facility was recorded.

This article¹⁸ examined the outcomes of patients diagnosed with DCI in a physician staffed helicopter retrieval service. Inconsistent use of the terms DCS and DCI make it difficult to determine if patients who surfaced suddenly were cerebral arterial gas emboli (CAGE) or CNS DCS. Duration of oxygen therapy was stated to be similar in all patients with reference to a table that does not contain that data. The significant improvement stated for oxygen saturation was clinically insignificant. There is no detail about the timing or indication for intubation in four patients. Follow up data on hospital treatment or outcome would have provided further insight but was not reported. All divers received oxygen therapy so there was no ability to compare outcomes with divers who did not receive oxygen. No analysis of the differences between the patients in relation to change in subjective symptoms was performed.

A French retrospective study examined pre-hospital variables of divers with spinal cord DCS and outcome.²⁰ The charts of 195 divers with symptoms suggestive of spinal DCS and time to recompression less than 24 h were reviewed. Median time to symptom onset was 10 min (IQR and range not reported) and median time to recompression was 2 h and 44 min. No prehospital management items (oxygen, hydration, aspirin or evacuation platform) were associated with outcome.

Normobaric oxygen use was high with 88% of the divers receiving oxygen. There was 57% (13/23) recovery in the no oxygen group and 67% (115/172) recovery in the oxygen group. This was not statistically different ($P = 0.45$). Divers with higher initial disease severity had worse outcomes. Time to HBOT of > 3 h led to an increase in sequelae but only in the group with higher initial disease severity.

The title of the article²⁰ does not align with the study objectives and presented results. The title states that the study examined the influence of prehospital management on the outcome of divers while the objectives stated in the article and the focus of the analysis is the influence of initial disease severity, time to HBOT and outcome. There is no description of the oxygen delivery devices used, time to onset of oxygen treatment from time of symptom onset or duration of oxygen treatment which would have provided greater insight. Though stated in the methods, there was no assessment of change in symptoms “under oxygen” prior to HBOT. Data on oxygen use and outcomes were presented in a table but not discussed in the text.

Discussion

There were few articles examining the clinical outcome in divers breathing oxygen. Many articles were not peer reviewed and published only as conference abstracts or proceedings. Published retrospective review articles were often only descriptive, presenting the incidence of oxygen use with minimal information on delivery device or outcomes.²¹⁻²⁶ This scoping review confirms that there is a paucity of research examining outcomes in scuba divers with DCS treated with normobaric oxygen with only five human studies included in this review and three animal studies. The results from the reviewed studies are equivocal and further good quality research in this area is required.

It has been promoted that near 100% oxygen should be delivered early to injured divers to enhance elimination of inert gas, decrease gas bubbles and improve tissue oxygenation.^{2,7} The use of normobaric oxygen treatment for DCS was suggested by Bert in the late 1800s⁵ with some uptake of its use in the 1960s.²⁷ It took until 1991 for an instructional oxygen delivery program⁶ to be developed and the encouragement of its common use by divers. DAN previously published the incidence of normobaric oxygen use in their yearly data²⁸ but that has ceased to be a focus of their annual report.

Paul Bert’s animal experiments performed in the 1800s discovered that after decompression nitrogen gas was found in the heart.⁵ He proposed that nitrogen was “liberated” in the form of “little” bubbles collecting in the heart. He reasoned that since the atmosphere was largely nitrogen, breathing pure oxygen would aid with the diffusion of the nitrogen through the lungs to be eliminated. Bert performed decompressions on dogs from 7-8 atmospheres with all animals having severe symptoms

on arrival back at surface pressure. He had the animals breathe oxygen and found symptomatic improvement as well as decreased cardiac and jugular bubbles. The animals did not survive, and he therefore recommended that oxygen therapy should be commenced with the addition of recompression. This research was the impetus for the use of normobaric oxygen in the treatment of inert gas bubble related disorders.

Oxygen therapy for divers with symptoms of DCS decreases symptoms and limits further disease progress. Oxygen reduces inflammation^{29,30} and increases inert gas washout and contributes to bubble involution.² Even though these bubbles are considered to be the primary vector of injury leading to the symptoms of DCS, these inert gas bubbles do not always lead to symptoms of DCS. Rather, they are an indicator of the diver's decompression stress, and are used as a surrogate marker of DCS, especially in human studies where it is unethical to cause DCS in a healthy diver. Since Bert's research, further animal studies have used direct visualization of tissue bubbles to assess the effectiveness of oxygen therapy.^{12,13} In one animal study¹², bubbles were initially found to increase in size while the animal was breathing oxygen then decrease in size and number. Divers should be made aware that this phenomenon may cause symptoms to initially worsen while breathing oxygen and to persevere with its use. Another study allocated animals with clinical signs of DCS to air or oxygen breathing.¹⁴ Acoustic Doppler was used to assess for vascular bubbles. Animals that breathed oxygen had improvement in their signs of DCS and a decrease in their bubble loads. One human study used acoustic Doppler to assess bubbles in divers after a decompression dive followed by air or oxygen breathing.¹⁵ Bubble grades were found to be lower in the divers who breathed normobaric oxygen. The results of this study require cautious interpretation due to methodological problems and the use of an older technique for bubble detection. Technological advances and reporting guidelines have substantially changed since its publication.¹¹ It is worthwhile repeating this type of study using the newer techniques for bubble detection.

One study that is commonly cited evaluated the benefit of normobaric oxygen prior to HBOT using a data base not designed for this purpose.¹⁶ No demographic data was presented therefore there is an inability to generalize the results to other populations. The discussion starts highlighting the improvement seen in divers receiving first aid oxygen versus those who did not, when the data presented stated that the improvement seen did not persist to discharge. The authors stated that the natural history of mild DCI is recovery, then stated that oxygen use decreases the need for more than one HBOT without stratifying by disease severity. Outcomes were not stratified by oxygen delivery device, and there was no evidence that an optimized delivery system had been used. Another study stated that the use of first aid oxygen did not influence outcome.³¹ However, the type of oxygen delivery devices used, timing and duration of oxygen therapy were not presented in the article. Another study by this research group sent out questionnaires to technical divers to assess self-

treatment.³² Only 6/27 (22%) used first aid oxygen. No details on the outcome of these divers, type of oxygen delivery system used, timing and duration of oxygen therapy were presented. There is little evidence in these retrospective studies that normobaric oxygen therapy improves final outcome. One non-peer reviewed document found that most divers receiving pre-recompression oxygen had severe symptoms and that normobaric oxygen delivery may mitigate the negative effects of delay to recompression.³³ However, the very nature of retrospective reviews often means many data points are missing. Understanding which delivery devices provide the best oxygenation could improve treatment and lead to better outcomes for injured divers.

Other research examined the efficacy of oxygen delivery systems rather than focusing on outcomes of injured divers. One study compared oxygen demand delivery devices with different breathing interfaces using healthy volunteers.³⁴ The demand valve system is easy for divers to use as they are familiar with this type of breathing apparatus. The study compared four different demand valve oxygen delivery systems using both a face mask and mouthpiece. Nine healthy male volunteers breathed oxygen for three min from eight different configurations in random order. Nitrogen washout was seen within the first min. Use of the mouthpiece did not provide significantly better results. Oxygen delivery of 100% was not achieved all the time for any of the devices. One system had a considerable air leak with the face mask and mouthpiece. One system was characterized as having inspiratory vibrations. The conclusion was that none of the systems were perfect for use in an emergency.

Dehydration has been identified as a risk factor for DCS and therefore IV hydration is often instituted once available.² Oxygen and fluids are often administered simultaneously to an injured divers so it is difficult to tease out the benefits of oxygen therapy alone when performing retrospective studies.^{13,18,20} One of the animal studies included in this review assessed bubble grades in groups breathing oxygen and receiving either IV saline or IV perfluorocarbon.¹³ Since previous research showed that bubbles were initially seen to increase in size while breathing normobaric oxygen, it was proposed to instill perfluorocarbon to increase the removal of nitrogen and improve tissue oxygenation.¹³ This technique has not been adopted in the clinical treatment of divers with DCS due to side-effects and long organ retention time.³⁵

The consensus guideline for the initial management of DCI recommends oxygen administration, hydration and a horizontal position.⁸ The supine position is recommended for treating injured divers with first aid oxygen as this position improves inert gas washout.³⁶ This position is also recommended in divers with suspected AGE to prevent cephalad distribution of arterial bubbles.⁸ Research using oxygen delivery devices for divers with DCI should be performed with participants in the supine and not the seated position.

Both AGE and DCS are caused by bubble generation although their mechanisms are significantly different, and this limits the generalization of the AGE outcomes to divers with DCS. AGE can occur in circumstances of lower inert gas load than serious DCS meaning that patients may respond differently to normobaric oxygen treatment. Two articles in this retrospective review evaluating clinical outcome included divers with DCS and AGE^{17,18} and the third focused on divers with spinal DCS.²⁰ Patients with AGE and spinal DCS tend to be sicker and with symptom onset earlier than patients with milder forms of DCS. Earlier commencement of oxygen breathing and request for transport in these divers may be due to clinical urgency. Reported efficacy of normobaric oxygen therapy in retrospective studies could be confounded by the severity of illness unless addressed in the analysis.

FUTURE RESEARCH

Though the evidence is limited, these articles provide a framework for further research into this area. Further research to identify the optimum delivery devices to provide high concentrations of oxygen and the uptake of their use may lead to an improvement in outcome. A prospective study collecting data on the type of oxygen delivery system used, gas flow rate, time oxygen started and stopped, reasons for stopping, air breaks, or change in symptoms of DCI would greatly improve the understanding of the benefits of normobaric oxygen. Outcome should be stratified by initial disease acuity and final diagnosis to better understand the difference between DCS and AGE. Early oxygen commencement is suggested in the treatment of DCI but advice on duration of administration is often vague.^{2,8} Nitrogen washout has been shown to be within a minute of the start of oxygen therapy³⁴ but bubbles have been shown to initially increase in size with oxygen treatment.¹³ Bubbles have been shown to disappear in an animal model 100 min after oxygen breathing if started immediately after surfacing.³⁷ Technological advances have occurred allowing for better assessment of bubbles in divers and may assist with answering the question of the optimal normobaric oxygen treatment duration.

LIMITATIONS

There is very little specific literature on normobaric oxygen treatment and clinical outcome in injured divers, with many articles only describing pre-hyperbaric oxygen treatment without any formal outcome analysis. Three articles^{13,18,20} included treatment with normobaric oxygen and IV fluids, however, due to the limited number of identified articles these were still included in this review. The clinical outcome articles limited to retrospective reviews. Older investigative techniques were poorly described and therefore it was difficult to interpret the results.¹³⁻¹⁵ Articles were of varying languages, but translation allowed for their inclusion. Unfortunately, the Rubicon Foundation research repository

ceased to exist after 2021 and some military research documents and many conference proceedings are no longer available. Including non-peer reviewed manuscripts may have given additional results.

Conclusion

There is a paucity of well conducted peer-reviewed articles examining normobaric oxygen treatment and clinical outcome in scuba divers with suspected DCI. Understanding which delivery devices provide the best oxygenation could improve treatment and lead to better outcomes for injured divers. Evidence was provided supporting a reduction in bubbles with oxygen breathing and retrospective reviews found that breathing oxygen led to a decrease in hyperbaric treatments required but no significant improvement in final outcome. This limited evidence does point to benefits for delivering high concentrations of normobaric oxygen prior to HBOT but more well controlled research is needed to confirm these initial findings.

References

- 1 Lippmann J. A review of snorkelling and scuba diving fatalities in Queensland, Australia, 2000 to 2019. *Diving Hyperb Med.* 2022;52(2):108–18. doi: 10.28920/dhm52.2.108-118. PMID: 35732283.
- 2 Mitchell SJ. Decompression illness: a comprehensive review. *Diving Hyperb Med.* 2024;54(1 Supp):1-53. doi: 10.28920/dhm53.4.supp.1-53. PMID: 38537300.
- 3 Francis TJR. The classification of dysbarism. In: Francis TJR, Smith DJ, eds. *Describing decompression illness: Proceedings of the 42nd workshop of the Undersea and Hyperbaric Medical Society*. Bethesda, MD: Undersea and Hyperbaric Medical Society. 1991.
- 4 Acott C. A brief history of diving and decompression illness. *SPUMS J.* 1999;29(2):98-109. [cited 2025 March 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/29June/Acott_SPUMSJ.29.2.98-109.pdf
- 5 Bert P. Barometric pressure [*La pression barometrique*]. Translated by M A Hitchcock and F A Hitchcock College Book Company, Colombus, Ohio, 1943.
- 6 Gunderson J. Appendix B. Training and education; pp. 81-4. In: Tillmans F, ed. *DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents*. Durham, NC: Divers Alert Network; 2020. [cited 2024 November 21]. Available from:
<https://www.ncbi.nlm.nih.gov/books/NBK582510/>
- 7 Lippmann J. First aid oxygen administration for divers. *SPUMS J.* 2003;33(4):192-8. [cited 2024 November 21]. Available from:
https://www.dhmjournal.com/images/IndividArticles/33Dec/Lippmann_dhm.33.4.192-198.pdf
- 8 Mitchell SJ, Bennett MH, Bryson P, Butler F, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med.* 2018;48:45-55. doi: 10.28920/dhm48.1.45-55.
- 9 Arksey H, O'Malley L. Scoping studies: towards a methodological framework. *Int J Soc Res Methodol.* 2002;8(1):19-32. doi: 10.1080/1364557032000119616.

- 10 Tricco AC, Lillie E, Zarin W, O'Brien KK et al. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. *Ann Intern Med*. 2018;169(7):467-473. doi: 10.7326/M18-0850. PMID: 30178033.
- 11 Møllerløkken A, Blogg SL, Doolette DJ, Nishi RY, Pollock NW. Consensus guidelines for the use of ultrasound for diving research. *Diving Hyperb Med*. 2016;46(1):26-32. PMID: 27044459. [cited 2025 February 25]. Available from: https://www.dhmjournal.com/images/IndividArticles/46March/Mollerlokken_dhm.46.1.26-32.pdf.
- 12 Hyldegaard O, Madsen J. Influence of heliox, oxygen and N₂O-0₂ breathing on N₂ bubble in adipose tissue. *Undersea Biomed Res*. 1989;16(3):185-93. PMID: 2741253. [cited March 10 2025]. Available from <https://www.uhms.org/uhm-search/chapter-1-influence-of-heliox-oxygen-and-n2o-o2-breathing-on-n2-bubbles-in-adipose-tissue/viewdocument/2558.html>.
- 13 Lynch PR, Krasner LJ, Vinciguerra T, Shafer TH. Effects of intravenous perfluorocarbon and oxygen breathing on acute decompression sickness in the hamster. *Undersea Biomed Res*. 1989;16(4):275-81. PMID: 2773159. [cited 2025 February 25]. Available from: <https://www.uhms.org/uhm-search/chapter-2-effects-of-intravenous-perfluorocarbon-and-oxygen-breathing-on-acute-decompression-sickness-in-the/viewdocument/2575.html>.
- 14 Loveman GAM, Seddon FM, Jurdi KM, Thacker JC, Fisher AS. First aid oxygen treatment for decompression illness in the goat after simulated submarine escape. *Aerosp Med Hum Perform*. 2015;86(12):1020-7. doi: 10.3357/AMHP.4306.2015. PMID: 26630048.
- 15 Blatteau J-E, Pontier J-M. Effect of in-water recompression breathing oxygen to 6 msw versus normobaric oxygen breathing on bubble formation in divers. *Eur J Appl Physiol*. 2009;106(5):691-5. doi: 10.1007/s00421-009-1065-y. PMID: 19424716.
- 16 Bond JG, Moon RE, Morris DL. Initial table treatment of decompression sickness and arterial gas embolism. *Aviat Space Environ Med*. 1990;61(8):738-43. PMID: 2205196. [cited 2026 April 30]. Available from: <https://asma.kglmeridian.com/meridian/asma/published/rest/pdf-watermark/v1/journals/ase/61/8/article-p738.pdf/watermark-pdf/>
- 17 Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiburger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. *Undersea Hyperb Med*. 2007;34(1):43-9. PMID: 17393938. [cited 2025 February 10]. Available from: https://www.uhms.org/images/DCS-and-AGE-Journal-Watch/longphre_surface_o2_uhm_2007.pdf.

- 18 Oode Y, Yanagawa Y, Omori K, Osaka H, Ishikawa K, Tanaka H. Analysis of patients with decompression illness transported via physician-staffed emergency helicopters. *J Emerg Trauma Shock*. 2015;8:26-9. doi: 10.4103/0974-2700.145396. PMID: 25709249 PMCID: PMC4335152.
- 19 Grover I, Reed W, Neuman T. The SANDHOG criteria and its validation for the diagnosis of DCS arising from bounce diving. *Undersea Hyperb Med*. 2007;34(3):199-210. PMID: 17672176. [cited 22 March 2025]. Available from: https://www.uhms.org/images/DCS-and-AGE-Journal-Watch/grover_umo_sandhog_criteria.pdf.
- 20 Andre S, Lehot H, Morin J, Louge P, de Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. *Emerg Med J*. 2022;39:747-52. doi: 10.1136/emered-2021-211227. PMID: 35135892.
- 21 Bessereau J, Genotelle N, Brun P-M, Aboab J, Antona M, Chenaitia H, Huon A, Annane D. Decompression sickness in urban divers in France. *Int Marit Health*. 2012;63(3):170-3. PMID: 23129100. [cited 22 April 2025]. Available from: https://journals.viamedica.pl/international_maritime_health/article/view/26137/20931
- 22 Harris RJD, Frawley G, Devaney BC, Fock A, Jones A. A 10-year estimate of the incidence of decompression illness in a discrete group of recreational cave divers in Australia. *Diving Hyperb Med*. 2015;45(3):147-53. PMID: 26415066. [cited 22 April 2025]. Available from: https://www.dhmjournal.com/images/IndividArticles/45Sept/Harris_dhm.45.3.147-163.pdf
- 23 Lundell RV, Arola O, Suvilehto J, Kuokkanen J, Valtonen M, Räisänen-Sokolowski AK. Decompression illness (DCI) in Finland 1999-2018: Special emphasis on technical diving. *Diving Hyperb Med*. 2019;49(4):259-65. doi: 10.28920/dhm49.4.259-265. PMID: 31828744 PMCID: PMC7039777.
- 24 Guillén-Pino F, Morera-Fumero A, Henry-Benítez M, Alonso-Lasheras E, Abreu-González P, Medina-Arana V. Descriptive study of diving injuries in the Canary Islands from 2008 to 2017. *Diving Hyperb Med*. 2019;49(3):204-8. doi: 10.28920/dhm49.3.204-208. PMID: 31523795.
- 25 Monnot D, Michot T, Dugrenot E, Guerrero F, Lafère P. A survey of scuba diving-related injuries and outcomes among French recreational divers. *Diving Hyperb Med*. 2019;49(2):96-106. doi: 10.28920/dhm49.2.96-106. PMID: 31177515 PMCID: PMC6704004.

- 26 Chevasutho P, Premmaneesakul H, Sujirata A. Descriptive study of decompression illness in a hyperbaric medicine centre in Bangkok, Thailand from 2015 to 2021. *Diving Hyperb Med*. 2022;52(4):277-80. doi: 10.28920/dhm52.4.277-280. PMID: 36525685 PMCID: PMC10017199.
- 27 Edmonds C. Underwater oxygen treatment of decompression sickness a review, *SPUMS J*. 1995;25(3):122-8. [cited 22 April 2025]. Available from: https://www.dhmjournal.com/images/IndividArticles/25Sept/Edmonds_SPUMSJ.25.3.122-128.pdf
- 28 Dovenbarger J, editor. 1990 report on diving accidents and fatalities. Durham, NC: Divers Alert Network; 1992. [cited 11 March 2025]. Available from: <https://apps.dan.org/publication-library/>
- 29 Wang Q, Zhang X, Suo Y, et al. Normobaric hyperoxia therapy in acute ischemic stroke: A literature review. *Heliyon*. 2024;10(1):e23744. Epub 20231220. doi:10.1016/j.heliyon.2023.e23744. PMID: 38223732; PMCID: PMC10787244.
- 30 Li C, Jiang M, Chen Z, et al. The neuroprotective effects of normobaric oxygen therapy after stroke. *CNS Neurosci Ther*. 2024;30(7):e14858. doi: 10.1111/cns.14858. PMID: 39009510; PMCID: PMC11250159.
- 31 Sokolowski SA, Räisänen-Sokolowski AK, Tuominen LJ, Lundell RV. Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcomes. *Diving Hyperb Med*. 2022;52(4):271-6. doi: 10.28920/dhm52.4.271-276. PMID: 36525684 PMCID: PMC10026386.
- 32 Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: A follow-up study on incidence and self-treatment. *Diving Hyperb Med*. 2022;52(2):78-84. doi: 10.28920/dhm52.2.74-84. PMID: 35732278.
- 33 Stipp W. Time to treatment for decompression illness. Research Report 550. UK Health and Safety Executive; 2007. [cited 2 May 2026]. Available from: https://www.researchgate.net/publication/369694059_Time_to_treatment_for_decompression_illness_httpswwwhsegovukresearchrrhtmrr550htm.
- 34 Hoffman U, Smerecnik M, Muth C-M. Administration of 100% oxygen in diving accidents-an evaluation of four emergency oxygen devices. *Int J Sports Med*. 2001;22:424-9. doi: 10.1055/s-2001-16247. PMID: 11531035.

35 Mayer D. *Effects of newly designed albumin-derived perfluorocarbon-based nanoparticles in the therapy of decompression induced gas embolisms in rats*. [Doctoral thesis]. Split: University of Split; 2023.

36 Balldin UI. Effects of ambient temperature and body position on tissue nitrogen elimination in man. *Aerosp Med*. 1973;44(4):365-70. PMID 4694843.

37 Brubaak A. Surface oxygen is an acceptable definitive treatment. *SPUMS J*. 2000;30(3):155-61. [cited 21 March 2025]. Available from:

https://www.dhmjournal.com/images/IndividArticles/30Sept/Brubakk_SPUMSJ.30.3.155-161.pdf.

Statement

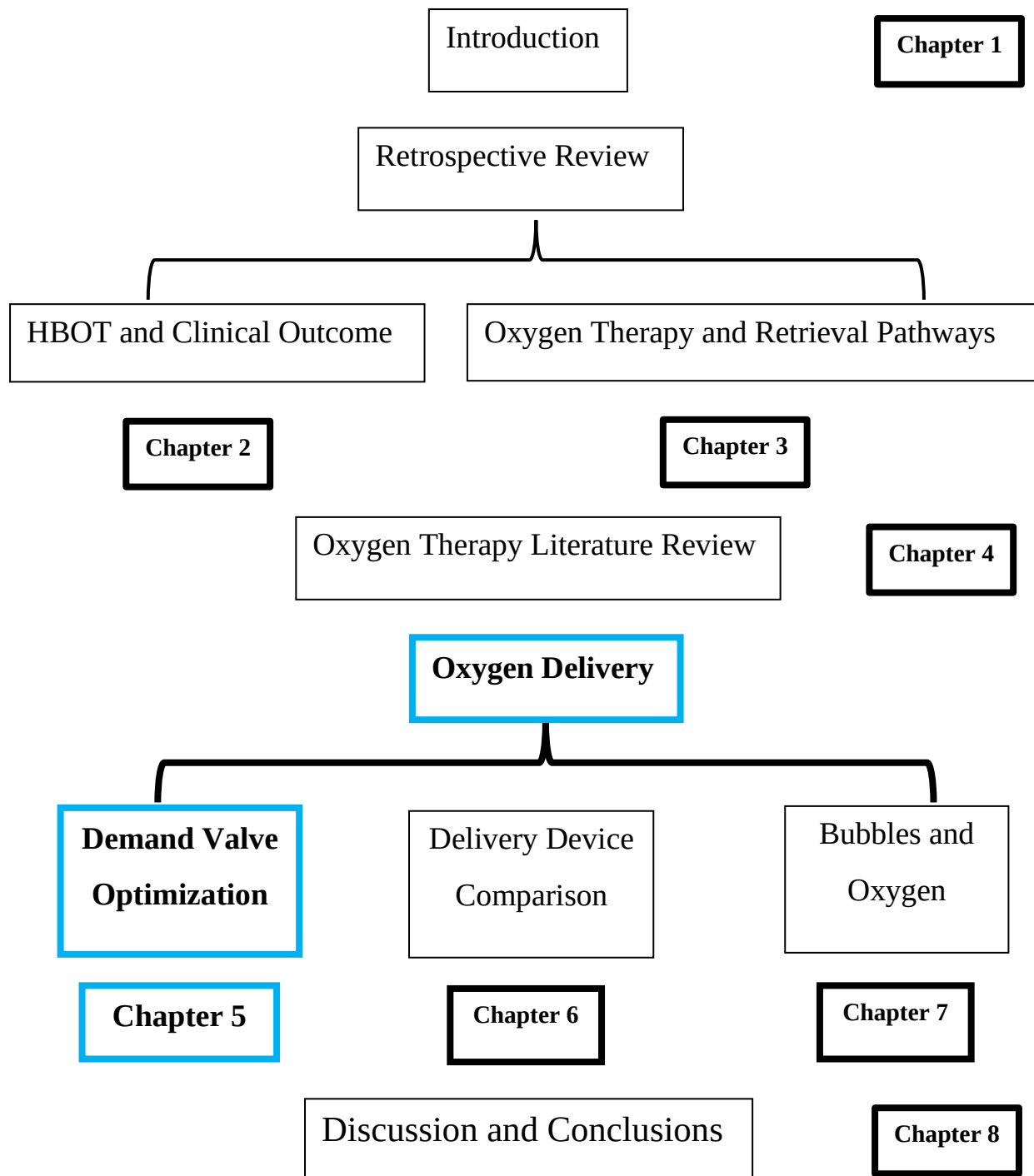
DFB conceived and designed the study with assistance from all authors. DFB collected the data and DFB and NWP performed the systematic review. DFB and MC analysed, and all authors interpreted the data. DB drafted the paper, and all others revised and approved the final version.

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CHAPTER 5 - COMPARISON OF TISSUE OXYGENATION ACHIEVED BREATHING OXYGEN FROM A DEMAND VALVE WITH FOUR DIFFERENT MASK CONFIGURATIONS

This chapter is an exact copy of the published journal article and the first of three sequential studies on oxygen delivery devices. Chapter 4 showed that there is some evidence that breathing oxygen reduces VGE and may decrease the number of required hyperbaric treatments. However, further evidence is required. The following three chapters present investigation of the effectiveness of oxygen treatment with Chapters 5 and 6 first evaluating the best device and oxygen flow rate for effective oxygen delivery and the final data chapter (Chapter 7) presenting preliminary evaluation of VGE grade breathing oxygen with the optimal delivery device compared to air following a simulated scuba dive. The focus of this chapter was to determine the mask configuration that provided the highest level of tissue oxygenation when breathing oxygen with a demand valve. The optimal mask configuration for use with the demand valve found in this study will then be used in the subsequent study.



Original articles

Comparison of tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations

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Key words

Decompression sickness; First aid; Masks; Medical kits; Oxygen; Transcutaneous oximetry; Scuba diving

Abstract

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Introduction: High concentration normobaric oxygen (O₂) is a priority in treating divers with suspected decompression illness. The effect of different O₂ mask configurations on tissue oxygenation when breathing with a demand valve was evaluated.

Methods: Sixteen divers had tissue oxygen partial pressure (P_{tc}O₂) measured at six limb sites. Participants breathed O₂ from a demand valve using: an intraoral mask (IOM®) with and without a nose clip (NC), a pocket face mask and an oronasal mask. In-line inspired O₂ (F_IO₂) and nasopharyngeal F_IO₂ were measured. Participants provided subjective ratings of mask comfort, ease of breathing and holding in position.

Results: P_{tc}O₂ values and nasopharyngeal F_IO₂ (median & range) were greatest using the IOM® with NC and similar with the IOM® without NC. O₂ measurements were lowest with the oronasal mask which also was rated as the most difficult to breathe from and to hold in position. The pocket face mask was reported as the most comfortable to wear. The NC was widely described as uncomfortable. The IOM® and pocket face mask were rated best for ease of breathing. The IOM® was rated as the easiest to hold in position.

Conclusion: Of the commonly available O₂ masks for use with a demand valve, the IOM® with NC achieved the highest P_{tc}O₂ values. P_{tc}O₂ and nasopharyngeal F_IO₂ values were similar between the IOM® with and without NC. Given the reported discomfort of the NC, the IOM® without NC may be the best option.

Introduction

Arterial gas embolism (AGE) and decompression sickness (DCS), collectively termed decompression illness (DCI), are risks for scuba divers. AGE results from pulmonary barotrauma introducing gas directly into the vascular system. DCS is caused by the formation of bubbles primarily from dissolved inert gas.¹ During a dive, an increase in the ambient pressure leads to a higher breathing gas pressure being delivered to the lungs, and more inert gas (nitrogen in air diving) dissolving in the blood and body tissues. This process is called on-gassing. At the end of a dive as the diver ascends, both the ambient pressure and the pressure

of the inspired inert gas decrease. The on-gassing process is reversed as tissue inert gas diffuses into the blood for carriage back to the lungs. However, if the pressure of dissolved gas in a tissue significantly exceeds ambient pressure (a condition referred to as ‘supersaturation’) the gas may form bubbles either in the extravascular space or within tissue capillaries. These bubbles are considered to be the primary cause of injury leading to the symptoms of DCS.

It follows from the above that arresting the growth of bubbles and encouraging their involution is a primary goal of treatment in DCS. Breathing a high concentration of oxygen (O₂) and therefore a lower partial pressure of inert

gas, creates a larger diffusion gradient between the blood and body tissues and blood and alveoli such that more inert gas moves from the tissue into the blood, and is then transported to the lungs to be exhaled.² Bubbles formed in decompressed divers without DCS have been demonstrated to resolve more quickly during O₂ breathing.³ Therefore, it is recommended that O₂ be given early to a diver with signs and symptoms of DCI, DCS and AGE, enhancing inert gas elimination from the body and supplying O₂ to hypoxic tissues.^{2,4-6}

The current pre-hospital care recommendation for divers with symptoms and signs of DCI is for O₂ delivery at the highest possible concentration (close to 100%).⁷ Divers Alert Network (DAN) has designed a variety of portable O₂ delivery units to provide divers with pre-hospital O₂.^{8,9} These units have two common components: (1) a constant flow capability for use with a non-rebreather mask (NRB) or other constant flow delivery device; and (2) a pressure-activated demand valve. Previous research comparing tissue oxygenation found that the NRB performed better than the demand valve with an oronasal mask.¹⁰ This research has been questioned as experts believe that the demand valve should be able to provide near 100% O₂ and therefore better tissue oxygenation than the NRB.

The present study used transcutaneous oximetry measurement (TCOM) to determine tissue oxygenation at multiple standardised sites in participants breathing O₂ from a demand valve using four different mask configurations. TCOM is a non-invasive technique that uses heated electrodes on the skin to measure the partial pressure of tissue oxygen (P_{tc}O₂).¹¹ The null hypothesis was that there would be no difference in the P_{tc}O₂ achieved after 10 minutes (min) of breathing O₂ with any of four different mask configurations.

Methods

Ethics approval was granted from the Townsville Health Service District Human Research Ethics Committee (HREC16/QTHS/196). The volunteers for this study were healthy non-smoking certified scuba divers of both sexes. Participants were recruited from James Cook University and the diving community in Townsville, Queensland, Australia. All participants were older than 18 years of age and had performed at least one dive within the previous year. Exclusion criteria included facial hair or anatomical abnormality that might impair mask seal, any medical condition or medication that may affect tissue oxygenation, or an allergy to the topical anaesthetic. All participants received a study information sheet and gave their written informed consent.

Participants were asked to refrain from consuming food or caffeine or performing heavy exercise for six hours prior to participating in the study. Age and sex were reported; height, weight and waist and hip circumferences were measured upon arrival for the study day. The participants were then

placed in a supine position on a hospital stretcher with their head slightly raised on one pillow and remained in this position for the duration of the study. The room temperature was maintained between 22.4 and 23.7°C; participants were covered with a blanket for comfort and to limit any vasoconstrictive effects of being cold.

Resting baseline measures included heart rate, respiratory rate, O₂ saturation and blood pressure. Topical lignocaine (5%) and phenylephrine (0.5%) (Co-Phenylcaine™ forte spray, ENT Technologies Pty Ltd., Hawthorne East, Australia) was sprayed into the right nares and an 8 French paediatric feeding tube (ConvaTec Ltd., Deeside, UK) was inserted. Position was verified visually with the tip of the tube placed just proximal to the soft palate and the tube then secured in place. The tube was attached to the E-sCO-OO module of a bedside monitor (GE Carescape Monitor B650, GE Healthcare Finland OY, Helsinki, Finland) allowing for both inspired O₂ (F_IO₂, paramagnetic) and end-tidal carbon dioxide (ETCO₂, infrared) measurements via a water trap (D-fend Pro+ Water Trap™, GE Healthcare Finland OY, Helsinki, Finland). The gas module was calibrated against room air before each mask configuration was used. The gas sampling rate was 120 ml·min⁻¹.

Tissue oxygenation was measured using the TCM400 Transcutaneous (tc) PO₂ Monitoring System (Radiometer, Copenhagen, Denmark) with tc Sensor E5250. Zero current calibration of the P_{tc}O₂ electrode using CAL2 gas (10% CO₂ with N₂ as balance) was performed prior to commencement of the study and calibration with atmospheric air occurred prior to each monitoring period. A 'humidity correction factor' was entered into the machine prior to each monitoring period. All assessments were performed by the same technician (AB). The TCM400 displayed P_{tc}O₂ values in units of mmHg.

Six sensors were used: three on the left arm and three on the left leg.¹⁰ One sensor was placed on the lateral aspect of the upper arm, mid-way between the acromial process and the olecranon process, one sensor 5 cm distal to the brachial crease on the lateral aspect of the lower arm and one sensor over the thenar eminence (palm hand). One leg sensor was placed 10 cm distal to the lateral femoral epicondyle (lateral leg); one 5 cm proximal to the lateral malleolus (lateral ankle) and one on the dorsum of the foot between the first and second metatarsal heads, attempting to avoid large superficial vessels. Participants rested quietly while the sensors were placed. They were requested to minimise talking during the study, but were not allowed to sleep. Initial normobaric, room air readings from all sensors were recorded after a minimum 20-min equilibration period that allowed all sensors to stabilize.

The participants were then asked to breathe O₂ for 10 min from a demand valve (L324-020, Life Support Products (LSP), Allied Healthcare Products, St. Louis, MO, USA)

Figure 1

Demand valve, spacer with side port, one-way valve configuration and intraoral mask

**Figure 3**

Study configuration with demand valve, spacer with side port, in-line and intra-mask gas sampling lines, nasopharyngeal catheter and pocket face mask (with subject's permission)



using each of four different mask configurations in randomized order:

- Intraoral mask (IOM®) with nose clip (NC) (NuMask®, Inc., Woodlands Hills, CA, USA) ([Figure 1](#));
- IOM® without NC;
- Adult soft silicone oronasal mask (Tru-Fit mask, Allied Healthcare Products Inc., St. Louis, MO, USA) ([Figure 2](#));
- Pocket face mask with air cushion (Sturdy Industrial CO., Ltd., Wugu Shiang, Taipei County, Taiwan) ([Figure 3](#)).

The oronasal, pocket face mask and demand inhalator valve which are provided in portable DAN O₂ units were used for this study. A flexible high-pressure O₂ hose was used to connect the demand valve to the hospital wall medical grade O₂ outlet (415 kPa). The demand valve was attached to a spacer with a side port allowing pressure and gas measurements. To measure the delivered O₂, a one-way valve was attached to the spacer and a T-piece with a one-way exhaust valve on the side ([Figure 1](#)). The side port was connected to the bedside monitor and a delivered O₂ percentage of 99% was obtained. The one-way valves and T-piece were removed and the single spacer with side port (in-line F_IO₂ measurement) was attached to each O₂ mask in turn during the study ([Figure 2](#)). A pressure line

Figure 2

Study configuration with demand valve, spacer with side port and oronasal mask



was attached to the side port and then to the bedside monitor via a BD DTXPlus™ pressure transducer (Argon Medical Devices Inc., Frisco, TX, USA). The monitor was configured to settings used for central venous pressure monitoring to give a high sensitivity in the lower range, and zeroed before each participant.

A single, new demand valve was used and the inspiratory opening pressure required to trigger the valve and the expiratory resistance pressure were verified prior to studying each new participant. The cracking pressure of the demand valve is 0 to -2 cm H₂O and exhalation pressure is 1.5 to 6.4 cm H₂O dependent on flow (LSP demand inhalator valve product insert). The pocket face mask has a nipple for the attachment of supplemental O₂, through which a gas sampling line (Microstream®, Oridion Medical Ltd., Jerusalem, Israel) was inserted and secured near the central opening of the mask to measure intra-mask F_IO₂ levels ([Figure 3](#)). Mask dead space was determined by measuring the amount of water required to fill each device. Fill levels were estimated by placing the masks on a mannequin's face and visual inspection of the intrusion of the facial features into the mask.

The order of the four O₂ mask configurations was randomised using the random number generator in Excel (Microsoft® Corporation, Redmond Washington, USA). The participants were asked to position and hold each mask for comfort and to ensure a tight seal to avoid leakage and to breathe deeply enough to trigger the demand valve as outlined in DAN educational material.^{8,9} In-line F_IO₂, nasopharyngeal F_IO₂, P_{tc}O₂ and other respiratory measures were recorded at the end of the 10-min breathing period. Nasopharyngeal gas sampling was intermittent and frequent throughout the

Table 1
Demographic and baseline measurements for the 16 participants; IQR - inter-quartile range

Characteristic	Median (IQR)	Range
Age (years)	27 (23, 30)	20–57
Body mass index (BMI) (kg·m ⁻²)	23 (22, 25)	17–29
Underweight (BMI < 18.5) (<i>n</i>)	1	
Normal (BMI 18.5–24.9) (<i>n</i>)	11	
Overweight (BMI 25.0–29.9) (<i>n</i>)	4	
Obese (BMI ≥ 30.0) (<i>n</i>)	0	
Waist-to-hip ratio		
Males (optimal < 0.82)	0.86 (0.84, 0.92)	0.84–0.92
Females (optimal < 0.71)	0.76 (0.74, 0.79)	0.69–0.88
Heart rate (beats·min ⁻¹)	66 (60, 72)	52–85
Systolic BP (mmHg)	109 (100, 116)	96–137
Diastolic BP (mmHg)	66 (58, 67)	52–87
Respiratory rate (breaths·min ⁻¹)	16 (12, 19)	12–22
Oxygen saturation (%)	97 (96, 98)	95–100
End-tidal CO ₂ (mmHg)	36 (34, 39)	32–47

study to prevent clogging of the catheter and in an attempt to capture peak values. Consistent repeated values were seen during each oxygen breathing session. After each 10-min O₂ breathing period, participants breathed room air for 10 min, allowing all P_{tc}O₂ levels to return to baseline before the next mask was trialled.¹² At the end of the data collection period all participants used a five-point Likert scale to rate each mask configuration on comfort, ease of breathing, and ease of holding the device in place. A final open-ended question asked about any adverse effects while breathing O₂.

ANALYSIS

Collected data were de-identified and entered into a preformatted Excel worksheet, then exported into Statistical Package for the Social Sciences version 23.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) for analysis.

Based on recent research, we expected mean P_{tc}O₂ values between 310 mmHg (forearm) and 421 mmHg (upper arm), with a sample standard deviation of 75 mmHg, when subjects breathed 100% oxygen.¹³ Using the mid-point of that range (365 mmHg), assuming a difference of 75 mmHg (smallest increase in P_{tc}O₂ at one sensor site breathing 100% oxygen with a hood¹³) would be clinically significant, and allowing for substantial correlation (*r* = 0.90) between the repeated

measures, we estimated that a sample size of 16 subjects would provide a power of 80% (with α = 0.05) to detect significant changes in tissue oxygenation.

Using G*Power (version 3.1.9.2)¹⁴ with medium effect size and the plan for a 2-way ANOVA, a sample size of 16 was calculated to give a power of 90% (α = 0.05) to detect a significant change in inspired O₂ values (notionally defined as 5%). We estimated this by using a 4 x 2 (4 masks x 2 O₂ values, in-line and nasopharyngeal F_IO₂) within and between factor analysis plan as no set values from previous research were available.

The Shapiro-Wilk test was used to evaluate normality of data distribution. None of the data were normally distributed. Thus, differences between median P_{tc}O₂, ET-CO₂, in-line, and nasopharyngeal F_IO₂ readings whilst breathing O₂ using the various masks and mask configuration ratings were analysed using non-parametric tests. Initial analysis was completed using the Friedman Test with *post hoc* paired analyses completed using the Wilcoxon Sign Rank test with Bonferroni correction. For the *post hoc* tests, a corrected *P*-value of 0.008 (0.05/6) was considered significant.

The primary outcome measure was a comparison of the median P_{tc}O₂ measurements recorded across the six

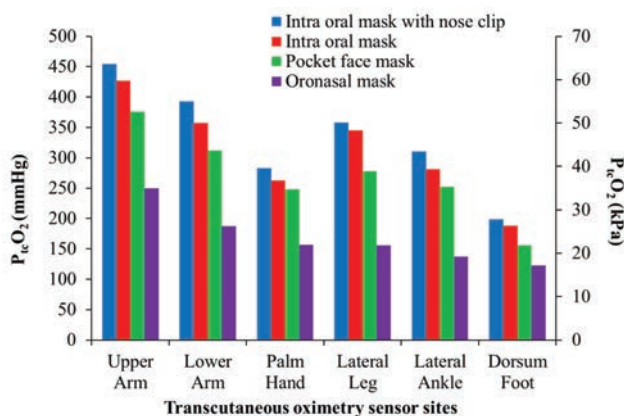
Table 2

Transcutaneous oxygen partial pressures (median and inter-quartile range shown in mmHg) while breathing oxygen using the demand valve with four different mask configurations; * *P*-values based on the Friedman test

Anatomical site	Baseline (room air)	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask	<i>P</i> -value*
Upper arm	69 (59, 75)	454 (437, 488)	427 (396, 474)	376 (347, 415)	250 (160, 326)	< 0.001
Lower arm	65 (59, 82)	393 (332, 437)	357 (324, 427)	312 (256, 383)	187 (135, 263)	< 0.001
Palm hand	77 (67, 81)	283 (229, 323)	263 (208, 285)	248 (160, 277)	157 (104, 217)	< 0.001
Lateral leg	61 (50, 75)	358 (283, 398)	345 (248, 394)	278 (243, 333)	156 (95, 294)	< 0.001
Lateral ankle	59 (51, 63)	310 (255, 329)	281 (238, 329)	252 (205, 286)	137 (110, 184)	< 0.001
Dorsum foot	64 (55, 78)	199 (175, 237)	188 (124, 228)	156 (132, 219)	123 (67, 137)	< 0.001

Figure 4

Transcutaneous oxygen partial pressures (median in mmHg) while breathing oxygen using the demand valve with four different mask configurations

**Table 3**

Statistically significant differences in transcutaneous oxygen partial pressures *post hoc* comparisons using Wilcoxon Sign Rank test with Bonferroni correction; *P* < 0.008 (0.05/6) considered significant; IOM® – intraoral mask

Post hoc analysis:

IOM® with nose clip ≈ IOM without nose clip
(all *P*-values > 0.01)

IOM with nose clip > Pocket face mask

IOM with nose clip > Oronasal mask

IOM without nose clip > Oronasal mask

Pocket face mask > Oronasal mask

(for all sensor sites)

IOM without nose clip > Pocket face mask
(for 3 of the 6 sensor sites)

Upper arm *P* = 0.003

Lower arm *P* = 0.001

Lateral leg *P* = 0.005

sensor sites after breathing O₂ for 10 min using each mask configuration. Secondary outcome measures included in-line and nasopharyngeal F_IO₂, ET_{CO}₂, mask comfort, ease of breathing and holding of each device.

Results

Sixteen healthy volunteers, 13 female and three male, met all inclusion criteria and completed the study protocol. Their demographic and baseline measures are shown in Table 1.

Figure 4 displays the median P_{tc}O₂ readings across all sensor sites and mask configurations. P_{tc}O₂ values were statistically different across each mask configuration for each sensor site (Table 2). The IOM® with NC delivered statistically better tissue oxygenation than the pocket face mask and the oronasal mask at all sensor sites and was similar to the oxygenation achieved with the IOM® without NC. Table 3 summarizes the *post hoc* comparison results.

Nasopharyngeal F_IO₂ was highest using the IOM® and similar results achieved with and without the NC. Nasopharyngeal F_IO₂ was lowest when breathing with the oronasal mask (Table 4). One participant's nasopharyngeal F_IO₂ values were lost due to ongoing catheter clogging. ET_{CO}₂ was statistically lower when participants breathed O₂ using the oronasal mask (Wilcoxon Signed Rank Test *P* < 0.008), but this almost certainly represents an artefactual reading due to an imperfect mask seal. In-line F_IO₂ did not exceed 97% with any of the mask configurations and was lowest using the oronasal mask (Table 4). Estimated mask assembly dead space is presented in Table 4. Actual individual pocket face and oronasal mask volumes would vary slightly depending on each participant's facial features.

The pocket face mask was rated as most comfortable (Table 5). Ease of breathing rating for each mask is listed in Table 6. Participant ratings for the holding of each mask configuration are presented in Table 7. On *post hoc* analysis no statistical

Table 4

Inspired oxygen and respiratory measurements while breathing oxygen using the demand valve with four different mask configurations and estimated mask assembly dead space (median and inter-quartile range); n/a – not applicable; ETCO₂ – end-tidal carbon dioxide;

* *P*-values based on the Friedman test

Parameter	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask	* <i>P</i> -value
In-line O ₂ (%)	95 (94, 96)	96 (94, 97)	94 (93, 95)	93 (89, 95)	< 0.001
Nasopharyngeal O ₂ (%) (<i>n</i> = 15)	95 (95, 96)	96 (95, 96)	84 (75, 88)	56 (37, 70)	< 0.001
Intra-mask O ₂ (%)	n/a	n/a	84 (72, 88)	n/a	
ETCO ₂ (mmHg)	34 (30, 38)	32 (30, 36)	35 (28, 38)	29 (23, 36)	0.001
Respiratory rate (breaths·min ⁻¹)	9 (8, 12)	10 (9, 10)	10 (8, 11)	10 (8, 12)	0.945
Mask assembly dead space (ml)	14	14	119	195	n/a

Table 5

Mask comfort rating for each mask configuration (*n* (%)); * *P*-value = 0.000, Friedman test

Comfort assessment	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very uncomfortable	2 (12.5)	0	0	3 (18.8)
Uncomfortable	5 (31.3)	3 (18.8)	1 (6.3)	2 (12.5)
Neither	5 (31.3)	4 (25.0)	0	5 (31.3)
Comfortable	4 (25.0)	8 (50.0)	7 (43.8)	5 (31.3)
Very comfortable	0	1 (6.3)	8 (50.0)	1 (6.3)
Median (IQR)*	3.0 (2.0–3.8)	4.0 (3.0–4.0)	4.5 (4.0–5.0)	3.0 (2.0–4.0)

Table 6

Ease of breathing rating for each mask configuration (*n* (%)); * *P*-value = 0.020, Friedman test

Breathing assessment	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very difficult	0	0	0	2 (12.5)
Difficult	0	0	1 (6.3)	6 (37.5)
Neither	8 (50.0)	4 (25.0)	3 (18.8)	2 (12.5)
Easy	2 (12.5)	5 (31.3)	5 (31.3)	4 (25.0)
Very easy	6 (37.5)	7 (43.8)	7 (43.8)	2 (12.5)
Median (IQR)*	3.5 (3.0–5.0)	4.0 (3.3–5.0)	4 (3.3–5.0)	2.5 (2.0–4.0)

Table 7Ease of holding rating for each mask configuration (*n* (%)); * *P*-value = 0.015, Friedman test

	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very difficult	0	0	0	1 (6.3)
Difficult	0	0	1 (6.3)	3 (18.8)
Neither	3 (18.8)	3 (18.8)	3 (18.8)	4 (25.0)
Easy	8 (50.0)	7 (43.8)	8 (50.0)	6 (37.5)
Very easy	5 (31.3)	6 (37.5)	4 (25.0)	2 (12.5)
Median (IQR)*	4.0 (4.0–5.0)	4.0 (4.0–5.0)	4 (3.25–4.75)	3.5 (2.25–4.00)

difference was found between each mask configuration for ease of breathing and holding of each mask configuration. The NC was frequently described as uncomfortable. The IOM® was described as easiest to use as it rested in the mouth whereas constant pressure was required to maintain a seal against the face with the two other masks.

Discussion

Oxygen is the primary first-aid treatment for divers suspected of having DCI.^{1,2,7} Oxygen has been shown in retrospective reviews to improve symptoms and decrease the subsequent number of hyperbaric treatments required.⁴ Of the commercially available O₂ masks for use with a demand valve delivery system designed for diver first aid, our study has shown that the IOM® with NC is the configuration that achieves the highest level of tissue oxygenation and nasopharyngeal F_IO₂.

DAN portable O₂ delivery units can provide a constant flow capability or operate as a pressure-triggered demand valve. The demand valve only delivers O₂ when the diver breathes in and, therefore, allows for conservation of O₂, dependent on the respiratory minute volume of the user. The ease of use, familiarity for divers, potential to deliver high inspired O₂ concentrations,¹⁵ as well as the potential for O₂ supply conservation, has led to the recommendation of the demand valve as the O₂ delivery method of choice in the pre-hospital treatment of DCI.²

However, previous research unexpectedly showed that the demand valve with oronasal mask provided less tissue O₂ than a constant flow NRB.¹⁰ P_{tc}O₂ readings whilst breathing O₂ via the demand valve with oronasal mask were anticipated to exceed those achieved with NRB at 15 L·min⁻¹.¹⁰ The previous contradictory findings¹⁰ were almost certainly explained by poor fit of the oronasal mask and subsequent entrainment of air. This assumption is supported by the current findings. Not only are the P_{tc}O₂ values lowest whilst

breathing O₂ using the oronasal mask, but the in-line and nasopharyngeal F_IO₂ values are also consistent with dilution of the O₂ with entrained air. The demand valve has been promoted to provide near 100% O₂;² however, this study highlights the need to assess the complete O₂ delivery system as different mask configurations provide different levels of O₂. It is important to remember that the one-way valve and filter provided for use with the pocket face mask and IOM® must be removed prior to use with the demand valve as leaving them in place leads to entrainment of air.

Oxygen therapy devices have traditionally been referred to as fixed or variable performance devices.¹⁶ When used appropriately, fixed performance devices deliver a constant fraction of O₂ to the patient's airway whereas the fraction delivered by variable performance devices can be affected by factors such as O₂ supply flow rate and the patient's respiratory minute volume. Demand regulators have traditionally been regarded as fixed performance devices based on the assumption that the composition of gas delivered to the patient's airway matches that delivered to the demand valve itself.¹⁶ However, our results show that even a demand regulator can, in fact, behave like a variable device depending on the interface between the valve and the patient. Use of an oronasal mask introduces variability to the behaviour of a demand valve depending on the adequacy of the seal on the patient's face, whereas when used with an IOM and NC the demand regulator likely behaves as a true fixed delivery device.

The IOM® with NC obtained the best P_{tc}O₂ results. Previous research comparing demand systems using both an oronasal mask and a mouthpiece with NC found no difference in inspired O₂ and nitrogen washout between the masks.¹⁷ We verified F_IO₂ of the demand system using one-way valves (99%), but these valves were removed for the study. In-line F_IO₂ measured during the study did not reach 99% and was significantly lower when breathing with the oronasal mask (Table 4). These lower in-line F_IO₂ levels are reflective

of gas contamination with exhaled air and possible air entrainment from mask leakage. The differences between the in-line $F_{I}O_2$ using the different masks in our study are subtle (Table 4). It is only when examining the delivery of the O_2 to the participants ($P_{tc}O_2$ and nasopharyngeal $F_{I}O_2$) that the differences between the masks become obvious. The IOM® out-performs both oronasal masks.

Divers are accustomed to breathing from a demand valve with a mouthpiece. This is reflected in their rating of the different mask configurations. The IOM® was commonly rated as easy to breathe from and easy to hold (Tables 6 and 7). The NC was reported as uncomfortable. The oronasal mask was ranked as the most difficult in terms of breathing ease and holding the device. Two subjects were noted to have difficulty breathing using the oronasal mask. When questioned, they stated that they had to hold the mask tightly and use larger breaths to trigger the demand valve. It is likely that poor mask fit and the large mask dead space (Table 4) contributed to a larger breath being required to adequately trigger the demand valve.

$ETCO_2$ was significantly lower when breathing with the oronasal mask though there were no differences in the respiratory rates while breathing with the different masks (Table 4). Entrained air from a sub-optimally fitting mask diluting the $ETCO_2$ is the most likely explanation and is consistent with the observed lower in-line $F_{I}O_2$ measured with this device.

There was a low number of male participants in this study due to a predominance of facial hair (an exclusion factor) as it was thought it could contribute to mask leak.¹⁸ Previous research showed no significant difference in $P_{tc}O_2$ by sex,¹³ but facial size may be a factor in mask fit.

Other investigators have explored closed-circuit O_2 delivery devices, other than demand valves, for the delivery of O_2 in DCI.^{19,20} None of these closed-circuit devices, however, are commonly used by recreational divers, partly due to increased complexity and operational requirements.^{2,21} Future research should compare continuous flow devices with demand valve and closed-circuit O_2 delivery systems.

LIMITATIONS

P_aO_2 was not measured but, rather, TCOM was used as a non-invasive method of measuring tissue oxygenation.²² Some DCS symptoms are presumably caused by tissue inert gas bubbles; therefore, a measurement estimating tissue oxygenation seems relevant to a study of first aid O_2 delivery. It could be argued that a higher $P_{tc}O_2$ must inevitably indicate higher values at all levels of the pre-tissue O_2 cascade, and that this, in turn, likely indicates greater drive for tissue inert gas elimination, bubble resolution and oxygenation of hypoxic tissues.^{2,5,6} However, although obvious, it is acknowledged that this is speculative as this study did not address the clinical efficacy of these devices in treating DCS.

The nasopharyngeal catheter provided valuable information on the oxygenation provided by each delivery system but may have compromised the seal of both the pocket face and oronasal masks. The catheter was secured to the nares and laid against the face, passing under the edge of the masks. The pocket face mask has an air-filled cushion which can easily mould around irregular facial features. The oronasal mask has a soft but more rigid edge and may have been more affected by the position of the catheter. However, participants who had difficulty sealing the oronasal mask felt air leaks at the apex of the mask at the bridge of the nose, not at the site of the catheter. Indeed, poor mask fit to facial contours and the large dead space likely contribute more to the poor performance of the oronasal mask than any small leak near the catheter.

Nasopharyngeal gas sampling was intermittent but frequent throughout the study with only the peak values recorded. Plotting any variable $F_{I}O_2/ETCO_2$ values obtained during the 10-minute oxygen breathing period was not possible.¹⁶

Conclusion

Of the commonly available O_2 delivery systems for use with the demand valve, the IOM® with NC is the device that achieved the highest $P_{tc}O_2$ values at the measured sites. We do not dispute previous findings that an oronasal mask could perform as well as a mouthpiece and noseclip,¹⁷ but this would require that great care be taken to ensure a perfect seal. Our results suggest that this would be very unlikely in field use of oronasal masks by divers. The IOM® is likely the most effective option of those tested. $P_{tc}O_2$ and nasopharyngeal $F_{I}O_2$ values were similar between the IOM® with and without NC. Given the reported discomfort of this, the IOM® without NC may be the best option.

References

- 1 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377:153–64. doi: 10.1016/S0140-6736(10)61085-9.
- 2 Lippmann J. First aid oxygen administration for divers. *SPUMS Journal*. 2003;33:192–8. Available from: <http://archive.rubicon-foundation.org/10050>. [cited 2018 May 17].
- 3 Blatteau J-E, Pontier J-M. Effect of in-water recompression with oxygen to 6 msw versus normobaric oxygen breathing on bubble formation in divers. *Eur J Appl Physiol*. 2009;106:691–5. PMID: 19424716.
- 4 Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiburger JJ. First aid normobaric oxygen for the treatment of recreational divers. *Undersea Hyperb Med*. 2007;34:43–9. PMID: 17393938. Available from: http://archive.rubicon-foundation.org/xmlui/bitstream/handle/123456789/9731/DHM_N37V4_8.pdf?sequence=1. [cited 2018 May 17].
- 5 Hyldegaard O, Møller M, Madsen J. Effect of He-O₂, O₂, and N₂O-O₂ breathing on injected bubbles in spinal white matter. *Undersea Biomed Research*. 1991;18:361–71. Available from: <http://archive.rubicon-foundation.org/2580>. [cited 2018 May 17]. PMID: 1746064.
- 6 Brubakk A. Surface oxygen is an acceptable definitive

- treatment. *Diving Hyperb Med.* 2000;30:155–61. Available from: <http://archive.rubicon-foundation.org/5866>. [cited 2018 May 17].
- 7 Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med.* 2018;48:45–55. doi: [10.28920/dhm48.1.45-55](https://doi.org/10.28920/dhm48.1.45-55). PMID: [29557102](https://pubmed.ncbi.nlm.nih.gov/29557102/).
 - 8 Lippmann J. Oxygen first aid, 4th ed. Melbourne: Submariner Publications; 2011.
 - 9 Bird N, Nochetto N. Emergency oxygen for scuba diving injuries: student handbook, 7th ed. Durham: Divers Alert Network; 2012.
 - 10 Blake DF, Naidoo P, Brown LH, Young D, Lippmann J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates. *Diving Hyperb Med.* 2015;45:79–83. PMID: [26165528](https://pubmed.ncbi.nlm.nih.gov/26165528/).
 - 11 Eberhard P, Mindt W, Jann F, Hammacher K. Continuous pO₂ monitoring in the neonate by skin electrodes. *Med Biol Eng.* 1975;13:436–42. PMID: [1195842](https://pubmed.ncbi.nlm.nih.gov/1195842/).
 - 12 Brown JT, Schur MS, McClain BC, Kafer ER. In vivo response time of transcutaneous oxygen measurement to changes in inspired oxygen in normal adults. *Can Anaesth Soc J.* 1984;31:91–6. PMID: [6692181](https://pubmed.ncbi.nlm.nih.gov/6692181/).
 - 13 Blake DF, Young DA, Brown LH. Transcutaneous oximetry: variability in normal values for the upper and lower limb. *Diving Hyperb Med.* 2018;48:2–9. doi: [10.28920/dhm48.1.2-9](https://doi.org/10.28920/dhm48.1.2-9). PMID: [29557095](https://pubmed.ncbi.nlm.nih.gov/29557095/).
 - 14 Faul F, Erdfelder E, Lang A-G, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioural, and biomedical sciences. *Behav Res Methods.* 2007;39:175–91. PMID: [17695343](https://pubmed.ncbi.nlm.nih.gov/17695343/).
 - 15 Hobbs GW, Natoli MJ, Pollock NW. Divers Alert Network emergency oxygen demand regulator validation trials. Centre for Environmental Physiology and Environmental Medicine, Duke University Medical Centre. Durham: Divers Alert Network Internal Report; 2000.
 - 16 Davis M. Oxygen therapy equipment: a theoretical review. *SPUMS Journal.* 1998;28:165–72. Available from: <http://archive.rubicon-foundation.org/5951>. [cited 2018 October 13].
 - 17 Hoffmann U, Smerecnik M, Muth CM. Administration of 100% oxygen in diving accidents-an evaluation of four emergency oxygen devices. *Int J Sport Med.* 2001;22:424–9. doi: [10.1055/s-2001-16247](https://doi.org/10.1055/s-2001-16247). PMID: [11531035](https://pubmed.ncbi.nlm.nih.gov/11531035/).
 - 18 Stobbe TJ, daRoza RA, Watkins MA. Facial hair and respirator fit: a review of the literature. *Am Ind Hyg Assoc J.* 1988;49:199–204. doi: [10.1080/15298668891379594](https://doi.org/10.1080/15298668891379594). PMID: [3287880](https://pubmed.ncbi.nlm.nih.gov/3287880/).
 - 19 Komesaroff D. Oxygen administration in diving accidents. *SPUMS Journal.* 1998;28[Suppl]:20–5. Available from: <http://archive.rubicon-foundation.org/5974>. [cited 2018 May 17].
 - 20 Wendling J. Normobaric oxygenation in dive accidents: a challenge for the developers of oxygen delivery systems. *SPUMS Journal.* 1997;27:101–4. Available from: <http://archive.rubicon-foundation.org/6063>. [cited 2018 May 17].
 - 21 Pollock NW, Natoli MJ. Performance characteristics of the second-generation remote emergency medical oxygen closed-circuit rebreather. *Wilderness Environ Med.* 2007;18:86–94. doi: [10.1580/06-WEME-OR-032R.1](https://doi.org/10.1580/06-WEME-OR-032R.1). PMID: [17590070](https://pubmed.ncbi.nlm.nih.gov/17590070/).
 - 22 Weaver LK. Transcutaneous oxygen and carbon dioxide tensions compared to arterial blood gases in normals. *Respir Care.* 2007;52:1490–6. PMID: [17971252](https://pubmed.ncbi.nlm.nih.gov/17971252/). [cited 2018 May 17]. Available from: <http://rc.rcjournal.com/content/respcare/52/11/1490.full.pdf>.

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Conflicts of interest

Professors Mitchell and Pollock are members of the Editorial Board of *Diving and Hyperbaric Medicine* but had no involvement in the peer review or publication decision-making process for this article.

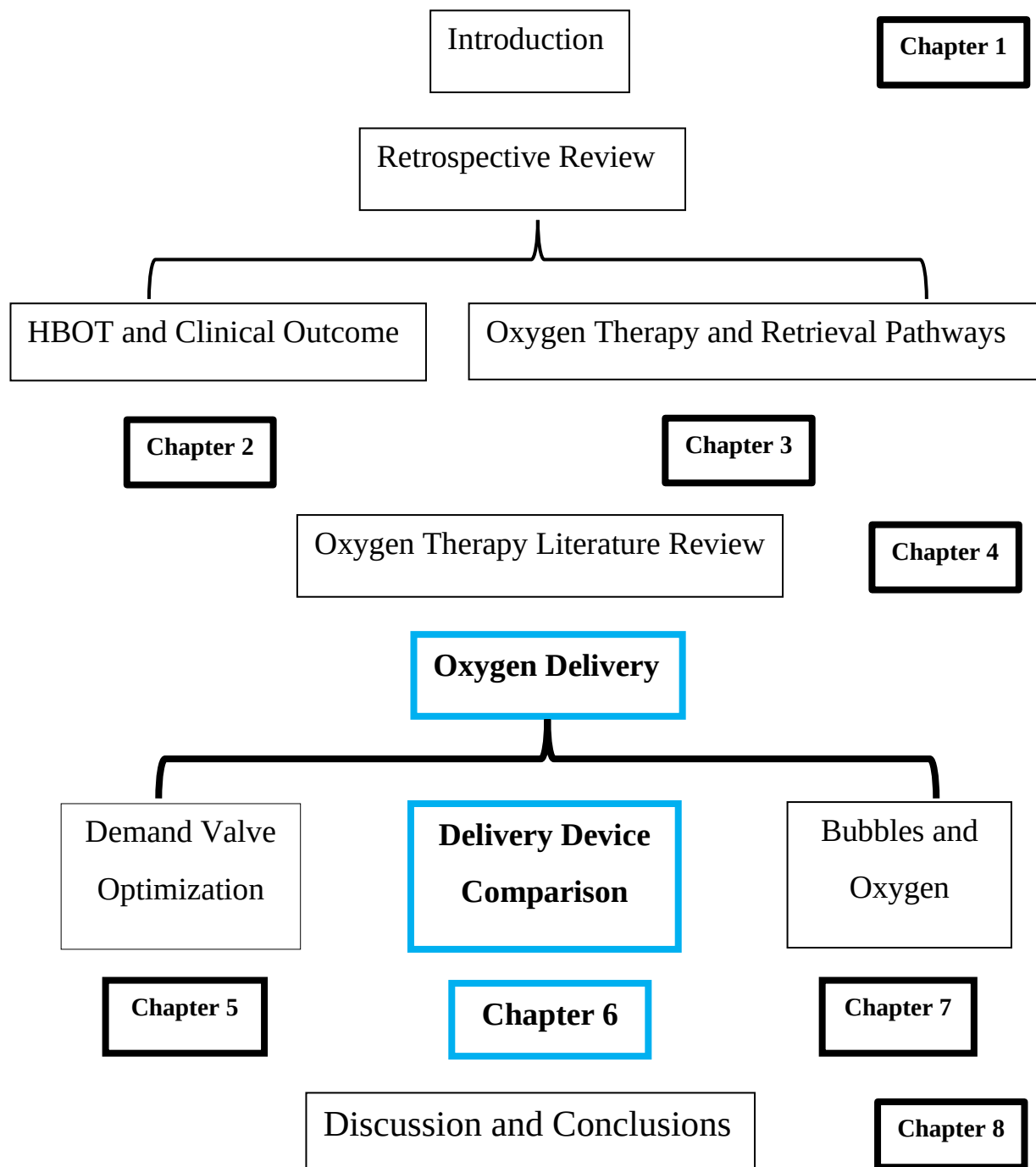
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CHAPTER 6 - COMPARISON OF TISSUE OXYGENATION ACHIEVED BREATHING OXYGEN USING DIFFERENT DELIVERY DEVICES AND FLOW RATES

This chapter is an exact copy of the published journal article. This paper is the second in the oxygen delivery series and compares oxygenation obtained breathing oxygen from the optimized demand valve from the previous study, a NRB mask at different flow rates and a medical oxygen rebreather system with two different mask configurations. The results from this study will inform the device selection for the third oxygen delivery study.



Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates

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Key words

Decompression sickness; Decompression illness; First aid; Masks; Medical kits; Oxygen; Transcutaneous oximetry; Scuba diving

Abstract

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Introduction: Divers with suspected decompression illness require high concentration oxygen (O_2). There are many different O_2 delivery devices, with few data comparing their performance. This study evaluated O_2 delivery, using tissue O_2 partial pressure (P_{tO_2}), in healthy divers breathing O_2 via three different delivery devices.

Methods: Twelve divers had P_{tO_2} measured at six limb sites. Participants breathed O_2 from: a demand valve using an intraoral mask with a nose clip (NC); a medical O_2 rebreathing system (MORS) with an oronasal mask and with an intraoral mask; and a non-rebreather mask (NRB) at 15 or 10 L·min⁻¹ O_2 flow. In-line inspired O_2 (F_{IO_2}) and nasopharyngeal F_{IO_2} were measured. Participants provided subjective ratings of device comfort, ease of breathing, and overall ease of use.

Results: P_{tO_2} values and nasopharyngeal F_{IO_2} were similar with the demand valve with intraoral mask, MORS with both masks and the NRB at 15 L·min⁻¹. P_{tO_2} and nasopharyngeal F_{IO_2} values were significantly lower with the NRB at 10 L·min⁻¹. The NRB was rated as the most comfortable to wear, easiest to breathe with, and overall the easiest to use.

Conclusion: Of the commonly available devices promoted for O_2 delivery to injured divers, similar P_{tO_2} and nasopharyngeal F_{IO_2} values were obtained with the three devices tested: MORS with an oronasal or intraoral mask, demand valve with an intraoral mask and NRB at a flow rate of 15 L·min⁻¹. P_{tO_2} and nasopharyngeal F_{IO_2} values were significantly lower when the flow rate using the NRB was decreased to 10 L·min⁻¹.

Introduction

High concentration oxygen (O_2) therapy is an important early first aid treatment for injured divers. Complete relief or improvement of the symptoms of decompression illness (DCI) has been seen in divers receiving pre-hospital normobaric O_2 therapy.¹ The current pre-hospital care recommendation for divers with symptoms and signs of DCI is for O_2 delivery at the highest possible inspired fraction (close to 100%).² However, there are many factors that need to be considered when choosing the most appropriate O_2 delivery system for a dive operation.^{3,4}

A variety of portable O_2 delivery units have been designed to provide divers with pre-hospital O_2 .^{3,5} These units incorporate one of two basic operating configurations: (1) a constant O_2 flow configuration used with a non-rebreather mask (NRB), medical O_2 rebreathing system (MORS) or other constant flow delivery devices; and (2) a patient triggered demand valve configuration. The recommended initial O_2 flow rate with the NRB mask for divers with suspected DCI has long been 15 L·min⁻¹.⁶ Divers Alert Network (DAN) America reduced its recommended O_2 flow rate to between 10 to 15 L·min⁻¹, to extend the duration of often limited O_2 supplies in the field, while still providing high levels of oxygenation.⁷ However, the effect of lower

flow rates on tissue oxygenation is unknown. A previous study comparing tissue oxygenation found that the NRB at 15 L·min⁻¹ performed better than the demand valve with an oronasal mask.⁸ However, a subsequent study showed that the demand valve provided the best tissue oxygenation when used with an intraoral mask and nose clip (NC);⁹ almost certainly because the intraoral mask eliminated leaks that were occurring with the oronasal mask.

The present study used transcutaneous oximetry measurement (TCOM) to determine tissue oxygenation at multiple standardised sites in participants breathing O₂ from a demand valve using an intraoral mask with a NC; a MORS with an oronasal mask and with an intraoral mask; and a NRB at 15 and 10 L·min⁻¹. The primary null hypothesis was that there would be no clinically significant difference in the partial pressure of transcutaneous tissue O₂ (P_{tc}O₂) achieved after 10 min of breathing O₂ with any of the different O₂ delivery devices or flow rates.

Methods

Ethics approval was granted from The Townsville Health Service District Human Research Ethics Committee (HREC16/QTHS/196). Healthy, non-smoking, adult certified scuba divers of both sexes were recruited for the study. Facial hair or anatomical abnormality that may impair mask seal, any medical condition or medication that may affect tissue oxygenation, or an allergy to topical anaesthetic were exclusion criteria. Written informed consent was provided by all participants prior to their participation.

Participants refrained from consuming food or caffeine or performing heavy exercise for six hours prior to participating in the study. Demographic data, anthropometric measurements, and resting baseline measurements were collected. Tidal volume (VT) was measured at rest using the EasyOne Spirometer (ndd Medical Technologies, Andover MA, USA) according to the manufacturer's instructions (average over 3–5 breaths). The participants rested in a supine position with their head on one pillow for the duration of the study. The test-room temperature was maintained between 22.4 and 22.9°C; to limit any vasoconstrictive effects of being cold, participants were covered with a blanket.

An 8 French paediatric feeding tube (ConvaTec Ltd., Deeside, UK) was inserted into the right nares after application of topical lignocaine (5%) and phenylephrine (0.5%) (Co-Phenylcaine™ forte spray, ENT Technologies Pty Ltd., Hawthorne East, Australia). Tube position was visually verified with the tip just proximal to the soft palate.⁹ The tube was then attached to the E-sCO-OO module of a bedside monitor (GE Carescape Monitor B650, GE Healthcare Finland OY, Helsinki, Finland) allowing for both inspired O₂ (F_IO₂, paramagnetic) and end-tidal carbon dioxide (E_TCO₂, infrared) measurements via a water trap (D-fend Pro+ Water Trap™, GE Healthcare Finland OY,

Helsinki, Finland). Room air gas calibration was completed before each breathing system was used. The gas sampling rate was 120 ml·min⁻¹.

TCOM is a non-invasive technique that uses heated electrodes on the skin to measure the P_{tc}O₂¹⁰ and was thought to provide a relevant measurement of tissue O₂ delivery in a study drawing an inference about tissue inert gas elimination. P_{tc}O₂ was measured using the TCM400 transcutaneous (tc) PO₂ Monitoring System (Radiometer, Copenhagen, Denmark) with tc Sensor E5250. Zero current calibration of the P_{tc}O₂ electrode was performed using CAL2 gas (10% CO₂ with N₂ as balance) prior to commencement of the study, and calibration with atmospheric air occurred prior to each monitoring period. A 'humidity correction factor' was entered into the machine prior to each monitoring period. All assessments were performed by the same technician. The TCM400 displayed P_{tc}O₂ values in units of mmHg (average of previous monitoring intervals).

Six sensors were used: three on the left arm and three on the left leg.⁹ Arm sensors were placed on the upper arm, lateral aspect of the lower arm, and the palm of the hand. Leg sensors were placed on the lateral leg, lateral ankle, and dorsum of the foot. Participants rested quietly while the sensors were placed. They were requested to minimise talking during the study as a method of control but were not allowed to sleep. Initial normobaric room air readings from all sensors were recorded after a minimum 20-min equilibration period that allowed all sensors to stabilize.

The participants were then asked to breathe O₂ for 10 min from the following devices in randomized order determined using the random number generator in Microsoft® Excel (Microsoft Corporation, Redmond Washington, USA):

- Demand valve (L324-020, Life Support Products, Allied Healthcare Products, St. Louis, MO, USA) with intraoral mask and NC held in place by the participant (NuMask® Inc., Woodlands Hills, CA, USA) (Figure 1);
- MORS (Wenoll-System, EMS GmbH, Möhrendorf, Germany) (Figure 2) with intraoral mask and NC held in place by the participant;
- MORS with air-cushion oronasal mask and a 4-strap mask holder (Figure 3);
- NRB mask at 15 L·min⁻¹ with elastic strap (Sturdy Industrial Co. Ltd, New Taipei City, Taiwan);
- NRB mask at 10 L·min⁻¹ with elastic strap.

The demand inhalator valve provided in portable DAN O₂ units was used for this study. A flexible high-pressure O₂ hose was used to connect the demand valve to the hospital wall medical grade O₂ outlet (415 kPa delivery pressure). The demand valve was attached to a spacer with a side port allowing pressure and gas measurements (Figure 1). A pressure line was attached to the side port and then to the bedside monitor via a BD DTXPlus™ pressure transducer (Argon Medical Devices Inc., Frisco, TX, USA). The monitor was configured to settings used for central venous

Figure 1

Configuration of demand valve, spacer with side port (allowing pressure and gas measurements), and intraoral mask

**Figure 2**

Medical oxygen rebreathing system (Wenoll-System)

**Figure 3**

Oronasal mask provided with the Wenoll-System and four-strap holder; oronasal mask secured in position on participant's face



pressure monitoring to give a high sensitivity in the lower range and zeroed before each participant. A single, new demand valve was used in the study and verification of inspiratory opening pressure required to trigger the valve and the expiratory resistance pressure was made prior to the commencement of each new participant. The demand valve configuration with intraoral mask and NC, from previous optimization trials, was used in this current research.⁹ Mask and circuit dead space was determined by measuring the amount of water required to fill each device. Mask fill levels were estimated by filling the masks with water and then placing a mannequin's face into the mask.

The NRB was examined to ensure there were three one-way valves in place and then primed with O₂ to inflate the reservoir bag. The NRB was positioned and adjusted to obtain the best seal possible. Participants were asked to

breathe normally, and the reservoir bag was monitored for persistent inflation during the breathing periods.

When using the demand valve participants were asked to breathe deeply enough to trigger the valve as outlined in DAN educational material.^{6,7}

The Wenoll MORS system was primed with 40 L·min⁻¹ of O₂ until the rebreathing bag was completely filled, and the oronasal mask was attached with a four-strap holder. The O₂ flow was 1.5 L·min⁻¹ during the 10-min breathing periods as outlined in the Wenoll-System operation manual.

In-line F_IO₂, nasopharyngeal F_IO₂, P_{ic}O₂, and other respiratory measures were recorded at the end of the 10-min breathing period, once P_{ic}O₂ had stabilized. In-line and nasopharyngeal F_IO₂ measurements were performed to determine if there is a difference between O₂ delivered by a device (in-line) and the O₂ reaching the upper airway (nasopharyngeal). Nasopharyngeal gas sampling was intermittent (every two min) throughout the O₂ breathing periods to prevent clogging of the catheter and to capture peak values. After each 10-min O₂ breathing period, participants breathed room air for 10 min, allowing all P_{ic}O₂ levels to return to baseline before the next device was trialled.¹¹ At the end of the data collection period all participants used a five-point Likert scale to rate each configuration on mask comfort, ease of breathing, and overall ease of use of each device. A final open-ended question asked about any adverse effects while breathing O₂.

ANALYSIS

All collected data were de-identified and entered into an Excel worksheet, and subsequently exported into Statistical Package for the Social Sciences version 25.0.0 (SPSS, IBM® Corporation, Armonk, New York, USA) for analysis.

Based on previous research when participants breathed 100% O₂, mean P_{ic}O₂ values between 199 mmHg (26 kPa) (dorsum of foot) and 454 mmHg (60 kPa) (upper arm) were expected.¹² Each sensor site generally has slightly different values, however, a decrease of 75 mmHg (10 kPa) across any of these sites was assumed to be clinically significant. Based on the values above and allowing for substantial correlation (r = 0.90) between the repeated measures, a sample size of 12 participants would provide a power of 90% (with α = 0.05) to detect clinically significant reductions in tissue oxygenation. In this context, 'clinically significant' was intended to mean a reduction in tissue O₂ delivery sufficient to indicate a potentially important corresponding reduction in the diffusion gradient for inert gas from tissues into blood. There are no published data which demonstrate how such gradients can be inferred from changes in tissue oxygenation, so a threshold tissue oxygenation change of 75 mmHg (10 kPa) (smallest increase in P_{ic}O₂ at one sensor site breathing 100% O₂ with a hood¹²) was agreed by consensus of the physiologists and clinicians involved in the study.

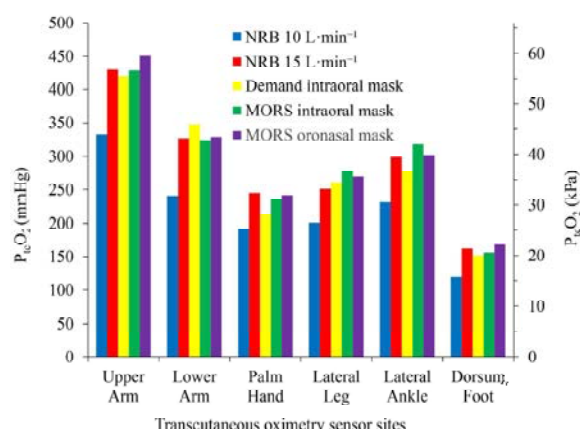
Table 1

Demographic and baseline measurements for the 12 participants breathing air. Optimal waist-to-hip ratios are < 0.82 for males and < 0.71 for females. BP = blood pressure. IQR = inter-quartile range

Characteristic	Median (IQR)	Range
Age (years)	30 (28, 32)	21–34
Body mass index (kg·m ²)	19 (15, 22)	15–24
Waist-to-hip ratio		
Males	0.86 (0.82, 0.87)	0.82–0.87
Females	0.74 (0.72, 0.80)	0.67–0.93
Heart rate (beats·min ⁻¹)	67 (59, 70)	50–94
Systolic BP (mmHg)	112 (111, 119)	100–128
Diastolic BP (mmHg)	66 (57, 73)	54–82
Respiratory rate (breaths·min ⁻¹)	14 (12, 16)	12–16
Tidal volume (ml)	745 (533, 913)	470–1130
Oxygen saturation (%)	97 (96, 98)	96–99
End-tidal CO ₂ (mmHg)	39 (37, 45)	36–46

Figure 4

Median transcutaneous oxygen partial pressures (mmHg) after breathing oxygen for 10 min with different devices and flow rates; NRB = non-rebreather mask; MORS = medical oxygen rebreathing system



The Shapiro-Wilk test was used to evaluate normality of data distribution. None of the data were normally distributed. Differences between median P_{tc}O₂, E_TCO₂, in-line, and peak and across device over time nasopharyngeal F_IO₂ readings using the various devices and flow rates were analysed using the Friedman test with *post hoc* paired analyses completed using the Wilcoxon signed-rank test with Bonferroni correction. For the *post hoc* tests, a corrected *P*-value of 0.005 (0.05/10) was considered significant for the P_{tc}O₂ values and a corrected *P*-value of 0.01 (0.05/4) for the 2-min nasopharyngeal F_IO₂ values.

The primary outcome measure was a comparison of the median P_{tc}O₂ measurements recorded across the six sensor sites after breathing O₂ for 10 min using each device and flow rate. Secondary outcome measures included in-line and

nasopharyngeal F_IO₂, E_TCO₂, and participant-rated mask comfort, ease of breathing and overall use of each device.

Results

Twelve healthy volunteers, nine females and three males, met all inclusion criteria and completed the study protocol. Their demographic and baseline measures breathing room air are shown in Table 1.

Figure 4 displays the median P_{tc}O₂ readings across all sensor sites and breathing devices and flow rates. Baseline P_{tc}O₂ values, median and IQR for each sensor site after breathing O₂ for 10 min are presented in Table 2. P_{tc}O₂ values were statistically different across each breathing device and flow rate for each sensor site (Table 2). *Post hoc* analysis showed there were no significant differences in P_{tc}O₂ values between the NRB 15 L·min⁻¹, demand valve and MORS with intraoral or oronasal mask. Some differences in median P_{tc}O₂ readings between devices at the same sites met the 75-mmHg (10 kPa) threshold for clinical significance, but only in comparisons between the NRB 10 L·min⁻¹ with other devices. The median P_{tc}O₂ readings achieved using the NRB 10 L·min⁻¹ were more than 75 mmHg (10 kPa) less than all other devices (including the NRB 15 L·min⁻¹) at the upper and lower arm sites, and at the lateral leg and ankle sites in comparison to the MORS with intraoral mask. No comparisons of the median P_{tc}O₂ between other devices met the threshold for clinical significance.

Peak nasopharyngeal F_IO₂ was highest breathing O₂ at 15 L·min⁻¹ with the NRB and lowest when breathing O₂ at 10 L·min⁻¹ with the NRB (Table 3). One participant's nasopharyngeal F_IO₂ value at 10 min while breathing using the demand valve was unattainable due to catheter clogging. There was a significant effect of time on nasopharyngeal F_IO₂ for the MORS (Figure 5). It was shown that for the MORS with intraoral mask, nasopharyngeal F_IO₂ was

Table 2

Transcutaneous oxygen partial pressures (median and inter-quartile range shown in mmHg) while breathing oxygen using the different devices and flow rates; * statistically significantly greater than NRB 10 L·min⁻¹ based on Wilcoxon signed-rank test with Bonferroni correction; # *P*-values based on the Friedman test; NRB = non-rebreather mask; MORS = medical oxygen rebreathing system

Site	Baseline (room air)	10 L·min ⁻¹ NRB	15 L·min ⁻¹ NRB	Demand with intraoral mask	MORS with intraoral mask	MORS with oronasal mask	<i>P</i> -value [#]
Upper arm	70 (61,77)	333 (285, 382)	429 (408, 464)*	420 (373, 465)	428 (385, 476)*	451 (375, 480)*	0.002
Lower arm	65 (58,70)	241 (217, 304)	327 (296, 405)*	348 (264, 370)	324 (286, 405)*	329 (313, 370)*	0.004
Palm hand	66 (63,74)	192 (151, 266)	245 (202, 275)	212 (179, 239)	236 (190, 293)*	242 (163, 289)	0.008
Lateral leg	57 (49,64)	201 (172, 233)	251 (217, 335)*	261 (208, 353)	278 (224, 350)*	270 (219, 330)*	0.004
Lateral ankle	62 (48, 67)	231 (152, 260)	299 (228, 336)*	277 (235, 342)	318 (221, 361)	302 (246, 319)*	0.002
Dorsum foot	54 (50, 65)	119 (88, 149)	162 (133, 226)*	152 (118, 220)	156 (93, 205)	168 (125, 198)*	0.008

Table 3

Inspired oxygen and respiratory measures while breathing oxygen using different devices and flow rates (median and inter-quartile range) and estimated mask and circuit dead space; NRB = non-rebreather mask; MORS = medical oxygen rebreathing system; n/a = not applicable; E_TCO₂ = end-tidal carbon dioxide; **P*-values based on the Friedman test

Parameter	10 L·min ⁻¹ NRB	15 L·min ⁻¹ NRB	Demand with intraoral mask	MORS with intraoral mask	MORS with oronasal mask	* <i>P</i> -value
In-line F _I O ₂ (%)	n/a	n/a	95 (92, 95)	93 (89, 95)	91 (88, 93)	0.045
Peak nasopharyngeal F _I O ₂ (%)	89 (75, 93)	97 (94, 98)	92 (88, 94)	91 (88, 95)	90 (88, 92)	0.013
E _T CO ₂ (mmHg)	39 (35, 43)	38 (34, 43)	38 (33, 41)	38 (32, 44)	39 (36, 43)	0.743
Respiratory rate (breaths·min ⁻¹)	12 (10, 15)	12 (10, 16)	10 (8, 12)	11 (8, 12)	12 (8, 14)	0.055
Mask + circuit dead space (ml)	95	95	14	14 + 350	136 + 350	n/a

significantly higher at time points 3, 4 and 5 (6, 8 and 10 min) compared to time 1 (*P* < 0.01). Nasopharyngeal F_IO₂ at all time points (4, 6, 8 and 10 min) was significantly higher than time point 1 breathing with the MORS and oronasal mask (*P* < 0.01). Both sets of results reflect a roughly linear increase in F_IO₂ over O₂ administration time. There was no statistical difference between the nasopharyngeal F_IO₂ values at each time point for any of the other breathing devices. Figure 6 illustrates the rise in P_{ic}O₂ values over the 10-min O₂ breathing periods, mirroring the rise in F_IO₂ values for the MORS.

E_TCO₂ was similar for all devices and flow rates. In-line F_IO₂ did not exceed 97% with any of the devices and was lowest using the MORS with oronasal mask (80%; Table 3). Estimated mask assembly and circuit dead space is presented in Table 3. Actual individual NRB and oronasal mask volumes would vary slightly depending on each participant's facial features.

Participant ratings for mask comfort are presented in Table 4. Ease of breathing rating for each device is listed

in Table 5. The NRB was rated as overall easiest to use (Table 6). On *post hoc* analysis no statistical difference was found between each device.

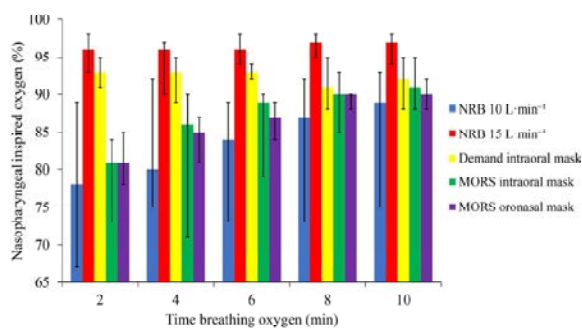
Discussion

High concentration O₂ is the primary first aid treatment for divers suspected of having DCI.^{2,3,13} O₂ has been shown in retrospective reviews to improve symptoms and decrease the subsequent number of hyperbaric treatments required.¹ Of the tested commercially available O₂ delivery systems designed for diver first aid, our study has shown that all systems can provide similar levels of tissue oxygenation and nasopharyngeal F_IO₂. However, when breathing with the NRB, an O₂ flow rate of 15 L·min⁻¹ is required to reach these levels.

Peak nasopharyngeal F_IO₂ was highest with the NRB with a flow rate of 15 L·min⁻¹ (Table 3) though 10-min P_{ic}O₂ values were similar for each device. This probably reflects the variability in breathing patterns of each participant and flow direction of the O₂. Nose breathing during use of the NRB

Figure 5

Median (IQR) nasopharyngeal inspired oxygen percentage recorded every two min for each delivery device and flow rate; NRB = non-rebreather mask; MORS = medical oxygen rebreathing system



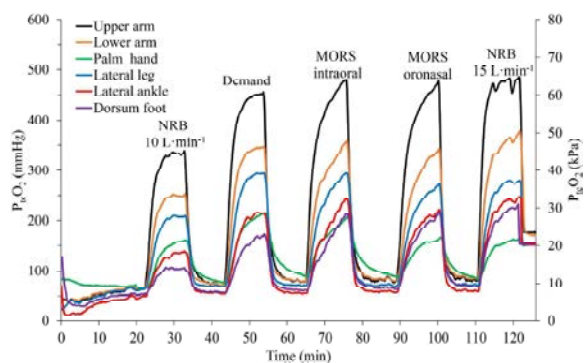
may explain the favourable nasopharyngeal F_{iO_2} results. When breathing with the MORS on the oronasal mask, some participants stated that they kept their mouths slightly open to ensure good fit of the mask, potentially bypassing the nasopharyngeal catheter through mouth breathing. Using the intraoral mask, the O_2 flow may have been directed slightly below the nasopharyngeal catheter through obligatory mouth breathing. This illustrates the importance of clearly describing the position of sampling ports in a research protocol and the potential variability in results if O_2 is measured at different sites. $P_{tc}O_2$ better reflects actual O_2 delivery to the body tissues whereas the nasopharyngeal F_{iO_2} is subject to the above possible confounders.

Portable O_2 delivery units can provide a constant flow capability or operate as a pressure-triggered demand valve. The demand valve only delivers O_2 when the diver inhales and therefore allows for conservation of O_2 , dependent on the respiratory minute volume of the user. The ease of use, familiarity for divers, potential to deliver high inspired O_2 concentrations,¹⁴ as well as the potential for O_2 supply conservation, has led to the recommendation of the demand valve as the O_2 delivery method of choice in the prehospital treatment of DCI.³ However, previous research unexpectedly showed that the demand valve with oronasal mask provided less tissue O_2 than a constant flow NRB.⁸ In the present study, $P_{tc}O_2$ readings whilst breathing O_2 via the demand valve with an intraoral mask and NC were similar to those achieved with a NRB at 15 L·min⁻¹. The previous contradictory findings⁸ were almost certainly explained by poor fit of the oronasal mask and subsequent entrainment of ambient air.⁹

The MORS provided similar oxygen levels regardless of the mask used. The oronasal mask provided with the system has an adjustable air-filled cushion to optimize mask fit and seal. The 4-point mask strap held the mask onto the face to aid with the seal. Fitting of the mask onto the participants' face prior to the study allowed for a good seal to limit if not eliminate any entrainment of ambient air. Oronasal masks

Figure 6

Transcutaneous oxygen partial pressures (mmHg) for one participant while breathing O_2 using different devices and flow rates over a complete iteration of the study. First 20-min equilibration period, followed by alternating 10-min O_2 and air breathing periods on different devices in randomized order. NRB = non-rebreather mask; MORS = medical oxygen rebreathing system



supplied with a demand valve system do not have the mask strap system, and the diver or first aider must therefore apply pressure to the mask to ensure an adequate seal. The technical difficulty of this almost certainly leads to breaks in the mask seal and entrainment of ambient air, especially because the user must generate negative pressure inside the mask to trigger the demand valve. While both oronasal or intraoral masks provide good O_2 delivery with the MORS, an intraoral mask may provide the highest levels of oxygenation with a demand valve.⁹

The NRB functions as a variable performance device with better oxygenation at a higher flow rate.¹⁵ Unfortunately, this means a greater consumption of O_2 . Demand regulators using an intraoral mask and NC behave as fixed performance devices,⁹ with O_2 consumption based on minute ventilation. The NRB and demand valve are both open systems with exhaled gas lost into the environment. A closed system is most beneficial for O_2 conservation as only low flow O_2 is required.^{5,16} MORS for first aid O_2 delivery are not commonly used by recreational divers, largely due to increased complexity and operational requirements^{3,17} and generally limited availability. However, it is possible that the growing popularity of closed-circuit rebreathers for diving will drive an increased interest in MORS systems for first aid use.

The Wenoll System MORS comes with an air-cushion mask (like a pocket face mask) held tightly in place with a 4-strap holder and a regulator mouthpiece. In this study an intraoral mask rather than the regulator mouthpiece was used with the MORS for better comparison with the demand system. Both masks provided good peak O_2 levels at 10 min, but the MORS took longer to reach peak inspired O_2 than the NRB and demand valve (Figure 5). This is consistent with previous research showing a seven-minute time frame to reach 98–100% inspired O_2 .¹⁶ It is possible that the MORS

Table 4Mask comfort rating for each delivery device (*n* (%)). MORS = medical oxygen rebreathing system; **P*-value = 0.052, Friedman test

Comfort assessment	Non-rebreather mask	Demand: intraoral mask	MORS: intraoral mask	MORS: oronasal mask
1. Very uncomfortable	0	0	0	0
2. Uncomfortable	0	4 (33.3)	3 (25.0)	3 (25.0)
3. Neither	2 (16.7)	2 (16.7)	1 (8.3)	3 (25.0)
4. Comfortable	3 (25.0)	4 (33.3)	6 (50.0)	5 (41.7)
5. Very comfortable	7 (58.3)	2 (16.7)	2 (16.7)	1 (8.3)
Median score (IQR)*	5.0 (4.0–5.0)	3.5 (2.0–4.0)	4.0 (2.3–4.0)	3.5 (2.3–4.0)

Table 5Ease of breathing rating for each delivery device (*n* (%)); MORS = medical oxygen rebreathing system; **P*-value = 0.061, Friedman test

Breathing assessment	Non-rebreather mask	Demand: intraoral mask	MORS: intraoral mask	MORS: oronasal mask
1. Very difficult	0	0	0	0
2. Difficult	0	3 (25.0)	2 (16.7)	1 (8.3)
3. Neither	0	2 (16.7)	1 (8.3)	4 (33.3)
4. Easy	3 (25.0)	1 (8.3)	5 (41.7)	3 (25.0)
5. Very easy	9 (75.0)	6 (50.0)	4 (33.3)	4 (33.3)
Median score (IQR)*	5.0 (4.3–5.0)	4.5 (2.3–5.0)	4.0 (3.3–5.0)	4.0 (3.0–5.0)

Table 6Overall ease of use for each delivery device (*n* (%)); MORS = medical oxygen rebreathing system; **P*-value = 0.009, Friedman test

Overall ease of use assessment	Non-rebreather mask	Demand: intraoral mask	MORS: intraoral mask	MORS: oronasal mask
1. Very difficult	0	0	0	0
2. Difficult	0	3 (25.0)	1 (8.3)	1 (8.3)
3. Neither	0	3 (25.0)	1 (8.3)	2 (16.7)
4. Easy	3 (25.0)	2 (16.7)	4 (33.3)	6 (50.0)
5. Very easy	9 (75.0)	4 (33.3)	6 (50.0)	3 (25.0)
Median score (IQR)*	5.0 (4.3–5.0)	3.5 (2.3–5.0)	4.5 (4.0–5.0)	4.0 (3.3–4.8)

may have delivered greater fractions of O₂ more quickly if a procedure to remove nitrogen (N₂) from the participants' lungs had been employed at the start of breathing on the MORS (typically, by exhaling to atmosphere completely, then inhaling O₂ from the system and exhaling it to the atmosphere for several breaths before breathing exclusively on the MORS). Examination of the P_{tc}O₂ values while breathing O₂ with the MORS showed a plateau at eight to 10 min and therefore the data reported here probably accurately reflect peak values.

Breathing high concentration O₂ eliminates N₂ from the inspired gas and enhances N₂ elimination from the body.³ Open circuit systems release exhaled gas into the environment, allowing for the elimination of N₂. In MORS, the higher O₂ flow rate in the first hour not only improves oxygenation¹⁶ but allows for excess gas in the breathing circuit to be automatically vented through the over-pressure valve, which serves to purge accumulated N₂ into the environment.⁵ Other suggestions for purging excess N₂ from the MORS circuit when used in injured divers include: periodic increase in O₂ flow rates and periodic use of a purge button, if equipped.^{5,16} Monitoring for colour change of the carbon dioxide (CO₂) absorber and spontaneous increase in tidal volume are ways to evaluate scrubber function.⁵

Limiting the usage time of the absorber can prevent CO₂ intoxication.⁵ Formal training in the use of a MORS for O₂ delivery is recommended.

The NRB was rated as the overall easiest to use (Table 6), even though divers are accustomed to breathing from a demand valve with a mouthpiece. Some participants commented on the change in their breathing patterns when using the demand valve, they used their 'diving breathing pattern' of slower deeper breaths. Although not statistically significant, there was a trend towards a slower respiratory rate when breathing with the demand valve (*P* = 0.055) (Table 3).

Three commonly available pre-hospital O₂ delivery systems were evaluated for O₂ delivery, comfort, ease of breathing, and overall use. There are many other factors that need to be considered when selecting the most appropriate O₂ delivery system for a dive operation.^{3,15} Remoteness of diving operations, and therefore a protracted time to arrive at medical care, may increase the need for a system that is more comfortable for the diver but also a system that provides a longer duration of O₂ delivery and CO₂ elimination. Cost, availability and O₂ supplies in different countries may also play an important part in the decision-making process.

Knowledge of individual country guidelines and training requirements are necessary to make educated decisions about appropriate O_2 delivery system selection.

LIMITATIONS

There was a low number of male participants in this study due to a predominance of facial hair. Facial hair was an exclusion factor, as it was thought it could contribute to mask leak.¹⁸ In real world use, facial hair in males may reduce the efficacy of O_2 delivery by a NRB in comparison to a device that does not rely on a facial seal, such as the demand valve or MORS used with an intraoral mask and NC. Previous research shows no significant difference in $P_{tc}O_2$ results by sex.¹²

Even though a higher $P_{tc}O_2$ value likely indicates a greater drive for tissue inert gas elimination, bubble resolution and oxygenation of hypoxic tissues,^{3,19,20} this study did not address the clinical efficacy of these O_2 devices in treating DCI or achieving bubble resolution.²¹ Similarly, the arbitrary nature of the consensus decision to use a 75 mmHg (10 kPa) $P_{tc}O_2$ threshold to power the study and to indicate a meaningful difference in O_2 delivery / outgassing gradient between devices is acknowledged. Therefore, although perhaps indicative, these data do not prove that one device will be associated with greater clinical efficacy than another.

The nasopharyngeal catheter provided valuable information on the oxygenation provided by each delivery system but may have compromised the seal of both the NRB and oronasal mask. The catheter was secured to the nares and laid against the face, passing under the edge of the masks. The oronasal mask has an air-filled cushion which can easily mould around irregular facial features. The NRB has a more rigid edge and may have been more affected by the presence of the catheter.

The oxygen breathing period was limited to 10 min based on previous research^{8,11} where $P_{tc}O_2$ values had stabilized at that time point. The TCM400 machine has a built-in arrow indicator that depicts upward or downward trends to help clinicians to identify stable peak values (when the arrows disappear). However, in visualizing the nasopharyngeal F_{IO_2} values it seems that the values were still rising for the MORS and NRB at 10 L·min⁻¹. Although there was no statistical difference in the values at eight and 10 min, extending the monitoring time beyond the 10 min O_2 breathing period could provide additional information.

Conclusion

The three tested O_2 delivery systems used to treat injured divers (MORS with an oronasal or intraoral mask, demand valve with an intraoral mask and NRB at a flow rate of 15 L·min⁻¹) delivered similar $P_{tc}O_2$ and nasopharyngeal F_{IO_2} values. $P_{tc}O_2$ and nasopharyngeal F_{IO_2} values were lower when the flow rate using the NRB was decreased from

15 to 10 L·min⁻¹. O_2 delivery and supply conservation are important factors to be considered when selecting an O_2 delivery system for a dive operation.

References

- Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiburger JJ. First aid normobaric oxygen for the treatment of recreational divers. *Undersea Hyperb Med.* 2007;34:43–9. Available from: <http://archive.rubicon-foundation.org/5515>. [cited 2018 May 17].
- Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med.* 2018;48:45–55. doi:10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.
- Lippmann J. First aid oxygen administration for divers. *SPUMS Journal.* 2003;33:192–8. Available from: <http://archive.rubicon-foundation.org/10050>. [cited 2018 May 17].
- Sayer MDJ, Küpper FC, van West P, Wilson CM, Brown H, Azzopardi E. Managing scientific diving operations in a remote location: The Canadian high Arctic. *Diving Hyperb Med.* 2013;43:239–43. Available from: https://www.researchgate.net/publication/260130983_Managing_scientific_diving_operations_in_a_remote_location_The_Canadian_high_Arctic. [cited 2019 April 18]. PMID: 24510334.
- Wendling J. Normobaric oxygenation in dive accidents: a challenge for the developers of oxygen delivery systems. *SPUMS Journal.* 1997;27:101–4. Available from: <http://archive.rubicon-foundation.org/6063>. [cited 2018 May 17].
- Lippmann J. *Oxygen first aid.* 4th ed. Melbourne: Submariner Publications; 2011.
- Bird N, Nochetto M. *Emergency oxygen for scuba diving injuries: student handbook.* 7th ed. Durham (NC): Divers Alert Network; 2012.
- Blake DF, Naidoo P, Brown LH, Young D, Lippmann J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates. *Diving Hyperb Med.* 2015;45:79–83. Available from: http://www.dhmjournal.com/images/Journals/45/DHM_Vol45_No2.pdf. [cited 2018 May 17]. PMID: 26165528.
- Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations. *Diving Hyperb Med.* 2018;48:209–17. doi: 10.28920/dhm48.4.209-217. PMID: 30517952. PMCID: PMC6355319.
- Eberhard P, Mindt W, Jann F, Hammacher K. Continuous pO_2 monitoring in the neonate by skin electrodes. *Med Biol Eng.* 1975;13:436–42. doi: 10.1007/BF02477116.
- Brown JT, Schur MS, McClain BC, Kafer ER. In vivo response time of transcutaneous oxygen measurement to changes in inspired oxygen in normal adults. *Can Anaesth Soc J.* 1984;31:91–6. Available from: <https://link.springer.com/article/10.1007/BF03011489>. [cited 2019 May 14]. PMID: 6692181.
- Blake DF, Young DA, Brown LH. Transcutaneous oximetry: variability in normal values for the upper and lower limb. *Diving Hyperb Med.* 2018;48:2–9. doi: 10.28920/dhm48.1.2-9. PMID: 29557095. PMCID: PMC6467822.
- Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet.* 2011;377:153–64. doi: 10.1016/S0140-6736(10)61085-9. PMID: 21215883.
- Hobbs GW, Natoli MJ, Pollock NW. Divers Alert Network

- emergency oxygen demand regulator validation trials. Centre for Environmental Physiology and Environmental Medicine, Divers Alert Network Internal Report. Durham: Duke University Medical Centre; 2000.
- 15 Davis M. Oxygen therapy equipment a theoretical review. SPUMS Journal. 1998;28:165–72. Available from: <http://archive.rubicon-foundation.org/5951>. [cited 2018 October 13].
 - 16 Komesaroff D. Oxygen administration in diving accidents. SPUMS Journal. 1998;28(Suppl):20–5. Available from: <http://archive.rubicon-foundation.org/5974>. [cited 2018 May 17].
 - 17 Pollock NW, Natoli MJ. Performance characteristics of the second-generation remote emergency medical oxygen closed-circuit rebreather. Wilderness Environ Med. 2007;18:86–94. doi: 10.1580/06-WEME-OR-032R.1. PMID: 17590070.
 - 18 Stobbe TJ, daRoza RA, Watkins MA. Facial hair and respirator fit: a review of the literature. Am Ind Hyg Assoc J. 1988;49:199–204. doi: 10.1080/15298668891379594. PMID: 3287880.
 - 19 Hyldegaard O, Møller M, Madsen J. Effect of He-O₂, O₂, and N₂O-O₂ breathing on injected bubbles in spinal white matter. Undersea Biomed Res. 1991;18:361–71. Available from: <http://archive.rubicon-foundation.org/2580>. [cited 2018 May 17]. PMID: 1746064.
 - 20 Brubakk A. Surface oxygen is an acceptable definitive treatment. Diving Hyperb Med. 2000;30:155–61. Available from: <http://archive.rubicon-foundation.org/5866>. [cited 2018 May 17].
 - 21 Blatteau J-E, Pontier J-M. Effect of in-water recompression with oxygen to 6 msw versus normobaric oxygen breathing on bubble formation in divers. Eur J Appl Physiol. 2009;106:691–5. doi: 10.1007/s00421-009-1065-y. PMID: 19424716.

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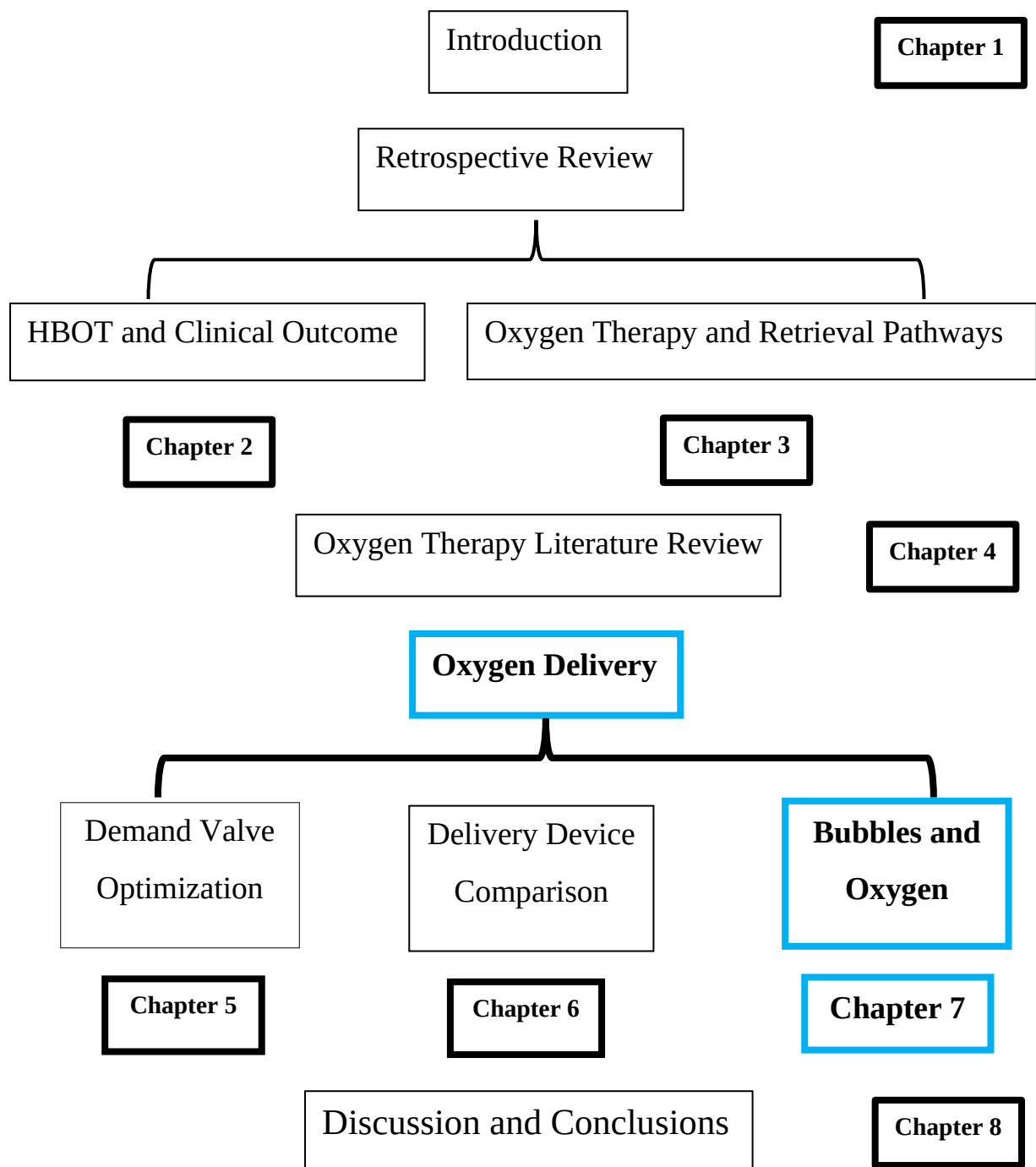
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CHAPTER 7 - EFFECT OF NORMOBARIC OXYGEN ADMINISTRATION ON VENOUS GAS EMBOLI GRADES AFTER A DRY CHAMBER DIVE

This is the third and final study in the oxygen delivery series. This chapter is written in the same format as the previous published journal articles, presenting preliminary results of VGE grade after breathing oxygen. The NRB mask with an oxygen flow rate of $15 \text{ L}\cdot\text{min}^{-1}$, identified in the previous study as providing the highest level of oxygenation, was used to investigate the effect of breathing normobaric oxygen versus air on VGE grade following a simulated dive in a dry hyperbaric chamber.



Effect of normobaric oxygen administration on venous gas emboli grades after a dry chamber dive

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Keywords

Aerobic capacity; Decompression illness; Decompression sickness; Echocardiography; First aid; Scuba diving

Abstract

Introduction: Decompression sickness (DCS) may occur when inert gas in supersaturated tissues become bubbles during decompression. High concentration oxygen should be provided early to divers with suspected DCS to accelerate the removal of inert gas from body tissues and bubbles. The non-rebreather (NRB) mask providing oxygen at 15 L·min⁻¹ has been shown to provide high tissue oxygenation. This study evaluated venous gas emboli (VGE) using transthoracic echocardiography (TTE) in divers breathing oxygen compared to air from a NRB mask after a dry dive in a hyperbaric chamber.

Methods: Healthy divers with no history of DCS were recruited. Maximal aerobic capacity testing was performed to set an aerobic capacity-indexed exercise intensity during the dives. Participants performed two no-decompression dives at 280 kPa for 50 min in a dry hyperbaric chamber riding a cycle ergometer. Participants breathed air after one dive and oxygen after the other dive in a double-blind randomized manner commencing 30 min after completion of the dive. TTE commenced after the participants exited the chamber and continued every 15 min for 120 min. VGE were graded using an expanded grading scale. Time to onset of non-zero VGE grades, time to max VGE grade, and VGE grade difference between air and oxygen breathing are reported.

Results: Eight male and four female divers participated, median age 32 (IQR 31, 53) years. No left sided heart bubbles were visualized and 50% of the divers had no VGE visualized in either arm of the study. Due to low VGE generation, the exercise intensity was increased to 75% VO_{2max} after six consecutive divers did not produce any VGE. Time to non-zero grade was at the first scanning time after the dive in the oxygen breathing session and at the 15 min post dive scanning time in the air breathing session. Time to max VGE grade was 45 min during oxygen breathing and 90 min during air breathing. Equal or higher VGE grades for 2/6 participants were detected during air versus oxygen breathing 45 to 120 min post dive. Participants in the oxygen breathing condition had no VGE at the 120 min period whereas VGE were still produced during air breathing at this time point.

Conclusion: In this study, 2/6 divers presented equal or higher VGE grades breathing air compared to oxygen at 45 to 120 min post dive. No participant breathing oxygen had VGE detected at 120 min but 3/6 participants breathing air had VGE detected at this time. Though this study was underpowered, these preliminary findings suggest a possible decrease in VGE formation with oxygen breathing compared to air. A high level of exercise during a dry dive will increase the likelihood of VGE generation.

Introduction

Scuba diving is an activity undertaken by many people around the world. A rare but recognized adverse outcome is decompression sickness (DCS). During a dive the ambient pressure and partial pressure of inspired gas is increased. Inert gases, such as nitrogen, are absorbed into body tissues during the dive. The depth and time of the dive, as well as other factors such as the mixture of the breathing gas used by the diver, effect the amount of inert gas absorbed. During ascent the ambient pressure is reduced, and tissues become supersaturated with inert gas. The dissolved inert gas may come out of solution in the form of bubbles. These bubbles accumulate in body tissues, lymphatic duct, and blood vessels and may precipitate symptoms of DCS.¹

Gas bubbles do not always lead to symptoms of DCS but are an indicator of decompression stress (factors contributing to an individual's susceptibility to DCS) and DCS probability in research where the generation of clinical DCS would be an unacceptable endpoint.² Bubbles can be detected trans thoracically using portable ultrasound machines that visualize the four chambers of the heart. Venous gas emboli (VGE) loads are graded using a semi-quantitative ordinal scale.³ Table 7.1 outlines one of the grading scales used. VGE and dissolved inert gas are eliminated from the body via the lungs through respiration. Breathing a higher concentration of oxygen increases the diffusion gradient between the alveoli and blood vessels such that more inert gas moves into the lungs to be exhaled. Oxygen should be given early to a diver with signs and symptoms of DCS, allowing them to accelerate inert gas elimination.^{1,4-6} In addition to helping displace dissolved gas and gas in bubbles, breathing a high concentration of oxygen also supplies oxygen to hypoxic tissues⁷ and reduces inflammation.^{8,9}

Table 7.1 Transthoracic echocardiography (TTE) expanded grading scale; Scores are reported as the highest of the three conditions (at rest, with right arm and leg movements) for a given scan

VGE Grade	Description
0	No observable bubbles
I	Occasional bubbles
II	At least one new bubble every four cardiac cycles
IIIa	At least one new bubble every cardiac cycle
IIIb	At least six new bubbles every cardiac cycle
IVa	At least one bubble·cm ⁻²
IVb	At least three bubbles·cm ⁻²
IVc	Near whiteout, individual bubbles still discerned
V	Whiteout, individual bubbles cannot be discerned

One previous study in 2009 examined VGE grades in divers breathing air, oxygen and in-water oxygen starting 10 min after a decompression dive.¹⁰ Divers performed three dives: one control dive, one dive followed by 30 min of normobaric oxygen breathing, and one dive followed by 30 min of in-water recompression (IWR) breathing oxygen. Acoustic Doppler was performed at 40-, 60- and 80-min post surfacing at rest and after two leg movements. VGE grades were found to be significantly lower after IWR breathing oxygen versus normobaric oxygen breathing. VGE were significantly less after breathing normobaric oxygen versus breathing air. This study administered oxygen at 15 L·min⁻¹ in the normobaric group, but the mask type was not described.¹⁰ Since this study was published, the newer technique of using echocardiography to grade bubbles has evolved with recommendations to perform measurements for 120 min post-dive.^{2,3} Further research has shown that the non-rebreather mask (NRB) at 15 L·min⁻¹ provided high levels of tissue oxygenation and reached peak tissue oxygen levels earlier than other delivery devices.¹¹ Potentially, the decompression dive study¹⁰ described above did not use an optimized normobaric oxygen delivery device or scanning technique. To our knowledge, no other published peer-reviewed human studies have assessed the effects of normobaric oxygen compared to air breathing to reduce decompression stress by assessing VGE.

The present study aimed to investigate the potential for normobaric oxygen to reduce VGE following a simulated dive compared to breathing air. A non-rebreather mask at 15 L·min⁻¹ was used to provide oxygen or air to divers after a simulated dive in a dry hyperbaric chamber. Transthoracic echocardiography (TTE) was performed to assess divers for VGE. The null hypothesis was that there would be no difference in VGE grade determined by TTE after 30 min of oxygen and air breathing via a NRB mask.

Methods

Ethics approval was granted from The Townsville Health Service District Human Research Ethics Committee (HREC16/QTHS/196) and James Cook University (H6859). Healthy, non-smoking, adult, certified scuba divers of both sexes were recruited to participate in the study. Divers with a history of DCS were excluded from participating. Excessive facial hair and anatomical abnormality that may affect mask seal were exclusion criteria. All participants were fit to dive with a current Australian Standard (AS) 2299.1 dive medical. Written informed consent was provided by all participants prior to their participation.

This was a prospective, randomized, double-blind, cross-over study. The study required participants

to attend three separate sessions. The first session assessed the aerobic capacity ($\text{VO}_{2\text{max}}$) of each participant in the Sports and Exercise Science laboratory at James Cook University. Medical pre-screening including vital signs was performed on the day of the $\text{VO}_{2\text{max}}$ test to ensure wellness. The determination of $\text{VO}_{2\text{max}}$ allowed the calculation of participant relevant exercise intensity to be used during the dive in the next sessions. The remaining two sessions were conducted in the hyperbaric medicine unit (HMU) at the Townsville University Hospital. The participants performed identical no-decompression dives following Defence and Civil Institute of Environmental Medicine (DCIEM) standard air decompression dive tables¹² in the hyperbaric chamber (Fink Engineering Pty Inc., Warana, Queensland, Australia). DCIEM dive tables are industry standard for occupational diving in Australia. The DCIEM no-decompression limits¹² were designed to produce low levels of VGE. The exercise periods in the present study were utilised to increase the uptake of inert gas and be provocative enough to overcome the DCIEM table conservatism and stimulate post-dive VGE production.^{13,14} Participants were asked to refrain from consuming caffeine or large amounts of food or performing heavy exercise for six hours prior to participating in all arms of the study. Participants had a minimum of a one-week break from hyperbaric exposures (wet or dry) prior to the first study chamber dive and a one-week break between the two chamber dives to minimize the potential influence of prior compression on study dives.

AEROBIC CAPACITY TESTING

Participants performed a maximal graded incremental test to determine $\text{VO}_{2\text{max}}$ using a stationary electronically braked cycle ergometer (Excalibur Sport PFM, Lode BV, Groningen, NL). A three-minute warm-up was performed before the test commenced. Male participants commenced the test at 100 watts (W) and females at 50 W and continued with increasing workload of 25 W every minute (min) until maximum effort was achieved. The participants were instructed to maintain a cadence of at least 70 revolutions per min (rpm) and were verbally encouraged to continue until exhaustion. The participants were required to breathe from a mask sealed over their mouth and nose with expired gas analysed by a calibrated metabolic gas cart (Cosmed Quark CPET, Omnia software Version 1.6.10 2020, Rome, Italy). Heart rate (Polar Electro Oy, Kempele, Finland) and Borg's rating of perceived exertion (RPE)¹⁵ (Table 7.2) were recorded every min. $\text{VO}_{2\text{max}}$ was determined based on at least three of the following criteria: (1) no modest increase in the VO_2 with increasing exercise intensity; (2) a respiratory exchange ratio greater than 1.10; (3) an $\text{RPE} \geq 17$ on the Borg scale; and (4) a heart rate above 90% of age predicted maximum ($220 - \text{age}$).¹⁶ The $\text{VO}_{2\text{max}}$ test results allowed determination of W and then calculation of kilopond (kp) ($[\text{W} \times 6 \text{ metres per revolution}] / 450 = \text{basket weight}$) to appropriately weight the basket of the exercise cycle ergometer for 50% $\text{VO}_{2\text{max}}$ for the second part of the study, with a plan to increase the exercise intensity, if required, to obtain VGE. An exercise intensity of 50% $\text{VO}_{2\text{max}}$ was chosen as a starting point based on a previous study that used two 5 min

stepping exercise periods to approximate 5 metabolic equivalents.¹⁷

Table 7.2 Borg's rating of perceived exertion (RPE) scale¹⁵

RPE	Description
6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

DRY CHAMBER DIVES

Participants presented to the HMU for the second and third study sessions. Demographic data (sex and age), anthropometric measurements (height, weight, and waist and hip circumferences) and resting baseline measurements (heart and respiratory rate, blood pressure, and oxygen saturation) were recorded. TTE was performed before the dive to determine the best scanning window for a 4-chamber view of the heart. The apical (Figure 7.1) and subcostal (Figure 7.2) sites are the most commonly used windows for a 4-chamber TTE scan. Participants were asked to change into cotton clothing and hospital booties were worn over their shoes. Participants were orientated to the hyperbaric chamber and seated on a basket loaded cycle ergometer (Ergomedic 874 E, Monark, Vansbro, Sweden). Weights were added to the basket as determined from the $\text{VO}_{2\text{max}}$ testing to set the exercise to 50% $\text{VO}_{2\text{max}}$. This initial level of exercise intensity was chosen with caution to prevent severe bubbling, as this technique had not been utilised previously. Participants were asked to pedal at 70 rpm displayed on a screen of the cycle ergometer. A metronome was available to assist the diver with keeping this cadence. Participants breathed room air during the dive. The chamber temperature was monitored, and adjustments made using the environmental control system to maintain 21°C. Participants completed all their dives within the same season to ensure similar outside temperatures and humidity. A certified hyperbaric nurse accompanied the participants in the chamber. Dives were to 280 kPa (18 msw) for 50 min with a compression rate of 60 $\text{kPa}\cdot\text{min}^{-1}$ (6 $\text{msw}\cdot\text{min}^{-1}$) and decompression rate of 45 $\text{kPa}\cdot\text{min}^{-1}$ (4.5 $\text{msw}\cdot\text{min}^{-1}$). The participants performed three exercising periods starting on arrival at 280 kPa. The protocol was 15 min exercising, 3 min rest, 15 min

exercising, 3 min rest, 11 min exercising and 4 min rest during ascent. The rest period allowed the participant to wipe away sweat and have a drink of water. Only one participant was compressed at a time. Each participant performed two dives breathing air or oxygen post-dive (see oxygen delivery section below). Divers served as their own controls to decrease the inter-diver variability known to exist in VGE production.³

Figure 7.1 Apical 4-chamber view

Apical 4-chamber view

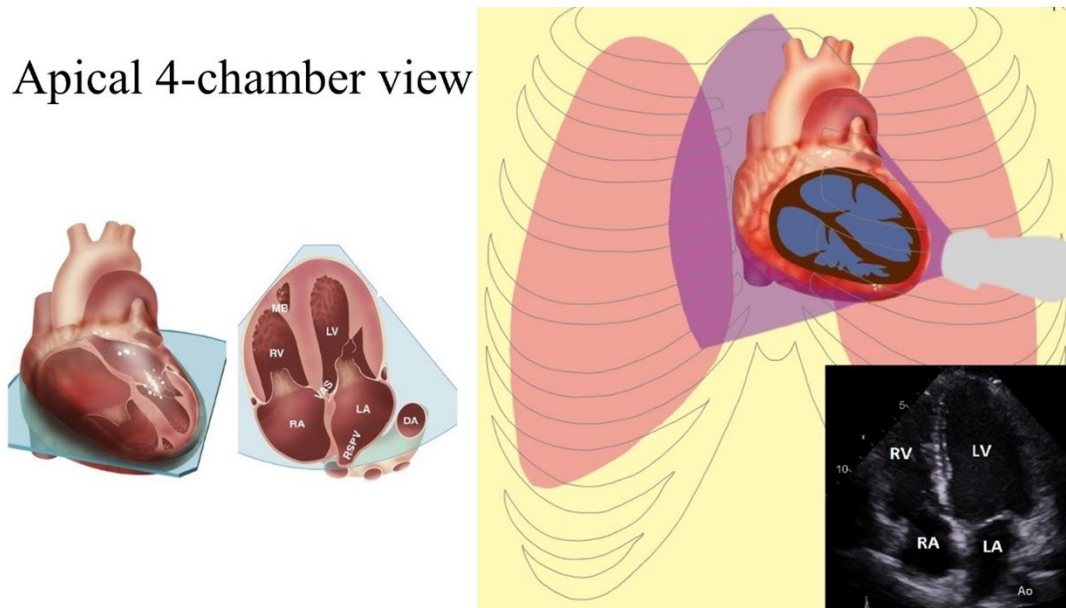
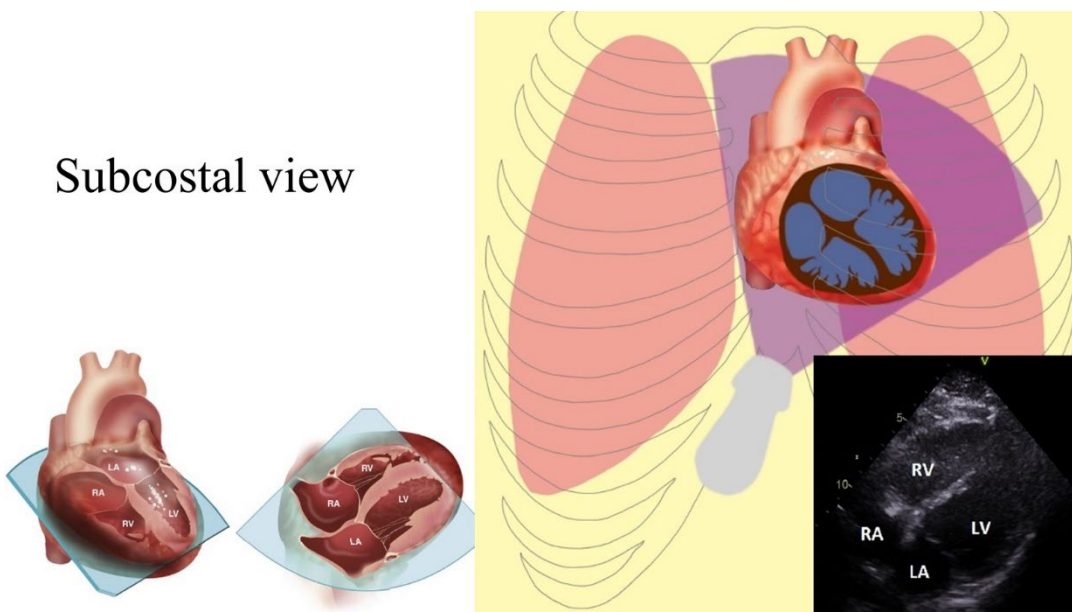


Figure 7.2 Subcostal 4-chamber view

Subcostal view



ECHOCARDIOGRAPHY

TTE was performed in a room adjacent to the hyperbaric chamber to decrease the movement performed by the participants after the dives. Participants walked slowly to the room and TTE commenced after resting for one minute. Scans continued in 15-min intervals to 120 min. During scanning, participants lay in the left lateral decubitus position on a hospital bed propped up on a bent left arm (Figure 7.3). Participants positioned themselves and scanning commenced a min after movement ceased. VGE for both the right and left heart were graded using the TTE expanded grading scale: at rest and with arm and leg movements (Table 7.1). The movements consisted of efforts to contract every muscle and move every joint in the target limb through full flexion and extension (Figures 7.4 and 7.5). These were performed three times in 5 seconds (s) before returning the limb to a neutral resting position. The ultrasound technician was an intensive care consultant with a diploma in diagnostic ultrasound from the Australasian Society for Ultrasound in Medicine and over 10 years of experience in clinical echocardiography. Due to COVID travel restrictions, initial on-line support for the echocardiographer was provided by video link from an experienced researcher from Canada (author NWP) with over 20 years of experience in performing TTE to grade VGE. Thirty second clips were recorded for review by the investigators. All the clips were reviewed by the echocardiographer and one of the investigators (DFB) and then by a blinded reviewer (NWP). Consensus was reached via face-to-face meetings, reviewing all scans with initial grading disagreement. Review allowed slowing the clip speed to 50% real-time to better distinguish between bubbles and noise. Scans were classified as uninterpretable if < 80% of the margins and structures were visualized or there was a loss of visualization of the luminal space for greater than 3 s in a 30 s clip. The goal was to have 90% of the total scan as interpretable. This was a conservative approach to evaluating scans. Participants lay quietly between scanning and were covered with a blanket to keep them comfortable.

OXYGEN DELIVERY

After each dive the participant randomly breathed either oxygen at 15 L·min⁻¹ for 30 min using a NRB mask starting after the 30-min TTE assessment or breathed air for the same time period with the same mask and flow rate. Oxygen breathing commenced 30 min post-dive as DCS symptom onset is often not immediately post-dive¹⁸ and to allow time for some bubbles to form. The participants breathed oxygen for 30 min as this is the approximate time a D size oxygen cylinder would last providing oxygen at 15 L·min⁻¹. The gas breathing order was randomized using a random number generator. Randomization for the participants was conducted in advance of the participant arriving at the HMU. Participants assigned to the oxygen arm after the first dive were pre-determined to be in the air arm after their next dive, and vice-versa. The researcher performing the TTE and the other researchers in the room were blinded to the gas assignment. Breathing gas allocation was done by the research nurse

and the mask tubing concealed under a sheet. The oxygen and air cylinders were the same size to assist with the blinding process. Unblinding was only performed after review of the echo clips by the independent reviewer and resolution of disagreements.

Figure 7.3 Participant position for transthoracic echocardiography apical 4-chamber view



Figure 7.4 Participant position for arm movement **Figure 7.5** Participant position for leg movement



STATISTICAL ANALYSIS

All collected data were de-identified and entered into Statistical Package for Social Sciences version 29.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) for analysis. Using G*Power¹⁹ with medium effect size and substantial correlation between the repeated measures ($r=0.90$), a sample size of 12 participants who generated VGE was calculated to give a power of 90% ($\alpha=0.05$) to detect a significant change in VGE between the two dives, oxygen and air. This was estimated by using a 2 x 9 (2 oxygen configurations and 9 TTE measurements) within and between factor analysis.

Skewness, kurtosis, histograms, and the Shapiro-Wilk test was used to evaluate normality of demographic, baseline measurements and aerobic testing data distribution. None of these data were normally distributed, therefore are presented as median, interquartile range (IQR) and range. VGE data, time to onset of non-zero VGE grades, time to max VGE grade, and median, mode and VGE grade range are presented. The max VGE grade for the combined conditions (rest and movements) was determined for each participant for each 15 min time period. The data analysis plan was to use non-parametric analyses (as the data are categorical and not continuous,) Pearson's chi-square and Fischer's exact test, to evaluate changes in grade of max VGE for each paired exposure. Post-hoc paired analyses using Pearson's chi-square were planned using data pooling: grade 0, grades I and II, grades III and IV, and grade V.³ A *P* value of 0.05 was accepted to indicate statistical significance for all tests. However, due to the small sample size statistical analysis was not performed on the VGE data and results are reported using descriptive statistics.

Results

Twelve healthy scuba divers, eight males and four females, met the inclusion criteria and completed the study protocol. Demographic, baseline measurements and maximal aerobic testing results are shown in Table 7.3. No participant had any signs or symptoms of DCS after the dives. There were no left-sided heart bubbles visualized and 50% of the divers had no VGE visualized in either arm of the study. Only one diver produced VGE following the dives with 50% VO_{2max} exercise intensity. This diver was the first participant and had produced VGE during a previous research study. No VGE were generated in the subsequent six divers, therefore the exercise intensity was increased to 75% VO_{2max} for the remaining five divers. There was 18% disagreement in initial VGE grading between the two reviewers (Cohen's weighted kappa 0.270), the majority in uninterpretable scans (11%). When the uninterpretable scans were excluded, the weighted kappa increased to 0.802. The final level of uninterpretable scans was 9% during the air breathing session (29/324) and 8% during the oxygen

breathing session (26/324). The apical 4-chamber view was used in 11 participants and the subcostal view was used in one participant.

Table 7.3 Demographics, baseline measurements and aerobic testing results for the 12 participants and six participants who generated VGE (median [IQR] and range); RPE-Borg's rating of perceived exertion

Characteristic	All	Generated VGE
Sex (<i>n</i>)	8 male/4 female	4 male/2 female
Age (years)	32 [31, 53] Range 28-67	52 [39, 62] Range 31-67
Body mass index (BMI) (kg·m ⁻²)	23.7 [21.8, 26.6] Range 19.5-28.0	24.8 [23.2, 26.9] Range 22.1-26.9
Waist-to-hip ratio		
Males	0.87 [0.84, 0.91] Range 0.84-1.00	0.91 [0.88, 0.98] Range 0.87-1.00
Females	0.75 [0.69, 0.82] Range .69-0.82	0.82 [0.81] Range 0.81-0.82
Resting heart rate (beats·min ⁻¹)	66 [60, 72] Range 52-85	67 [59, 80] Range 54-98
Resting systolic BP (mmHg)	127 [118, 133] Range 108-142	132 [113, 135] Range 108-142
Resting diastolic BP (mmHg)	78 [71, 86] Range 44-95	76 [71, 90] Range 69-95
Resting respiratory rate (breaths·min ⁻¹)	16 [16, 16] Range 12-20	16 [15, 20] Range 12-20
Resting oxygen saturation (%)	98 [98, 99] Range 97-100	99 [98, 99] Range 98-99
VO _{2 max} (mL·kg ⁻¹ ·min ⁻¹)	40.5 [36.1, 51.9] Range 34.8-63.0	42.3 [39.4, 56.0] Range 34.8-56.0
RPE at VO _{2 max}	17 [16, 18] Range 16-20	16 [16, 18] Range 16-18

Right sided heart VGE grades for all 12 participants for each scanning period are in Appendix B and C. Further analysis was performed on the six participants who generated VGE; the five divers who performed exercise at 75% $\text{VO}_{2\text{max}}$ and one diver who performed exercise at 50% $\text{VO}_{2\text{max}}$. The apical 4-chamber view was used in these 6 participants. Time to non-zero grade was immediately after the dive in the oxygen breathing session and at 15 min in the air breathing session (Table 7.4). Time to max VGE grade was 45 min during oxygen breathing and 90 min during air breathing (Table 7.5). Median and mode VGE grades were 0 for both the air and oxygen breathing sessions. VGE range was from 0 to IVa in the oxygen breathing arm and from 0 to IVb in the air breathing arm. There were no IVc or V bubble grades.

Maximum VGE grades for each participant who generated VGE for each time period are shown in Table 7.6. VGE grades were higher during the air breathing session for all time periods from 60 to 120 min post dive (Table 7.7). Maximum VGE grades for each participant in the two groups for the 45 to 120 min time periods are shown in Table 7.8. Pooling the data did not add any insight into the analysis. Participants in the oxygen breathing group had no VGE by the 120 min time period whereas the air breathing group continued to produce VGE (Table 7.5).

Table 7.4 VGE grades for six participants who generated VGE prior to breathing air or oxygen for 15-min time periods from 0 to 30 min post-compression at rest and maximum grade after either right arm or right leg movements

Time period	VGE grade							
	0	I	II	IIIa	IIIb	IVa	IVb	Uninterpretable
0 min rest								
Air	5	0	0	0	0	0	0	1
Oxygen	5	0	0	0	0	0	0	1
0 min movement								
Air	5	0	0	0	0	0	0	1
Oxygen	4	0	1	0	0	0	0	1
15 min rest								
Air	5	0	0	0	0	0	0	1
Oxygen	5	0	0	0	0	0	0	1
15 min movement								
Air	4	1	0	0	0	0	0	1
Oxygen	4	1	0	0	0	0	0	1
30 min rest								
Air	5	0	0	0	0	0	0	1
Oxygen	6	0	0	0	0	0	0	0
30 min movement								
Air	2	1	0	0	1	1	0	1
Oxygen	4	1	1	0	0	0	0	0

Table 7.5 VGE grades in six participants who generated VGE breathing air or oxygen for 15-min time periods from 45 to 120 min post-compression at rest and maximum grade after either right arm or leg movements

Time period	VGE grade							
	0	I	II	IIIa	IIIb	IVa	IVb	Uninterpretable
45 min rest								
Air	3	3	0	0	0	0	0	0
Oxygen	5	0	0	0	0	0	0	1
45 min movement								
Air	2	2	0	1	0	1	0	0
Oxygen	1	2	2	0	0	1	0	0
60 min rest								
Air	4	2	0	0	0	0	0	0
Oxygen	5	0	0	0	0	0	0	1
60 min movement								
Air	4	0	0	1	0	1	0	0
Oxygen	4	2	0	0	0	0	0	0
75 min rest								
Air	3	2	1	0	0	0	0	0
Oxygen	6	0	0	0	0	0	0	0
75 min movement								
Air	4	0	1	0	0	1	0	0
Oxygen	4	2	0	0	0	0	0	0
90 min rest								
Air	5	1	0	0	0	0	0	0
Oxygen	6	0	0	0	0	0	0	0
90 min movement								
Air	4	0	1	0	0	0	1	0
Oxygen	6	0	0	0	0	0	0	0
105 min rest								
Air	3	2	0	0	0	0	0	1
Oxygen	5	1	0	0	0	0	0	0
105 min movement								
Air	3	2	0	1	0	0	0	
Oxygen	5	1	0	0	0	0	0	0
120 min rest								
Air	5	0	1	0	0	0	0	0
Oxygen	6	0	0	0	0	0	0	0
120 min movement								
Air	4	1	0	0	1	0	0	0
Oxygen	6	0	0	0	0	0	0	0

Table 7.6 Number of participants categorized by maximum VGE grades, breathing gas and time of measurement; one participant uninterpretable scans for 0 to 30 min

Time period	VGE grade $n = 5$						
	0	I	II	IIIa	IIIb	IVa	IVb
0 min							
Air	5	0	0	0	0	0	0
Oxygen	5	0	1	0	0	0	0
15 min							
Air	4	1	0	0	0	0	0
Oxygen	4	1	0	0	0	0	0
30 min							
Air	2	1	0	0	1	1	0
Oxygen	3	1	1	0	0	0	0
	VGE grade post-dive oxygen or air breathing $n = 6$						
45 min							
Air	2	2	0	0	1	1	0
Oxygen	1	2	2	0	0	1	0
60 min							
Air	3	1	0	1	0	1	0
Oxygen	4	2	0	0	0	0	0
75 min							
Air	3	0	2	0	0	1	0
Oxygen	4	2	0	0	0	0	0
90 min							
Air	4	0	1	0	0	0	1
Oxygen	6	0	0	0	0	0	0
105 min							
Air	3	2	1	0	0	0	0
Oxygen	5	1	0	0	0	0	0
120 min							
Air	3	1	1	0	1	0	0
Oxygen	6	0	0	0	0	0	0

Table 7.7 Maximum VGE grades for participants who generated VGE air versus oxygen for the 45-to-120-min time periods

	Time period					
	45 min	60 min	75 min	90 min	105 min	120 min
Air	IVa	IVa	IVa	IVb	IIIa	IIIb
Oxygen	IVa	I	I	0	I	0

Table 7.8 Number of participants categorized by maximum VGE grades for the 45-to-120-min time period

	VGE grade						
	0	I	II	IIIa	IIIb	IVa	IVb
Air	1	2	1	0	1	0	1
Oxygen	1	2	2	0	0	1	0

Discussion

VGE are generated after a dive and thought to be the inciting factor in DCS.¹ Normobaric oxygen delivered as close as possible to 100% should be administered to divers with early signs and symptoms of DCS.^{1,20} Normobaric oxygen therapy is thought to assist with the removal of inert gas located in the body tissues and VGE. It has been hypothesised that oxygen may decrease VGE generation and assist with involution or distribution of VGE in an injured diver potentially improving their condition.^{1,7} The overall small sample size and low number of participants that generated detectable bubbles limited the ability to perform statistical analysis. However, the preliminary data from the six participants who generated measurable VGE indicated that the VGE levels were generally lower after 30 min of oxygen breathing versus air breathing.

Previous research has shown time to max VGE earlier in oxygen breathing sessions than air breathing sessions.¹⁰ This could represent improved removal of inert gas with oxygen breathing and therefore decreased VGE formation. In the previously mentioned study¹⁰, max VGE grades were reported to be at 40 min after breathing normobaric oxygen whereas in our study the max VGE was at 45 min post-dive when breathing oxygen. This small difference is surely due to the different scanning periods used in the two studies. In our study, 2/6 divers presented equal or higher VGE grades breathing air compared to oxygen at 45 to 120 min post dive. No participant breathing oxygen had VGE detected at 120 min but 3/6 participants breathing air had VGE detected at this time. Our findings suggest that further studies are warranted with a sufficient number of participants and statistical power to answer whether normobaric oxygen may decrease post-dive VGE load.

The NRB mask with an oxygen flow rate of 15 L·min⁻¹ was used in this study as it has been found to provide high levels of oxygenation.^{11,21} Normobaric oxygen therapy was suggested as a treatment for DCS in the late 1800s²² with some uptake of its use documented in the 1960s.²³ In 1991 the Divers Alert Network (DAN) developed an oxygen first aid training program focusing on the delivery of oxygen in diving emergencies.²⁴ Since then there has been minimal published research on the clinical outcome of divers receiving oxygen therapy²⁵ with most research focusing on the efficacy of oxygen delivery systems.^{11,21,26-28} Ensuring the use of optimal oxygen delivery devices may lead to improved outcomes in injured divers and should be used in future studies.

TTE allows the direct visualization of bubbles in the heart, allowing for differentiation between the right and left heart chambers.² This is a benefit over using acoustic Doppler where this differentiation is not possible. Newer portable ultrasound machines are easier to use and have increased the ability to perform field research into DCS prevention.²⁹ Guidelines for the use of ultrasound in diving research have been published³ and should be followed to ensure robust research protocols and the ability to compare research results. Comparison between studies needs to be done with caution, with particular attention to time to first scan, time between scans and duration of scanning periods.³ In this study, pre-dive scanning to locate an optimal cardiac window allowed for immediate scanning after decompression and having only one participant in each dive allowed for 15 min intervals between subsequent scans. Early and frequent measurements are recommended to ensure capture of VGE onset and max values improving the value of studies.³⁰

The majority of participants in this study did not start to produce bubbles until 30 min after the dive (Table 7.4). This was most evident during the movement scans. TTE is performed at rest and after provocative movements to elicit bubble showers.^{2,3,31} It is thought that bubbles seen after movement of a limb may indicate regional origins of the bubbles.² This is an interesting concept making one think the type of exercise used in this study would increase nitrogen on-gassing in the lower limbs and therefore a higher grade of bubbles visualized during leg movements. This was not seen in the results (Appendix B and C). It is possible that a pressure wave could mobilize bubbles off vessel walls and visualization of bubbles could simply reflect the magnitude of the effort. Bubbles appearing immediately upon exercise might be mobilized from areas closer to the heart due to the pressure wave and not come from the working limb. Movement guidelines are published² and these should be standardized in a study and clearly reported.³

DCIEM dive tables are industry standard in Australia and were used to plan the simulated no-decompression research dive because of their safety profile. The tables are known to produce low levels of VGE³² but it was hoped that using set relative exercise intensity would increase uptake of

inert gas and the chance of VGE production. The use of an exercise intensity at 50% $\text{VO}_{2\text{max}}$ did not produce VGE, except in one participant who had produced bubbles in a previous study. The length of time performing exercise was longer than previously used¹⁷, but an increased exercise intensity of 75% of $\text{VO}_{2\text{max}}$ was still required to successfully generate VGE. Furthermore, VGE generation is reportedly higher during ‘wet’ dives^{13,14} and therefore during ‘dry’ dives like during our study, an increased exercise intensity may assist with higher levels of VGE formation.

LIMITATIONS

This study was underpowered with half of the participants not producing post-dive VGE in either arm of the study. Due to COVID ceasing all research in the health system, financial and time constraints, enrolment in the study ceased after recruitment of 12 participants with only six of these participants producing detectable bubbles, thus reducing the sample size. Mathematical modeling has shown that larger paired sample sizes of 30 to 50 provide adequate power for a one grade difference in VGE.³³ Updated consensus guidelines are expected to be published in the December 2025 Diving and Hyperbaric Medicine journal.

The original use of an exercise intensity of 50% $\text{VO}_{2\text{max}}$ for the in-chamber workload did not produce adequate levels of detectable VGE in six participants therefore, these data were not included in the final analysis. Inter-diver variability in VGE generation is known^{3,31} but the use of a fitness-indexed or absolute workload maybe more relevant depending on the research question. Initial disagreement between reviewers was predominately with uninterpretable scans due to the primary researcher attempting to grade all scans. After review, less than 10% of the scans were uninterpretable which was within the study goal but may have contributed to missed scans with bubbles. Participants were asked to make similar lifestyle choices prior to each compression, but changes in diet, exercise, sleep, or other activities may have contributed to differences in VGE with repeated trials.

FUTURE RESEARCH

The type and relative exercise intensity required to induce safe levels of bubbling during a dry dive should be further investigated. This would help ensure an adequate exercise challenge during a dive to generate VGE but with no cases of DCS. Continuing recruitment for this study to a sample size of 30 participants would increase the ability to detect a statistical difference between the two breathing gas groups.³³ Time to commencement and duration of normobaric oxygen therapy should be considered in future studies. Further investigation into the efficacy of different delivery devices to reduce VGE would supplement previous research into oxygen delivery device performance.¹¹

Conclusion

In this study, 2/6 divers presented equal or higher VGE grades breathing air compared to oxygen at 45 to 120 min post dive. No participant breathing oxygen had VGE detected at 120 min but 3/6 participants breathing air had VGE detected at this time. Though this study was underpowered, these preliminary findings suggest a possible decrease in VGE formation with oxygen breathing compared to air. An exercise intensity of 75% $\text{VO}_{2\text{max}}$ during a dry dive may increase the likelihood of VGE generation.

References

- 1 Mitchell SJ. Decompression illness: a comprehensive review. *Diving Hyperb Med.* 2024;54(1 Supp):1-53. doi: 10.28920/dhm53.4.supp.1-53. PMID: 38537300.
- 2 Pollock NW. Use of ultrasound in decompression research. *Diving Hyperb Med.* 2007;37(2):68–72. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/37June/Pollock_dhm.37.2.68-72.pdf
- 3 Møllerløkken A, Blogg SL, Doolette DJ, Nishi RY, Pollock NW. Consensus guidelines for the use of ultrasound for diving research. *Diving Hyperb Med.* 2016;46(1):26-32. PMID: 27044459. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/46March/Mollerlokken_dhm.46.1.26-32.pdf.
- 4 Hyldegaard O, Møller M, Madsen J. Effect of He-O₂, O₂, and N₂O-O₂ breathing on injected bubbles in spinal matter. *Undersea Biomed Res.* 1991;18:361-71. PMID: 1746064. [cited 2025 June 3]. Available from: [https://www.researchgate.net/publication/21409854_Effect_of_He-O₂_O₂_and_N₂O-O₂_breathing_on_injected_bubbles_in_spinal_white_matter](https://www.researchgate.net/publication/21409854_Effect_of_He-O2_O2_and_N2O-O2_breathing_on_injected_bubbles_in_spinal_white_matter).
- 5 Brubakk A. Surface oxygen is an acceptable definitive treatment. *SPUMS J.* 2000;30(3):155-61. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/30Sept/Brubakk_SPUMSJ.30.3.155-161.pdf.
- 6 Gennser M, Loveman G, Seddon F, Thacker J, Blogg SL. Oxygen and carbogen breathing following simulated submarine escape. *Undersea Hyperb Med.* 2014;41:387-92. PMID: 25558547. [cited 2025 June 3]. Available from: <https://www.uhms.org/uhm-search/uhm-journal-volume-41/number-5/oxygen-and-carbogen-breathing-following-simulated-submarine-escape.html>.
- 7 Lippmann J. First aid oxygen administration for divers. *SPUMS J.* 2003;33(4):192-8. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/33Dec/Lippmann_dhm.33.4.192-198.pdf.
- 8 Wang Q, Zhang X, Suo Y, et al. Normobaric hyperoxia therapy in acute ischemic stroke: A literature review. *Heliyon.* 2024;10(1):e23744. Epub 20231220. doi:10.1016/j.heliyon.2023.e23744. PMID: 38223732; PMCID: PMC10787244.

9 Li C, Jiang M, Chen Z, et al. The neuroprotective effects of normobaric oxygen therapy after stroke. *CNS Neurosci Ther.* 2024;30(7):e14858. doi: 10.1111/cns.14858. PMID: 39009510; PMCID: PMC11250159.

10 Blatteau JE, Pontier JM. Effect of in-water recompression with oxygen to 6 msw versus normobaric oxygen breathing on bubble formation in divers. *Eur J Appl Physiol.* 2009;106:691-5. doi: 10.1007/s00421-009-1065-y. PMID: 19424716.

11 Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Leggat, PA, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates. *Diving Hyperb Med.* 2020;50(1):34-42. doi: 10.28920/dhm50.1.34-42. PMID: 32187616 PMCID: PMC7276271.

12 Defence and Civil Institute of Environmental Medicine. DCIEM diving manual. Richmond, BC, Canada. Universal Dive Techtronic, Inc. 1992.

13 Weathersby PK, Survanshi SS, Nishi RY. Relative decompression risk of dry and wet chamber air dives. *Undersea Biomed Res.* 1990;17(4):333-48.

14 Møllerlækken A, Breskovic T, Palada I, Valic Z, Dujic Z, Brubakk AO. Observation of increased venous gas emboli after wet dives compared to dry dives. *Diving Hyperb Med.* 2011;41(3):124-8. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/41Sept/Mollerlokken_dhm.41.3.124-128.pdf

15 Borg GAV. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc.* 1982;14(5):377-81. PMID: 7154893. [cited 2025 June 3]. Available from: https://journals.lww.com/acsm-msse/abstract/1982/05000/psychophysical_bases_of_perceived_exertion.12.aspx

16 American College of Sports Medicine. Guidelines for exercise testing and prescription. 11th ed. Philadelphia (PA): Wolters Kluwer; 2022.

17 Harris RJD, Doolette DJ, Wilkinson DC, Williams DJ. Measurement of fatigue following 18 msw dry chamber dives breathing air or enriched air nitrox. *Undersea Hyperb Med.* 2003;30(4):285-91.

18 Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. *Diving Hyperb Med.* 2024;48(4):308-15. doi: 10.28920/dhm54.4.308-319. PMID: 39675739.

- 19 Faul F, Erdfelder E, Lang A-G, Buchner A. G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*. 2007;39:175-91. doi: 10.3758/bf03193146. PMID: 17695343.
- 20 Mitchell SJ, Bennett MH, Bryson P, Butler F, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med*. 2018;48(1):45-55. doi: 10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.
- 21 Köhler A, Zoll FM, Ploner T, Hammer A, Joannidis M, Tilg H, Finkenstedt A, Hartig F. Oxygenation performance of different non-invasive devices for treatment of decompression illness and carbon monoxide poisoning. *Front Physiol*. 2022;13:885898. doi: 10.3389/fphys.2022.885898. PMID: 35557974 PMCID: PMC9090223.
- 22 Bert P. Barometric pressure [*La pression barometrique*]. Translated by M A Hitchcock and F A Hitchcock College Book Company, Colombus, Ohio, 1943.
- 23 Edmonds C. Underwater oxygen treatment of decompression sickness a review, *SPUMS J*. 1995;25(3):122-8. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/25Sept/Edmonds_SPUMSJ.25.3.122-128.pdf
- 24 Gunderson J. Appendix B. Training and education; pp. 81-4. In: Tillmans F, editor. DAN annual diving report 2020 edition – a report on 2018 diving fatalities, injuries and incidents. Durham, NC: Divers Alert Network; 2020. [cited 2025 June 3]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK582510/>
- 25 Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiburger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. *Undersea Hyperb Med*. 2007;34(1):43-9. PMID: 17393938. [cited 2025 June 3]. Available from: https://www.uhms.org/images/DCS-and-AGE-Journal-Watch/longphre_surface_o2_uhm_2007.pdf.
- 26 Hoffman U, Smerecnik M, Muth C-M. Administration of 100% oxygen in diving accidents-an evaluation of four emergency oxygen devices. *Int J Sports Med*. 2001;22:424-9. doi: 10.1055/s-2001-16247. PMID: 11531035.

27 Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Pollock NW. A comparison of the tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations. *Diving Hyperb Med.* 2018;24;48(4):209-17. doi: 10.28920/dhm48.4.209-217. PMID: 30517952.

28 Komesaroff D. Oxygen administration in diving accidents. *SPUMS J*;1998;28(3 Suppl):20-5. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/28Suppl/Komsaroff_SPUMSJ_28_3Suppl.20-25.pdf.

29 Hjelte C, Plogmark O, Silvanus M, Ekström M, Frånberg O. Risk assessment of SWEN21 a suggested new dive table for the Swedish armed forces: bubble grades by ultrasonography. *Diving Hyperb Med.* 2023;53(4):299–305. doi: 10.28920/dhm53.4.299-305. PMID: 38091588. PMCID: PMC10944666.

30 Blogg SL, Gennser M. The need for optimization of post-dive ultrasound monitoring to properly evaluate the evolution of venous gas emboli. *Diving Hyperb Med.* 2011;41(3):139-49. [cited 7 June 2025] Available from:
https://www.dhmjournal.com/images/IndividArticles/41Sept/Blogg_dhm.41.3.139-146.pdf

31 Doolette DJ, Murphy FG. Within-diver variability in venous gas emboli (VGE) following repeated dives. *Diving Hyperb Med.* 2023;53(4):333-9. doi: 10.28920/dhm53.4.333-339. PMID: 38091593 PMCID: PMC10944664.

32 Gawthrope IC, Summers M, Macey DJ, Playford DA. An observation of venous gas emboli in divers and susceptibility to decompression sickness. *Diving Hyperb Med.* 2015;45(1):25-9. [cited 2025 June 3]. Available from:
https://www.dhmjournal.com/images/IndividArticles/45March/Gawthrope_dhm.45.25-29.pdf

33 Doolette DJ, Gault KA, Gutvik CR. Sample size requirement for comparison of decompression outcomes using ultrasonically detected venous gas emboli (VGE): power calculations using Monte Carlo resampling from real data. *Diving Hyperb Med.* 2014;44(1):14-19. [cited 2025 November 23]. Available from:
https://www.dhmjournal.com/images/IndividArticles/44March/Doolette_dhm.44.1.14-19.pdf

Statement

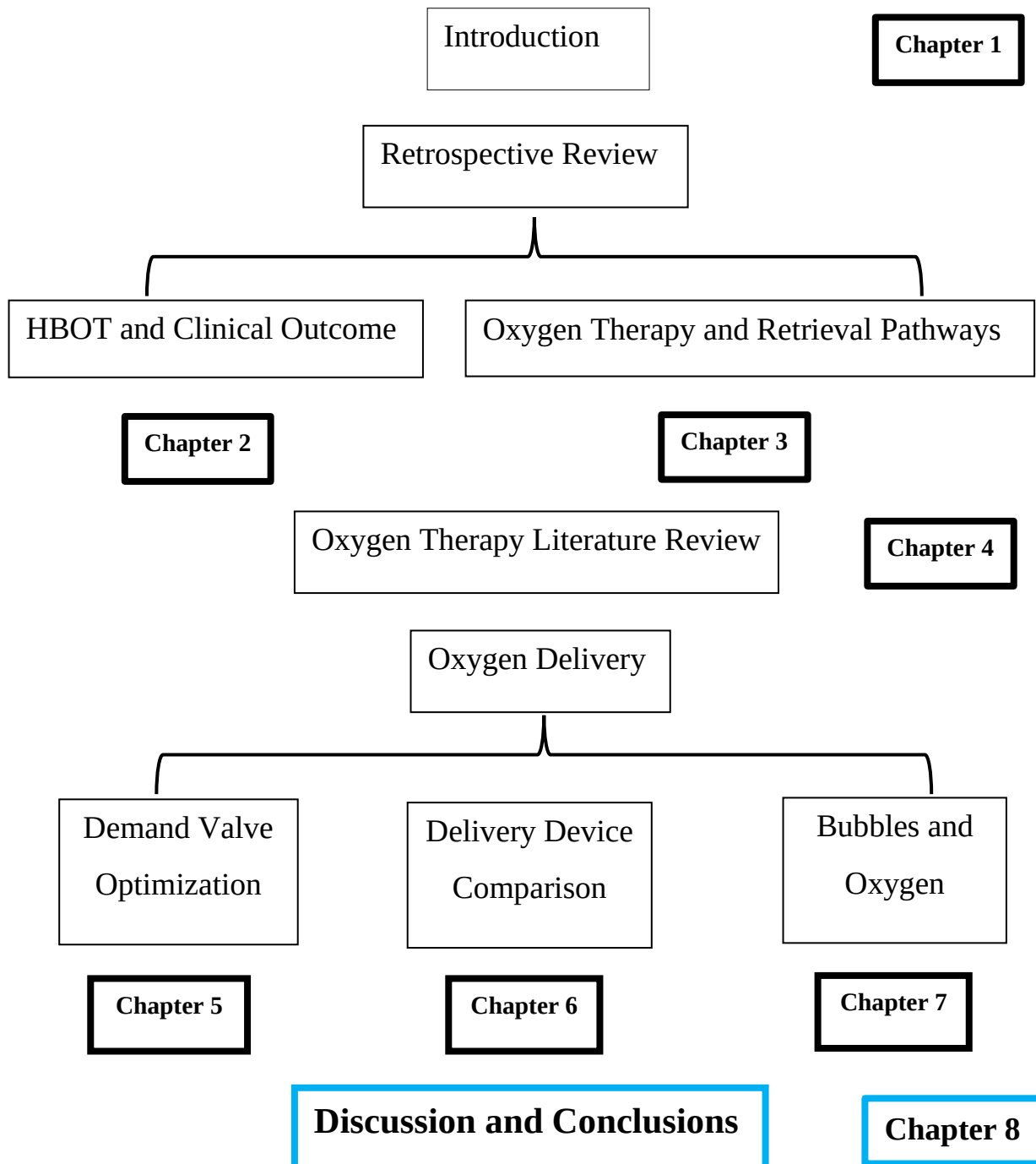
DB conceived and designed the study with input from all authors. DB, MC and AB collected the data. DB, DL and NP analysed, and all authors interpreted the data. DB drafted the manuscript, and all authors revised and approved the final version.

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CHAPTER 8 - GENERAL DISCUSSION AND CONCLUSIONS

This chapter will highlight and integrate the significant findings of the preceding chapters and conclusions that can be drawn from the data. Directions for future research will be suggested and recommendations for clinical practice outlined.



8.1 Problem Statement

Even though diving is a relatively safe sport there are still deaths and injuries each year. During the years from 1970 to 2018 there were 116 diving deaths reported in Australia.¹ Previous published Australian data has focused on mortality reporting and analysis of the chain of events leading to these fatalities.² In 2018, 112 injured divers were treated for decompression illness in Australia (Hyperbaric Technicians and Nurses Association, unpublished data), almost as many injured divers in a year as deaths over 50 years. There has been very little research focusing on the care of injured divers prior to arrival at a hyperbaric medical facility. This thesis aimed to examine the current prehospital care of injured divers in North Queensland, analysing oxygen therapy and retrieval pathways, and determine the optimum first aid oxygen delivery system.

8.2 Key Findings

The findings presented in this thesis were focused on prehospital care in one setting from Queensland, Australia: a retrospective review analysing the retrieval pathways and outcomes of injured divers and prospective studies to define the optimal oxygen delivery device. The main finding of the retrospective review showed that divers with higher initial disease grade had worse outcomes. The results of the study also identified cases that were staged inappropriately, cases that did not require transfer to a hyperbaric facility and adverse effects of oxygen therapy. These findings will provide areas of focus for education for retrieval and emergency physicians and provide insight for the appropriate urgency and staging of diving retrievals. The commonly available oxygen delivery devices promoted for use in injured divers were studied. It was found that when breathing oxygen using the demand valve an intraoral mask (image Appendix E) provided the highest oxygenation and when breathing oxygen using the NRB mask an oxygen flow rate of 15 L·min⁻¹ was required to provide high levels of oxygenation. The medical oxygen rebreather system was found to provide high levels of oxygen with either the air cushion oronasal mask or an intraoral mask. The significance of these results is that there is now clearer guidance on mask selection and oxygen flow rate for providing high levels of first aid oxygen to injured divers.

8.3 Implications of Findings of the Retrospective Studies

Many factors have been suggested to contribute to the risk of decompression sickness but clear associations between these factors and disease severity have been difficult to demonstrate. The incidence of these factors is often reported in retrospective reviews.³⁻⁸ It is important to identify factors contributing to the risk of DCI in order to change diving practice to reduce injury risk. The research questions proposed for this section of the thesis included:

RQ 1. How many injured divers were treated at the Townsville University Hospital in the last 15 years and what were the demographics, medical and dive history, symptoms and clinical outcomes of these divers?

RQ 2. What was the retrieval method and pathway used to transfer the injured divers to Townsville?

RQ 3. Did the injured diver receive normobaric oxygen prior to HBOT and if so, what oxygen delivery device was used?

Chapter 2 of this thesis provides an in-depth examination of the treated injured divers characteristics. Divers presenting to the Townsville HMU were young and just over half of them male. Most were certified with a wide range of reported previous diving experience. Just under half of the divers had a history of a medical or surgical condition and many used a medication (acute or chronic) in the 48 hours before or after the incident dive.

The current retrospective review revealed poor documentation of many of the possible risk factors in the medical records making any association with these factors and outcome difficult. Documentation was improved when the Queensland Diving Injury Assessment form was used (Appendix A). The findings of this thesis indicated that divers with higher initial disease severity had poorer outcomes, and this was the only factor associated with poorer outcomes. Highlighting the implications of severe initial disease grade to clinical coordinators is essential to ensure expeditious retrievals.

Incident dive details including dive site location allowed for assessment of appropriateness of retrieval destination and asset location.⁹ Dive site locations are many and often visited for different objectives, such as open water dive certification courses to seasonal minke whale migration. The distance to the GBR is shorter from Cairns and easier to reach for day trips. Townsville has two commonly dived sites, the new Museum of Underwater Art and the Yongala shipwreck, and Mackay is near the Whitsunday Islands with numerous dive sites. Rotary wing aeromedical assets are located in these three cities allowing for retrieval from difficult locations. Divers with severe initial disease grade were diving near all these cities. The retrospective review identified that divers within a primary retrieval radius of Townsville were diving near these three major cities; 10 were diving in the Cairns area, three in the Townsville area and three in the Whitsundays. Thus, the geographical location of Townsville provides an ideal central location, almost equidistant from Cairns and Mackay, for access to hyperbaric medicine services for injured divers. Townsville is a major regional centre in North

Queensland with a tertiary care hospital and medical school. Importantly, Townsville is of sufficient size to attract and retain hyperbaric and diving medicine specialists, an issue that can be a problem in non-metropolitan areas.¹⁰

Delay to recompression for injured divers varied by severity of disease and distance from Townsville. Extreme retrieval distances were common for international retrievals. The referral pattern to Townsville from international sites has become less over the years as DAN Asia Pacific worked with medical facilities near top dive destinations to install hyperbaric chambers, thus negating the need for medical evacuation to Australia. In the current retrospective review, divers with longest delays to HBOT had mild disease and presented late for medical review. The second reason for long delays to HBOT was extreme distance from Townsville. A larger proportion of divers with severe initial disease grade had extreme retrieval distances, 500 to > 1,700 km. Even though the distances were extreme, divers with severe initial disease grade had shorter time to HBOT commencement. Previous research has shown that similar to the current study, severe initial disease grade was associated with poorer outcomes, but outcome was improved if HBOT was commenced within three hours.³ No diver in the current study received HBOT within three-hours. The substantial delay to HBOT in the current study precluded any meaningful evaluation of effect of time to HBOT on clinical outcome. However, it seems extremely unlikely that with the vast geographical diving area surrounding Townsville that a three-hour goal for HBOT could be attained. Nonetheless, the retrospective review revealed that no diver died and that the severe cases had shorter time to HBOT.

Normobaric oxygen was commonly delivered to divers prior to HBOT. This may be related to workplace legislation in Queensland requiring all dive masters and instructors to be certified in oxygen administration and all dive vessels to have first aid oxygen available (Workplace health and safety Queensland, personal communication, 2023). Poor documentation of delivery device, oxygen flow rate and change of symptom with use limited the further evaluation of individual devices. Updating the Diver Injury Assessment form (Appendix A) to include these details would allow for improved capturing of this data. Furthermore, ensuring the use of optimal oxygen delivery devices found in the second section of this thesis may lead to improved outcomes in injured divers and should be encouraged by those providing care to these divers.

Pulmonary oxygen toxicity was identified as an adverse effect in 33/306 (10%) of the injured divers.⁹ This oxygen toxicity incidence is higher than previously published but not unsurprising with the high use of pre-hospital oxygen available in Queensland, vast retrieval distances and overnight stays in

peripheral hospitals prior to arrival in Townsville for HBOT. Pulmonary oxygen toxicity was a clinical diagnosis made by the treating hyperbaric physician with no formal pre-defined diagnostic criteria. Divers with symptoms of pulmonary oxygen toxicity had a longer duration of normobaric oxygen treatment prior to HBOT than divers without documented oxygen toxicity. Current prehospital recommendations state to have an oxygen “supply sufficient to cover the duration of the most plausible evacuation scenario”.¹¹ In our cohort of divers, the median time to HBOT commencement was 38 h and median time of normobaric oxygen delivery was 10 h.⁹ These time frames should be presented at future workshops examining prehospital recommendations for oxygen treatment of injured divers. One case of pulmonary oxygen toxicity identified in this retrospective review, was of a diver who had normobaric oxygen, then HBOT on a Pacific Island prior to aeromedical retrieval to Townsville for further care. During the international retrieval, the repeated removal of the oxygen mask by the diver was not recognized as oxygen toxicity and the diver was sedated to enforce the wearing of the mask.

Normobaric oxygen treatment is not common between HBOT. However, divers may also have saltwater aspiration leading to hypoxia when breathing air between or after HBOT. Judicious use of oxygen to maintain minimum oxygen saturations at 92% should be recommended rather than the first aid recommendation of using a NRB at 15 L·min⁻¹. Divers may experience pulmonary oxygen toxicity symptoms during or after completing HBOT for DCS. In this cohort of injured divers, the final diagnosis was mild pulmonary oxygen toxicity. The substantial (10%) incidence of pulmonary oxygen toxicity identified in Chapter 3 classifies this as a common adverse effect. Even though the risk of long-term sequelae is low, the symptoms can be distressing for the diver. Divers with symptoms of pulmonary oxygen toxicity need to be counselled and provided with reassurance on the low likelihood of any long-term sequelae.

These adverse effects of oxygen treatment highlight the importance of treating oxygen like a drug, ensuring the administration of the proper dose. This was one of the knowledge areas identified from the retrospective review as requiring education for retrieval and emergency physicians. Oxygen therapy is not a one dose fits all divers. A drug monograph for oxygen is presented in Appendix F and should be part of the training package for divers, retrieval, emergency and diving physicians. The guidelines have stated that 100% oxygen be given to divers as soon as possible after symptom onset and continued for the entire retrieval.¹¹ There are some subtleties that need to be considered when delivering oxygen to divers. The above-mentioned pulmonary oxygen toxicity issues highlight some areas to be considered when providing oxygen therapy. The case identified in the retrospective review: If a diver has already received HBOT then the original inert gas burden is most definitely

resolved. If the diver is hypoxic during aeromedical transfer, low levels of oxygen should be provided, but the use of a NRB mask at 15 L·min⁻¹ is not required. Saltwater aspiration may accompany DCI, especially after CAGE or other event that led to a decreased level of consciousness. This often resolves with time but use of oral antibiotics and low dose oxygen for hypoxia may be required. If a diver develops symptoms of oxygen toxicity reconsider the current oxygen therapy. During HBOT an air break may be beneficial. During normobaric oxygen therapy the dose and duration of the treatment needs to be considered. Reassess the diver. If the symptoms of DCS have resolved, then consider cessation of the oxygen. If DCS symptoms persist, educate the diver about oxygen toxicity and discuss the risk and benefits of continued treatment. When discharging the diver after treatment, ensure the diver understands the nature of oxygen toxicity and that oxygen toxicity in its early stages is reversible and extremely unlikely to cause long term sequelae.¹²

Not all injured divers in this study required recompression and therefore, did not need transfer to a hyperbaric facility. Some divers that did not receive HBOT had a non-diving final diagnosis, but some had a diving related injury that did not require recompression. Immersion pulmonary oedema was identified as a problem not commonly seen, presenting with severe respiratory distress and not requiring HBOT. Education for medical practitioners and retrieval physicians often focuses on DCS and AGE. Since the findings of this study, presentations by the PhD candidate on immersion pulmonary oedema have been given to the Townsville clinical coordinators, retrieval and emergency physicians. This was an opportunity to increase awareness of this problem to non-diving physicians, leading to better outcomes for these patients and better allocation of the limited resources (retrieval assets and HBOT). Insight into the reasons for a primary retrieval provided by the retrospective review has led to a more comprehensive understanding of the acutely unwell diver and identified divers that do not require HBOT. Thus, early advice from diving physicians can assist with better treatment decisions and appropriate destination choice.

The retrospective review identified two divers who were doing decompression diving and identified as inappropriately staged overnight during their retrieval to Townsville. Decompression diving, by its nature, carries a higher load of inert gas. The lack of awareness of decompression diving was identified as a knowledge gap for retrieval and emergency physicians. The changing face of recreational diving to include diving once thought to be in the realm of 'technical divers' (nitrox, trimix and closed-circuit rebreathers) presents new challenges for those caring for injured divers. Diving physicians need to remain current, have a good understanding of the diving being performed in their catchment area, and ensure this information is provided to all the members along the retrieval

pathway. Diving physicians should assist with curriculum updates especially for clinical coordinators, retrieval and emergency physicians.

8.4 Implications of Oxygen Delivery Studies

Normobaric oxygen therapy is thought to assist with the removal of inert gas located in the body tissues and bubbles, provide oxygen to hypoxic tissue and reduce inflammation.^{13,14} It has been hypothesized that oxygen may decrease bubble generation, evolution or distribution in an injured diver potentially improving their condition.^{12,15} The purpose of this section of the thesis was to identify the oxygen delivery device that provides the highest concentration of oxygen for the treatment of divers with suspected DCS. The first study (Chapter 4) aimed to systematically review the literature surrounding normobaric oxygen treatment of injured divers to find any evidence that oxygen contributes to symptom reduction and improved clinical outcome. The systematic review research question was:

RQ 4. What is the published literature surrounding VGE reduction or clinical outcome in injured scuba divers or animal models after treatment with normobaric oxygen prior to hyperbaric oxygen therapy?

Current guidelines for the administration of first aid oxygen for injured divers have been written based on expert opinion.¹¹ No systematic review or other strong evidence that could guide the clinician regarding the use of normobaric oxygen therapy in injured scuba divers was found. There was a paucity of well conducted peer-reviewed articles examining normobaric oxygen treatment and clinical outcome in divers with suspected DCI. Most articles identified for this review examined the efficacy of oxygen delivery systems rather than focusing on outcomes of injured divers. Understanding which delivery devices provide the best oxygenation could improve treatment and lead to better outcomes for injured divers. Evidence was provided supporting a reduction in bubbles with oxygen breathing and guidance on delivery device selection. Retrospective reviews found that breathing oxygen led to a decrease in hyperbaric treatments required but no significant improvement in final outcome. This limited evidence does point to benefits for delivering high concentrations of normobaric oxygen prior to HBOT but more well controlled research is needed to confirm these initial findings.

The two studies following the systematic review (Chapters 5 and 6) were the first to use transcutaneous oximetry (TCOM) to assess the efficacy of currently available oxygen delivery

systems promoted for use with injured divers. TCOM is a non-invasive way to measure tissue oxygen delivery, decreasing risk to study participants compared to invasive arterial catheter placement. Common DCS symptoms are thought to be caused by peripheral tissue inert gas bubbles, therefore measuring tissue oxygenation seems ideal for a study of first aid oxygen treatment for injured divers. The results of these studies provide research evidence of the oxygenation levels with the varied devices and flow rates. The research questions posed for these two studies were:

RQ 5. In healthy divers, when delivering oxygen with a demand valve which oxygen mask configuration provides the highest peripheral tissue oxygen partial pressure determined using transcutaneous oximetry?

RQ 6. In healthy divers, when delivering oxygen with commonly used devices and flow rates is there a difference in peripheral tissue oxygen partial pressure determined using transcutaneous oximetry?

The demand valve has been promoted as an ideal oxygen delivery device as oxygen is only delivered on inspiration decreasing wastage and increasing the duration of delivery.¹² Previous research in 2015 showed that when breathing oxygen with a demand valve using a silicone oronasal mask, lower levels of oxygenation were attained than when breathing oxygen with a NRB mask with an oxygen flow rate of 15 L·min⁻¹.¹⁶ The results of this study were presented at a diving medicine conference and the results criticized by a conference attendee stating there must be a flaw in the study as the demand valve should provide higher levels of oxygenation. This previous research was the instigating factor for the studies presented in this thesis. The replicated study in this thesis showed that the silicone oronasal mask provided with the oxygen system and used in the previous study¹⁶, had a poor fit and provided the lowest levels of tissue oxygen. The use of an intraoral mask overcame the poor facial fit and was found to provide the highest level of oxygenation of the masks tested. This intraoral mask has been promoted as providing the highest oxygen level with the publication of articles in dive magazines by the PhD candidate, both in English (Appendix D) and French (Appendix E), and with the presentation of the results to divers and medical personnel. The important practical application of the oxygen delivery study findings is exemplified by recent email correspondence with a representative from DAN America (personal communication, Francois Burman) which confirmed that the silicone oronasal mask is no longer sold with their systems. The intraoral mask used in this study is no longer available. The author of the thesis is working in conjunction with a commercial diver/researcher to develop a new intraoral mask based on the feedback from the participants in the demand valve optimization study and will be the focus of future research.

Oxygen delivery flow rate using the NRB mask has historically been 15 L·min⁻¹ to provide high levels of oxygenation. In order to conserve oxygen, the recommended flow rate was changed to 10 to 15 L·min⁻¹.¹⁷ The oxygen delivery study presented in this thesis (Chapter 6) found that tissue oxygenation was significantly lower when the oxygen flow rate was decreased to 10 L·min⁻¹ and that the NRB mask with oxygen at 15 L·min⁻¹ provided the highest level of tissue oxygenation of all devices tested. The common advice is that the NRB mask can provide high levels of oxygen even when the oxygen flow rate is decreased, as long as the reservoir bag does not deflate. This was found to be untrue. The reservoir bag did not deflate in either arm of the study, with the oxygen flow rate of 10 or 15 L·min⁻¹. The results show that if the goal of oxygen therapy is to provide near 100% oxygen for divers with symptoms of DCS¹¹, the NRB mask needs to be used with a flow rate of 15 L·min⁻¹. To further disseminate this important information, in addition to the peer-reviewed publication of this study, these results have also been published in dive magazines, both in English and French, and presented to divers and medical personnel. Publishing results in both medical journals and dive magazines will widen the spread of these research results to the varied providers of oxygen therapy and thus have the potential to improve the outcomes for injured divers.

A medical oxygen rebreather system, promoted for use in Europe¹⁸ but with little awareness of its use in the recreational, scientific or commercial diving industries in Australia and Canada, was studied for this thesis (Chapter 6). A rebreather is a closed loop breathing system that contains (1) a counterlung, (2) a carbon dioxide absorbent material, (3) tubing contains one-way valves and (4) and an oxygen supply.¹⁹ The counterlung holds the exhaled gas until it is inhaled again. The absorbent material, primarily soda lime, absorbs the carbon dioxide in the exhaled gas. The tubing contains one-way valves to ensure the exhaled gas moves in the correct direction. Oxygen is added to replenish the amount metabolised by the user but with substantially lower flow rates (0.8 to 1.5 L·min⁻¹) than other free flow devices. The system permits the rebreathing of the substantial unused oxygen content of each breath. When dive sites are remote and retrieval distances vast, this is an ideal system to extend oxygen delivery time. However, prior to publishing the study presented in Chapter 6, there was very little research investigating the effectiveness of oxygen rebreather systems as studies focused on system function.^{20,21} Production of a previous DAN America designed systems was stopped due to difficulties obtaining the scrubber canisters.²¹

The results presented in Chapter 6 of this thesis found that the oxygen rebreather system could provide oxygenation comparable to both the demand valve with intraoral mask and the NRB mask with an oxygen flow rate of 15 L·min⁻¹. The use of a rebreather system considerably extends the oxygen duration of a standard first aid oxygen D size cylinder (425 L) from 28 min with a flow rate of

15 L·min⁻¹ to almost 8 h. Providing awareness of this device and promoting its use started in early 2023 with the PhD candidate presenting as an invited speaker at a dive medical technician (DMT) course in Kelowna, BC, Canada. Since then, the candidate has become a DMT instructor accredited by the International Marine Contractors Association (IMCA) and annually teaches on the DMT course. The candidate was recently an invited speaker at the Canadian Underwater Conference and Exhibition (March 2025), presenting her research results and providing hands on use of the rebreather system to commercial divers from across Canada. Increasing the awareness of the oxygen delivery devices available for treating injured divers, appropriate mask selection and optimal flow rate will assist dive operators in selecting the system appropriate for their site and the mask and oxygen flow rate to provide near 100% oxygen. Oxygen mask selection and flow rate are important factors in ensuring the provision of high levels of oxygenation to injured divers. Enhanced understanding of the best oxygen delivery device could lead to improved treatment and outcomes in injured divers.

The current consensus guidelines recommend providing near 100% normobaric oxygen to injured divers but admit that there is limited research to support this advice.¹¹ Indeed, in the retrospective review presented in this thesis, poor documentation of the oxygen delivery device used, and any symptom change as a result of breathing oxygen meant that it was not possible to analysis device efficacy. Therefore, it was necessary to investigate whether VGE could be attenuated by breathing normobaric oxygen in comparison to breathing air. This was addressed in Chapter 7 and the research question for this study was:

RQ 7. In healthy divers, does administration of normobaric oxygen after a simulated dive change venous gas emboli grade determined by transthoracic echocardiography compared to breathing air?

The results of the study presented in Chapter 7 revealed that simulating dives in a hyperbaric chamber enabled the assessment of VGE grades after participants breathed air and oxygen. In this study, VGE were consistently higher during air breathing than oxygen breathing, with measured bubbles persisting for a longer period of time in the air breathing arm, but the overall low number of participants that generated detectable bubbles limits the ability to fully interpret these results. This study provided some initial data on decreased VGE grades after breathing oxygen via a NRB mask with oxygen flow rate of 15 L·min⁻¹, but further research is required to verify these findings.

The results also provided insight into the exercise intensity required to induce VGE in healthy divers during a dry dive, with an initial 50% VO_{2max} intensity producing bubbles in only one diver. Increasing the exercise intensity to 75% VO_{2max} caused bubbles in all the remaining participants. The

exercise periods in the present study were utilised to increase the uptake of inert gas and be provocative enough to overcome the DCIEM table conservatism and stimulate post-dive VGE production.^{22,23}

8.5 Strengths and Limitations

This thesis presents the first study assessing the retrieval pathways and appropriateness of primary retrieval destinations and staging for divers treated at the Townsville HMU. This research has provided valuable information supporting Townsville as an ideal location for a hyperbaric facility and identified areas for education to clinical coordinators, retrieval and emergency physicians.

This thesis also presents the first studies to use TCOM to assess tissue oxygen levels for assessing oxygen delivery device efficacy. TCOM is non-invasive and therefore decreases risk to study participants. The most common presenting symptoms in our cohort of injured divers were paraesthesia and arthralgia/myalgia.²⁴ These DCS symptoms are thought to be caused by tissue inert gas bubbles. The goals of normobaric oxygen therapy for injured divers is to eliminate nitrogen in the breathing gas to accelerate its elimination from the body, reduce bubble size and provide oxygen to hypoxic tissues.¹⁵ Increasing the concentration gradient from inert gas bubbles to the surrounding tissues facilitates the off gassing of inert gasses. Therefore, measuring peripheral tissue oxygenation seems ideal for a study of first aid oxygen delivery in injured divers where off gassing is the primary goal.

This thesis presents the first study to use a commercially available intraoral mask for oxygen delivery using a demand valve system designed for the treatment of injured divers. Previous advice has been to use a scuba regulator mouthpiece with the demand valve²⁵ or oxygen rebreather system (provided with the Wenoll-System). The regulator mouthpiece opening is oval shaped to fit on a scuba second stage. The intraoral mask opening is circular and compatible with both the demand valve regulator and the rebreather system tubing ensuring a good fit. The intraoral mask sits in the mouth between the teeth and the gums in a fashion similar to a regulator mouthpiece. Compatibility with oxygen delivery systems and a familiar oral shape for divers makes this an ideal mask for use with the demand valve or rebreather system.

TTE allows for direct visualization of bubbles in the heart chambers, allowing for differentiation between the right and left heart chambers.²⁶ VGE enter the right side of the heart and travel to the

lungs. VGE and dissolved inert gas are eliminated from the body via the lungs through respiration. Movement of bubbles from the right atria to the left atria of the heart, for example with a patent foramen ovale, can result in these bubbles being released into the arterial system causing AGE. It is, therefore, important when designing a new dive protocol for generating VGE for research purposes that safe levels of VGE are generated and the risk of DCI is low. Using TTE in this thesis allowed for the direct visualization of both right and left heart bubbles allowing for the increase of exercise intensity, from 50% $\text{VO}_{2\text{max}}$ to 75% $\text{VO}_{2\text{max}}$, to generate VGE but with the assurance that bubbles were not seen in the left heart chamber. TTE is thus recommended for future studies investigating VGE.

All research has limitations. The retrospective studies presented in this thesis were limited by incomplete records and missing data. Missing data may have contributed to the difficulty to detect associations between initial disease severity, contributing factors, timelines, and clinical outcomes. Poor documentation limited the ability to assess the efficacy of oxygen delivery devices. There was no documentation on tasking deliberations or resource availability, therefore, determination of the appropriateness of destination and retrieval timeliness was decided based on the available medical and retrieval notes. Improved documentation of tasking deliberations would provide a better understanding of the choice of retrieval destination and timelines. This would allow the determination of any knowledge gaps for the coordination staff in the areas of divers needing urgent retrieval to a hyperbaric facility and those able to be managed locally.

The oxygen delivery studies were performed on healthy individuals and did not address the clinical efficacy of the devices in treating DCS. Participants only breathed oxygen for 10 min from each device. Longer breathing periods may provide different mask comfort ratings. The simulated chamber dive study enabled the evaluation of VGE grade after breathing oxygen compared to air but was underpowered as half the participants did not produce post-dive VGE in either arm of the study. Therefore, statistical analysis was not undertaken on the data and the data presented as preliminary findings using descriptive analysis. Time constraints did not allow for further recruitment to this study. However, it does provide preliminary evidence for the effectiveness of oxygen in reducing VGE and thus provides a basis for future research.

8.6 Directions for Future Research

There is a paucity of research into the prehospital treatment of injured divers. Data presented in this thesis indicate the importance of initial disease severity and clinical outcome and provide a starting point for further research in this area. A detailed retrospective analysis of the severe cases identified in

the retrospective should be performed to identify any dive practice or other factors that contribute to disease severity. This could determine areas that could be modified to improve outcome as initial disease severity was the only factor associated with a poor outcome in the retrospective study.

A prospective study would greatly improve data collection and our understanding of factors contributing to initial disease severity and outcome. It is hoped that the new Australasian Diving Safety Foundation sponsored Australia-wide injured diver data base will provide some insight into identifying high risk dive sites, risk factors, prehospital treatment and outcomes, helping to focus educational opportunities.

The incidence of clinically significant pulmonary oxygen toxicity was 10% in this study and therefore classified as a common adverse effect. It is not clear whether imposing air breaks during oxygen delivery would compromise DCI treatment, or at what time point in a long evacuation process that air breaks should occur. Further research should be conducted to clarify the timing and duration of air breaks during normobaric oxygen delivery treatment.

Since the commencement of the candidate's doctoral program, the intraoral mask used in the oxygen studies is no longer available. A new intraoral mask is being designed based on the feedback of the study participants to the researchers. Further research should be performed using this mask to ensure it functions similarly to the one promoted in this thesis.

8.7 Recommendations

The inciting event that launched this series of studies was the Divers Alert Network changing the recommendation for the oxygen flow rate when using a NRB mask from 15 L·min⁻¹ to 10 to 15 L·min⁻¹.²⁷ The rationale for this change was to conserve oxygen but with no research on the delivery of oxygen at the lower flow rate. It was found that peripheral tissue oxygenation was statistically lower when breathing using a NRB at 10 L·min⁻¹.²⁸ Since the goal of prehospital treatment for injured divers is to provide near 100% oxygen¹¹, the higher flow rate should be recommended. However, many other factors have been identified during this PhD.

During the oxygen studies, TCOM values were seen to precipitously drop in two participants. This was probably due to vasoconstriction caused by the high levels of oxygen. This drop was short lived

but illustrates the fact that treatment with high levels of oxygen may in fact decrease perfusion leading to a decrease in oxygenation and inert gas off gassing. This is now part of a SPUMS diploma project for another clinician (An investigation into the pathophysiological vasomotor changes associated with hyperoxia and how these may influence normobaric oxygen supplementation in the management of patients with suspected decompression illness).

Clinically diagnosed pulmonary oxygen toxicity was seen in 10% of the injured divers treated in Townsville over a 15-year period.⁹ Divers travelled vast distances and received considerable amounts of oxygen prior to arriving in Townsville for assessment and HBOT. Current recommendations are to ensure enough oxygen for continued administration to cover a plausible evacuation.¹¹ Certainly if a diver is hypoxic (at altitude during aeromedical retrievals or with concurrent pulmonary problems) oxygen should be delivered, but if not hypoxic should there be a time frame for reassessment and cessation of oxygen? With the loss of urgent care support at many facilities in the USA (data presented and discussed at UHMS meetings), long transfer times for HBOT for injured divers may become an issue in other jurisdictions.

There is also a change in diving practices that may contribute to earlier pulmonary oxygen toxicity. Divers are breathing nitrox with concentrations of oxygen in gas mixtures of 28 to 32% commonly used. Rebreather circuits are also being used by recreational divers allowing a variable concentration of oxygen to decrease decompression obligations. These divers are still at risk of DCI and may be at risk of earlier onset of pulmonary symptoms due to their increased exposure to oxygen during dives.

Oxygen is a drug and thus would benefit from being treated as such with recommendations to provide the dose, route of administration and length of treatment. Taking this approach, the findings from this thesis have been integrated with previous literature to develop a drug monograph for medical oxygen for the treatment of injured divers (Appendix F).

The data presented in this thesis suggests there would be benefit from the revision and promotion of the use of the Queensland Diving Injury Assessment form (Appendix A) to collect information on oxygen delivery device, flow rate and duration of treatment. Including these details would allow for improved capturing of data on oxygen use and correlation with clinical outcome. Other recommended changes to the form are to divide thermal stress into cold and overheated and include phase of the dive at which this occurred. Current diving practice would indicate that a 60 min surface interval between

dives is now considered standard, therefore, this item should be changed from 120 to 60 min. Lastly, documenting the incident dive location would assist in identifying high risk dive sites and allow for improved analysis of retrieval pathways and time to HBOT.

8.8 Conclusions

The North Queensland hyperbaric medicine unit is ideally located midway along the GBR in Townsville with coastal aeromedical bases in Thursday Island, Cairns, Townsville and Mackay appropriately placed in areas of high diving. Most injured divers received oxygen first aid and were transported to Townsville for definitive care with a variable number of stages. Many different retrieval assets and legs were necessary when dive sites were remote and distances vast. These findings have provided valuable insight into the retrieval pathways of divers transported to Townsville and identified areas of education for retrieval physicians. All three available oxygen delivery systems studied and promoted for use with injured divers can provide high oxygenation levels when used with the appropriate mask and oxygen flow rate: demand valve with an intraoral mask, NRB mask at an oxygen flow rate of $15 \text{ L} \cdot \text{min}^{-1}$, and oxygen rebreather system with either an air cushion or intraoral mask. Though underpowered, preliminary findings suggest a possible decrease in VGE formation with oxygen breathing compared to air following a simulated dive as assessed using TTE. These results have provided an understanding of the varied systems available and will provide valuable information for divers and medical personal to assist with device selection in diving emergencies.

References

- 1 Lippmann J. A review of snorkelling and scuba fatalities in Queensland, Australia, 2000 to 2019. *Diving Hyperbaric Medicine*. 2022;52(2):108-18. doi: 10.28920/dhm52.2.108-118. PMID: 35732283.
- 2 Lippmann J. Diving-related fatalities in Victoria, Australia, 2000 to 2022. *Diving Hyperb Med*. 2025;55(1):35-43. doi: 10.28920/dhm55.1.35-43. PMID: 40090024.
- 3 Andre S, Lehot H, Morin J, Louge P, de Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. *Emerg Med J*. 2022; 39:747-52. doi: 10.1136/emermed-2021-211227. PMID: 35135892.
- 4 Gempp E, Blatteau J-E. Risk factors and treatment outcomes in scuba divers with spinal cord decompression sickness. *J Crit Care*. 2010;25:236–42. doi: 10.1016/j.jcrc.2009.05.011. PMID: 19682840.
- 5 Blatteau, J-E, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: influence of time to recompression. *Diving Hyperb Med*. 2011;41(3):129–34. PMID: 21948497. Available from: https://www.dhmjournal.com/images/IndividArticles/41Sept/Blatteau_dhm.41.3.129-134.pdf.
- 6 Haas RM, Hannam JA, Sames C, Schmidt R, Tyson A, Francombe M, et al. Decompression illness in divers treated in Auckland, New Zealand, 1996-2012. *Diving Hyperb Med*. 2014;44(1):20–5. PMID: 24687481. Available from: https://www.dhmjournal.com/images/IndividArticles/44March/Haas_dhm.44.1.20-25.pdf.
- 7 Thaler J, Pignel R, Magnan M-A, Pellegrini M, Louge P. Decompression illness treated at the Geneva hyperbaric facility 2010-2016: a retrospective analysis of local cases. *Diving Hyperb Med*. 2020;50:370–6. doi: 10.28920/dhm50.4.370-376. PMID: 33325018. PMCID: PMC8038901.
- 8 Danker R, Gall N, Freidman G, Arad J. Recompression treatment of Red Sea diving accidents: A 23-year summary. *Clin J Sport Med*. 2005;15:253–6. doi: 10.1097/01.jsm.0000168074.89744.4f. PMID: 16003040.
- 9 Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review. *Diving Hyperb Med*. 2025;55(2):79-90. doi: 10.28920/dhm55.2.79-90. PMID: 40544136.
- 10 Martin P, Walker C, Sangelaji B, Elliott J, O'Malley L, Van Erp A, Chong HS, Coglán A, Fallon T. Using archival employee data to examine retention patterns for healthcare workers in non-metropolitan Australia: a survival analysis. *BMC Health Serv Res*. 2025;25:605. doi.org/10.1186/s12913-025-12725-6.

- 11 Mitchell SJ, Bennett MH, Bryson P, Butler F, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med.* 2018;48(1):45-55. doi: 10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.
- 12 Mitchell SJ. Decompression illness: a comprehensive review. *Diving Hyperb Med.* 2024;54(1 Supp):1-53. doi: 10.28920/dhm53.4.supp.1-53. PMID: 38537300.
- 13 Wang Q, Zhang X, Suo Y, et al. Normobaric hyperoxia therapy in acute ischemic stroke: A literature review. *Heliyon.* 2024;10(1):e23744. Epub 20231220. doi:10.1016/j.heliyon.2023.e23744. PMID: 38223732; PMCID: PMC10787244.
- 14 Li C, Jiang M, Chen Z, et al. The neuroprotective effects of normobaric oxygen therapy after stroke. *CNS Neurosci Ther.* 2024;30(7):e14858. doi: 10.1111/cns.14858. PMID: 39009510; PMCID: PMC11250159.
- 15 Lippmann J. First aid oxygen administration for divers. *SPUMS J.* 2003;33(4):192-8. [cited 2024 November 21]. Available from:
https://www.dhmjournal.com/images/IndividArticles/33Dec/Lippmann_dhm.33.4.192-198.pdf
- 16 Blake DF, Naidoo P, Brown LH, Young D, Lippmann J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates. *Diving Hyperb Med.* 2015;45(2):79-83. (Partial retraction March 2016). [cited 27 June 2025]. Available from:
https://www.dhmjournal.com/images/IndividArticles/45June/Blake_dhm.45.2.79-83.pdf
- 17 Bird N, Nochetto M. Emergency oxygen for scuba diving injuries: student handbook. 9th ed. Durham: Divers Alert Network; 2021. [cited 27 June 2025]. Available from: <https://dan.org/wp-content/uploads/2021/08/dan-emergency-oxygen-handbook-v3.pdf>
- 18 The Diving Medical Advisory Committee. Medical equipment to be held at the site of an offshore diving operation. DMAC 15 Rev. 5. London; February 2025. [cited 21 June 2025]. Available from: <https://www.dmac-diving.org/guidance/DMAC15.pdf>
- 19 Wendling J. Normobaric oxygen in dive accidents: a challenge for the developers of oxygen delivery systems. *SPUMS J.* 1997;27(2):101-4. [cited 27 June 2025]. Available from:
https://www.dhmjournal.com/images/IndividArticles/27June/Wendling_SPUMSJ.27.2.101-104.pdf
- 20 Komesaroff D. Oxygen administration in diving accidents. *SPUMS J.* 1998;28(3):20-5. (Supplement). [cited 27 June 2025]. Available from:
https://www.dhmjournal.com/images/28Suppl/Komsaroff_SPUMSJ_28_3Suppl.20-25.pdf

- 21 Pollock NW, Natoli MJ. Performance characteristics of the second-generation remote emergency medical oxygen closed-circuit rebreather. *Wilderness Environ Med.* 2007;18(2):86-94. doi: 10.1580/06-WEME-OR-032R.1. PMID: 17590070.
- 22 Weathersby PK, Survanshi SS, Nishi RY. Relative decompression risk of dry and wet chamber air dives. *Undersea Biomed Res.* 1990;17(4):333-48. PMID: 2396332.
- 23 Møllerlækken A, Breskovic T, Palada I, Valic Z, Dujic Z, Brubakk AO. Observation of increased venous gas emboli after wet dives compared to dry dives. *Diving Hyperb Med.* 2011;41(3):124-8. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/41Sept/Mollerlokken_dhm.41.3.124-128.pdf
- 24 Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. *Diving Hyperb Med.* 2024;54(4):308-15. doi: 10.28920/dhm54.4.308-319. PMID: 39675739.
- 25 Davis M. Oxygen therapy equipment a theoretical review. *SPUMS J.* 1998;28(3):165-72. [cited 27 June 2025]. Available from: https://www.dhmjournal.com/images/IndividArticles/28Sept/Davis_SPUMSJ.28.3.165-172.pdf
- 26 Pollock NW. Use of ultrasound in decompression research. *Diving Hyperb Med.* 2007;37(2):68–72. [cited 2025 June 3]. Available from: https://www.dhmjournal.com/images/IndividArticles/37June/Pollock_dhm.37.2.68-72.pdf
- 27 Bird N, Nochetto N. Emergency oxygen for scuba diving injuries: student handbook, 9th ed. Durham: Divers Alert Network; 2021. [cited 2 December 2025]. Available from: <https://dan.org/wp-content/uploads/2021/08/dan-emergency-oxygen-handbook-v3.pdf>
- 28 Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Leggat, PA, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates. *Diving Hyperb Med.* 2020;50(1):34-42. doi: 10.28920/dhm50.1.3.34-42. PMID: 32187616.

Assessing Potential Diving Related Illness

90% of illnesses after diving are dive-related

The attached form is a guide for assessing unwell divers

Unique specialities like Diving Medicine are difficult especially if your experience is limited. This guide aims to be user friendly and straight forward. Add any information you feel necessary and do not feel obligated to complete every detail regarding the patient's clinical picture.

History taking should be self-explanatory

The front of the form deals with diving history, risks for diving illnesses and general medical history. The back of the form outlines examples of symptoms experienced by the patient and related examination findings. The focus of assessment is mainly neurological and musculoskeletal systems. Some history hints include:

- Use the patient's log book or computer to get the dive profile information.
- A **Decompression (deco) Stop** is a planned stop in accordance with dive tables.
- A **Safety Stop** is a recommended stop done even if a 'deco' stop is unnecessary.

Examination is essentially the same for any patient

Specific dive-related examinations include the Sharpened Romberg Test and looking for **Barotrauma** (LUNGS-pneumothorax / surgical emphysema and EAR). **Do not perform this test if you suspect CAGE (Cerebral Arterial Gas Embolism). Keep patient supine.**

Sharpened Romberg Test (SRT)✧

Sharpened Romberg Test, when performed properly, is very beneficial to diver assessment:

Patient stands with feet in heel to toe position and arms crossed over the chest. Once stable, the patient should close their eyes and attempt to maintain that position for 60 seconds. If the patient fails to maintain the position, by movement of either arms or feet or by opening their eyes, the time taken to failure is noted. If not achieved, the patient is to attempt up to 3 further trials of 60 seconds. If any attempt is successful, no further testing is required.

Record results in the 'SRT results' section (page 2) 'Diving Injury Assessment' form.

If you are suspicious of an illness requiring Hyperbaric Therapy or you need advice, please contact:

Diving Emergency Services (funded by DAN)
24hr Emergency Hotline: **1800 088 200**

OR

Hyperbaric Medicine Unit
The Townsville Hospital: **07 4433 1111**
Royal Brisbane and Women's Hospital: **07 3636 8111**

If the patient needs to attend a Hyperbaric Unit

- He / she must travel either by land (<200 metres altitude) or by aircraft pressurised to sea level equivalent. Discuss transport options with the Hyperbaric Team. **You** will need to organise transport. If person comes by personal car please ensure they are not the driver.
- Administer 100% oxygen (with air breaks as per advice).
- Keep the patient well hydrated (either oral or IV fluids).
- Tell the patient that:
 - » The first treatment takes a minimum of almost 5 hours;
 - » Subsequent treatments are once a day;
 - » Anywhere from 1–8 treatments may be necessary (average is 2–4);
 - » Treatment is usually as an outpatient;
 - » Present to the hospital main entrance and ask for directions to the Hyperbaric Medicine Unit;
 - » Illness may impact on air travel after treatment.
- Please ensure patient's belongings are transported with them.

Photocopy the completed 'Diving Injury Assessment' form to send as your referral letter

Assessing Potential Diving Related Illness

Decompression Illness (DCI) results from the development of bubbles within the body.

DCS - Decompression Sickness ('The Bends')*

DCS is a subset of DCI. This term specifically refers to the formation of bubbles from dissolved nitrogen or another inert gas. It is a complex process, which may potentially involve many systems, with the spinal cord and peripheral nerves thought to be commonly involved (e.g. musculoskeletal, neurological, dermatological etc).

Symptom onset is often within 30–60 minutes of surfacing; nearly all within 24 hours and unlikely after 48 hours.

- **Mechanisms are through Nitrogen bubbles:**

- » Forming in tissues - causing pressure on and compression of various structures;
- » Forming in vessels - causing altered blood flow;
- » Activating inflammatory proteins, cascades and reactions.

- **Hyperbaric Oxygen Therapeutic mechanisms include:**

- » ↑ Pressure → ↓ Nitrogen bubble size;
- » Oxygen to hypoxic tissues
- » Accelerating nitrogen elimination;
- » Anti-inflammatory.

- **Symptoms of DCS include:**

- | | | | |
|------------------------|----------------------------------|-------------------|----------------------|
| » Rash | » Dizziness | » Weakness | » Headache |
| » Difficulty breathing | » Paralysis | » Joint pain | » Nausea |
| » Pruritus | » Paraesthesia / tingling | » Extreme fatigue | » Difficulty walking |
| » Visual disturbances | » Altered level of consciousness | | |

CAGE - Cerebral Arterial Gas Embolism*

CAGE is a subset of DCI. This term refers specifically to the introduction of bubbles to the arterial circulation by pulmonary barotrauma or PFO. **If you suspect CAGE keep the patient supine.**

Rapid neurological syndrome as a result of gas bubbles in the arteries supplying the brain.

Associated with rapid ascent, breath holding leading to barotrauma or a large nitrogen load.

Clinical picture includes any rapid onset with altered neurology (e.g. ↓ GCS, power, co-ordination) within 10 mins of surfacing due to the passage +/- trapping of bubbles in the cerebral circulation. Divers usually show an initial improvement or recovery. Systemic response is one of cerebral vasodilation and ↑ BP with relapse within hours. This delayed deterioration is due to bubble activated inflammatory reactions and damage to vessel walls.

The air bubbles enter the circulation as a result of shearing and tearing of pulmonary tissue. Surprisingly enough, the incidence of CAGE and pneumothorax / mediastinum together is quite low but still must be considered. There is also the possibility of nitrogen venous bubbles entering the arterial circulation via a patent Foramen Ovale (28%–30% of the population), but most venous bubbles are 'filtered' by the pulmonary circulation. Bubbles in larger vessels distribute according to buoyancy, therefore if diver is vertical they can go to the brain. Obviously though, intravascular bubbles are not limited to the cerebral circulation.

- **DCI definition: includes DCS and CAGE - describing the clinical picture in terms of:**

- » Acute / chronic
- » Evolution (progressive, stable / static, relapsing, resolving etc);
- » Organ system involved (neurological - central or peripheral, musculoskeletal etc);
- » +/- degree of severity (mild / moderate / severe);
- » Evidence of barotrauma (ear - middle / inner, pulmonary-pneumothorax / mediastinum / CAGE);
- » e.g. acute, stable, mild neurological DCS.

It is very difficult to differentiate between Cerebral DCS and CAGE. Sometimes both may be present. However, it is unnecessary to differentiate between the two as treatment is the same.

Photocopy the completed 'Diving Injury Assessment' form to send as your referral letter



Diving Injury Assessment

Facility:

(Affix identification label here)

URN:

Family name:

Given name(s):

Address:

Date of birth:

Sex: ☐ M ☐ F ☐ I

Staff should be familiar with the Clinical Practice Guideline 'Diving Injuries' prior to commencement. Refer to 'Assessing Potential Diving Related Illness' for instructions.

Supine position if significant barotrauma or suspect CAGE (Cerebral Arterial Gas Embolism)

☐ Yes ☐ No

Dive profile

Date	Depth (m)	Dive time (min)	Surface interval (min)	Safety stop (min)

Breathing gas: ☐ Air ☐ Nitrox (..... % O₂) ☐ Other (specify:)

Diving apparatus: ☐ Scuba ☐ Rebreather ☐ Surface supply

Risk factors for decompression sickness / barotrauma

Rapid ascent	☐ Yes ☐ No	Altitude >200m since dive	☐ Yes ☐ No
Breath holding during ascent	☐ Yes ☐ No	Repetitive dives in single day (>3)	☐ Yes ☐ No
Difficulty equalising ears	☐ Yes ☐ No	Reverse profiles (shallow then deep)	☐ Yes ☐ No
Previous decompression sickness	☐ Yes ☐ No	Multiple ascents	☐ Yes ☐ No
Dehydration	☐ Yes ☐ No	Inadequate safety stops	☐ Yes ☐ No
Obesity	☐ Yes ☐ No	Inadequate surface interval (<2 hours)	☐ Yes ☐ No
Excessive physical exertion during dive	☐ Yes ☐ No	Multiple days of diving (>3)	☐ Yes ☐ No
Dive in excess of table / computer	☐ Yes ☐ No		

History:

.....

.....

.....

Symptoms

ALOC	☐ Yes ☐ No	Paraesthesia	☐ Yes ☐ No
Weakness	☐ Yes ☐ No	Severe lethargy	☐ Yes ☐ No
Nausea or vomiting	☐ Yes ☐ No	Arthralgia / myalgia	☐ Yes ☐ No
Poor concentration	☐ Yes ☐ No	Palpitations	☐ Yes ☐ No
Behavioural change	☐ Yes ☐ No	Chest pain	☐ Yes ☐ No
Vertigo	☐ Yes ☐ No	Dyspnoea	☐ Yes ☐ No
Poor balance or ataxia	☐ Yes ☐ No	Rash	☐ Yes ☐ No
Hearing loss or tinnitus	☐ Yes ☐ No		

Time to onset of symptoms post dive:

Symptoms improved by (e.g. O₂, fluid, re-dive):

.....

Symptoms worsened by:

Management prior to attending emergency department:

.....

.....

.....

Photocopy the completed 'Diving Injury Assessment' form to send as your referral letter

DO NOT WRITE IN THIS BINDING MARGIN

v2.00 - 10/2012



SW210

DIVING INJURY ASSESSMENT



Diving Injury Assessment

(Affix identification label here)

URN:

Family name:

Given name(s):

Address:

Date of birth:

Sex: ☐ M ☐ F ☐ I

Examination

Pulse: BP: Temp: RR: O₂Sats:

Neurological - peripheral nerves

Tone ☐ NAD ☐ Abnormal
Power ☐ NAD ☐ Abnormal
Reflexes ☐ NAD ☐ Abnormal
Sensation ☐ NAD ☐ Abnormal

If abnormal, specify:

Neurological - cranial nerves

II-XII ☐ NAD ☐ Abnormal

Neurological - cerebellum

Nystagmus ☐ NAD ☐ Abnormal
Finger nose ☐ NAD ☐ Abnormal
Dysdiadokinesis ☐ NAD ☐ Abnormal
Heel - shin ☐ NAD ☐ Abnormal
Gait ☐ NAD ☐ Abnormal ☐ N/A

Higher mental function

Serial 7s ☐ NAD ☐ Abnormal
MMSE / 30
SRT results 1: / 60 2: / 60
(see Assessing Potential 3: / 60 4: / 60
Diving Related Illness
for instructions) ☐ N/A

Joints and muscles

Local tenderness ☐ NAD ☐ Abnormal

ENT

Tympanic membrane ☐ NAD ☐ Abnormal

Other

Abd ☐ NAD ☐ Abnormal
Chest ☐ NAD ☐ Abnormal
Rash ☐ Yes ☐ No

Other examination findings:

Investigations (CXR if any evidence of pulmonary barotrauma):

Management

Oxygen ☐ Yes ☐ No Case discussed with Hyperbaric Medicine Unit ☐ Yes ☐ No
Oral fluid ☐ Yes ☐ No Intravenous fluid ☐ Yes ☐ No

Differential diagnosis:

Dr Signature:

Printed name:

Time:

Date:

Photocopy the completed 'Diving Injury Assessment' form to send as your referral letter

Appendix B Venous gas emboli (VGE) grades for all 12 participants prior to breathing air or oxygen (raw data) for 15-min time periods from 0 to 30 min post-compression; RA - after right arm movements; RL - after right leg movements

Time period	VGE Grade							
	0	I	II	IIIa	IIIb	IVa	IVb	Uninterpretable
0 min rest								
Air	11	0	0	0	0	0	0	1
Oxygen	11	0	0	0	0	0	0	1
0 min RA								
Air	9	0	0	0	0	0	0	3
Oxygen	9	0	1	0	0	0	0	2
0 min RL								
Air	10	0	0	0	0	0	0	2
Oxygen	10	0	0	0	0	0	0	2
15 min rest								
Air	11	0	0	0	0	0	0	1
Oxygen	11	0	0	0	0	0	0	1
15 min RA								
Air	9	1	0	0	0	0	0	2
Oxygen	8	1	0	0	0	0	0	3
15 min RL								
Air	8	1	0	0	0	0	0	3
Oxygen	9	1	0	0	0	0	0	2
30 min rest								
Air	11	0	0	0	0	0	0	1
Oxygen	12	0	0	0	0	0	0	0
30 min RA								
Air	9	0	0	0	1	1	0	1
Oxygen	10	1	0	0	0	0	0	1
30 min RL								
Air	8	1	0	1	0	0	0	2
Oxygen	9	1	1	0	0	0	0	1

Appendix C Venous gas emboli (VGE) grades for all 12 participants breathing air or oxygen (raw data) for 15-min time periods from 45 to 120 min post-compression; RA-after right arm movements; RL-after right leg movements

Time period	VGE Grade							
	0	I	II	IIIa	IIIb	IVa	IVb	Uninterpretable
45 min rest								
Air	9	3	0	0	0	0	0	0
Oxygen	11	0	0	0	0	0	0	1
45 min RA								
Air	10	0	0	1	0	1	0	0
Oxygen	6	1	2	0	0	0	0	3
45 min RL								
Air	7	2	1	1	0	0	0	1
Oxygen	8	2	0	0	0	1	0	1
60 min rest								
Air	10	2	0	0	0	0	0	0
Oxygen	11	0	0	0	0	0	0	1
60 min RA								
Air	10	0	0	1	0	1	0	0
Oxygen	10	2	0	0	0	0	0	0
60 min RL								
Air	9	0	0	2	0	0	0	1
Oxygen	10	0	0	0	0	0	0	2
75 min rest								
Air	9	2	1	0	0	0	0	0
Oxygen	12	0	0	0	0	0	0	0
75 min RA								
Air	9	0	1	0	0	1	0	1
Oxygen	11	1	0	0	0	0	0	0
75 min RL								
Air	9	0	1	0	0	1	0	1
Oxygen	11	1	0	0	0	0	0	0
90 min rest								
Air	10	1	0	0	0	0	0	1
Oxygen	12	0	0	0	0	0	0	0
90 min RA								
Air	10	0	1	0	0	0	1	0
Oxygen	12	0	0	0	0	0	0	0
90 min RL								
Air	9	0	1	0	0	0	0	2
Oxygen	12	0	0	0	0	0	0	0
105 min rest								
Air	9	2	0	0	0	0	0	1
Oxygen	11	1	0	0	0	0	0	0
105 min RA								
Air	9	1	0	1	0	0	0	1
Oxygen	12	0	0	0	0	0	0	0

105 min RL								
Air	9	1	1	0	0	0	0	1
Oxygen	10	1	0	0	0	0	0	1
120 min rest								
Air	11	1	0	0	0	0	0	0
Oxygen	12	0	0	0	0	0	0	0
120 min RA								
Air	10	1	0	0	1	0	0	0
Oxygen	12	0	0	0	0	0	0	0
120 min RL								
Air	10	0	1	0	0	0	0	1
Oxygen	11	0	0	0	0	0	0	1



In this test, the intraoral mask (NuMask) delivered the highest tissue oxygen levels and was easiest to use.

Evaluating Oxygen Delivery with a Demand Valve

By Denise Blake, M.D., and Neal Pollock, Ph.D.

Oxygen administration is an important early first aid measure for divers with suspected decompression illness (DCI). The goal is to deliver oxygen at the highest possible oxygen concentration while being mindful of oxygen supply limits. Oxygen systems commonly used for dive accidents can have two delivery options: a pressure-activated demand valve or constant flow for use with a nonrebreather mask.

The demand valve has been promoted as an easy-to-use system that conserves the oxygen supply since it releases oxygen only when the user inhales. Recent research, however, found that the nonrebreather mask set at 15 liters per minute can provide better tissue oxygenation than the demand valve with an oronasal mask.¹ Researchers suspected that poor mask fit led to the underperformance of the demand valve. This

article will summarize an evaluation of different mask configurations to determine if they influence the oxygen delivery fraction when breathing with the demand valve.²

METHODS

Sixteen healthy volunteer divers (both male and female) tested four different mask configurations: an adult soft silicone oronasal mask (Tru-Fit mask by Allied Health Care Products Inc.), a pocket face mask with air cushion (Sturdy Industrial Co.), and an intraoral mask (NuMask) with a nose clip and without a nose clip.

The dead space volume, representing the volume of gas that will be breathed (or rebreathed) from a sealed mask, was measured prior to testing. Oxygenation was measured in three different ways: the fraction delivered to the mouth (inspired oxygen fraction (FiO_2)), the fraction reaching the back of the throat (nasopharynx) and tissue



STEPHEN FRANK

oxygen levels measured through skin transcutaneous oximetry. Sensors were taped on six standard sites on the arms and legs, the skin was warmed to ensure good blood flow, and the levels of tissue oxygen were recorded at regular intervals throughout the oxygen breathing periods. After the oxygen breathing, the participants rated each mask configuration on comfort, ease of breathing and ease of holding the device in place.

RESULTS

The oxygen fraction delivered to the mouth was similar for all configurations. The nasopharyngeal FiO_2 was significantly higher with the intraoral mask (both with and without the nose clip) than with the pocket face mask or silicone oronasal mask. The tissue oxygen levels achieved were statistically similar for the intraoral mask both with and without the nose clip. The silicone oronasal mask delivered statistically lower tissue oxygen levels (Figure 1).

Participants rated the pocket face mask as the most comfortable and the nose clip as very uncomfortable. They described the intraoral mask (like a scuba regulator mouthpiece) as the easiest to use since it rests in the mouth when the subjects are lying on their backs. Constant pressure was required to maintain a seal with the other two masks. The intraoral mask had a very small dead space volume (14 milliliters), meaning little gas would be rebreathed. The dead space

was eight times larger for the pocket face mask and 14 times larger for the silicone oronasal mask.

DISCUSSION

The demand oxygen delivery system was designed to provide effective first aid oxygen to injured divers. Evaluating tissue oxygen levels is the best way to assess oxygen delivery effectiveness. Our findings confirm that the type of mask used with the oxygen delivery system can affect performance. Both the intraoral mask and the pocket face mask were superior to the silicone oronasal mask in performance. The intraoral mask with a nose clip offered the best performance but not statistically better than the intraoral mask without a nose clip.

Divers are accustomed to breathing through a mouthpiece, so the intraoral mask is easy to use, and facial hair and face shape do not interfere with the fit. It is easy to hold in place and continued to maintain a good seal under a wide range of conditions.

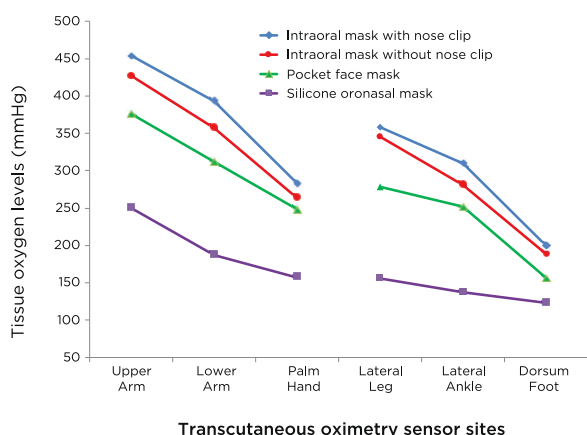
The large dead space volume of the oronasal mask has both positive and negative effects. While the large volume can serve as a reservoir for the oxygen supply, it also means that there is more rebreathing of expired gas, diluting the fresh oxygen supply with expired carbon dioxide. Fidgeting with the mask can introduce more surrounding air that can dilute the inspired oxygen fraction. There is much less expired gas rebreathing or ambient air dilution with the intraoral mask.

The intraoral and pocket face masks come with a filter and one-way valve that allows the masks to be used for mouth-to-mouth resuscitation in a nonbreathing diver. The filter and one-way valve assembly need to be removed before attaching these masks to the demand valve.

CONCLUSION

We tested three commonly available oxygen masks used with a demand valve for first aid oxygen delivery. Of these, the intraoral mask with a nose clip obtained the highest tissue and inspired oxygen levels in healthy divers when used with a demand valve. Oxygen values were similar for the intraoral mask with and without the nose clip. Given the reported discomfort of the nose clip, the use of the intraoral mask without the nose clip may be the best of the options tested. **AD**

Figure 1. Tissue oxygen levels while breathing oxygen using a demand valve with four different mask configurations



REFERENCES

1. Blake DF, Naidoo P, Brown LH, Young D, Lippmann J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates. *Diving Hyperb. Med.* 2015; 45(2):79-83.
2. Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations. *Diving Hyperb. Med.* 2018; 48(4):209-17.

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Évaluation de l'apport en oxygène avec la valve à la demande



Texte : Denise Blake, M.D.,
et Neal W. Pollock, Ph. D.
(traduction de
Denise Blake et
Margaux Hein)

Photos : Denise Blake
et Margaux Hein

La communauté des plongeurs comprend que l'administration d'oxygène est une mesure de premiers secours importante pour les plongeurs soupçonnés d'accident de décompression. L'objectif est d'apporter de l'oxygène à la concentration la plus élevée possible, tout en tenant compte des limites d'approvisionnement en oxygène. Cet article résume une évaluation de différentes configurations de masques pour déterminer si cela peut influencer la fraction d'apport en oxygène lors de la respiration avec la valve à la demande.

Les systèmes d'administration d'oxygène¹ couramment utilisés pour les accidents de plongée peuvent avoir deux options de distribution : 1) à débit constant pour une utilisation avec un masque sans recycleur et 2) une valve à la demande activée par la pression.

La valve à la demande a été présentée comme un système à la fois simple d'utilisation et pouvant conserver l'apport d'oxygène, car il ne libère de l'oxygène que lorsque l'utilisateur inspire. Cependant, des recherches récentes ont montré que le masque sans recycleur réglé à 15 litres/min peut fournir une meilleure oxygénation des tissus que la valve à la demande avec un masque bucco-nasal

(Blake et collab., 2015). Nous étions portés à croire que le mauvais ajustement du masque entraînait la sous-performance de la valve à la demande.

Méthode

Quatre configurations de masque différentes ont été testées auprès de 16 plongeurs volontaires sains (hommes et femmes) :

- un masque bucco-nasal en silicone souple pour adultes (masque Tru-Fit fabriqué par Allied Health Care Products);
- un masque facial de poche avec coussin d'air (par Sturdy Industrial Co.);
- un masque intraoral (NuMask) avec une pince nasale; et
- un masque intraoral sans pince nasale (voir Photo 1).

Le volume de l'espace mort, qui représente le volume de gaz qui sera respiré (ou réinspiré) à partir d'un masque scellé, a été mesuré avant les tests. L'oxygénation a été mesurée de trois manières différentes : 1) par la fraction transmise à la bouche (fraction inspirée de l'oxygène [FiO_2]); 2) par la fraction atteignant l'arrière de la gorge (nasopharynx); et 3) par les niveaux d'oxygène tissulaire mesurés par l'oxymétrie transcutanée de la peau (voir graphique à la page suivante).

Des capteurs ont été collés sur six emplacements standards sur les bras et les jambes. La peau a été réchauffée pour assurer une bonne circulation sanguine et les niveaux d'oxygène tissulaire ont été enregistrés à intervalles réguliers pendant les périodes de respiration d'oxygène. À la fin de la respiration d'oxygène, on a demandé aux participants d'évaluer chaque configuration de masque en matière de confort, de facilité de respiration et de maintien du dispositif en place.

Photo 1 : Masque intraoral (en haut à gauche), masque bucco-nasal (en haut à droite) et masque facial de poche (en bas à droite) utilisés avec la valve à la demande. La pince nasale avec le masque intraoral n'est pas montrée.



Photo 2 : Masque intraoral avec la pince nasale, la valve à la demande et les deux cathéters pour mesurer l'oxygène inspiré.



¹ Tous les détails sont disponibles dans la version originale de l'article de Blake et collab., 2018.

Résultats

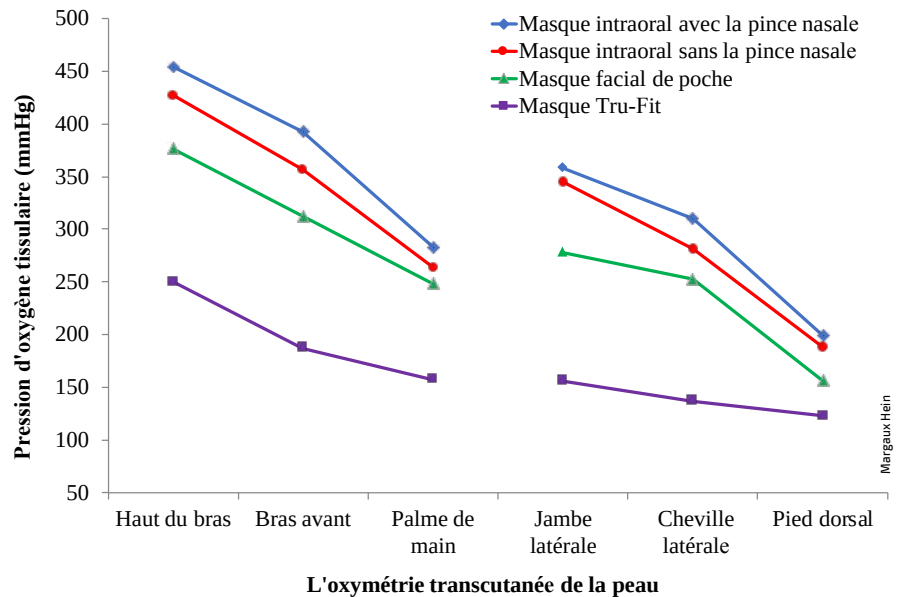
La fraction d'oxygène administrée à la bouche était similaire pour toutes les configurations. La FiO_2 nasopharyngée était significativement plus élevée avec le masque intraoral (avec et sans pince nasale) qu'avec le masque facial de poche ou le masque bucco-nasal. Les niveaux d'oxygène tissulaire obtenus étaient statistiquement similaires pour le masque intraoral avec et sans pince nasale. Le masque bucco-nasal a fourni des taux d'oxygène tissulaire statistiquement moins élevés (voir graphique ci-contre). Le masque facial de poche a été jugé comme étant le plus confortable, tandis que la pince nasale a été jugée comme étant très inconfortable. Le masque intraoral (comme un embout de régulateur de plongée) a été décrit comme étant le plus facile à utiliser, car il reposait dans la bouche lorsque le sujet était couché sur le dos. Une pression constante était nécessaire pour maintenir l'étanchéité avec les deux autres masques. Le masque intraoral avait un très petit volume de l'espace mort (14 ml), ce qui signifie que peu de gaz serait réinspiré. L'espace mort pour le masque facial de poche était 8 fois plus grand, et pour le masque bucco-nasal, 14 fois plus grand.

Discussion

Le système de distribution d'oxygène à la demande a été conçu pour fournir de l'oxygène de premiers secours efficace aux plongeurs blessés. L'évaluation des niveaux d'oxygène dans les tissus est le meilleur moyen d'évaluer l'efficacité de l'administration d'oxygène. Nos résultats confirment que le type de masque utilisé avec le système peut avoir un impact sur la performance. Le masque intraoral et le masque facial de poche ont des performances supérieures à celles du masque bucco-nasal. Le masque intraoral avec pince nasale offrait les meilleures performances, mais pas statistiquement meilleures que le masque intraoral sans pince nasale.

Les plongeurs sont habitués à respirer à travers un embout buccal; le masque intraoral est donc facile à utiliser. Les poils et la forme du visage n'interfèrent pas avec l'ajustement. Il est facile à maintenir en place et continuerait à maintenir une bonne étanchéité dans un large éventail de conditions.

Le grand volume de l'espace mort des masques bucco-nasaux a des effets à la fois positifs et négatifs. Bien que le volume important puisse servir de réservoir pour l'apport en oxygène, cela signifie également que le gaz expiré est davantage réintroduit, ce qui dilue l'apport d'oxygène frais avec du dioxyde de carbone expiré. Le fait de bouger avec le masque peut introduire plus d'air ambiant, qui peut diluer la fraction d'oxygène inspirée. Il y aurait beaucoup moins de réinhalation de gaz expiré ou de dilution dans l'air ambiant avec le masque intraoral.



Les masques intraoral et de poche sont livrés avec un filtre et une valve unidirectionnelle qui permettent aux masques d'être utilisés pour la réanimation bouche à bouche chez un plongeur qui ne respire pas. L'ensemble filtre/valve unidirectionnelle doit être retiré avant de fixer ces masques à la valve à la demande.

Résultats d'oxygène tissulaire en respirant de l'oxygène à l'aide de la valve à la demande avec quatre configurations de masque différentes.

Conclusion

Trois masques à oxygène couramment utilisés avec une valve à la demande pour l'administration d'oxygène de premiers secours ont été testés. Parmi ceux-ci, le masque intraoral avec pince nasale utilisant la valve à la demande a obtenu les taux les plus élevés d'oxygène tissulaire et d'oxygène inspiré chez des plongeurs en bonne santé. Les valeurs en oxygène étaient similaires entre le masque intraoral avec et sans pince nasale. Compte tenu de l'inconfort rapporté de la pince nasale, l'utilisation du masque intraoral sans la pince nasale pourrait être la meilleure des options testées. ■

RÉFÉRENCES

- BLAKE D. F., NAIDOO, P., BROWN, L. H., YOUNG, D. et LIPPMANN, J. A comparison of the tissue oxygenation achieved using different oxygen delivery devices and flow rates [Comparaison de l'oxygénation des tissus obtenue à l'aide de différents dispositifs de délivrance d'oxygène et de débits différents], *Diving and Hyperbaric Medicine*, vol. 45, n° 2, 2015, p. 79-83.
- BLAKE, D. F., CROWE, M., LINDSAY, D., BROUFF, A., MITCHELL, S. J. et POLLOCK, N. W. Comparison of tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations [Comparaison de l'oxygénation des tissus réalisée en respirant de l'oxygène à partir d'une valve à la demande avec quatre configurations de masque différentes], *Diving and Hyperbaric Medicine*, vol. 48, n° 4, 2018, p. 209-217.

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Appendix F Drug monograph for medical oxygen for treatment of injured divers

Mode of action: Breathing a high concentration of oxygen increases the diffusion gradient between the alveoli and blood vessels such that more inert gas moves into the lungs to be exhaled. This will also supply oxygen to hypoxic tissues.

Indications: Divers with suspected decompression sickness or arterial gas embolism, collectively called decompression illness. Divers with immersion pulmonary oedema, suspected saltwater aspiration or near-drowning. Divers with suspected lung barotrauma including pneumothorax, subcutaneous or mediastinal emphysema.

Precautions: Oxygen should never be used near an open flame or anything that can produce intense heat, flames or sparks.

Adverse effects: Pulmonary oxygen toxicity - pleuritic chest pain, substernal heaviness, burning on inhalation, shortness of breath, and coughing. Incidence 10%.¹ Symptoms may commence after 12 hours of normobaric oxygen use. Long-term sequelae are rare. May occur during hyperbaric oxygen treatment.

Central nervous system (CNS) oxygen toxicity - nausea, visual changes (flashing lights, tunnel vision), muscular twitching especially lips or hands, tinnitus, and seizures. CNS oxygen toxicity requires a high partial pressure of oxygen such as provided in a hyperbaric chamber or during in-water recompression.

Dosage: Near 100%. Titrate the dose according to response to treatment. Assess the diver for a change in their symptoms (resolution, improvement or ongoing symptoms). Monitor for signs and symptoms of oxygen toxicity if treatment is prolonged. See above for signs and symptoms of oxygen toxicity.

Route of administration: Inhaled

Administration advice: Three devices are commonly available for prehospital oxygen administration for injured divers.

1. Non-rebreather mask: use an oxygen flow rate of 15 L·min⁻¹ for divers with suspected DCI.
2. Demand valve: use with an intraoral mask or air-filled cushion mask (pocket face mask).
3. Medical oxygen rebreather system: use with either an intraoral mask or an air-filled cushion mask. Follow the device operating instructions for oxygen flow rates and timing. Gas economy makes this an attractive choice for resource-limited environments.

Practice points: Prolonged duration of oxygen delivery may require a change in device selection for comfort and ease of administration.

Use the non-rebreather mask or medical oxygen rebreather system during sleep.

A supine position is better for nitrogen off-gassing in a diver with decompression sickness.² This position is also recommended in divers with suspected AGE to prevent cephalad distribution of arterial bubbles.³

Oxygen treatment should continue until arrival at a hyperbaric facility. Ensure adequate oxygen supplies for a likely evacuation scenario especially in remote locations with long distances to medical care. If evacuation duration is lengthy, assess the diver for signs and symptoms of oxygen toxicity. If oxygen toxicity is present, obtain advice from a diving medicine specialist on the risk and benefits of continued treatment. Modification to treatment may be advised including the addition of air breathing periods.

If oxygen supplies are limited, a flow rate of 10 L·min⁻¹ can be used for divers with suspected hypoxia as seen in immersion pulmonary oedema, saltwater aspiration or near drowning. This lower flow rate is inadequate for divers with suspected DCI.

References

- 1 Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Oxygen treatment and retrieval pathways of divers with diving-related conditions in Townsville, Australia: a 15-year retrospective review. *Diving Hyperb Med.* 2025;55(2):79-90. doi: 10.28920/dhm55.2.79-90. PMID: 40544136.
- 2 Balldin U. Effects of ambient temperature and body position on tissue nitrogen elimination in man. *Aerospace Med.* 1973;44:365-70. PMID: 4694843.
- 3 Mitchell SJ, Bennett MH, Bryson P, Butler F, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med.* 2018;48(1):45-55. doi: 10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.