

Joint position statement on immersion pulmonary oedema and diving from the South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC) 2024

Neil Banham¹, David Smart², Peter Wilmshurst³, Simon J Mitchell^{4,5,6}, Mark S Turner⁷, Philip Bryson⁸

¹ Department of Hyperbaric Medicine, Fiona Stanley Hospital, Perth, Australia

² Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Australia

³ Cardiology Department, Royal Stoke University Hospital, Stoke on Trent, United Kingdom

⁴ Department of Anaesthesiology, University of Auckland, Auckland, New Zealand

⁵ Department of Anaesthesia, Auckland City Hospital, Auckland, New Zealand

⁶ Slark Hyperbaric Unit, North Shore Hospital, Auckland, New Zealand

⁷ Bristol Heart Institute, Bristol, United Kingdom

⁸ TAC Healthcare Group, Wellheads Industrial Estate, Aberdeen, United Kingdom

Corresponding author: Dr Neil Banham, Department of Hyperbaric Medicine, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch WA 6150, Australia

neil.banham@health.wa.gov.au

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Abstract

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This joint position statement (JPS) on immersion pulmonary oedema (IPO) and diving is the product of a workshop held at the 52nd Annual Scientific Meeting of the South Pacific Underwater Medicine Society (SPUMS) from 12–17 May 2024, and consultation with the United Kingdom Diving Medical Committee (UKDMC), three members of which attended the meeting. The JPS is a consensus of experts with relevant evidence cited where available. The statement reviews the nomenclature, pathophysiology, risk factors, clinical features, prehospital treatment, investigation of and the fitness for future compressed gas diving following an episode of IPO. Immersion pulmonary oedema is a life-threatening illness that requires emergency management as described in this statement. A diver with previous suspected or confirmed IPO should consult a medical practitioner experienced in diving medicine. The SPUMS and the UKDMC strongly advise against further compressed gas diving if an individual has experienced an episode of IPO.

Introduction

This joint position statement is the product of a workshop held at the 52nd Annual Scientific Meeting of the South Pacific Underwater Medicine Society (SPUMS) from 12–17 May 2024, and following consultation with the United Kingdom Diving Medical Committee (UKDMC), three members of which attended the meeting. The joint position statement is a consensus of experts with relevant evidence cited where available.

The purpose of the statement is to provide medical practitioners with guidance regarding immersion pulmonary oedema (IPO) and diving, and in particular the emergency management of IPO and return to diving following a

diagnosed episode of IPO, or an event in which there is a high degree of suspicion.

The statement must be interpreted in consultation with a medical practitioner experienced in diving medicine and will be subject to review based on new evidence becoming available.

Definition

Immersion pulmonary oedema is acute pulmonary oedema that occurs in divers, snorkellers and swimmers whilst immersed. Some individuals have developed pulmonary oedema when surface swimming and on other occasions when scuba diving, which suggests that the main pathogenic mechanisms are related to immersion.^{1,2}

It may affect the following people when immersed:

- Divers breathing compressed gas using scuba, 'hookah', other surface supply, and rebreather apparatus
- Snorkelers and breath-hold divers
- Swimmers, particularly triathletes, open water swimmers and combat swimmers with head out immersion

Nomenclature

Immersion pulmonary oedema is also known as:

- Immersion pulmonary edema (IPE) (US spelling)
- Swimming induced pulmonary oedema/edema (SIPO/SIPE)
- Scuba divers pulmonary oedema/edema (SDPO/SDPE)

Pathophysiology

The hydrostatic effect of head out immersion on peripheral veins causes redistribution of blood centrally, which increases heart size and significantly increases cardiac filling pressures, including pulmonary capillary pressure.³ At the same time, because the lung centroid is below the surface of the water, respiration is with a continuous negative airway pressure equal to the vertical distance between the lung centroid and the surface of the water.⁴

This combination of increased pulmonary capillary pressure and negative airway pressure creates a pressure gradient for transudation of fluid from pulmonary capillary blood into the alveoli. However, given these physiological changes when immersed are essentially ubiquitous while IPO is relatively uncommon, it seems that for frank pulmonary oedema to develop additional factors are usually required (see risk factors below).

In a diver, the hydrostatic effects of immersion on redistribution of blood are identical to head out immersion, but the effects on intrapulmonary pressures are dependent on the type of breathing equipment used, the relation between the diver's lung centroid and the breathing gas source, and gas density.⁵ An increase in external inspiratory resistance from breathing equipment and increased resistance to flow through airways due to dense gas will cyclically exaggerate any negative airway pressures during inspiration. As in those with head-out immersion, additional factors are usually required before frank pulmonary oedema develops.

There is individual predisposition to immersion pulmonary oedema as indicated by the fact that individuals can get recurrent episodes.^{1,2,6-11}

In addition, some studies have shown that divers affected have haemodynamic differences compared with divers who have never had IPO.^{1,10}

Occasionally individuals that have had IPO experience pulmonary oedema in other extreme circumstances, which is in keeping with an increased susceptibility.^{1,12}

It is also clear that IPO can be fatal.^{8,13}

Risk factors

A number of risk factors for IPO have been identified:

Intrinsic:

- Previous episode of immersion pulmonary oedema^{1,2,6-11}
- Female sex^{11,14}
- Older age¹¹
- Hypertension and / or pre-existing cardiovascular disease^{1,2,13,15-17}

Extrinsic:

- Colder waters^{1,14,17,18}
- Equipment – causing excess negative inspiratory pressures from regulators, rebreathers^{2,5,19-23}
- Severe exertion^{5,7,9,24}
- Excessive hydration⁹

Ascent: In divers, particularly those using open circuit, symptoms related to hypoxia may occur or worsen on ascent and/or after surfacing as the inspired partial pressure of oxygen (PO₂) decreases.

Symptoms, signs and diagnosis

The following are the main clinical signs and symptoms of IPO, but the diagnosis does not require the presence of them all:

- Cough
- Dyspnoea (shortness of breath)
- Expectoration of frothy sputum (which may be blood stained/ pink) or haemoptysis
- Moist or rattling breath sounds and wheeze
- Chest tightness
- Cyanosis and hypoxaemia
- Confusion and agitation
- Unconsciousness
- Cardiorespiratory arrest

NOTE: Signs and symptoms are predominantly respiratory with secondary effects due to respiratory failure and hypoxia e.g., chest tightness, weakness, vomiting. There is a wide range of severity of symptoms in affected individuals.^{21,22,25-27}

The clinical diagnosis of IPO takes into account the temporal relationship during immersion, history and onset of signs and symptoms of the affected individual:

- Suspect if there has been a previous episode of IPO
- Suspect if there is an episode of dyspnoea during

immersion (in particular without aspiration), with any of the above signs and symptoms, and when the casualty has 'moist' breathing and crepitations on auscultation, evidence of arterial hypoxaemia clinically (i.e., cyanosis) or on oximetry. Because swimming is usually in the prone position, auscultatory signs of IPO are often predominantly or entirely in the anterior chest.²⁸ IPO may be unilateral and if so, the right lung is most commonly affected²⁸

Differential diagnoses include aspiration and near drowning, pulmonary decompression sickness (the 'chokes'), and pulmonary barotrauma.

Sometimes, the diver or swimmer has recovered before being medically assessed.

INDICATIONS OF IPO NOTED BY DIVE BUDDIES:

- Diver is coughing
- Diver appears to be more breathless, is breathing more rapidly (may be apparent from exhaled bubbles when on open circuit) or is using breathing gas at a faster rate than is appropriate for the degree of exertion involved in the dive
- Diver mistakenly believes they are out of breathing gas, or their breathing equipment is malfunctioning (may be apparent if a diver switches to own back up regulator, flushes their equipment or requests gas supply from buddy)
- Agitation, panic, and compulsion to ascend

Recommended pre-hospital treatment of IPO

- Immediately terminate the dive and leave the water as soon as possible
- Ascend safely but omit 'safety' decompression stops and, if the casualty is very breathless or distressed, consider omitting compulsory stops. If compulsory stops are omitted, it is important to give normobaric oxygen on the surface, observe for signs of decompression sickness and inform the local recompression facility
- At surface, establish positive buoyancy but avoid over inflation of buoyancy compensator
- Rescue and remove affected individual from the water as quickly as possible²¹
- Targeted ABCD assessment
- Provide O₂ – highest concentration possible²¹
- Maintain chest upright / supported propped-up position for breathing efficiency
- Remove tight diving gear and / or wetsuit
- Keep the casualty warm
- Transfer to a hospital emergency department
- Intravenous access but restrict intravenous fluids
- Non-invasive ventilation with constant positive airway pressure (CPAP) if possible or positive end-expiratory pressure (PEEP) (early if available)^{29,30}

- If the casualty is severely affected, consider providing assisted ventilation in the field via bag-valve-mask with PEEP valve if available
- Vasodilators (provided blood pressure is normal or high)
- Patients with IPO are not usually fluid overloaded. Therefore, diuretics are not a first line treatment of IPO and should only be considered a second line treatment after use of non-invasive ventilation and vasodilator medication

Investigations

The following investigations may support the diagnosis of IPO.

- Oximetry confirmation of hypoxaemia^{18,28}
- Point of care ultrasound findings consistent with pulmonary oedema^{5,18,28}
- Chest X-ray findings of pulmonary oedema¹⁸
- High resolution CT chest findings of pulmonary oedema
- Echocardiography soon after admission with IPO may show left ventricular dysfunction, particularly in older individuals.³¹ Occasionally an individual with IPO will have echocardiographic or other evidence of Takotsubo cardiomyopathy^{32,33}

Future suitability for diving

When assessing future fitness to undertake compressed gas diving or open water swimming following an episode of IPO, the following must be considered.

THE POSSIBILITY OF UNDERLYING CARDIOVASCULAR DISEASE

Immersion pulmonary oedema could be an indication of underlying cardiovascular disease especially in older divers, and the diagnosis warrants a detailed cardiovascular and respiratory assessment by specialists in conjunction with specialists in diving medicine.^{2,15,31}

Detailed cardiovascular assessment is advised, including 24-hour blood pressure monitoring and echocardiogram, to identify treatable pre-existing cardiovascular disease.

If hypertension is present, further tests to exclude a primary cause such as renal artery stenosis should be considered.²

Other tests to exclude myocardial ischaemia and dysfunction, such as stress echocardiogram, exercise stress test, myocardial perfusion scan, CT coronary angiogram and stress cardiac magnetic resonance imaging scan should be considered, as should a test of fitness that includes peripheral oxygen saturation monitoring.

RISK OF RECURRENCE

IPO has a high risk of recurrence.^{1,2,6–11} Fatal recurrence is documented.^{8,13}

Recurrent episodes can occur even when thorough investigations have found no abnormal results. However, what constitutes a thorough work-up before clearance to undertake diving has not been clearly defined.

Any decisions about returning to compressed gas diving require careful consideration of the risks applicable to the specific individual and should be made in consultation with a diving medicine physician and / or a cardiologist with an interest in diving medicine. It is recognised that some centres have significant experience and expertise in assessing these individuals.^{4,13,21,22,34}

SPUMS/UKDMC statements regarding returning to compressed gas diving after an episode of IPO

STATEMENT 1

SPUMS and UKDMC strongly advises against further compressed gas diving if an individual has experienced an episode of IPO.

STATEMENT 2

All divers who have had an episode of IPO should be fully investigated to identify any disease that predisposed to the condition because it may have implications for the individual unrelated to future diving. (e.g., IPO has occurred in divers with significant coronary disease, cardiac valve disease, cardiomyopathy, renal artery stenosis, etc).

Investigations should be overseen by physicians and cardiologists experienced in diving medicine.

STATEMENT 3

If divers choose to dive again despite the advice in Statement 1, they must be fully informed of the risk, including that a recurrent episode of IPO may be fatal.^{8,13,35}

If divers choose to dive again despite the advice in Statement 1, they should only do so after satisfactory treatment / resolution of any disease or risk factors identified during the full investigation recommended in Statement 2.

If divers choose to dive again despite the advice in Statement 1, they should be made aware of potential risk mitigation strategies such as: wearing high quality well-fitting thermal protection, avoiding heavy exertion, avoiding overhead environments or virtual ceilings (decompression diving), only diving if O₂ is immediately available after surfacing, avoiding dive locations remote from tertiary

medical services, avoiding pre-dive overhydration, avoiding back-mounted counter lungs (rebreather divers).

STATEMENT 4

Depth limitation is not an acceptable risk mitigation strategy to prevent IPO. There is no known association between IPO and decompression sickness. It should be noted that if a diver develops IPO during a deep dive, it will take longer to surface and exit the water, particularly if decompression stops are required. In addition, an ascent from depth involves additional risks to casualties suffering from IPO, particularly when the diver is using open circuit breathing apparatus. First, the inspired pO₂ will decrease during the ascent. Second, ascents are usually performed head-up, which results in negative pressure breathing and that will exacerbate the development of IPO.

STATEMENT 5

A diver who has had IPO should be advised that there is a known association between experiencing IPO and subsequently developing hypertension.^{1,2,36} Therefore they should receive life-long regular blood pressure checks – due to the risk of developing hypertension.

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Dr Turner acts as a consultant and proctor for St Jude Medical, Medtronic and Edwards Lifesciences, as a consultant and lecturer for Gore Medical and performs PFO closures on private patients. The other authors declare that they have no conflicts of interest.

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