Review article

Diving fatality investigations: recent changes

Carl Edmonds and James Caruso

Abstract

(Edmonds C, Caruso J. Diving fatality investigations: recent changes. *Diving and Hyperbaric Medicine*. 2014 June;44(2):91-96.)

Modifications to the investigation procedures in diving fatalities have been incorporated into the data acquisition by diving accident investigators. The most germane proposal for investigators assessing diving fatalities is to delay the drawing of conclusions until all relevant diving information is known. This includes: the accumulation and integration of the pathological data; the access to dive computer information; re-enactments of diving incidents; post-mortem CT scans and the interpretation of intravascular and tissue gas detected. These are all discussed, with reference to the established literature and recent publications.

Key words

Diving deaths, investigations, autopsy, radiological imaging, review article

---

Background

The investigation of diving fatalities has changed markedly over the last few decades, more than many conventional pathologists and diving physicians have appreciated. The 19th-century caisson physicians who investigated the dysbaric causes of death demonstrated the value of autopsy diagnoses. Later, the diving pathologists were not so fortunate – having to cope with a delayed recovery of an often damaged body, as well as the supervening complications of drowning, marine animal trauma, decomposition, post-mortem decompression artefact (PMDA) and resuscitation effects. Drowning was the autopsy diagnosis in about 80% of cases, with about 10% of cases attributed to gas embolism.[1] A review of the techniques used to investigate diving fatalities, including the interpretation of autopsy findings, was indicated. A fully annotated description of the changes and the reasons for them has recently been published in the forensic pathology literature.[2] A summary is presented here for diving physicians.

When the first edition of *Diving and Subaquatic Medicine* was printed in 1976, the autopsy, which had been the gold standard for post-mortem diagnosis, was being complemented by assessments from the clinical and diving data.[3,4] The publication focused on clinical interpretations, the dive profile and equipment testing. A new approach to diving accident scenarios has since been introduced, influencing fatality assessments.[5] A similar approach has been adopted in recent years in the annual reports of Australian diving-related fatalities.[6]

The causes of diving deaths have been extensively reported since the advent of scuba diving as a popular sport.[1,4–7] Reports acknowledge the importance of early liaison between diving experts, technicians, diving clinicians and pathologists if inappropriate conclusions are to be avoided. If the autopsy is performed in isolation from other sources of the diving data, interpretations may be problematic.

The investigation of diving deaths has followed this documented scenario:

1. Recovery, handling and observations associated with the body and all related equipment;
2. Witness and other informed statements;
3. The formal autopsy for immersion victims (techniques for aberrant gas identification and some procedural modifications for autopsies on divers are recommended to maximize the value of the postmortem examination[2,7–10]);
4. Technical assessments of the functioning of the diving equipment;
5. Gas analysis by reputable laboratories;
6. Coronial or other enquiries.

Approaches differ from country to country. In the Australian and New Zealand context (unlike the medical examiners system in the USA), it is usually the coroner who receives all of the documents and reports. While the prudent forensic pathologist would try to access these reports and would invite the diving physician to attend the autopsy, this is often the exception rather than the rule.

This paper deals with recent modifications to these diving fatality investigations and encompasses some autopsy techniques and laboratory tests, post-mortem CT scanning (PMCT), interpretation of extraneous gas in the deceased, dive computer data and the re-enactment of diving incidents. Frequent modifications of diving equipment and diving techniques (closed and semi-closed rebreathers, technical diving, varying dive profiles and newer decompression algorithms, etc.) dictate the constant up-dating of test procedures and are thus beyond the scope of this paper.

Autopsy techniques

These are well documented in current texts.[7,9] Some of the techniques previously proposed for diving autopsies have slipped into disuse because of difficulty in performing them or problems with their interpretation.[9–15] These are mentioned later, together with more valuable developments such as: the accumulation and integration of other contributing data, the use of dive computer information, re-enactments of diving incidents, post-mortem CT scans (PMCT) and the interpretation of intravascular and tissue gas detected by these.[2] Possibly the most important proposal for the autopsy investigation is to delay the drawing of conclusions until all possible diving information is known.

Fluid in the trachea and main bronchi is often observed in (but not specific to) drowning, as it is present in some cardiac deaths. Frothy airway fluid is present in both drowning and scuba divers' pulmonary oedema (SDPE).[15,16] The formal autopsy, where the chest cavity is manually opened, more clearly demonstrates the hyper-expanded lungs of drowning and the overweighted lungs of all three disorders.

Middle ear haemorrhage is often described as evidence of drowning.[2,14] The presence of middle ear/sinus mucosal congestion or haemorrhage is a frequent observation in clinical diving medicine, with associated symptoms and radiological validation. Symptoms usually limit the conscious diver from descending further, but when the victim is unconscious and descending and there is still circulatory activity, such barotrauma is to be expected.[7] Thus, this observation may simply imply descent whilst unconscious, not drowning as such. This explanation of middle ear and sinus barotraumas of descent is a far more feasible one than an inexplicable indication of drowning per se.

Diatom identification from various parts of the body, the airways and the incriminated water environment, despite having some potential value, has serious limitations and is rarely undertaken. Species recognition only implies water aspiration whilst alive, not death from drowning. Similarly, strontium, chloride, haemoglobin and other biochemical analyses have not had widespread acceptance and usage.[15]

One biochemical investigation that may differentiate other causes of death from sea water drowning, is the elevation of vitreous sodium and chloride levels.[17] It is not known whether this can differentiate non-fatal sea-water aspiration from drowning. False negative and false positive results need to be quantified.

While serological and immunoglobulin assessments to identify injuries from venomous marine animals have theoretical value, their use has not reached the international acceptability that was once predicted. In the appropriate setting, morbid anatomy and histology of skin and tissue wounds, and microscopic identification of nematocysts, are still of value.

Conventional diving autopsy techniques allow for the demonstration and examination of gas in atypical sites, such as the cardiac chambers, vessels, pleura, peritoneum and in other tissues. This can be achieved by opening these sites underwater or by perfusing vessels with inserted gas traps.[7–9] These techniques are not always easy or reliable, but they allow for the aspiration of the gas and analysis of its composition, which may indicate its source. PMCT is now recommended for demonstration of gas, prior to a diving autopsy, which itself can cause gas artefacts.

Conducting the autopsy at depth in a hyperbaric environment, which is possible under certain saturation diving conditions, avoids the disruptive influence of PMDA (or post-mortem 'off-gassing'). Such an autopsy location would be logistically and technologically problematic for most pathologists, even though some may argue it should be the 'gold standard' in identifying gas-related diving diseases.[7] In most diving fatalities the diver is either already at the surface or is brought to the surface with the hope of resuscitation. Transferring the body back to depth is not only impractical but fails to eliminate this artefact.

Post-mortem computerized tomography (PMCT)

Post-mortem X-rays were of value but these have been superseded by PMCT and other imaging techniques. These are becoming more commonplace as an adjunct, or even replacement, to the formal autopsy for detecting the origin, site and volume of abnormal gas spaces, as well as other pathology of the respiratory tract. It is more reliable in detecting gas spaces, more sensitive, less invasive, less time consuming and less offensive to various cultural and ethnic groups than a formal autopsy.[2,12,14] PMCT is performed as soon as possible, preferably within hours of the incident, and should precede any formal invasive autopsy procedure.

Evidence of pulmonary interstitial oedema is seen with the drowning syndromes (aspiration, near-drowning and drowning), cardiac disease and SDPE. SDPE has now been identified as a cause of diving deaths, but with an unknown incidence and similar lung pathology to drowning.[16] On PMCT a ground-glass appearance is observed in all these diagnoses. High attenuation particles, indicating sand or other sediment, may be present on PMCT in any of the drowning or aspiration syndromes, in the airways or para-nasal sinuses.[14] Frequently haemorrhage or effusion is detected in the middle ear and para-nasal or mastoid sinuses with PMCT.[2,14] This is an indication of possible barotrauma of descent – a consequence of descent by an unconscious diver.

Extraneous gas in the diving autopsy

Perhaps the most valuable but controversial aspect of the PMCT is the observation and interpretation of extraneous gas spaces in the diver's body. The techniques previously embraced to demonstrate abnormal gas in the diving autopsy

were introduced because gases are influential in the causes of death from diving and hyperbaric exposures, especially with gas embolism induced by pulmonary barotrauma (PBt) and decompression sickness (DCS).

The interpretation of gas detected radiologically and with newer scanning techniques has been marred in controversy. Some authors have embraced the newer technologies with enthusiasm whilst others have denigrated them as being valueless or misleading. This quandary has been addressed recently and is clarified if one understands the aetiology of the gas.[2] It requires a knowledge of infrequently accessed literature and an understanding of the processes that cause gas formation. Unfortunately, the presence of gas from processes that are not related to the cause of death have complicated its interpretation. These include PMDA, putrefaction, trauma and resuscitation effects.

It is often concluded that gas embolism caused a diver's death despite the diver being in a situation where this development was impossible. In 12 out of 13 diving fatalities autopsied at the NSW Institute of Forensic Medicine, intravascular gas was detected.[13] In some, the history and autopsy findings were inconsistent with pre-morbid gas embolism.

Extraneous gas may be detected post mortem in many anatomical sites: pleural, peritoneal, gastric, hepatic, muscular and intravascular. Interpretation relies not only on the site but also the volume and composition of the gas.[2] The potential causes are as follows.

Post-mortem decompression artefact
Boycott, Damant and Haldane in 1908 warned that "*the presence of bubbles in vivo must be inferred from their discovery post-mortem with considerable caution. The supersaturation of the body may be such that the separation of the gas bubbles may take place after death.*"[18]

The bubbling is mainly from inert gas, previously breathed by the diver and then dissolved in the blood and tissues.[12,13] PMDA can develop if the diver dies at depth or soon after ascent, if his body still retained supersaturated, dissolved gas. From deep and/or prolonged dives, it can produce extensive surgical emphysema, be present in all tissues and replace blood from both venous and arterial vessels (gas angiograms) and both sides of the heart. A PMCT scan should include the thighs, where gas is easily seen in the intra-muscular fascial layers. There are few other explanations for this observation.

Well-controlled animal experiments, across different species validate and quantify the concept of PMDA or off-gassing.[19–21] Animals that die at sea level and are then exposed to pressure do not subsequently develop PMDA. Nor do those that die immediately after exposure to pressure. Only those who were exposed to pressure whilst still alive and thus had a functioning circulation are so affected after surfacing. The depths and durations in these animal experiments were designed to parallel typical profiles of human compressed-air divers. There was a latent period of about an hour until the PMDA became evident, and then a progression of this effect over the subsequent 1 to 8+ hours. Another pertinent observation was the presence of small local areas of gas pockets adjacent to trauma from resuscitation and invasive procedures (see later).

Thus the animal experiments confirmed clinical experiences but were in disagreement with a popular belief that deep diving, in excess of 40 metres' sea water (msw), may be necessary for PMDA to develop. Excessive depths were not reached in most of the animal experiments described above, nor in human divers and caisson workers described by others.[22,23] Logically one can understand that, while a deep or decompression dive is not required to initiate this phenomenon, the amount, likelihood, extent and speed of development of PMDA is a consequence of both depth and duration of the hyperbaric exposure.

Decompression sickness
Although uncommon, death can occur from DCS, with gas bubbles developing within any tissue, owing to excessive exposure to pressure (depth) and too rapid an ascent. Histopathological signs include haemorrhage, necrosis and tissue reaction or inflammation around the tissue gas bubbles, differentiating it from PMDA; but histopathology is frequently not sought.[24,25] It is more evident in lipid tissues, including myelin sheaths of peripheral nerves, induced by the nitrogen breathing of compressed air divers.

Over a century ago DCS deaths were far more frequent and the pathology not usually complicated by resuscitation – which may cause local gas artefacts and re-distribution of intra-vascular gas. Hoff, reporting on autopsies performed on divers and caisson workers, observed that the less acute DCS cases tended to have gas in the right ventricle of the heart, whereas, in those who died very soon after decompression or from explosive decompression, gas was present in both the arterial and venous systems, with widespread gross distribution throughout many tissues.[22] This latter group is more likely to have complicated their DCS findings with the effects of PMDA and/or barotrauma.

Sir Leonard Hill, in his literature review, noted that Von Schrotter observed gas in the vascular system in 11 of his 18 well-described autopsies on DCS victims, whilst Keays described it in eight of his 12 victims.[23] Paul Bert showed in animal experiments that gas from decompression collected in the venous system and the right heart and also that the composition of this gas reflected tissue gas pressures. Hill described further autopsies of DCS in caisson workers who were exposed to pressures equivalent to 19–34 msw for over 3 hours, then had a very slow ascent (so PBt was an unlikely complication). In these cases, with typical DCS symptomatology preceding the death, the gas was often observed in the venous system at autopsy. It collected in the right heart in seven of the 10 cases. No arterial and left-heart

gas involvement was reported in the cases that died after a delay of some hours (when PMDA was unlikely).

Of relevance, but not specifically addressed by Hill, was the excessive volume and widespread extent of the gas in four workers who died of DCS within an hour of surfacing. In these cases there was also gross gas in the arterial system, the left heart, the viscera, subcutaneous tissues, thighs and even the cerebral ventricles. DCS cases that succumb very soon after ascent are vulnerable to supervening PMDA, obscuring many of the DCS features, but not all. Necrotic areas around obstructed vessels, lesions in myelin sheaths and skin manifestations may still be detected.

Gas embolism following pulmonary barotrauma
This is well documented in diving medical texts and is initially observed as air (nitrogen/oxygen) or gas bubbles in the systemic arterial system. It arises from lung rupture allowing inhaled gas to pass into the pulmonary veins, then the left heart and the arterial system. Because gas emboli are redistributed partly by buoyancy in the larger vessels, they tend to travel to the brain in the ascending diver and with the erect posture after surfacing. Some of the emboli may obstruct the smaller arterioles, or involve multiple generations of arterioles. Many, however, pass through to the venous system and thus to the right heart and pulmonary arteriolar filter. This occurs with continuation of life and circulation, including effective resuscitation efforts. The arterial bubbles may persist and obstruct, especially in small arteries such as the circle of Willis, and indicate the pathological diagnosis and the cause of death. Gas within the venous system does not invalidate this. The association of lung damage, pneumothoraces, pneumoperitoneum and mediastinal emphysema are strongly supportive of a PBt origin for the embolism, as is a history of rapid ascent followed by unconsciousness.

Resuscitation-induced gas (artefact)
Resuscitation efforts may admit small volumes of gas into the venous system or cause local subcutaneous emphysema over the affected sites.[21,26,27] This rarely simulates the large volumes seen with PMDA or even PBt. Knowledge of the resuscitation scenario and the usually small amounts of gas, as well as its location, should suffice to exclude this as a contributor to death in non-traumatised patients, but it may show up in the CT scans.

Invasive and traumatic events, including head injury, intravenous cannulation, endotracheal intubation, external cardiac compression, etc., can induce local gas artefacts that may be misinterpreted. Subcutaneous gas or surgical emphysema at the site of thoracic compression can be produced from resuscitation. Shiotami et al has quantified this using PMCTs, with 71% of non-traumatic CPR fatalities containing some cardiovascular gas and 7.5% with cerebral gas, compared to zero in non-CPR cases.[27] The vast majority of bubbles were grade 1 (< 5 mm diameter) and were in the right heart or systemic venous system. Because gas can be introduced during the autopsy, they recommended that the PMCT be performed first.[27]

Lung damage from resuscitation efforts and/or paradoxical embolism from arterio-venous anastomoses, such as a patent foramen ovale, may explain the small and uncommon intra-arterial gas bubbles seen in some cases. Resuscitation may thus occasionally redistribute intravenous gas, such as from DCS, into the arterial system. The use of oxygen during resuscitation may reduce the volume and number of gas bubbles detected. This is the basis of our current first-aid resolution of the bubbles induced in diving accidents (DCS, PBt) and continued resuscitation, even after death, could have a similar effect if circulation is maintained.

Putrefaction (decomposition)
This is well described in general medical texts. It is evident after about 24 hours if the body is not refrigerated, although the onset varies from 3 to 72 hours, depending on the environmental conditions and the gas volumes being detected.[7,8,13,27] Some recommend that the diver's protective clothing (usually a wetsuit) should be removed early, before the body is refrigerated, to more rapidly reduce the body temperature and thus delay decomposition.

Putrefaction causes a foul-smelling gas initially evident in the gastro-intestinal tract, the portal veins and liver. Hydrogen, carbon dioxide, hydrogen sulphide and methane may be present. Because divers who die underwater are exposed to environmental cooling influences, it is likely that putrefaction may be more delayed. It is this gas that causes many submerged divers to float to the surface a few days after death.

Drowning
In addition to aspirating fluid, drowning often results in the swallowing of air and water into the gastrointestinal tract, explaining the tendency of near-drowning victims to vomit.[7,14,15] Ascent may increase the volume of gas, according to Boyle's Law, distending the stomach. The composition of the gas (usually nitrogen and oxygen) is in approximately the same proportions as in the air or other gases being breathed. In some cases, individuals may regurgitate and aspirate stomach contents, but typically this is not a factor in the drowning process, although it is not uncommon during resuscitation.

Dive computer records

Often, the description of a fatal dive is vague, sanitised and inaccurate, especially from the diving companions and dive operators who may have a conflict of interest in the results of the investigation. Also, the deceased is often alone prior to or at the time of the incident, denying the investigator of relevant diving data. Over the last few decades, the use of dive computers has become ubiquitous. These accurately depict the details of the fatal dive. Depths, dive durations, ascent rates, the number of ascents, decompression staging

and decompression stress, dive profiles (reverse or forward), water temperature, gas pressures and gas consumption are all informative and accessible by downloading with suitable computer software. If the dive computer is gas-integrated, i.e., the changing breathing gas pressure is being recorded and integrated into the database, then, knowing the scuba tank size, the diver's gas consumption can be extrapolated for the various sectors of the dive profile. In addition, similar information, together with gas sensor data to record oxygen pressures, is accessible from rebreather sets.

This information allows an assessment of the likelihood of PBt, DCS, SDPE, panic, fatigue, aspiration, gas toxicity, cold effects, etc. As previous diving data is also stored in the dive computer, this may imply a predisposition to diving accidents. It may indicate the diver's experience, rapid ascents, inadequate decompression, deep diving, 'low-on-air' situations, etc. As well as the dive computer data from the deceased diver, that of his companions and rescuers may also be downloaded by an impartial, competent technician.

Re-enactment of the diving incident

This term does not refer to the laboratory testing of diving equipment. That is conducted routinely after diving fatalities, to ensure compliance with the manufacturers' or other's specifications (usually performed by technicians in a diving equipment laboratory). The re-enactment is a more recent and totally different concept used to demonstrate the functioning of the equipment under the conditions prevailing during the time of the fatality.

Laboratory testing of equipment will determine whether it meets certain performance criteria and that it can be used as intended. It does not imply that it did not contribute to the death. Thus, a diving regulator may be functional, producing a water-tight seal and adequate inspiratory gas flows under normal conditions, with an experienced diver breathing gently in an upright position; however, under different conditions, it may malfunction. Examples of such conditions are excessive air consumption from anxiety or extreme exertion, negative buoyancy or swimming against strong currents, at great depths or with the diver in a different spatial orientation. That information can only be elicited with a re-enactment, which can also detect hazards such as other equipment problems, potential entrapments, hazardous environments, technique difficulties and personal demands.

The concept of re-enactment of the diving incident was introduced in 1967.[28] It followed the unexplained deaths of two divers using re-breathing equipment. It was designed for internal use by experts in the Royal Australian Navy in Australia, which was the primary organisation that investigated such accidents at that time. The concept, which has become more widespread and is now often employed by police divers, is designed for the situation where a death has occurred but where there is no convincing explanation for the fatality.[11,28] The purpose is to observe the presence of adverse situations that had previously not been evident, and which may help to clarify the fatal incident and/or prevent future ones. It is carried out only after all the other dive investigations (including the autopsy) are complete and involves the following:[2]

- A detailed and accurate knowledge is required of the dive plan, dive profile, environmental conditions, buoyancy status, equipment used, and breathing gas pressures, composition and volume that existed at the time of the unexplained death.
- An accurate replication of the above is made by expert divers of a similar stature to the deceased, using the same or equivalent equipment and performing a similar dive in similar circumstances. Sample ports allowing for repeated gas sampling and analysis may be added when re-breathing equipment is involved.
- The divers need to have access to redundant emergency equipment to be used if necessary.
- Diving medical support, full resuscitation facilities and a rescue dive team must be available on site. It can be a hazardous exposure and attention must be paid to the ethical issues.
- Observer divers record the re-enactment using underwater video. Full documentation of the experiences and observations is made independently by each participant and this is compared to the video records.
- The fatal dive profile is replicated, but terminated prior to a catastrophic event.

If more than one potential scenario is present for the fatal dive, then more than one re-enactment may be required. In this event, any findings may not represent the actual situation existing at the time of the fatality, and should only be considered as possibilities to explore, not actualities.

A variety of observations may clarify the original assumptions and encompass demanding conditions, entrapment, water aspiration, disorientation, resistance to breathing, equipment inadequacy, gas toxicities (carbon dioxide, hyperoxia, hypoxia, narcosis), etc.

Conclusions

In most countries there are no analogous diving units to those that investigate aircraft accidents. Thus, the typical practice is for the investigation of the diving accident to be performed by police only moderately knowledgeable in the investigatory aspects of diving accidents, a local clinician who has little training in diving medicine, and a pathologist who is overworked and less then amenable to varying the standard conventional techniques. The result is often a mistaken diagnosis without an explanation of the causative sequence of events. There is thus a loss of valuable information and a failure to learn from the mistakes of the past.

A common error is for the diver fatality investigators to conclude a cause of death based on their own sphere of expertise before all the data are available. There needs to be a

close integration of data acquisition from all parties involved in the investigation. The pathologist needs to be aware of the specific requirements for a diving autopsy, as well as those required with aquatic/submersion fatalities.[2,7–9,29] The use of more sophisticated scanning techniques, their interpretation and the possible integration with the formal autopsy findings, conventional equipment testing and gas analysis, the dive computer data and, occasionally, re-enactment findings, requires a multi-disciplinary team approach.

References

1 *DAN's Annual reviews of recreational scuba diving injuries and fatalities*. 1989–2009. Durham, NC, USA, Divers Alert Network. Available from: <http://diversalertnetwork.org/medical/report/index.asp>.

2 Edmonds C, Caruso JL. Recent modifications to the investigation of diving related deaths. *Forensic Sci Med Pathol*. 2014;10:83-90. DOI 10.1007/s12024-013-9491-x.

3 Edmonds C, Lowry C, Pennefather J, editors. *Diving and subaquatic medicine*, 1st ed. Sydney: Diving Medical Centre; 1976.

4 Walker D. *Report on Australian diving deaths 1972 - 1993*. Melbourne: JL Publications; 1998.

5 Denoble HPJ, Caruso JL, Dear GL, Peiper CF, Vann RD. Common causes of open circuit recreational diving fatalities. *Undersea Hyperb Med*. 2008;35:393-406.

6 Lippmann J, Lawrence CL, Fock A, Wodak T, Jamieson S. Provisional report on diving-related fatalities in Australian waters 2009. *Diving Hyperb Med*. 2013;43:194-217.

7 Edmonds C, Lowry C, Pennefather J, Walker R, editors. *Diving and subaquatic medicine*, 4th ed. London: Arnolds; 2002.

8 Lawrence C, Cooke C. *Autopsy and the investigation of scuba diving fatalities*. Guidelines from the Royal College of Pathologists of Australia; 2003. Republished as: *SPUMS Journal*. 2006;36:2-8. Available from: <http://archive.rubicon-foundation.org/xmlui/bitstream/handle/123456789/10163/DHM_V36N1_2.pdf?sequence=1>.

9 Caruso JL. Pathology of diving accidents. In: Brubakk AO, Neuman TS, editors. *Physiology and medicine of diving*, 5th ed. Edinburgh: Saunders; 2003. Chapter 14.

10 Caruso J. Autopsy protocol for recreational diving fatality. In: Vann RD, Lang MA, editors. *Recreational diving fatalities*. Proceedings of the Divers Alert Network 2010 April 8–10 Workshop. Durham, NC: Divers Alert Network; 2011.

11 Edmonds C. A forensic diving medicine examination of a highly publicised diving fatality. *Diving Hyperb Med*. 2012;42:224-30.

12 Wheen LC, Williams MP. Post-mortems in scuba diving deaths: The utility of radiology. *Journal of Forensic and Legal Medicine*. 2009;16:273-6.

13 Lawrence C. Interpretation of gas in diving autopsies. *SPUMS Journal*. 1997;27:228-30.

14 Levy AD, Harcke HT, Getz JM, Mallak CT, Caruso JL, Pearse L, et al. Virtual autopsy: two- and three-dimensional multidetector CT findings in drowning with autopsy comparison. *Radiology*. 2007;243:862-8.

15 DeMaio VY, DeMaio D, editors. Death by drowning. *Forensic pathology*, 2nd ed. Oxford, UK: CRC press. 2001. Chapter 15.

16 Edmonds C, Lippmann J, Lockley S, Wolfers D. Scuba divers' pulmonary oedema: recurrences and fatalities. *Diving Hyperb Med*. 2012;42:40-4.

17 Cala AD, Vilain R, Tse R. Elevated postmortem vitreous sodium and chloride levels distinguish saltwater drowning (SWD) deaths from immersion deaths not related to drowning but recovered from saltwater (DNRD). *Am J Forensic Med Pathol*. 2013;34:133-8. doi: 10.1097/PAF.0b013e3182868ee1.

18 Boycott, DM, Damant, GCC, Haldane, JS. The prevention of compressed air illness. *J Hygiene*. 1908;8:342-443.

19 Brown CD, Kime W, Sherrer EL Jr. Postmortem intravascular bubbling: a decompression artefact? *J Forensic Sci*. 1978;23:511-8.

20 Cole AJ, Griffiths D, Lavender S, Summers P, Rich K. Relevance of postmortem radiology to the diagnosis of fatal cerebral gas embolism from compressed air diving. *J Clin Pathol*. 2006;59:489-91.

21 Laurent PE, Coulange M, Bartoli C, Luciano M, Cohen F , Rolland PH, et al. Appearance of gas collections after scuba diving death: a computed tomography study in a porcine model. *Int J Legal Med*. 2013;127:177-84.

22 Hoff EC. *Decompression sickness. Pathological lesions. Post-mortem findings. A bibliographical sourcebook of compressed air, diving and submarine medicine*. NAVMED 1191, vol 1. Washington, DC: Department of Navy; 1948. p. 142-62.

23 Hill L. *Caisson sickness and the physiology of work in compressed air*. New York: Longmans, Green and Co; 1912.

24 Gersch I, Catchpole H. Decompression sickness. Physical factors and pathological consequences. In: Fulton JF, editor. *Fulton's decompression sickness*. Philadelphia: Saunders; 1951. Chapter 7.

25 Albano G. *Principles and observations on the physiology of the scuba diver*. Arlington: Office of Naval Research; 1967. p. 236-62.

26 Shiotami S, Kohno M, Ohashi N, Atake *S*, Yamazaki K, Nakayama H. Cardiovascular gas on non-traumatic postmortem computerised tomography (PMCT): the influence of cardiopulmonary resuscitation. *Radiation Medicine*. 2005;23:225-9.

27 Shiotami S, Ueno Y, Atake S, Kohno M, Mototsugu K, Suzuki M, et al. Nontraumatic postmortem computed tomographic demonstration of cerebral gas embolism following cardiopulmonary resuscitation. *J Radiol*. 2010;28:1-7.

28 Edmonds C. *Reappraisals of a diving disaster*. Royal Australian Navy School of Underwater Medicine Report 4/68. Sydney: RAN Submarine and Underwater Medicine Unit; 1968.

29 Teather RG. *Encyclopaedia of underwater investigations*, 2nd ed. Flagstaff AZ: Best Publishing; 2013.

Conflicts of interest: nil

Submitted: 20 October 2013
Accepted: 04 April 2014

Carl Edmonds is a Consultant in Diving Medicine, Sydney, Australia
James Caruso, is Division Director, Chief Medical Examiner/ Coroner, Denver Office of the Medical Examiner, Denver, Colorado, USA

Corresponding author:
Carl Edmonds, OAM
Phone: *+61-(0)2-9976-5556*
E-mail: *<puddle@bigpond.net.au>*