Atom bombs, circle circuits, and CO_2: links between diving and anaesthesia

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There are many reasons why the specialties of anaesthesia and diving medicine are complimentary, not least of which is the strong relevance of respiratory physiology to both fields. This presentation will describe several of our recent attempts to answer questions relating to carbon dioxide (CO_2) homeostasis that are both of high practical relevance to the diving community and of potential interest to anaesthetists interested in the related physiology.

Prologue: Atom bombs

It was known in the 1930s that fluorination of anaesthetic drugs would produce less soluble (but nevertheless potent) agents that would be more stable and less metabolised. However, fluorine halogenation is technically difficult and fluorine chemistry was insufficiently advanced to produce such compounds. The impetus for advancement occurred during the Second World War for two reasons [1]. First, hydrogen fluoride could be used to accelerate the reactive steps in producing high octane aviation fuel. Second (and more importantly) uranium hexafluoride was found to be an obligatory reactant in the production of enriched uranium. The means of producing the former was developed as part of the secret Manhattan Project tasked to develop the atomic bomb. The fluorine chemistry component of this work was of great interest to (and partly funded by) drug companies. A number of potential anaesthetic agents were synthesised at the time, but none were suitable for use in humans. Nevertheless, this work indisputably accelerated development of Halothane which emerged in clinical use in 1951.

The Manhattan Project was successful in developing a nuclear weapon. The first detonation of an atomic bomb took place in the Nevada Desert on 16 July 1945, and the Hiroshima bomb was the second ever nuclear explosion (and regrettably the first use of a nuclear weapon on a human target), taking place only weeks later on 6 August. Nagasaki was the third nuclear explosion on 9 August.
The link with diving came after an interlude in nuclear testing following the war. Testing was resumed in 1946 with the fourth and fifth explosions both taking place at Bikini Atoll in the Pacific Ocean. These tests were designed to evaluate the effect of a nuclear blast on surface ships, and a large fleet of captured or surplus warships were anchored in the blast zone. In the end, only 19 of over 70 ships sank at Bikini.

These ships, lying in the sheltered enclosed waters of the Atoll, are now collectively considered one of the most iconic wreck diving sites on the planet, but getting to Bikini and diving the wrecks is logistically difficult and a true expedition-style undertaking. Of greatest interest among the wrecks is the aircraft carrier USS Saratoga. She is sitting intact and upright on the bottom at 52m depth. The flight deck is at 23m. We have explored the Saratoga extensively. It is a dangerous, silty, no-light environment inside the wreck which is becoming progressively unstable. Traversing the corridors must be undertaken with great care because there are numerous entrapment hazards, not least of which is the potential for silting and loss of visibility as a consequence of indiscriminate use of fins. One of our targets (understandably) has been the Sick Bay / Dental Surgery area which is down several decks and a long corridor traverse from the nearest entrance.

Circle circuits

Exploration of the Bikini wrecks is undertaken almost exclusively using underwater breathing apparatus based on circle circuits. Just as in an anaesthetic machine, these so-called "rebreather" devices recycle expired gas through a carbon dioxide absorbent and into a counter-lung from which it is subsequently re-inhaled. Oxygen levels in the circuit are typically maintained by a system incorporating galvanic fuel cells to measure the PO2 in the circulating gas. When the PO2 falls below a user-specified threshold, a microprocessor opens an electronic valve allowing more oxygen to flow into the circuit until the PO2 is restored. Gas is also added from a cylinder of nitrogen or helium-containing "diluent" gas during increases in depth to maintain the volume of the circuit (and to dilute the oxygen) as water pressure increases, but once at the target depth the only gas consumed is the exact amount of oxygen metabolised by the diver. The rebreather can therefore be seen as the ultimate low flow anaesthetic machine (without the anaesthetic), and one of their great advantages is parsimony with gas supply [2].
Rebreathers are complex devices with many failure points, and there are many accidents. Gas-related accidents are designated by "the three H's": hypoxia, hyperoxia, and hypercapnia. This presentation will focus on the hazard of hypercapnia. Hypercapnia in diving is undesirable because it can, of itself, cause unpleasant and debilitating symptoms of dyspnoea, headache, confusion, anxiety, and panic. In addition, hypercapnia is synergistic with nitrogen in producing narcosis, and it markedly increases the risk of cerebral oxygen toxicity during typical exposures to the elevated inspired PO2s typically chosen for deep dives; probably because of cerebral vasodilation with a consequent increase in the oxygen "dose" delivered to the brain.

The presence of a CO2 absorbent in a rebreather (commonly referred to in diving circles as a CO2 "scrubber") is an obvious failure point that, if faulty, expired, or incorrectly installed might precipitate hypercapnia due to CO2 rebreathing. Anaesthetists generally rely on their assistants to replace the CO2 absorbent in a timely manner, and medical absorbent material often reminds of the need for this by changing colour as it becomes consumed. In addition, the use of capnography provides a real time means to readily identify a CO2 absorbent failure. This would be indicated by failure of the capnography trace to return to zero during the inhalation phase of the respiratory cycle. Rebreather divers cannot see their CO2 absorbent so indicator material is no use, and although inhaled CO2 detection devices have been produced, their use is not yet widespread. Thus, the mainstay of prevention of CO2 rebreathing in rebreather diving is adherence to some simple rules and procedures. The rules include: checking the one way valve operation in the rebreather mouthpiece prior to assembly; packing the absorbent material into the canister meticulously; and installing the canister correctly; discarding the absorbent material after its recommended usage period (typically 3 – 4 hours); and "pre-breathing" the rebreather after assembly but before diving.

Pre-breathing a rebreather loop

The pre-breathe procedure has been of interest to us. Every rebreather diver is trained to sit quietly and breathe on the loop for 5 minutes prior to entering the water. This serves multiple purposes, but the one most heavily emphasised (and the reason for the recommended 5 minute duration) is based on the assumption that if there is a problem with CO2 rebreathing the prebreathe will result in symptoms of hypercapnia (such as shortness of breath) thus warning the diver not to proceed with the dive. This assumption had never been tested formally.

We conducted a study in which divers were randomised to perform a 5 minute prebreathe on a loop with a normal scrubber, no scrubber, or a scrubber with a fault allowing partial rebreathing of exhaled gas. The subjects were: blinded to the scrubber condition; instructed to treat the prebreathe as normal; and told to terminate the prebreathe before the 5 minutes was complete if they developed symptoms of hypercapnia (which they were reminded of). We monitored inspired and end tidal CO2, tidal volume, respiratory rate, and minute volume in all prebreathes. Despite the obvious expectation associated with participation, none of 20 subjects terminated when prebreathing with the normal scrubber. Only 2 of 20 subjects terminated when prebreathing with the partial fault despite the P_ICO2 being approximately 20mmHg. Fifteen of 20 subjects terminated when prebreathing with the absent scrubber, but remarkably 5 did not terminate, despite a $P_{ET}CO2$ in the 60s. We concluded that the 5 minute prebreathe is not a sensitive test for scrubber problems; particularly in the partial failure condition [3].

Hypercapnia caused by deranged control of ventilation

All FANZCA Part 1 examination candidates would be familiar with the simple relationship between some measure of CO2 state (alveolar CO2 or arterial CO2), CO2 production and alveolar ventilation, as described by the simple equation:
PACO2 = VCO2 / VA

Since VCO2 is effectively uncontrollable (unless physical activity is intentionally moderated) this equation exemplifies the critical role of control of ventilation in CO2 homeostasis. Under normal circumstances ventilation is automatically regulated by the respiratory controller, and not surprisingly the classical VE / VCO2 curve (in which ventilation is plotted against either CO2 production, arterial CO2 or end tidal CO2) appears as a straight upwardly sloping line; thus, as arterial CO2 increases, so does ventilation in an attempt to bring the arterial CO2 back to normal. In anaesthetic physiology teaching we have become familiar with the concepts that the curve can be shifted to the right and its slope reduced by drugs (particularly opiates) and hyperoxia, and shifted to the left by hypoxia.

Diving physiologists have become familiar with other fascinating phenomena in relation to this curve; in particular, the substantial inter-personal variability that can be masked by population curves, and the fact that this variability is maximally unmasked by increases in the work of breathing, particularly during exercise. Thus, in the face of an increase in work of breathing, it is as though the respiratory controller has a choice either to drive more work to maintain CO2 homeostasis, or to avoid the work and allow the arterial CO2 to drift upwards. Since the use of underwater breathing apparatus imposes an increase in the work of breathing (particularly at deep depths when the respired gas is dense) hypercapnia due to inadequate ventilation (sometimes referred to as "CO2 retention") is a well-recognised phenomena [4]. Importantly, this mechanism is distinct from hypercapnia caused by CO2 scrubber failure and consequent CO2 rebreathing although the two problems can interact; if a rebreather diver with a tendency to retain CO2 is breathing dense gas at depth and there is CO2 breaking through the scrubber, then dangerous hypercapnia will develop more quickly. We saw relevant examples in our prebreathe study described above. The divers were not exercising and at atmospheric pressure the gas was not abnormally dense, but the rebreather as configured for the experiment (including an anaesthetic antibacterial filter) did impose a higher work of breathing than normal. Among the subjects exposed to the absent scrubber condition some increased their ventilation substantially as their end tidal CO2 rose, whereas others did not increase ventilation at all even when their end tidal CO2 increased to greater than 60 mmHg.

Guidance on tolerable gas density

In view of the predilection for CO2 retention among some divers as work of breathing increases it is surprising that there are no published guidelines for gas density in planning deep dives. This is important because it is a variable that divers can manipulate by including more helium (a light, non-narcotic but expensive gas) in their breathing mix. Although the database was not compiled specifically for this purpose, we are about to publish an analysis of outcomes for a database of human test dives utilising various underwater breathing apparatus operated with gases of different density on a graded exercise protocol [5]. These tests, performed at a UK testing house for diving equipment under the supervision of Mr TG Anthony, resulted in "dive completion" or "dive failure" where one of the outcomes designated as "failure" was the development of an end tidal CO2 greater than 8.5 kPa (64 mmHg). There was a clear inflection of risk at gas densities above 6 g/L. Indeed, the proportion of dives failing because of hypercapnia rose from 7% at densities between 4.1 and 5 g /L, to 8.5% between 5.1 and 6 g/L, and to 41% between 6.1 and 7 g/L. On this basis we recommend that divers plan breathing gases to have a density that is ideally below 5.2 g/L and to treat 6.2 g/L as an absolute maximum.

CO2 retention during rest in shallow depths

This threat of CO2 retention during diving has been particularly troublesome for deep divers who spend hours decompressing from their dives whilst breathing high pressures of inspired oxygen (typically a PiO2 between 1.3 and 1.6 ATA). Although oxygen toxic seizures are rare at these PO2s, they do occasionally occur and the risk is thought to increase as the duration of exposure lengthens. Thus, the final hours of decompression are, in theory, the period of highest risk. This is a greatly feared

complication because a seizure underwater may result in drowning (not to mention the inevitable failure to complete prescribed decompression).

Since hypercapnia markedly increases the risk of cerebral oxygen toxicity, there has been interest in whether CO2 retention is likely during decompression. The final hours of decompression are typically spent resting in shallow depths, thus there is no exercise and the respired gas is not dense. These factors should lower the risk of CO2 retention, but the possibility had not been investigated in a real world study mainly because it is virtually impossible to establish real time end tidal CO2 monitoring in a typical diving environment.

This brings the narrative back to Bikini Atoll where we were diving the wrecks sunk by the 1946 nuclear weapon tests. We took advantage of the necessity for decompressions under a stable working platform in ideal surface conditions to conduct a simple field study in which we measured end tidal CO2 in divers immediately on surfacing after a very brief ascent from their final 3m decompression stop [6]. Data were gathered from 34 dives, and compared to resting end tidal CO2 levels from the same subjects at the surface two hours after the dive. In-water and post-dive resting end tidal CO2 readings did not differ, and were almost invariably less than a nominal normal value of 45 mmHg. One subject returned a surfacing end tidal CO2 of 48 mmHg. Thus, perhaps not surprisingly, we did not find a systematic tendency to CO2 retention in decompressing divers after their shallowest stop in ideal conditions. While this is reassuring, we cannot exclude the possibility of sporadic CO2 retention under these circumstances by predisposed individuals.

Epilogue: exploring the medical spaces on USS Saratoga

Over several expeditions to Bikini Atoll we have found our way into and explored the Sick Bay area on USS Saratoga. There is a very small operating room which is tight, obstructed by equipment and dangerous. I was unable to find any obvious anaesthesia equipment. The hospital ward area is more open and unobstructed, but there are few items of interest (unless sinks and urinals can be categorised as such). The dental surgery is by far the most fascinating. There are 3 largely intact work stations with drills, elaborate cascading spit bowl systems, instrument racks, and chairs with head clamps! It is an intriguing museum-piece exemplar of the approach to care at that time. Unfortunately, somewhat wasted on an anaesthetist.

References

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