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WHY EXERCISE IS NOT ALWAYS GOOD FOR YOU TWO CASE REPORTS

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Key Words

Carbon dioxide, medical conditions and problems, physiology, risk, unconsciousness.

Introduction

Royal Australian Navy Clearance Divers are required to pass stringent physical fitness standards, regardless of age. As a consequence, individuals often push themselves to their physical limits attempting to meet these standards, particularly during the selection tests to join this select and elite branch. At times individuals exhibit the mental toughness necessary to push through their own pain barriers, with devastating physical consequences. Two such cases are presented.

Case 1

Diver A was a 31 year old male, considerably older than most trainee divers, highly motivated, with a somewhat obsessional desire to become a Clearance Diver. He was well educated, fluent in a number of languages and had worked as a recreational dive instructor before enlisting in the Navy. He claimed that, in the two years before joining the Navy, he had performed approximately 1,600 dives without mishap. He was extremely proud of his underwater ability and was in good health, but said that he had never been a good runner.

He was a participant on the Clearance Diving Acceptance Test Course. This is a punishing course, where candidates are tested both physically and psychologically. The candidates do not undertake many dives during the course, which is designed more to assess teamwork skills and attitude during extreme situations. The course was being conducted in Pittwater, approximately 40 km north of HMAS PENGUIN, when the report came one Saturday night that he had collapsed. In fact this was the third time he had "collapsed", but this information had not been passed to senior medical staff.

The history I received that night was that he had collapsed during a PT session, which was said to be fairly testing. He was alleged to have lost consciousness, was frothing at the mouth, tachycardic, profusely sweating, with his eyes rolling back. It is at your peril that you ignore the statement of a senior diving instructor who says "Ma'am, he looked like he was going to die".

I was a bit alarmed by the story, however I was then informed some two hours had elapsed since his collapse and he had since made a good recovery. He had not dived for the 24 hours before his collapse, and in the previous week had only undertaken one 6-metre dive on air. He had complained to the training staff that whenever he had to run very hard, he became dizzy, his head spun, he developed blurred vision and difficulty breathing. His symptoms always resolved on completion of exercise.

By the time I saw him he had fully recovered and was normal to examination. An ECG showed sinus rhythm, with a few ventricular ectopics, which were considered to be benign. An echocardiograph revealed no structural abnormality. A maximal exercise stress test was performed; he exercised for 17 minutes, and only stopped because of calf pain. When he exercised, the number of ventricular ectopics decreased. A malignant arrhythmia was not considered to be a likely explanation for his symptoms.

Routine spirometry, lung volumes, transfer factor and arterial blood gas measurements were all normal. He then went on to a progressive cardio-respiratory exercise test. He exercised well, to 200 watts, but went into respiratory failure during the test. Despite a substantial fall in arterial oxygen saturation, to a PO_2 of 43 mmHg, and an increase in end-tidal PCO_2 to 63 mmHg his respiratory rate remained at 9 breaths per minute. By the end of the test his symptoms of blurred vision and dizziness were reproduced.

His ventilatory responsiveness to carbon dioxide was then tested. He had a reasonable response to carbon dioxide, but the pattern of his hyperventilation was unusual, with an increase in tidal volume but no accompanying increase in frequency. It appeared that he was voluntarily holding down his respiratory frequency. The patient then admitted that he was extremely proud of his ability to skip breath and said that for all his 1,600 dives he only took three breaths a minute. The respiratory physician said that he did not believe the patient's voluntary reduction in respiratory frequency presented a hazard to him diving, although he becomes significantly hypoxaemic and hypercapnic at the extremes of exertion. I disagree with this opinion for a number of reasons.

The normal response to hypercapnia is an increase in respiratory rate and the development of a headache. These symptoms can cause significant distress. It is known that carbon dioxide retainers have a higher threshold before experiencing the effects of raised carbon dioxide partial pressure. Our patient's threshold was much increased, so he did not experience those warning signs. It is also known that hypercapnia may potentiate oxygen toxicity and nitrogen narcosis, may precipitate oxygen convulsions and, by itself, has been associated with loss of consciousness.[1,2]

Situations likely to aggravate hypercapnia include exercise, an increased breathing resistance, use of carbon

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dioxide absorbent systems, hyperoxia and nitrogen narcosis. These are conditions faced everyday by a clearance diver diving with closed circuit rebreathers.

We considered this patient to be at increased risk of developing both carbon dioxide and oxygen toxicity when using closed circuit breathing sets. The patient believed he could "unlearn" his ventilatory response, however, we were unwilling to accept this as, in the face of extreme provocation during the exercise test, he had been unable to increase his ventilation even in the presence of life threatening acidosis (a consequence of his severe hypoxia and hypercapnia).

He was made permanently unfit to dive using closed circuit diving sets, which meant he could not be a Clearance Diver in the Navy. His fitness for open circuit diving poses an interesting problem, particularly in view of his uneventful diving past. He was discharged from the Navy intending to resume his recreational diving instructor career.

Case 2

Diver B was a 24 year old male in good health, who had not dived before. He was a student on the Scuba Air Diving Course and had passed all the requirements of the Royal Australian Navy diving medical. He denied taking any medication or tablets on day 1 of the course. This was later determined to be inaccurate. He was in the last week of the three week course and was assessed as being one of the better students, performing well in both the diving and physical fitness activities.

B collapsed during a physical training session involving, while wearing overalls, a 200 m harbour swim followed by an 8 km run back to the Naval base. The history related by his course mates was that B had become distressed after running 3-4 km. He stopped, fell to the ground and then got up complaining of thirst. A fellow student took his pulse, which was said to be 180 per minute. Unfortunately, he was encouraged to continue by one of his classmates. He fell again. His classmate thought "We're holding everyone back" so he piggybacked the victim up the hill. At the top of the hill B was given a drink of water but did not feel much better. B continued the run, becoming increasingly distressed, but kept repeating, "I have to get home. I have to keep going."

When he reached the base, he suddenly sprinted but was veering from side to side on the road. He reached the Diving School, where his distress was immediately recognised and he was carried to the Submarine and Underwater Medicine Unit.

On arrival he was unconscious, with a Glasgow coma score of 7. He was in severe respiratory distress with a respiratory rate of 40. He had a sinus tachycardia of 180. He was peripherally shut down and sweating profusely. His temperature was 38.4°C on arrival and was never measured any higher. He was intubated, his ventilation controlled and intravenous fluids started. Arterial blood gases, while on 100% oxygen, revealed a profound metabolic acidosis (pH 7.17, PCO_2 43 mmHg, PO_2 371 mmHg, HCO_3 15.8 mmol/l, base excess (BE) –11.8 mmol/l and O_2 saturation 99.9%). He was transferred to the Royal North Shore Hospital.

A chest X-ray and CT scan of his brain were normal. His sinus tachycardia slowed with fluid replacement although he was also given adenosine. His clinical condition settled quickly and he was extubated later that afternoon.

He had, however, suffered a severe systemic insult as he went on to develop acute hepatic failure, disseminated intravascular coagulation and renal failure (alanine amino transferase (ALT) peaked at 12,000 U/l and aspartate amino transferase (AST) 9,830 U/l on day 3 post injury, platelets 79 x 10^9/l, the international normalised rate (INR) was 4.1 and creatinine peaked at 0.2 mmol/l). He developed widespread ST segment changes on his ECG. Creatine kinase peaked at 1,323 and was said to be of skeletal muscle origin. Despite this there was no evidence of rhabdomyolysis. Echocardiography and gall bladder ultrasound were reported as normal examinations. Serology for hepatitis A, B and C and HIV was negative. Urinary drug screens (including paracetamol) were negative although it was then discovered B had been taking creatine supplementation, in accordance with the manufacturer's guidelines (1 teaspoonful a day) in the two weeks before and during the course.

B eventually improved with conservative management, but not before the liver transplant team was consulted and were on stand by. At the time of discharge from hospital, some 14 days after the incident, all biochemical parameters had returned to normal except his ALT and AST. These had dropped considerably but it took another couple of months more before they returned to normal.

His discharge diagnosis was heat stroke, in association with severe physical exertion. Whether or not the creatine supplement played a part in his presentation was uncertain although the consultant physician believed B would be fit to return to complete his diving course within a few months.

There is some difficulty in accepting this diagnosis. Heat stroke, by definition is thermo-regulatory failure, usually associated with warm environmental conditions and core body temperatures over 41°C. While some sufferers can be sweating on presentation, more usually they present with dry skin, having ceased sweating. Contributing

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factors include dehydration and antihistamines with anticholinergic activity. On the day of B's run the temperature was only 19°C and it was drizzling. His temperature was never recorded higher than 38.4°C. He had been well hydrated before the exercise that morning, he had no underlying illness and was on no medication, other than the creatine. He was accustomed to the level of exercise undertaken.

What about the role of creatine? Creatine is found naturally in skeletal muscle, the heart, brain, testes and other tissue. It exists in its free form (1/3), and as phosphocreatine (2/3). Creatine plays an important role in anaerobic ATP production during maximal anaerobic burst type activity. During intense muscle contraction, ADP is rapidly phosphorylated by phosphocreatine to produce ATP that is utilised within the myofibrils of skeletal muscle. This reaction can produce ATP for 10–20 seconds of maximal exercise, after which, other mechanisms are needed for ATP production (anaerobic glycolysis or aerobic oxidation of carbohydrates and fats).

The supposed basis of the ergogenic effect of creatine supplementation is an increased storage pool of phosphocreatine in skeletal muscle, an enhanced resynthesis of phosphocreatine during recovery periods after intense exercise and possibly an increase in skeletal muscle protein synthesis.

Typically creatine supplementation is a loading dose, of 20 g daily for 5 days, followed by a maintenance dose of 2 g a day. The loading regime can increase a person's total creatine stores by 17-22%. Without a loading phase a daily dose of 3 grams will achieve a similar increase after about 28 days.

Creatine is ergogenic, for repeated 6 to 30 second bouts of maximal, stationary cycling (recovery periods of 20 second to 5 minutes). It is not considered ergogenic for single or repeated swimming and running sprints, but it may increase strength as a result of increased protein synthesis in muscle. That is not yet proven. It does not benefit sub-maximal or endurance exercise. The individual response can vary greatly in the extent of increase in muscle creatine concentration, as well as the performance results.

There is limited data available regarding the safety of creatine.[3,4] It is not considered a drug, is distributed as a food agent and therefore claims regarding performance and safety do not need to be substantiated. It causes weight gain initially, thought to be as a result of water retention. There has been no reported relationship between creatine supplements and muscle dysfunction, or gastrointestinal symptoms. However, diarrhoea and gastrointestinal pain have occasionally been reported as side effects of the loading dose. Cramping, strains, tears and muscle tightness have been reported with creatine use. These effects have been thought to be associated with the water retention, which increases muscle compartment pressure leading to risk of muscle dysfunction. Increased levels of creatine have been measured in both serum and urine with supplementation, however, normal kidneys manage this load without compromise.

Short-term supplementation of 10 days or less has no effect on cardiac ejection fraction or blood pressure. Creatine is normally found in the brain and in the CSF. Some studies have suggested that there might be a relationship between seizure disorders and increased brain creatine. However it is not known whether oral creatine supplements affect brain levels. Creatine is also found normally in the testes where both forms are involved in sperm production. As endogenous creatine synthesis is suppressed during periods of supplementation it is postulated that Sertoli cell function may be affected. Long term effects of this suppression are unknown.

It is possible that in B's case the risk of dehydration was increased a consequence of creatine induced fluid shift into skeletal muscle.

B, although having made a complete physical recovery, was somewhat anxious about returning to rigorous exercise. He underwent psychological review and an individualised, supervised, PT program was arranged for him. Then he had a formal exercise test to prove to him, in the safety of a hospital environment, that exercise was safe. Resting lung function was normal. He achieved his maximum predicted exercise workload, at a maximum oxygen uptake (VO_2) of 96% of predicted and a heart rate of 90% of predicted. However, his maximum ventilation achieved was only 74% of predicted with a peak end-tidal CO_2 of 46 mmHg. His respiratory rate did not increase with exercise but he had an increase in tidal volume. This suggests reduced sensitivity to CO_2, as with Diver A, but milder. It was considered unethical to repeat exercise testing to the limits of exhaustion.

There remains the question of fitness to dive. Multi-system organ failure had followed routine exercise in a man with a mildly reduced sensitivity to carbon dioxide and who was taking creatine supplementation. In coming to a decision about his future we used similar criteria to Case 1. A person with reduced sensitivity to carbon dioxide is at increased risk of carbon dioxide toxicity, cerebral oxygen toxicity, nitrogen narcosis, and loss of consciousness. So B was declared to be fit for open circuit diving, but unfit for closed circuit diving. He has returned to exercise and to his ship at sea. But, at the time of writing, has not yet returned to repeat his diving course.

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ADJUNCTIVE THERAPY IN DECOMPRESSION ILLNESS: PRESENT AND FUTURE

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Key Words
Decompression illness, drugs, treatment.

Historical background

When attempting to evaluate the value of adjunctive treatment, it is instructive to examine case descriptions from an era when none were available. In Edward Ellsberg's book, *Men Under the Sea*, is a dramatic description of air recompression therapy of a diver with a severe case of the bends, experienced during the salvage of the submarine *S-51* in 1925.[1]

> [After a 60 minute dive to 132 feet, and two hours of in-water decompression]…the tenders finished undressing the divers, leaving the *Falcon's* fantail a mess of wet lead shoes, lead belts, helmets, hoses, and sodden diving rigs, and the three, still in their underwear, hurried below for supper, already somewhat late.
>
> Five minutes later, seated on a bench before the heavily laden mess table, L'Heureux, still as merry as ever, suddenly collapsed, pitched forward on the table, unconscious!

No need to ask questions in that company. "The bends" and a bad case of it! No one wasted time in futile first aid measures. Hastily his shipmates seized the silent figure of L'Heureux, unceremoniously rushed him up the steep ladder to the deck above…

In through the round steel door of the recompression tank went L'Heureux's inert form, one diver dragging his head, another pushing on his legs. Running from the wardroom came Surgeon Flotte, to dive through the opening almost on L'Heureux's heels. The door clanged shut behind him. On went the compressed air, hissing gently into the chamber as was customary. Hastily Surgeon Flotte felt L'Heureux. No sign of heartbeat. The man was completely out, might die at any moment, might perhaps already be dead from bubbles of air clogging his heart. It was no time for routine measures. At any cost those bubbles must be reduced to a size small enough to pass through the heart valves, to allow circulation to continue. And only high air pressure could compress them enough for that.

Dr. Flotte sprang for the air valve and twisted it wide open (apparently, in those days the insider tender operated the chamber). Immediately the low hiss of the incoming air changed to a loud roar and, under the terrific pressure of the high pressure air banks, air started to pour into that recompression chamber. The needle on the caisson gauge jumped like a race horse getting away from the barrier, continued rapidly round the dial. Twenty pounds, 40, 50. Dr. Flotte's ears began to ring. That was as high in pressure as we had ever gone before on anybody. But no stop now. Sixty pounds. Blood oozed in Flotte's nose and mouth, but still he kept the roaring in full blast. He must get the pressure up on L'Heureux, never mind himself. Seventy pounds, with the valve wide open, the needle still racing up the dial. Eighty pounds, a higher pressure by far than anybody on that diving job had ever before been subjected to, either on the bottom or in the tank, and, worst of all for Flotte, taken in one swift rush!

Eighty pounds (55 m equivalent depth) was enough. Flotte shut off the air. Dizzy from the sudden impact of high pressure, ears ringing excruciatingly, he bent over L'Heureux, tore off his shirt. The diver's chest was covered with purple splotches, the result of the bursting of a myriad small blood vessels from expanding air. But that was a minor result of "the bends." The major question was circulation. Had he got those heart bubbles down before L'Heureux's heart had stopped forever?

Flotte bent over his chest, listened, then smiled wanly. His heroic treatment had succeeded. A faint heartbeat became perceptible, L'Heureux began to breathe again. The bubbles, compressed to one-sixth their previous size by the sudden application of