of sports diving in remote areas presents a major area of concern.

In such settings, the initial transportation of the casualty in a 30° head-low tilt and administration of 100% oxygen continuously until arrival at definitive chamber management will be beneficial. Adequate fluid administration begun early and maintained during transportation to the chamber would help towards eventual successful recompression therapy. Steroids may also be useful in the treatment of cerebral oedema.

At the time of arrival at the chamber, a pre-treatment examination is done to gauge the seriousness of the patient's condition. Clinical examination, including a neurological assessment, is mandatory. In the absence of chest X-ray the physician should at least clinically exclude a pneumothorax. If present, a chest tube with Heimlich valve or underwater seal is required prior to any recompression. Conscious patients can clear their own ears but in the comatose or unconscious patients bilateral myringomtomies may be performed.

Royal Navy Table 63 or US Navy Table 6A may be used for recompression therapy. This is the generally accepted treatment for CAGE. The suspected cases are taken directly to 50 m (6 ATA) and treated according to the requirements of the tables.

Conclusion

The clinical case of HC presents itself as a unique example of how much work there still needs to be done in the field of CAGE. Although the neurological team felt HC was a hopeless case destined either for death or a persistent vegetative state, hyperbaric treatment was nevertheless instituted.

The hyperbaric treatment challenged conventionally accepted criteria for delayed management in that it was started 58 hours after the initial hypoxic event. Regardless of this, HC did over a period of weeks to months show considerable improvement in his clinical state. Whether this would have been the natural outcome had HBO not been used is open to question.

Far from being an area of consensus and understanding, CAGE is an area for further study and active debate. Our task of understanding relates not only to the underlying pathological process but also to the ideal treatment of CAGE.

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MASSIVE HAEMOPTYSIS AND AGE A CASE REPORT

A Santos

Introduction

We have the opportunity to review the theory of Caisson disease (decompression sickness or DCS) and diving related maladies. These are the particular risks for us as we attempt to transcend what is described as the teleological barrier between land and sea. I would like to present a case for demonstration, particularly the clinical aspects of diving medicine and its varied presentation. In America we try to differentiate the various entities in diving medicine into two major groups, DCS and pulmonary barotrauma. In differentiating these groups we usually label joint pain and spinal cord symptoms, with loss of motor and sensory

function below the umbilicus, as DCS. We divide pulmonary barotrauma into two groups, arterial gas embolism (AGE) and air in spaces in the body where it is not supposed to be, skin, lungs, hemithorax, mediastrinum, and even the peritoneum. Going a bit further we can say that AGE is primarily a disease of ascent and DCS is a disease of exposure over a period of time. In addition there is otic barotrauma, a disease of descent, which is commoner than anything else.

We have a difficult time in diving medicine as it is hard to differentiate between those types of diving maladies. Any one patient may present with both kinds of problem at any one time. Haemoptysis is, for us, an unusual event in AGE, massive haemoptysis is even more rare. So I am presenting a case report of a patient who had massive haemoptysis.

Case history

A 40 year old male made his first ever scuba dive, in Mexico, to 4.5 m (15 feet) for approximately 5 minutes. Then he panicked. Later he said that he had had difficulty with the regulator and could not breathe as well as he thought he should. He removed his weight belt and made a breath-hold ascent to the surface. On the surface it was noticed that he was breathing freely but had a depressed level of consciousness. He began to expectorate large quantities of blood. He was taken to a nearby hospital and given intravenous fluids and his vital signs were monitored. He was noted to have a respiratory rate of approximately 30-40 breaths/minute and his pulse rate was 120-130/minute. He was transferred to a second hospital where he promptly went into cardiopulmonary arrest. He was resuscitated , given 4 ampoules of bicarbonate, was intubated and successfully recovered after 3 minutes. His deep tendon reflexes at that time were intact and he was felt to be stable. By this time he had brought up about 800 ml of blood, which was actually measured and sent to the University of California from where transport was arranged for him by the Divers Alert Network (DAN).

He was transported by jet, at 1 ATA. I think that sea level transfer is essential. We have a similar problem in that we dive in Imperial Valley which is below sea level and we have to get back over 5,000 foot mountains to San Diego. When he arrived at the University of California, San Diego (UCSD) he had an adequate urine output and his vital signs were much the same, respiration. 35-40, pulse 105 and seemed to be not too bad. He complained of double vision, blurred vision and back pain which we attributed to being on a trauma board for 10-11 hours. We examined him and found marked cerebellar dysfunction which was the main neurological problem. Interestingly, he had very little in the way of motor findings, or sensory findings. In his right arm there was some sensory deficit, but marked past pointing when trying to do finger to nose and having him follow the examiner's finger. He was placed in the chamber after arterial blood gases were obtained. During transport he had been placed on 6 l/min oxygen and his PaO_2 as he came through the door was measured at 43 mm Hg. On 10 l/min of oxygen the PaO_2 jumped to 79 mm Hg.

We have been in the habit of taking chest X-rays before putting people in the chamber at UCSD. This patient had bilateral diffuse alveolar infiltrates, in addition his ECG showed that he had elevated P waves and, in retrospect, probably pulmonary hypertension. Back pressure pushing through the right ventricle into the right atrium creates the effect seen on the ECG. Precordial ST segment and T wave changes were also present but he had no evidence of myocardial infarction. His chest X-ray showed no evidence of preumothorax. He had a diffuse ground glass appearance to both hemithoraces and he was intubated. He had no subcutaneous emphysema anywhere. In retrospect again, there was a line which represented the reflection of the pleura on the pulmonary artery separated from the artery by an airspace and there was another airspace that pushed the pleura away from the aorta. There was also a suggestion that there might be a preumomediastinum as well as air along the left cardiac silhouette. The chest X-ray finding is interesting and it is unusual to have a diffuse generalised process. It was something we did not expect. The alternatives for that picture include aspiration, diffuse alveolar haemorrhage and even shock lung.

We took him into the chamber after this 45 minute work up and treated with a USN table 6A. In our unit we usually treat patients on a table 6A for what we suspect is AGE. This is a controversial topic. Table 6A is initially to 50 msw (165 fsw) on air then back to 18 msw (60 fsw) on oxygen with airbreaks. In the chamber he bled a couple of hundred ml then promptly closed up and subsequently did not bleed at all. This is what was interesting. We had no trouble with him after that. He rapidly improved and he was extubated the following day. He was on 50% oxygen and there was improvement in his neurological deficit especially the right arm sensory deficit as well as the past pointing. However his dysdiadochokinesis and his cerebellar dysfunction remained. The chest X-ray gradually cleared. There is no evidence of subcutaneous emphysema and there was no crepitus. He was treated again 30 hours post injury, this time with table 6. Improvement in cerebellar dysfunction was noted at this point and he had a CT scan of his head which was read as completely normal. By the 3rd day his chest X-ray had cleared further. He began to walk alone and was recompressed 50 hours post injury to 14 msw (45 fsw). This is the pressure where most of our clinical hyperbaric patients are treated. We treated him a further 3 times and as there was no further neurological improvement we stopped.

After the last treatment the neurological deficit was a wide based gait, his heel toe walk was abnormal and there was a residual abnormality of rapid alternating movement

of his left arm. A PaO_2 breathing air of 86 showed the resolution of his pulmonary problem. The haematocrit had dropped significantly suggesting a loss of about 25% of his red cell volume. The CPK peaked at about 5000 on the 3rd post injury day in the skeletal muscle band and this may have been ischaemic injury to the skeletal muscle. His ECG prior to discharge was normal and the CPK in the heart muscle band was not elevated so there was no evidence of myocardial infarction.

CPK is an interesting enzyme and we are studying if it can be used as an adjunct to diagnosing AGE. There may be evidence that ischaemic muscle injury has occurred. Such injury releases the enzyme and it can be detected biochemically. A significant muscle injury may represent massive or significant AGE.

Just prior to discharge he became shaky, had visual problems and felt uncomfortable. Most of this, I think, was apprehension about going home and the fact that he was an anxious individual. Other interesting results were a normal EEG while the nuclear magnetic resonance (NMR) scan showed cerebellar and cerebral cortical volume losses without any evidence of focal deficit. This goes back to what Dr How was saying earlier. Our patient had a normal CT scan and EEG but an abnormal NMR scan. It may be that the Though the NMR is more expensive it may be more sensitive to the specific neurological injury that we can not find clinically. He was discharged and went home to Alaska on his 6th post injury day.

Discussion

The mechanism of pulmonary barotrauma is something we have covered. It is an ascent injury that occurs from the lung actually rupturing. It can create a situation in which the gas runs either down the outside of the vessels or inside the vessels.

The pulmonary injury resolved fairly rapidly both on the chest X-ray and the arterial blood gases. In a series of our patients with AGE problems we found that the chest X-rays have not been very helpful. However looking at them retrospectively we can see small areas of pneumomediastnum but they are very subtle findings. They certainly were not of the magnitude that would require clinical treatment. From the submarine escape papers published, they did not do chest X-rays prior to recompression before about 1964. In those days the patient was picked up and put directly in the chamber. Today I think our cardiac resuscitative measures before and while we are in the chamber are a bit better. Their findings of evidence of pulmonary barotrauma on chest X-ray, whenever they were taken, was not extensive. That was surprising as one would expect from submarine escape training that there would have been more radiographic evidence.

Haemoptysis is a real problem for thoracic surgeons and physicians. In most non-diving patients it is is related to pulmonary arteriole rupture and some sort of bronchial pathology which leads to relatively rapid bleeding into the bronchus. This becomes a problem in airway maintenance. Another mechanism we can suggestis pulmonary hypertension, especially in this patient where there was evidence of pulmonary hypertension based on his ECG, leading to rupture of a pulmonary arteriole in a localised fashion. A third theory is that a diffuse form of bleeding occurs. The reason that I mention pulmonary hypertension is because this patient bled for about 12 hours following the incident and to get bleeding of that magnitude for that length of time there must be a higher than normal head of pressure in the pulmonary tree. We define haemoptysis usually in terms of both amount and time. Serious haemoptysis is 800 ml/24 hours. This is significant only if the patient drowns in his own blood. Submarine escape training is the primary model for arterial gas embolism. The trainees ascend from up to 100 feet at a rate 6-8 times that we normally use on scuba. So it is a pure ascent related thing. Even with these, haemoptysis is relatively rare.

Summary

I have presented a case of AGE complicated by massive haemoptysis. His cardiopulmonary arrest may have been due to the fact that his airway was not well controlled. Intubation is the key form of treatment at this time. Such patients do not need bronchoscopy, pulmonary angiography, bronchial artery angiography or CT scan when they prtesent but do need immediate recompression. This patient with his neurological insults and delayed recompression did quite well. He is presently active.

Questions and answers

Dr C Lourey

Do you have any idea what percentage of patients who have cardiopulmonary bypass get AGE? It is my impression that the incidence of AGE varies from nil in cardiothoracic units where there is no hyperbaric facility to a significant number in units where there is a hyperbaric facility.

Dr A. Santos

That goes back to the history of cardiothoracic surgery. The worst thing a cardiothoracic surgeon can do is to pump air into the patient. One takes blood from the venous system, oxygenates it, then pumps the oxygenated blood back into the aortic arch. If one does not watch the level in the oxygenator one can pump air into the patient. Unfortunately most thoracic surgeons know very little about hyperbaric medicine. There have been a number of patients in this situation and the surgeons did not know what to do about it. In thoracic surgery the patient completely

antiocoagulated, sometimes they get steroids and they are fully oxygenated. What a perfect set up to be taken to a chamber to be fixed and one would think the survival should be excellent.

Unfortunately one of my first experiences was a 39 year old woman operated on for an atrial septal defect. She got some air because of the technique and they did not do anything about it, they just basically let her go. It is only recently that a chamber has been put in that city. That represents the sort of problem that cardiac surgery can present. There is one surgeon in the US who operates in the chamber. He has been jumping up and down for years and no one has ever listened to him. Cardiothoracic surgeons are not the only ones in medicine who create that sort of a problem. People on dialysis have the problem with their arteriovenous fistulae when the dialysis machine infuses air into the patient. Neurosurgery also does the same thing to people sitting up when, especially in the posterior fossa approach, air can be sucked into a vein because of the low venous pressure. These require the same sort of recompression treatment.

Dr A Slark

I have a suspicion that this sort of case presents great concern to the people managing it because the physician is alarmed at the sight of blood and the surgeon always wants to do something, actively to put his finger on the leak. As soon as you put a patient in a chamber you have taken him away from the person who can actively stop the bleeding. Did you have a great deal of discussion about this particular pattern of management in this most unusual case?

Dr A.Santos

No. The uniqueness of the situation dictated that he just go to the chamber and no one thought any more about it. It is only one case so one can either postulate nothing or conversely one can postulate everything. It is certainly a bad time. One needs to have somebody in there with him, one needs to maintain the airway in the chamber and if something does go awry or persists too long one needs to do something about it. With diffuse pulmonary haemorrhage that would be very tough problem. Conversely if it was bronchial artery rupture we now have the capability of putting a catheter into the site of the rupture and embolising gel foam or clots into the artery and stopping the bleeding that way.

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HYPERBARIC MEDICINE IN CHRISTCHURCH, NEW ZEALAND, 1979-88

Michael Davis

Introduction

In 1980 at the SPUMS meeting in Singapore, I reported on the establishment of a new hyperbaric chamber at The Princess Margaret Hospital (TPMH), Christchurch, and our first year of activities[1]. The chamber was then, and still is, the only hospital-based multi-place chamber in New Zealand. Monoplace chambers exist in hospitals Dunedin (currently non-operational), Auckland and Wellington (operational). Until a decade ago, the only chamber actively treating divers was that at the Naval Base in Auckland, and even today about 80% of all decompression sickness (DCS) and cerebral arterial gas embolism (CAGE) cases in New Zealand are treated there.

The Christchurch facility's history is an interesting one. In brief, the chamber and a small compressor were donated to the Hospital Board by the local diving community in 1979 following a vigorous fund-raising effort. The whole thing was installed on the cheap, the Board undertaking to house and maintain the unit, but no funds were earmarked to establish a clinical service, staffing was to be on a voluntary basis.

The chamber was generally regarded to be a White Elephant, and Harry Guy (a respiratory physiologist now working for NASA) and I to be a pair of eccentric quacks. However, the referral of 33 patients in the first 15 months brought about something of a crisis, forcing the Hospital Board to address rather more constructively the provision of a "service", however rudimentary. Board policy was and still is that we may provide an acute emergency service only, and that there would be no additional staffing establishment to do it! Nevertheless, within these limits the Board has been supportive in many small ways over the years to maintaining our rudimentary service. At a time of severe financial constraints in health care in New Zealand, the establishment of formal hyperbaric medical services is likely to remain a low priority compared to many other perceived needs.

Despite the voluntary nature of the service, we are surprisingly well staffed. Medical cover is currently provided by 7 doctors, 3 consultants (2 anaesthetists and a geriatrician), 3 registrars and a family physician, all on a ring-round basis, there being no obligation to make oneself available. Only the family physician is paid on a fee-for-service basis by the Hospital Board, the rest of us do it for free, over and above our usual commitments. Only I had previous hyperbaric or diving medical experience.

We have 10 chamber operators. Four are Board employees, whilst the other six are local divers on whom