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CEREBRAL ARTERIAL GAS EMBOLISM

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Introduction

Pulmonary barotrauma arises from excessive changes in pressure affecting the lungs. This is widely known among the diving fraternity as "burst lungs". It is a clinical manifestation of Boyle's Law acting upon the effects of a burst lung. The clinical effects of pulmonary barotrauma such as pulmonary tissue damage, pneumothorax, surgical emphysema and cerebral arterial gas embolism (CAGE) are often serious and potentially life-threatening. Among recreational divers, CAGE is the commonest cause of death, which is often attributed to drowning. Leitch et al[1] reported an incidence of CAGE of 7 per 100,000 dives in a group of military divers. In the same study, CAGE was responsible for 31% of the diving accidents. Although with proper training and equipment the incidence is small, there is still a real risk in cases of panic surfacing, chronic smokers and those with a history of lung disease and previous pneumothorax. The various forms of pulmonary barotrauma are shown in Figure 1.

Pathophysiology

Cerebral arterial gas embolism is the most serious form of pulmonary barotrauma and early recognition and treatment is urgent. It results when air emboli are introduced directly into the blood circulation. CAGE can result in an acute anoxic episode with diffuse generalised ischaemia culminating inevitably in death if untreated.

The distribution of gas introduced into the arterial circulation is dictated by the posture of the diver and often exacerbated by the profuse liberation of air bubbles into the circulation. The air emboli distribution is dependant on the local perfusion pressure which is an interaction of the mean arterial blood pressure (MABP), cerebrovascular resistance and intracranial pressure. The interaction is made more complex by the variation of cerebrovascular resistance with mean arterial blood pressure, such that cerebral blood flow remains constant over a range of arterial pressures.[2]

Air embolus entrapment in a cerebral arteriole will occur when the forces opposing the embolus movement exceed the local perfusion pressure. These forces are related to the embolus size. The larger the embolus, the more likely is entrapment. For a given vessel this relates to embolus length, which is directly related to embolus volume and inversely related to the square of the diameter of the vessel it is occupying. Gorman[2] found that the cerebrovascular reaction to gas embolism differed from that to a solid embolus. In air emboli, localised vasodilation was seen while with solid emboli vasoconstriction was seen. In his animal experimental studies, cerebral arterial air embolism caused an increase of 42% + 28.13 SD in the cerebral arteriole diameters.

In gas embolisation a significant proportion of gas entering the cerebral circulation will pass through without causing vessel occlusion. In Gorman's study[2] spontaneous redistribution occurred in 5 out 30 animals (17%).

Entrapment of air emboli occurred in arterioles of 50-200 micrometers in diameter. The most frequently involved cerebral arterioles are those having diameters of about 150 micrometers. If an air embolus that had a length exceeding 500 micrometers entered an arteriole of this size entrapment with local circulatory arrest was inevitable. Conversely, if the length was less than 500 micrometers entrapment never occurred, with the embolus progressing without interruption.

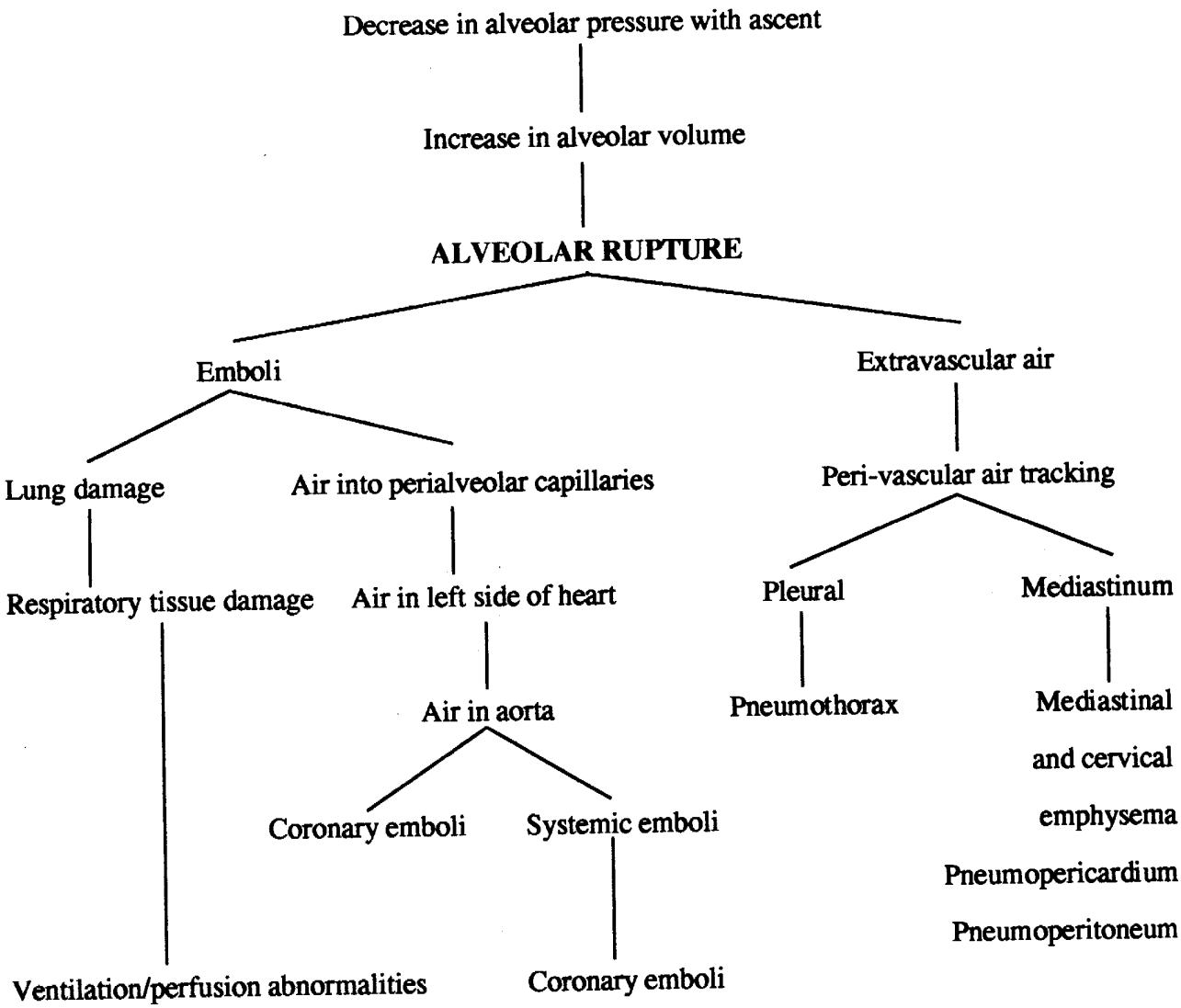
Pathological sequence of air embolism

Once CAGE causes vascular occlusion, a number of pathological processes are initiated. The following events take place:[2]

(a) Permeability of the blood-brain barrier increases.
(b) Ischaemia of tissues.
(c) Post-obstructive vessel coagulopathy.
(d) Microfoci of haemorrhage.
(e) Cerebrovascular autoregulation is lost.
(f) Changes in metabolism.

An increase in the blood brain barrier permeability results in increased brain water content, i.e. cerebral oedema. Tissue ischaemia results in infarction. The cortical gray-white junction is particularly vulnerable to ischaemia from air embolism due to its angio-architecture which causes preferential lodging of emboli in this border zone. The gray-white junction occupies a watershed between the penetrating arteries of the cortex and the deep penetrators nourishing the white matter.[3,4] Ischaemic hypoxia that is neither sufficiently prolonged nor profound enough to cause a classic watershed infarction can give rise to necrosis in the deep cortical areas alone. The passage of air emboli through vessels is associated with arterial thrombosis. This is thought to be mediated via platelets rich in serotonin and adenosine diphosphate (ADP) and aggravated by agents such as collagen, thrombin, adrenaline and noraderanaline. Gas bubbles in themselves function as platelet agonists. Scanning electron microscopy has shown interaction between air microbubbles and platelets. It shows adhesion of platelets

FIGURE 1



to the bubble surface with formation of aggregates.[5] Microbubbles act as a foreign surface and a resultant platelet clumping causes a fall in platelet concentration with formation of platelet micro-thrombi. Endothelial oedema and formation of platelet thrombi cause progressive arteriolar occlusion, small focal haemorrhages and cellular metabolic disruption. The process is further aggravated by haemoconcentration secondary to increased vascular permeability.

Case presentation

Numerous studies have shown the beneficial effects of hyperbaric oxygen in the management of ischaemic disorders of the brain and in cerebral oedema.[6] The following is a clinical case treated at the Naval Medicine and Research Centre, Singapore (NMRC). I would like to discuss this case to establish a clinical approach in the management of CAGE that results from pulmonary barotrauma.

Clinical History

HC was a 42 year old Chinese male, seen at NMRC in September 1984. Prior to the incident he was a sports diver for one and a half years with no previous professional training in diving. He used to dive about once a month for a few days at a time. He had a history of viral pneumonia in December 1983 which resolved with treatment but no other past history of note.

On 2 September 1984 at 1300 hours after diving for about half an hour to a depth of 30 metres off the coast at Mersing, he was seen to suddenly surface. He was apparently able to inflate his life jacket. However, when the safety boat (sited 60 metres away) reached him, he was found to be unconscious with blood stained froth discharging from his mouth. His scuba tank was found to be emptied of air. There was no spontaneous pulse and respiration. CPR was started. After about 12 minutes he regained spontaneous pulse and respiration but remained unconscious. He was

placed on his side and evacuated to a hospital in Mersing (6 hours away). At Mersing Hospital, he was noted to be in coma. Pulse was 102/min, BP 120/70, respiration 28/min. His pupils were sluggish reacting to light. He responded to pain. He was intubated and given I/V dextrose, ampicillin, decadron and frusemide. I/M anti tetanus toxoid was also administered.

He was evacuated to Singapore by helicopter. During the flight he had 2 fits. He arrived at the Department of Neurosurgery, Tan Tock Seng Hospital, on 3 September 1984 at 1200 hours. He was assessed to be in Coma III. His pupils were noted to be unequal. The left pupil was 5 mm and the right pupil 4.5 mm. He showed a decerebrate response to pain. He had a febrile episode which was investigated. A provisional diagnosis of diffuse anoxic brain damage secondary to a diving accident was made by the neurosurgical team.

Investigation

Table 1 shows the investigations done during his stay in hospital.

On admission his urea was mildly elevated at 79 m eq/l. This was probably due to dehydration as he had a raised haematocrit. Septic workout in the form of lumbar puncture, urinanalysis, chest X-ray and sputum analysis were done for his fever and raised total white count. Klebsiella was isolated from sputum culture and his endotracheal tube and he was treated with the appropriate antibiotics. His fever subsequently resolved and total white count returned to normal. Chest X-ray was reported to have evidence of infective changes.

The blood gas picture showed a compensated metabolic acidosis with a low PCO_2, low HCO_3 and a negative base excess. The anion-gap on 3 September 1984 was 10 m eg/l and in the presence of normal K^+ levels was consistent with early renal failure. This was probably of pre-renal origin and hydration helped reverse the picture. His blood coagulation profile was normal and platelets were within normal range without evidence of consumptive coagulopathy. CT scans of the head done on admission and 9 days later were reported as normal. A repeat scan one month later showed features consistent with resolving cerebral oedema or cerebral atrophy.

Hyperbaric Treatment

He was referred to the Naval Medicine and Research Centre on 4 September 1984 at 2300 hours about 58 hours after the diving accident. A clinical diagnosis of pulmonary barotrauma with air embolism was made based on the history.

Hyperbaric treatment was instituted with oxygen using an extended Table 6A for a duration of 7 hours. No definite changes were noted in his coma state immediately after treatment. On the third day following treatment, he was noted to be in a “lighter state” by ward staff and the neurologist. Four days later he was able to withdraw from pain. Seven days after the treatment he was able to open to his eyes. In the following days to weeks, his condition gradually improved. Auditory evoked potentials done on 11 September 1984 showed signs of brainstem demyelination. This test is useful in the diagnosis of small vessel lesion, pontine myelinoses and hypoxic brain damage.

TABLE 1

INVESTIGATIONS

Full Blood Count

Date	**3/9**	**5/9**	**7/9**	**8/9**	**13/9**	**18/9**	**28/9**
WBC	25.7	15.8	19.7	16.9	18.4	15.1	10.1
RBC	5.41	5.41	4.52	4.65	3.90	3.63	3.52
Hb	16.2	16.3	13.7	13.7	11.8	11.0	10.9
Hct	51.6	52	43.3	43	37.9	34.2	34.0
MCV	93	94	93	90	94	92	94
P	98	86	96	83	98	91	86
L	2	12	4	10	2	8	12
M	-	2	-	7	-	1	2
E	-	-	-	-	-	-	-
Platelet	-	-	-	250	-	310	-

TABLE 1 (CONTINUED)

Urea and Electrolytes

Date	**3/9**	**5/9**
Urea	79	48
Nat	149	145
K+	4.7	4.3
Cl-	119	110
NCO_3	-	28.1
G	2.8	1.3
G/U	170	120

Blood Gases

Date	**4/9**	**5/9**	**6/9**
O_2%	Air	40%	40%
pH	7.39	7.375	7.457
PCO_2	33.6	29.7	33.0
PO_2	77.9	135.6	77.0
HCO_3	20.0	17.0	23.1
TCO_2	21.0	17.9	24.1
Base excess	-3.8	-6.5	-2
SBE	-4.1	7.1	-0.4
O_2 saturation %	94.1	98.2	94.9
SBC	21.1	19.1	24.5

PT:	14 seconds	Control	13 seconds
PTT:	30 seconds	Control	38 seconds or less

Urine Microscopy (3/9/84)

Packed with RBC AP +++ Granular casts +

Lumbar Puncture (4/9/84)

CSF appearance	clear
Cell count	11
Sugar	129
Total protein	less than 10
Globulin	negative

No torula or AFB organism

Liver Function Tests (16/2/85)

TP	6.5 g/ds
Alb	3.9 g/ds
Bil	0.3 mg/dl
Alk PO4 alt	69 u/l
ALT	28 u/l

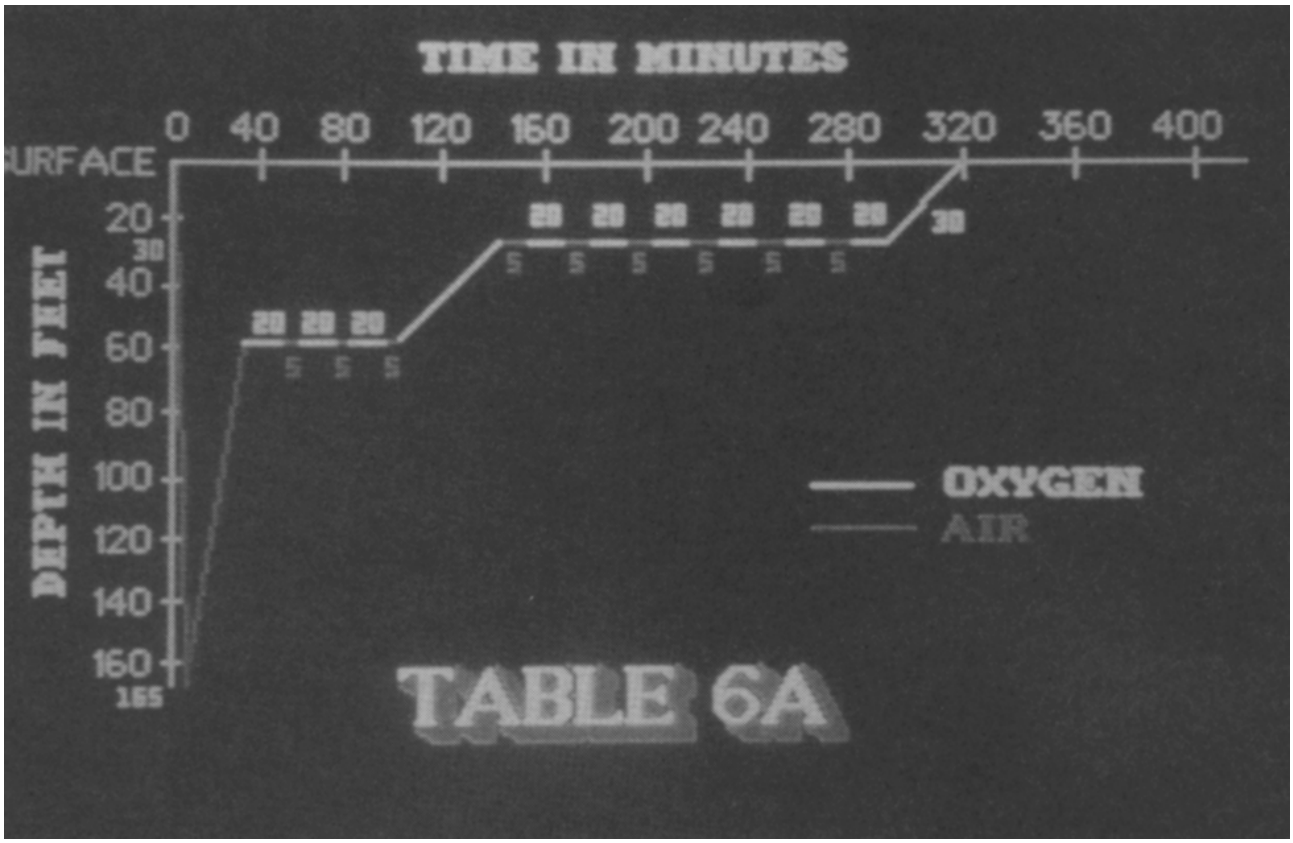
Auditory evoked potential (11/9/84)
Showed brainstem demyelination.

Culture and sensitivity tests

Blood	5/9/84	Negative
CSF	4/9/85	Negative
Sputum	5/9/85	grew Klebsiella
ETT Swab	5/9/85	grew Klebsiella
Blood	7/9/84	Negative
Urine	8/9/84	grew Klebsiella
Blood	22/9/84	Negative

Radiology

Chest X-ray	3/9/84	Ward film taken. Bilateral infective changes.
CT Scan	3/9/84	NAD
	12/9/84	Normal
	8/10/84	Compared to that of 3/9/84 the scan shows that the lateral ventricles and cerebral sulci are slightly larger, consistent with resolving cerebral edema or cerebral atrophy.



Progress

By early October he was able to open his eyes spontaneously and blink at a visual threat. Pupils were equal and reactive, corneal and gag reflexes were present and Doll's eye manoeuvre positive. By early December he had regained control of bladder and bowel function and was able to respond to verbal commands. There was generalised hypertonus and reflexes were brisk but power had improved. Speech therapy was started soon after in late December and over the next few months with a regular programme at the rehabilitation centre he improved considerably.

Today, though confined to a wheelchair, he is able to carry out most of his daily activities such as toilet, dressing, eating and movement. His speech is mildly impaired and he has some loss of recent memory but apart from this, he is able to function well. His last tested IQ was 98 which falls within average intelligence category.

Disscussion

The commonest presenting symptoms and signs of air embolism arising from pulmonary barotrauma are the loss of consciousness, sensation and the loss of motor power depending on the area of brain involvement. A study of 117 cases of CAGE in divers done by Leitch[1] is shown in Table 2.

TABLE 2

PRESENTING SIGNS AND SYMPTOMS OF CAGE

(prepared from information in Leitch and green.[1])

Unconsciousness		38.5%
Power Loss		
(a)	Generalised	6.8%
(b)	Legs	16.2%
(c)	Unilateral	26.5%
Sensory changes		
(a)	Unilateral	19.7%
(b)	Bilateral	22.2%
Loss of co-ordination		11.0%
Visual changes		21.4%
Confusion/disorientation		36.7%
Speech problems		11.1%
Convulsions		
(a)	Generalised	8.5%
(b)	Focal	2.6%

Other symptoms included somatosensory changes, personality changes, retrograde amnesia, vertigo and deafness.

In the case of HC, unconsciousnss and convulsions were noted as presenting symptoms.

The clinical presentation was that of anoxic encephalopathy secondary to cerebral air embolsim. Anoxic encephalopathy is commonaly seen in other conditions such as:

(a) Suffocation
(b) Carbon monoxide poisoning
(c) Neuromuscular disease
(d) Myocardial Infarction, with secondary circulatory collapse.

The severity of the clinical presentation depends on the degree of anoxia.[7] In mild anoxia consciousness returns after a short period of coma and full recovery is usual.

In moderately severe anoxia the patients are profoundly comatose with decerebrate postures. In the first 24-48 hours, death may terminate this state in a setting of rising temperatures, deepening coma and circulatory collapse. Such individuals usually survive, if at all, in a state of irreversible coma or persistent vegetative state. If coma lasts for more than 5 days the patients almost never recover.

In severe anoxia complete unawareness and unresponsiveness is noted with the abolition of brainstem reflexes. Natural respiration cannot be sustained. Only cardiac action and blood pressure are maintained. EEG shows no electrical activity (i.e. isoelectric). This is the brain death syndrome.

HC fell into the second group of moderately severe anoxia based on his clinical presentation and the natural course of events should have been progression to death or at best a persistent vegetative state.

Hyperbaric oxygen was used to combat his ischaemic cerebrovascular condition and his traumatic brain oedema. The use of hyperbaric (HBO) produces more dissolved oxygen in plasma resulting in improvement of hypoxia in the ischaemic tissue. It is also known that progression of brain oedema can be suppressed by cerebral vasoconstriction due to high PO_2 in combination with the increased availability of oxygen.

In animal experiemental studies the use of HBO therapy up to 3 hours after artificially induced ischaemia, via bilateral carotid artery ligation, significantly prolonged survival time compared with that in non-treated ischaemic animals.[3] The mechanism of this has been attributed to oxygenation of ischaemic tissue, suppression of brain oedema and reduction in red cell agglutination. In addition, ATP in the treated group tended to be higher than in the non-treated group. The metabolic data suggest that prolonged survival in these treated animals is due to suppression of further increase in lactate.

In the management of pulmonary barotrauma with air embolism, it has been recognised that the time from onset of symptoms to adequate recompression therapy is the main determinant of success in treatment. Any delay in therapy allows progress of the blood-bubble interaction which leads to intravascular coagulation, capillary leakiness, oedema, haemoconcentration and infarction.

In a review of 117 cases of CAGE Leitch[1] noted 21% of individuals recovered completely without treatment. In those treated, the relationship between delay and response to treatment is shown in Table 3.

Cure was defined as apparently complete recovery without signs or symptoms at any time following treatment. In our case HC was started on treatment 58 hours after the diving accident. From the study above it was noted that likelihood of cure was reduced as the delay to treatment increased. The cut-off time for favourable outcome was about 4 hours. The likelihood of achieving a cure was reduced to less than 50% when the delay exceeded 4 hours.

While commercial or naval diving operations usually have access to on-the-scene recompression chambers, the growth

TABLE 3

RESPONSE TO TREATMENT IN CAGE CASES

Delay to treatment	Cases cured	Cases not cured	Total cases treated
< 1 hour	16	8	24
1 - 2 hours	13	2	15
2 - 3 hours	12	4	16
3 - 4 hours	7	4	11
4 - 5 hours	3	4	7
> 5 hours	7	9	16
Totals	**58**	**31**	**89**

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