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Hyperbaric Oxygen Therapy for Central Nervous System Damage Induced by Air Embolism

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SUMMARY

The clinical experiences of hyperbaric oxygen therapy for decompression sickness with spinal cord involvement and cerebral air embolism were reported. From 1977 to 1980, 17 cases of decompression sickness with spinal cord involvement among SCUBA divers and 3 cases of cerebral air embolism were treated in our hyperbaric unit. In cases of decompression sickness, the duration between the onset of accident and the start of treatment were from 3 hours to 4 days and the half of them had complete paraplegia. In these cases, there were 4 cases with permanent impairment and the half of them had minimal neurologic residue after the treatments. In cases of cerebral air embolism, 2 cases following induction of diagnostic pneumomediastinography were treated immediately after accidents with complete recovery. A case as a result of open heart surgery was treated after 25 hours delay with clinically almost complete recovery.

These cases confirm the fact that hyperbaric oxygen therapy may still be of use even if the onset of therapy is delayed and also applicable to the central nervous system damage from other causes suspected hypoxia.

INTRODUCTION

Air embolism is a serious disorder and damage to the central nervous system which sometimes cannot be corrected, especially in delayed cases. Reported causes include accidental (iatrogenic) arterial gas embolism, such as perfusion for heart surgery; heart procedures (especially on the left side or in the presence of septal defects); intravenous therapy (especially with pressure injection or the presence of a patent foramen ovale etc.); placement or use of central venous catheters; neurosurgery (especially in the upright position); diagnostic or surgical procedures involving the lung; surgery of the aorta or cervical arteries, head and neck procedures in surgery; hemodialysis; abdominal or retroperitoneal gas insufflation; liver transplantation, and diving accidents.[1]

The successful immediate therapy with hyperbaric oxygen for these cases has been reported, but the treatment for severe delayed cases are still difficult requiring comprehensive intensive care.[1)~12)]

The purpose of this report is to present our clinical experiences of hyperbaric oxygen therapy (HBO) for cerebral air embolism and severe decompression sickness with spinal cord involvement.

CASE MATERIAL AND METHOD OF TREATMENT

From 1977 to 1980, our hyperbaric unit treated 3 cases of cerebral air embolism and 17 cases of decompression sickness with spinal cord involvement among SCUBA divers.

The treatment was conducted in the compressed air multiplace chamber with oxygen supplied by a built-in breathing system.[13]

1. Cases of decompression sickness:

The interval between the onset of accident and the start of treatment of these 17 cases were from 3 hours to 4 days. The half of them were moved by helicopter or cessna from isolated islands in Okinawa, keeping the flight altitude as low as safely possible but not greater than 200 m. grand level. It is said that the dominance in the neurological manifestations of acute decompression sickness is the spinal cord; the most commonly affected area is the middle third of the spinal cord.[14] The most common levels of proximal involved spinal cord segments in our cases were at the level of C4, T5 and L1, and 6 cases had complete paraplegia; shown in Table 1.

Table 1 Cases of decompression sickness (signs and symptoms at admission)

Case	Age (yrs.)	Delay # (hrs.)	Sensory ## Disturbance	Moter Disturbance		Recto-urinary Disturbance	By air Transportation
1	21	12	C4	complete paraplegia	(flaccid)	+	+
2	45	8	C4	〃	〃	+	+
3	28	4	T5	〃	(spastic)	+	+
4	43	12	T7	〃	(flaccid)	+	+
5	36	20	T5	〃	〃	+	
6	24	24	L1	〃	〃	+	+
7	43	24	C4	right hemiparesis		+	+
8	40	5	C4 (left)				
9	30	18	L1				
10	36	72	L1 (right)				
11	45	24	T10				
12	31	3	L1				
13	24	5	T5				
14	25	48	T5	slight impaired gait		+	+
15	40	24	T9	〃		+	+
16	34	3	L1	〃			
17	45	96	L1				+

interval between accident and therapy

level of spinal cord involvement (upper involved segment)

The treatment was in accordance with the U.S.Navy minimal pressure oxygen treatment Table 6[15] in conjunction with drug and fluid therapy, such as corticosteroid, lower molar dextran, Urokinase etc. Table 5[15] or 2.8 atmosphere absolute (ATA) of HBO were administered once a day for a week following several Table 6 treatments and afterwards 2 ATA of HBO continuously as long as definite improvement was seen. The treatment tables used are shown in Fig. 1.

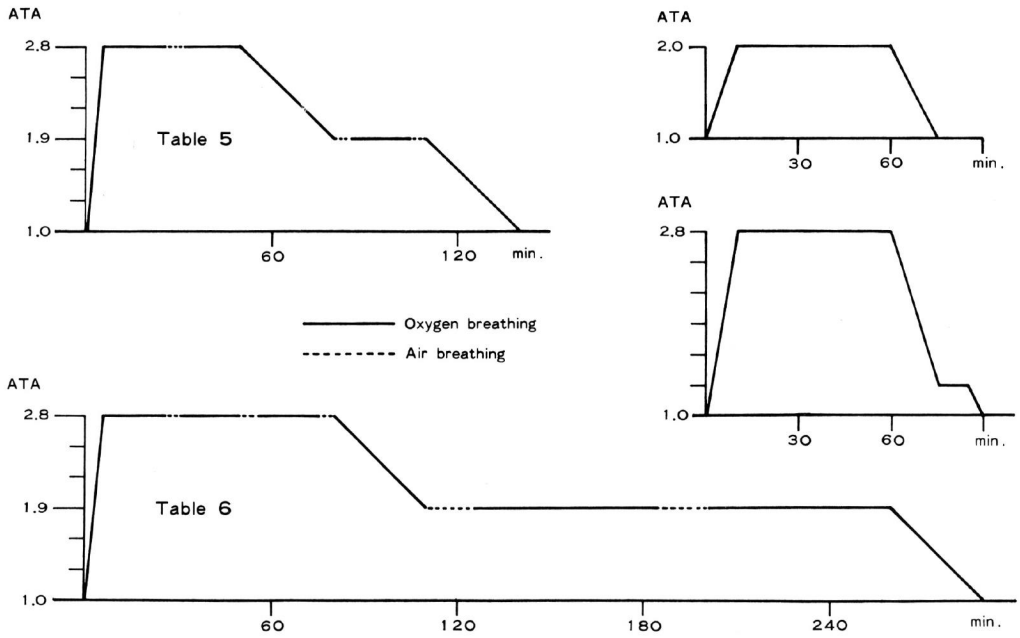

Fig. 1 Hyperbaric oxygen therapy Table used for cases of air embolism.

The results of treatment: There were 4 cases with permanent impairments, such as neurogenic bladder and impaired gait. Also half of the cases had minimal neurologic residue, mostly partial sensory disturbances at the time of discharge. There wes no fatality. The result of each case is shown in Table 2.

Table 2 Hyperbaric oxygen therapy for cases of decompression sickness

Case	Hyperbaric Treatment (No.)	Treatment Results (at discharge)
1	64	slight claudication ⟶ recovery
2	90	urinary disturbance, aided walking (crutched)
3	54	aided walking (crutched)
4	67+ (9)	urinary disturbance, aided walking (crutched)
5	77+ (3)	paraparesis (wheel-chair)
6	35	
7	14	slight claudication ⟶ recovery
8	5	
9	9	
10	1	
11	13	
12	1	
13	5	
14	30	
15	16	
16	15	
17	4	

( ) : treatment at other facility

2. Cases of cerebral air embolism:

Case 1, 21 year-old male and Case 2, 65 year-old male received diagnostic pneumomediastinography. Soon after the injection of air, patients showed confusion and left-sided hemiparesis. Immediately after intravenous pentobarbital injection, the treatment in the hyperbaric chamber was started within 30 min. In Case 1, during the compression, the patient showed recovery and complete recovery with 2 ATA of HBO. In Case 2, complete recovery was obtained by Table 5, but slight paresis occured in the ward which disappeared after another Table 5 treatment. The details are shown in Table 3.

Table 3 Cases of cerebral air embolism following diagnostic pneumomediastinography
(A) 21 year-old male with myasthenia gravis suspected
(B) 65 year-old male with mediastinal tumor suspected

A)

Sept. 7, 1978	Pneumomediastinography (about 400 ml of air used)		
	immediatly after Injection		confusion, left-hemiparesis
		barbiturate Injection	
	after 20 min.		left-arm paresthesia
	after 30 min.	2 ATA of HBO	
	after HBO		complete recovery

B)

May 24, 1979	Pneumomediastinography (about 400 ml of air used)		
	immediately after injection		confusion, right-arm cramp
			left-hemiparesis
		oxygen inhalation	
		barbiturate injection	
	after 30 min.	Table 5	
	after treatment		complete recovery
	at ward		left-leg paresthesia
May 26		2.8 ATA of HBO	complete recovery

Case 3, 35 year-old male underwent open heart surgery under cardiopulmonary bypass for correction of ruptured aneurysm of Valsalva's sinus and VSD. Two hours postoperatively in ICU, the patient developed a series of general convulsions despite administrations of anticonvulsants and muscle relaxant and remained comatose. The patient had been maintained under mild hypothermia and mechanical ventilation with high oxygen concentration. Twenty five hours after the suspected occurance of air embolization where air was introduced into arterial line by compressing cold cardioplegia solution, the patient was transfered to the hyperbaric chamber and HBO was given once a day. After the 2nd HBO, convulsions disappeared, and the patient regained full consiousness after the 3rd HBO. However, retrograde amnesia and right arm paresis persisted for about one week, which disappeared by continuous daily 2 ATA of HBO. After repeated HBO, the patient had only minimal neurologic residue and was discharged one month following the operation with clinically almost complete recovery. The details are shown in Table 4. During the HBO therapy, no abnormality was seen on the CT-scan of the brain, but the EEG showed dominant activity slower than normal throughout. It improved gradually, but diffuse abnormality still remained even at the time of

discharge, as shown in the Fig. 2.

Table 4 Case of cerebral air embolism following open heart surgery

35 year-old male with raptured aneurysm of Valsalva's sinus and VSD

Day	Hyperbaric Oxygen Therapy and Other Treatments	Main Symptoms
operation	mild hypothermia, IPPB	unconsciousness, convulsion, right-hemiparesis
post-op, 1	2.0 ATA of HBO 〃 〃	〃 〃 〃
〃 2	2.8 ATA 〃 〃 〃	〃 (−) 〃
〃 3	2.8 ATA 〃 normothermia, spon, resp,	conscious 〃
〃 4		retrograde amnesia (up to 15 yrs), right-hand paresthesia
〃 5	2.0 ATA 〃	〃 (〃 12 〃), 〃 〃
〃 6	2.0 ATA 〃	〃 (〃 5 〃), right-hand paresis
〃 7	2.0 ATA 〃	right-hand paresthesia
〃 8	2.0 ATA 〃	
〃 16	2.0 ATA 〃	slight right-hand paresthesia
〃 20		
〃 35		discharged with clinically complete recovery

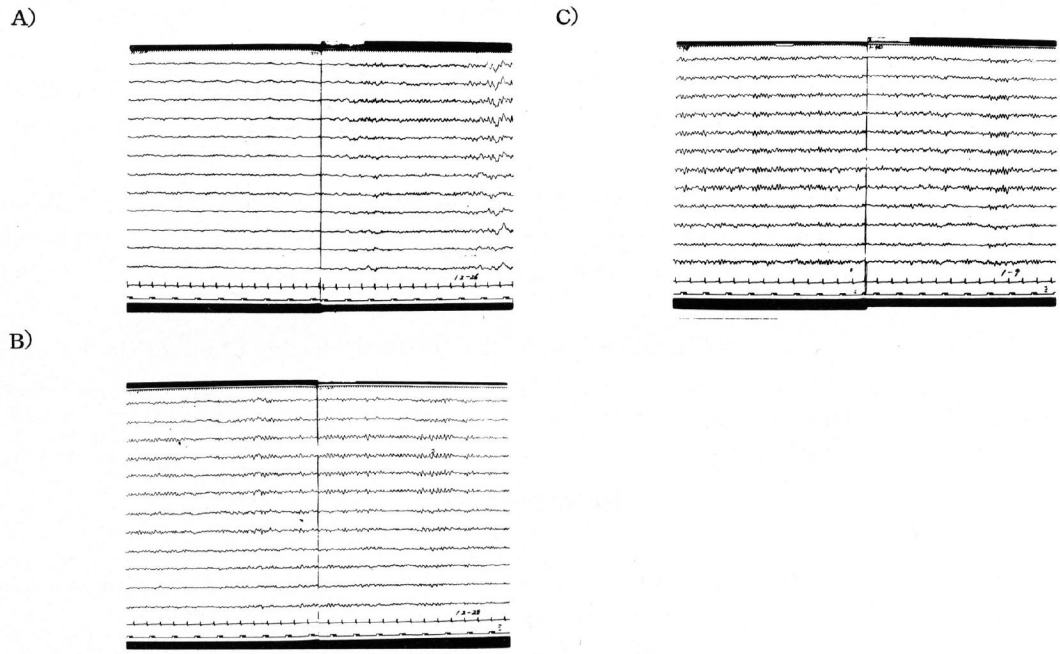

Fig. 2 EEGs of cerebral air embolism Case 3.

A) 6 days after operation

B) 8 days after operation

C) 20 days after operation

DISCUSSION

Hyperbaric oxygen therapy has been established as an effective treatment for air embolism as well as decompression sickness, which are resulting from nitrogen bubbles both intra and extra vascular, and many successful hyperbaric treatments have been reported.[1]~[12] Applied early, this therapy is the only specific and the mechanical compression alone restores circulation, even when the embolization is massive[16], as Case 1 of cerebral air embolism. Sometimes the outcome without hyperbaric oxygen therapy can be favorable, especially during extracorporeal perfusion for open heart surgery in which the patient suffering air embolism has been protected by prior heparinization, corticosteroids, mild hypothermia etc[11]. In delayed cases, however, residual damage or failure is increasingly more likely[6], because the secondary effects triggered by bubbles undoubtedly play a large role in the patient's poor neurologic status, such as sludging of the blood, increased platelet aggregation, micro-thrombi, hemocncentration and the certain production of profound tissue hypoxia and hypoxic edema.

In this point of view, hyperbaric oxygen therapy can be favorable, because the large amounts of oxygen disolved in the plasma may maintain viability in tissus by diffusing more readily into poor micro-circulation area, in conjunction with decrasing intracranial pressure and edema. This is probably what happened in our patient Case 3 and delayed cases of decompression sickness with spinal cord involvement.

CONCLUSION

The amount of air embolized and the duration between embolization and the onset of treatment cleary affect the severity of injury, and although hyperbaric oxygen therapy should be initiated as soon as possible. after the accident.

Our clinical experiences reported, however, confirms the fact that hyperbaric oxygen therapy may still be of use even if the onset of therapy is delayed and also applicable to the central nervous system damage from other causes suspected hypoxia.

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