

Hyperbaric Oxygen in the Treatment of Posthanging Cerebral Anoxia

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Mathieu D, Wattel F, Gosselin B, Chopin C, Durocher A. Hyperbaric oxygen in the treatment of posthanging cerebral anoxia. *J Hyperbaric Med* 1987; 2(2):63-67.—Between 1971 and 1981, 170 patients, who had attempted suicide by hanging, were admitted in our emergency unit. On admission, 19 were conscious or confused and 151 unconscious: 54 in grade I coma, 26 in grade II, 30 in grade III, and 31 in grade IV. Brain death was pronounced in 10 patients. Supportive and anticerebral edema measures were provided and, for 136 patients 278, 90-min hyperbaric oxygen (HBO) courses at 2.5 ATA. Thirty-four patients with only minor neurologic problems received normobaric oxygen. Of the 170 patients, 132 recovered completely without any residual (78%), 8 recovered with neurologic sequelae (5%), and 30 died (17%). Neurologic recovery was obtained after an average of 0.8 HBO course in patients with grade I coma, 1.5 HBO courses in patients with grade II, 2.5 HBO courses in patients with grade III, and 3.9 HBO courses in patients with grade IV. In no case where coma persisted was recovery obtained after the 5th course. No patient with neurologic depression above grade III coma died and 10 patients died through complications unrelated to the cerebral anoxia. Results are significantly better when HBO treatment begins before the 3rd h after the unsuccessful hanging (recovery without neurologic sequela: 85 vs. 56%, $P < (0.05)$).

hyperbaric oxygen, cerebral anoxia, hanging, strangulation

Introduction

Hanging leads to cerebral anoxia and death by gradual or acute, partial or complete failure of brain oxygen supply. Hyperbaric oxygen therapy has been proposed in the treatment of cerebral anoxia from various origins (cardiac arrest, carbon monoxide poisoning, asphyxia).

In a previous work, we showed that in unsuccessful hanging, HBO is associated with a neurologic recovery both quicker and of better quality than normobaric oxygen (NBO) therapy (1). In this paper, we report our 10 yr experience with HBO in the treatment of postcerebral anoxia.

Material and Methods

Patients

Between 1971 and 1981, 170 patients (135 males, 35 females) were admitted in our emergency unit after unsuccessful hanging. Ages ranged from 10 to 83

yr (mean \pm SD: 41.5 \pm 13.5 yr). Hanging was accidental in 4 cases (all children) and self-attempted in all others, of which 9 were prisoners and 71 suffered from psychiatric disease. Drug intoxication was associated in 26 cases; for 15 patients it was the second attempt.

On admission, 19 patients were conscious or slightly obtunded; 151 had an altered conscious level. Using the Glasgow coma scale, 54 were in grade I, 26 in grade II, 30 in grade III, 31 in grade IV. Brain death was pronounced in 10. Agitation was common in grade I and II comas. Extrapyraximal rigidity, Babinski reflex, and convulsions could be observed in grade III and IV.

Arterial blood gases (during spontaneous ventilation in room air) showed moderate hypoxemia whatever the coma grade. Metabolic acidosis appeared in grade III coma and was more important in grade IV coma (Table 1).

Comparisons between groups were made by the chi-square method.

Treatment

Supportive measures (intubation, assisted ventilation, bicarbonate and fluid therapy) were applied according to the patient state.

Steroids or β -1-24-tetracosactide was used in every comatose patient until the recovery of consciousness or the 3rd d.

Hyperbaric oxygen therapy was provided for 90 min in a monoplace chamber compressed to 2.5 ATA. A Vickers pure oxygen hyperbaric chamber was used until 1978; today a COMEX-PRO air hyperbaric chamber is in use, the patient breathing pure oxygen by facial mask or assisted ventilation. HBO was repeated until the conscious level returned to normal. When several HBO courses were needed, they were separated by 6-h intervals. Between two courses of HBO, patients received normobaric oxygen (5 liter/min). Only patients with impaired conscious levels were selected for HBO, but 15 patients in grade I coma on admission recovered before HBO was available. Altogether 278 HBO courses were provided to 136 patients.

Results

One hundred thirty-two patients out of 170 (77.5%) recovered without any residual; 30 (17.5%) died; 8 (5%) recovered with neurologic sequelae (chronic

Table 1: Arterial Blood Gases on Admission (65 Patients Breathing Room Air Spontaneously, Mean Values \pm 1 SD).

	Coma Grade (No. Patients)			
	I (13)	II (15)	III (21)	IV (16)
pH	7.37 \pm 0.10	7.40 \pm 0.12	7.30 \pm 0.15	7.24 \pm 0.10
PaO ₂ mmHg	78.9 \pm 11.4	73.4 \pm 14.1	65 \pm 7.1	60.6 \pm 10.2
PaCO ₂ mmHg	37.6 \pm 6.3	39.7 \pm 8.4	36.7 \pm 3.6	35.6 \pm 9.4

coma 3 cases, locked-in syndrome 2 cases, extrapyramidal rigidity 1 case, hemiplegia 1 case, hemiasomatognosia 1 case).

Every patient in grade I and II coma on admission recovered, but 2 out of 30 grade III coma patients (7%) and 8 out of 31 grade IV coma patients (26%) did not improve under HBO. The 10 patients pronounced brain dead were, of course, not improved by HBO and died (Table 2).

Cervical trauma was responsible for 21 complications: laryngeal edema 5 cases, dysphagia 1 case, dysphonia 2 cases, phrenic paralysis 5 cases, brachial plexus paralysis 3 cases, vertebral fracture 1 case, periglottic abscess 1 case. None of these led to permanent sequelae.

After admission, complications occurred in 10 patients and were the cause of death even when the neurologic state improved: pulmonary edema 5 cases, pulmonary infection 4 cases, cardiac arrest 1 case.

A relationship with HBO could only be invoked in 3 of these complications; 2 pulmonary edema appeared during the course of HBO, and the cardiac arrest could not be resuscitated in time because of the monoplace chamber.

Discussion

The usefulness of HBO in the treatment of cerebral anoxia is still controversial, and definitive studies on this point are difficult because of the heterogeneity of the clinical situation leading to cerebral anoxia. Hanging produced a cerebral anoxia of peculiar interest for three reasons. First, carotid blood flow interruption is the predominant mechanism of anoxia; second, no hypoxia preceded the cerebral anoxia; and third, the delay between hanging and the first HBO course can be easily determined. For these points, posthanging cerebral anoxia can be considered a good clinical model to study the effect of HBO on cerebral anoxia.

Experimentally, some facts favor the use of HBO in cerebral anoxia. First, by its hemodynamic effects on the cerebral circulation, HBO reduces intracranial pressure (2-6) which is elevated in the postanoxia period, in particular

Table 2: Neurologic Evolution Under Oxygen Therapy

Conscious Level	Normal or Obnubilated, <i>n</i> 19	Coma Grade (No. Patients)					
		I (15)	II (39)	III (26)	IV (30)	V (10)	
Oxygen therapy	NBO	NBO	HBO	HBO	HBO	HBO	HBO
Complete recovery	19	15	38	25	26	19	—
Recovery with sequelae	—	—	1	1	2	4	—
Failure	—	—	—	—	2	8	10

after hanging where cerebral venous return is first stopped by the neck compression. By this effect, HBO counters the major factor of maintenance of the cerebral anoxia. Second, HBO favors oxygen diffusion to cells allowing metabolism repairing (7), in particular, the recovery of aerobic metabolism by the cerebral cell which reduces the quantity of intracerebral lactic acid (8). Third, HBO enhances the activity of the pentose shunt in the neuroglia cells which also favors the neuronal metabolic repairing (9).

In a previous work about unsuccessful hanging (1), we showed that HBO was associated with a recovery both quicker and of better quality than normobaric oxygen therapy. In this paper, we confirm the efficacy of HBO in this indication and conclude the following. First, neurologic state on admission influences the evolution under HBO; recovery without sequelae is the rule in grade I (53/54) and II (25/26); less common in grade III (26/30) and IV (19/31). Only 1 patient out of 54 (2%) in grade I coma on admission maintained neurologic sequelae compared with 4 out of 31 in grade IV (13%). Neurologic recovery was obtained after 0.8 HBO course in grade I coma patients, 1.5 HBO courses in grade II coma, 2.5 HBO courses in grade III coma, and 3.9 HBO courses in grade IV coma. In no case where coma persisted was recovery obtained after the 5th HBO course.

It appears also that the delay between hanging and the first HBO course influences the neurologic evolution. If the first HBO course begins before the 3rd h after hanging, neurologic recovery without sequelae is obtained in 94 out of 111 (85%), compared with 14 out of 25 (56%) if the delay is longer ($P < 0.05$). Although indirect, this fact argues in favor of the usefulness of HBO in unsuccessful hanging, and it is remarkable that such a "golden period" has been found also for other types of cerebral anoxia (10) or spinal cord injury (11).

Conclusion

From the study of 170 patients with posthanging cerebral anoxia we concluded that HBO therapy is a useful tool in the management of these patients. Best results are obtained when HBO therapy begins within the 3rd h after hanging.

References

1. Voisin C, Wattel F, Pruvost Ph, et al. Intérêt de l'oxygénothérapie hyperbare dans le traitement de la strangulation par pendaison. A propos de 35 observations. *Maroc Med* 1973; 53:302-305.
2. Ashfield R, Drew CF. Clinical use of the hyperbaric oxygen bed. *Postgrad Med J* 1969; 45:643-647.
3. Harper AM, Jacobson I, Macdowall DG. The effect of hyperbaric oxygen on the blood flow through the cerebral cortex. In: Ledingham I, ed. *Proceeding of the second international congress on hyperbaric oxygenation*. London: Livingstone, 1965:184-189.

4. Jacobson I, Lawson DB. The effect of hyperbaric oxygen on experimental cerebral infarction in the dog. *J Neurol Surg* 1963; 20:849-859.
5. Ketty SS, Schmidt CF. The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J Clin Invest* 1948; 27:484-489.
6. Ledingham I, Macdowall, Harpep AM. Cerebral cortical blood flow under hyperbaric conditions. In: Brown I, Cox B, eds. *Proceeding of the third international conference on hyperbaric medicine*. National Academy of Sciences, Washington, DC: 1966:243-249.
7. Lambertsen CJ, Kough RH, Cooper DY, Emmel AL, Loeschicke, Schmidt CF. Oxygen toxicity: effects in man of oxygen inhalation at 1 and 3.5 atmospheres upon blood gas transport, cerebral circulation and cerebral metabolism. *J Appl Physiol* 1953; 5:471-478.
8. Hollin SA, Espinosa OE, Sukoff MH, et al. The effect of hyperbaric oxygenation on cerebrospinal fluid oxygen. *J Neurosurg* 1968; 29:229-235.
9. Brue F. La toxicité de l'oxygène hyperbare. Physiopathologie, cytotoxicité protection, activation du cycle des pentoses et auto-défense cellulaire. *Ann Anesthesiol Fr* 1967; 8:359-365.
10. Sukoff MH, Ragatz RE. Use of hyperbaric oxygen for acute cerebral edema. *Neurosurgery* 1982; 10:29-38.
11. Higgings AC, Pearlstein RD, Mullen JB, Nashold BS. Effects of hyperbaric oxygen therapy on long-tract neuronal conditions in the acute phase of spinal cord injury. *J Neurosurg* 1981; 55:501-510.