北醫學報 第二十卷 1991年六月
P. 23-29

Hyperventilation in the Management of Severe Head Injury

SHIH-TSENG LEE and DAVID J. FAIRHOLM*

ABSTRACT

Forty severe closed head injury patients were selected, randomized into 2 groups and treated with (A) spontaneous ventilation and (B) controlled hyperventilation. There was no significant difference in the incidence of increased intracranial pressure or patient outcome among the two groups. The incidence of pulmonary complications was higher in the hyperventilated patients than in those receiving spontaneous ventilation. In this study, controlled hyperventilation offered no specific advantage in the management of those patients with severe head injury who can ventilate spontaneously.

Despite vigorous surgical and intensive medical therapy for severe head injury, the mortality and morbidity remains unsatisfactorily high[1-5]. The severity of the primary injury and the variable importance of hematomas as mass lesion, cerebral edema, increased intracranial pressure and cerebral perfusion pressure, have led to multiple modes of therapy. In 1959 Lundberg et al[6], demonstrated that hyperventilation was a useful aid in reducing increased intracranial pressure (ICP) in patients with intracranial hypertension. There have since been many reports contending that long term controlled hyperventilation can improve the outcome of patients after severe head injury[7-11]. Jennett, however, in a large scale retrospective review of head injury management did not support the use of prolonged hyperventilation[2]. The purpose of this study is to evaluate in a controlled, randomized, prospective way the effect of controlled hyperventilation in the management of patients with severe head injury.

MATERIAL AND METHODS

A series of 40 consecutive patients with closed head injury were selected for this study. Patients with multiple injuries, major systemic diseases and acute surgical intracranial hematomas were excluded. Patients requiring intubation and ventilation for respiratory conditions were also excluded. The study group therefore was confined to those patients with cerebral edema in isolated severe head injury (Glas-

Department of Neurosurgery Taipei Municipal Chung Hsiao Hospital, Taipei, Taiwan, R.O.C.
* Department of Neurosurgery Vancouver General Hospital, University of British Columbia, Canada
Received for Publication: November 19, 1990.

gow coma scale 5 to 8) without other complicating factors.

A CT scan was performed within 1 hour of admission in all patients. Mannitol (1.0 gm/kg) was given immediately prior to CT scanning. Every patient received a cranial subarachnoid screw for continue intracranial pressure monitoring.

All patients were placed on an identical management protocol for increased ICP with the exception of their type of ventilation. Intracranial pressure and systemic arterial pressure (SAP) were monitored continuously, and cerebral perfusion pressure (CPP) was calculated continuously for 5 days or longer, if necessary. Mannitol, 1 gm/kg by IV bolus, was given if the ICP rose above 25 mmHg for 5 minutes and was repeated after 30 minutes if the pressure still remained above 25 mmHg. Intravascular volume was maintained by fluid and colloid infusion and attempts were made to maintain the CPP above 60 mmHg in all patients.

The patients were randomized by a paramedical person not associated with the study into two groups using random digits:

(A) Spontaneous ventilation.

(B) Controlled hyperventilation.

In Group A the airway patency was maintained by positioning and aggressive chest physiotherapy. The PaO2 was maintained between 70 and 150 Torr by inhalation of air or oxygen through a face mask. In the hyperventilated group, the arterial carbon dioxide tension (PaCO2) was maintained between 24 and 30 Torr, and arterial oxygen tension (PaO2) between 70 and 150 Torr by mechanical volume cycle ventilator (MA-1). Blood gas determinations were performed regularly at least twice daily or more often when indicated. Intubation was carried out with high volume low pressure silastic cuffed tubes. Cuff pressure was maintained below 20 cm of water. Diazepam (valium) 2.5-10 mg, meperidine (Demerol) 30-40 mg, and Pancuronium (Pavulon) 2-4 mg were used as needed or in combination to synchronize respirations with the ventilator and to prevent fluctuations in ICP due to fighting and bucking.

General medical measures were used to maintain cardiovascular and renal homeostasis. Intravascular volume was determined by clinical means and by monitoring central venous pressure. Regular chest X-rays and sputum cultures along with the blood gas determinations were used to monitor the respiratory status. Antibiotics were only used to treat established infections with positive cultures. A repeated CT scan was done in the face of a prolonged elevation in ICP, or if there was evidence of neurological deterioration. Following the acute study period in the intensive care unit, the patients were transferred to the regular wards for ongoing care and rehabilitation as their condition permitted. The outcome was evaluated according to the standard classification as described by Jennett and Bond. Six months after injury was chosen as an end point because it has been concluded that most functional recovery will have occurred by that time[12]. Chi-Square, or one way analysis of variance and Fisher's exact test were performed in the data interpretation.

RESULTS

Baseline admission data were similar among the 2 groups of patients (Table I). There was no significant difference in the age, sex ratio and GCS score between the two group of patients. ICP was classified as normal (0-10

Table I. Basic Clinical Data of 60 Patients with Severe Head Injury

	A	B	P * value
Age (mean ± SD) (Years)	33.5 ± 11.45	33.7 ± 12.25	> 0.1
Sex Ratio (M:F)	15:5	16:4	> 0.1
Coma scale			
5	5	5	> 0.1
6	3	2	
7	8	6	
8	4	7	
Total	20	20	

A: Spontaneous ventilation.

B: Controlled hyperventilation.

* One way analysis of variance.

Table II. Relationship between Ventilation and Highest ICP

	ICP				
	Normal 0-10 mmHg	Mildly increased 11-25 mmHg	Moderately increased 260-40 mmHg	severely increased ≧41 mmHg	Total
A	3	5	4	8	20
B	3	6	4	7	20
Total	6	11	8	15	40

P > 0.1

mmHg), mildly increased (11-25 mmHg), moderately increased (26-40 mmHg), and severely increased (above 40 mmHg). Increased intracranial pressure (11 mmHg or higher) was present in 83% of all patients, equally distributed in both groups.

The relationship between ventilation and highest increased intracranial pressure is shown in Table II. There is no significant difference in the incidence of increased ICP among two groups in spite of their different ventilation treatment.

Forty percent of Group A, and 35% of Group B patients died or became vegetative. Conversely, 35% of Group A, and 45% of Group B, showed good recovery or had only mild neurological deficits. Although the early mortality rate seems higher in the spontaneous ventilation group, differences in the results are not statistically significant among the two group of patients according to their outcomes as shown in Table III.

Respiratory tract complication (pneumonia and subglottic stenosis) were more frequent in those cases who had undergone controlled ventilation (Table IV).

Table III. Six Month Outcome Related to Hyperventilation

Group	Grade				
	1 or 2	3	4	5	Total
A	7	5	1	7(4)	20
B	9	4	3	4(2)	20
Total	16	9	4	11	40

P>0.1

( ): Mortality within first week

Grade 1: Good recovery.

Grade 2: Moderately disabled

Grade 3: Severely disabled

Grade 4: Vegetative

Grade 5: Dead

Table IV. Respiratory Complications

Group	Respiratory tract Complication		
	* Pneumonia	Subglottic stenosis	Total
A	2	0	2
B	9	2	11

*: P< 0.05

DISCUSSION

Controlled hyperventilation is frequently used as an adjuvant therapy for severely head injured patients. Experimental studies showed that hyperventilation reduces PaCO2 and produces alkalosis in the CSF. This has several effects on the vessels, including a direct effect upon vascular smooth muscles causing constriction of arterioles and subsequent reduction of cerebral blood flow and cerebral volume within the cerebral vascular bed. This effect decreases the ICP possible[13-17]. Because of the above mechanism, Heffner and Sahn suggested controlled prolonged hyperventilation as a therapeutic modality in several neurological conditions associated with increasing ICP[18]. McGillicuddy also recommended use of controlled hyperventilation as an initial emergent teatment for comatose patients[19]. If the cerebral resistance vessels are dilated by the autoregulatory response to intracranial hypertension, the vasoconstriction produced by the hypocapnia may be balanced by the relief of venous compression resulting from decreased ICP. Resistance across the cerebrovascular bed may remain constant or may decrease. In this circum-

stance, hyperventilation may produce increase in cerebral blood flow, even though the resistant vessels constrict normally to the decrease in the arterial carbon dioxide tension[14,20-22]. As a result, within a few hours of starting hyperventilation, intracranial pressure will gradually rise, and may go even higher[10,22-25]. In addition, there may be focal complete vasomotor paralysis where the vessels are unresponsive to hypocapnia and shunting of blood to them may result. Such phenomenon could create ischemia in normal brain tissue[20,23,26,27].

In our study, hyperventilation did not improved the mortality and morbidity in this group of severe head injury patients.The incidence of increased ICP was also not reduced by controlled hyperventilation.

In spite of carefully controlled endotracheal cuff pressures, sub-epiglottic granulation stenosis occurred in 2 cases due to prolonged intubation for hyperventilation. Forrest[28] has pointed out that hyperventilation in associated with a reduction of pulmonary compliance and the lung stability index. There is also a decrease in the total volume of the lung to a degree which is dependent on the duration of hyperventilation. Hypocapnia could inhibit the surfactant producing function of type II alveolar pneumocytes either directly, or secondary to a rise in pH. This produces a continuing effect of impaired surface film regeneration. Following initial film disintegration by overinflation, repeated mechanical overinflation soon depletes hypophase within the surface film. The above mechanisms, by increasing surface tension forces, promote premature alveolar collapse. This may be the reason for the higher incidence of pneumonia in hyperventilated patients, despite intensive chest care.

Before starting this study we had expected better results in the hyperventilated patients. The results of the study did not support our presupposition. The incidence of increased ICP was not reduced, the mortality was not reduced or the degree of recovery improved, and a higher incidence of pulmonary complication occurred. The data from this report do not favor the use of controlled hyperventilation for treatment of severe head injury patients who can breath spontaneously without respiratory difficulty.

ACKNOWLEDGMENT

The author would like to thank Miss Yun Shu for her secretarial assistance.

REFERENCES

1. Becker DP, Miller JD, Ward JD: The outcome from severe head injury with early diagnosis and intensive management.J Neurosurg 47: 491-502, 1977.
2. Jennett B, Teasdale G, Fry J, et al: Treatment of severe head injury. J Neurol Neurosurg Psych 43:289-95, 1980.
3. Levati A, Fairna ML, Vecchi G, et al. Prognosis of severe head injuries. J Neurosurg 57:779-83, 1982.
4. Aberico AM, Ward JD, Chol SC, et al: Outcome after severe head injury--Relationship to mass lesions, diffuse inury, and ICP course in pediatric and adult patient. J Neurosurg 67:648-56, 1987.
5. Lee ST, Lui TN, Chang CN, et al: Features of head injury in a developing country--Taiwan 1977-1987. J Trauma 30:194-9, 1990.
6. Lundberg N, Kjallquist A, Bien C: Reduction of increased intracranial pressure by hyper-

ventilation. A therpaeutic aid in neurological surgery. Acta Psychiat Scand 34(suppl):139, 1959.

7. Rossanda M: Prolonged hyperventilation in treatment of unconscious patients with severe brain injuries. Scand J Clin Lab Invest 102 (suppl):XIII, E, 1968.
8. Gordon E: Controlled respiration in the management of patients with traumatic brain injuries. Acta Anaesth Scand 15:193-208, 1971.
9. Rossanda M, Selenati A, Villa L: Role of automatic ventilation in treatment of severe head injuries. J Neurol Sci 17:265-70, 1973.
10. Rossanda M, Collice M, Porta M, et al: Intracranial hypertension in head injury. Clinical significance and relation to respiration. In: Lundberg N, Ponten U, Brock M eds. Intracranial pressure II. New York: Springer-Verlag, 475-9, 1975.
11. Gordon E: Managment of acute head injuries by controlled ventilations. In: Laurado R ed. Recent progress in anesthesiology and resuscitation. Amsterdam: Excerpta Medica. 784-9, 1975.
12. Jennett B, Teasdale G: Management of Head Injuries. Philadelphia: Davis Co., 308-11, 1981.
13. Lassen NA: Clinical implications of cerebral blood flow studies. In: Brode M, Fieschi C, Ingvor DH, Lassen NA, Schuman K, eds. Cerebral blood flow: clinical and experimental results. New York: Springer-Verlag 278-85, 1969.
14. Raichle ME, Plum F: Hyperventilation in cerebral blood flow. Storke 3:566-75, 1972.
15. Czernick Z, Jurkiewica J, Kunicki A: The effect of hypocapnia on normal and increased intracranial pressure in cats and rabbits. In: Lundberg N, Ponten U, Brock M, eds. Intracranial pressure II. New York: Springer-Verlag, Berlin, Heidelberg, pp. 471, 1975.
16. Kindt GW, Gosch HH: Arterial PCO2 effect at various levels of intracranial pressure. In: Brock M, Dietz H, eds.Intracranial pressure. New York: Springer-Verlag, Berlin, Heidelberg; pp. 210-3, 1975.
17. Leech PJ, Miller JD: The effect of mannitol, steroids and hypocapnia on the intracranial volume, pressure response. An experimental and clinical study. In: Lunberg N, Ponten U, Brock M, eds. Intracranial pressure II. New York: Springer-Verlag, pp. 361-4, 1975.
18. Heffner JE, Sahn SA: Controlled hyperventilation in patients with intracranial hypertension: application and management. Arch Intern Med 143:765-9, 1983.
19. McGillicuddy JE: Cerebral protection: pathophysiology and treatment of increased intracranial pressure. Chest 87:85-93, 1985.
20. Crockart HA, Coppel DL, Morrow WFK: Evaluation of hyperventilation in treatment of head injuries. Br Med J 4:634-40, 1973.
21. Langfitt TW: Increased intracranial pressure and the cerebral circulation. In: Youmans JR, ed. Neurological surgery; vol 2. Philadelphia: W.B. Saunders Co., pp. 901-3 1982.
22. Levy DE: Controlled hyperventilation. Arch Intern Med. 143:2342, 1983.
23. Paul RL, Polanco O, Turney SZ, et al: Intracranial pressure responses to alterations in arterial carbon dioxide pressure in patients with head injuries.J Neurosurg 36:714-20, 1972.

24. James HE, Langfitt TW, Kumar VS: Treatment of intracranial hypertension: Analysis of 105 consecutive cases of continued recordings of intracranial pressure. Acta Neurochir 36:189-200, 1977.
25. Hahn JF: Cerebral edema and neurointensive care. Pediatr Clin North Am 27:587-92, 1980.
26. Lassen NA: The luxury-perfusion syndrome and its possible relation to acute metabolic acidosis localized within the brain. Lancet 11:1113-6, 1966.
27. Reid RA: Hyperventilation and cerebral protection. Chest 88:798-99, 1985.
28. Forrest JB: The effect of hyperventilation on pulomnary surface activity. Br J Anaesth 44:313-20, 1972.

以高度換氣法治療嚴重頭部受傷病患

李石增　*費宏德

爲了瞭解以高度換氣法治嚴重頭部受傷病患的療效及對長期預後的影響，我們將 40 位無呼吸困難的嚴重頭部受傷病患(昏迷指數小於 8)以隨機方式分爲 2 組。A 組爲對照組不採用高度換氣法治療，B 組則使用高度換氣法治療 5 天。所有病患皆採用顱內壓測量術並採取相同之治療計劃。結果顯示 2 組病患顱內壓上升之頻率，死亡率及預後皆不達統計學上有意義差異($P>0.1$)。肺部感染及呼吸道合併症在高度換氣組有明顯的增加($P<0.05$)。

本研究顯示對於嚴重頭部受傷病患若無呼吸困難使用高度換氣治療法應採用較審愼之態度。

臺北市立忠孝醫院神經外科*加拿大溫哥華大學醫院神經外科
民國七十九年十一月十九日受理