

Methods and Devices

The Use of Continuous Flow of Oxygen and PEEP During Apnea in the Diagnosis of Brain Death

A. Perel, M. Berger and S. Cotev

Respiratory Intensive Care Unit, Department of Anesthesiology, Hadassah-Hebrew University Medical School, Ein Karem, Jerusalem, Israel

Accepted: 23 March 1982

Abstract. The establishment of apnea for the diagnosis of brain death by disconnecting the patient from the ventilator may lead to dangerous hypoxemia at the end of the test period. We established apnea for 4 min in 8 patients with suspected brain death, both by disconnecting them from the ventilator after 10 min ventilation with $FIO_2 = 1.0$ (method "A"), and by leaving them attached to an IMV ventilator circuit with a continuous flow of 100% O_2 and PEEP of 4–8 cm H_2O without mechanical ventilation (method "B"). PaO_2 decreased during the apneic period by 143 ± 65 (SD) mmHg using method "A", and by 48 ± 28 mmHg using method "B" ($p < 0.002$). The changes in $PaCO_2$ and pH were similar following both apneic methods.

We conclude that it is safer to test for apnea by leaving the patients on a continuous flow of 100% oxygen and low PEEP than to disconnect them from the ventilator.

Key words: Brain death – Ventilation – PEEP – Apneic oxygenation

The absence of spontaneous ventilation is one of the main criteria for the diagnosis of brain death [1]. Patients suspected of brain death are generally maintained on mechanical ventilation and the ventilator has to be disconnected in order to establish apnea. The duration of the apnea needed to increase $PaCO_2$ to levels that must stimulate ventilation ranges from 3 to 20 min according to various authors [1, 5, 6]. During this time, a life-threatening hypoxemia may develop with a potential to further damage the brain and other organs. To prevent this hypoxemia it is common to ventilate the patients with 100% O_2 prior to the test, add CO_2 to the inspired air to hasten the rate of rise of

$PaCO_2$, or supply additional oxygen during the test [1, 5, 6, 8].

As many brain dead patients have pulmonary dysfunction due to head injury [3, 7], additional lung contusion, aspiration pneumonia or previous chronic lung disease, the disconnection from the ventilator can be especially hazardous. For this reason we have compared, in brain-death patients, the effect on arterial oxygenation of disconnecting the ventilator after previous oxygenation with 100% O_2 , to using a continuous flow of oxygen with low PEEP through an IMV system during the apneic period.

Materials and Methods

Eight patients with suspected brain death were tested for the presence of spontaneous ventilation. There were six males and two females, with ages ranging from 8 to 45 years. All patients suffered severe cranial trauma at least 24 h prior to the test. All were deeply comatose and had definite clinical evidence of irreversible structural brain damage. None of the patients were under the influence of depressant drugs, and were kept normothermic with normal blood electrolytes and acid-base balance.

All patients were ventilated using Bennett MA-1 ventilators, adapted for intermittent mandatory ventilation (IMV) [4]. The absence of spontaneous ventilation was verified in each patient by two methods: Method "A" consisted of normoventilation with an $FIO_2 = 1.0$ for 15 min at the end of which an arterial blood gas analysis was performed. The patients were then disconnected from the ventilator for 3 min and then another arterial blood sample was taken for blood gas analysis, and the patients were put back on the ventilator.

Table 1. Mean arterial blood gas tensions and pH at baseline, and after 3 min of apnea, using method "A" (disconnection from the ventilator) and method "B" (continuous flow of 100% O₂ and PEEP)

	Method "A" (disconnection)		Method "B" (O ₂ and PEEP)	
	Baseline	Apnea	Baseline	Apnea
PaO ₂ (mmHg)	276 ± 119	132 ± 97	266 ± 124	227 ± 127
PaCO ₂ (mmHg)	32 ± 8	56 ± 12	33 ± 8	54 ± 17
pH	7.42 ± 0.06	7.28 ± 0.05	7.40 ± 0.05	7.24 ± 0.06

Method "B" consisted of the same procedure except that apnea was established with the patients remaining attached to the IMV circuit with PEEP of 4–8 cm H₂O, high fresh gas flow of 100% O₂, but without mechanical ventilations.

All the patients were tested using both methods with a 15 min interval between tests, and the test sequence being chosen arbitrarily. Changes in PaO₂, PaCO₂ and pH at the end of the apnea period following each method were compared by the paired Student's t-test.

Results

Spontaneous respiration did not occur in any patient during either apnea test. The mean baseline and post-apnea arterial blood gas tensions with both apneic methods are shown in Table 1. Using method "A" (disconnection) PaO₂ fell by 143 ± 65 (SD) mmHg, while with method "B" (100% O₂ flow and PEEP) PaO₂ decreased by only 48 ± 26 mmHg after 3 min of apnea. The difference between these changes was statistically significant ($p < 0.002$). Four out of the 8 patients exhibited PaO₂ values below 90 mmHg after 3 min of apnea using method "A", while all 8 patients had PaO₂ values above 90 mmHg using method "B" (Table 2). The changes in PaCO₂ and pH (Table 1) were similar using either method.

Table 2. PaO₂ values of the 8 patients at baseline and after 3 min of apnea using methods "A" and "B"

Patient no.	PaO ₂ (mmHg) during method "A" (disconnection)		PaO ₂ (mmHg) during method "B" (O ₂ and PEEP)	
	Baseline	Apnea	Baseline	Apnea
1	201	101	190	224
2	256	54	183	92
3	419	181	420	364
4	194	139	206	209
5	147	75	173	114
6	223	81	213	171
7	270	75	242	177
8	493	350	500	467

Discussion

The absence of spontaneous respiration is an essential criterion for the diagnosis of brain death [1, 8]. As all patients subjected to the diagnosis of brain death are invariably being supported by mechanical ventilation (most are hyperventilated to reduce intracranial hypertension), their ventilator support has to be stopped to establish the presence of apnea. In many of the recommendations that appeared in the literature in this regard, the actual method of stopping mechanical ventilation has not been specified. The duration of apnea needed to establish brain death is also unclear, and the test period was recommended to be from 3 to 20 minutes according to various authors [1, 5, 6, 8]. There is, however, a general consensus about the importance of post-apnea blood gas monitoring, but the PaCO₂ level that must be reached by the end of test period is also under debate, with values ranging between 40 to 60 mmHg. The danger of confusing brainstem death with posthyperventilation apnea has been recognized [2], and therefore all patients must be normocapnic at the beginning of the test, or the fact that they were hyperventilated be taken into account, and the test period lengthened. Since PaO₂ can decrease to hazardous values during apnea, the common practice is either to ventilate the patients with 100% O₂ prior to the test, or supply additional oxygen by a catheter via the edotracheal tube during the test [5, 6, 8]. These methods are satisfactory for most cases but both studies that reported the results of these methods included one patient each who developed either extreme hypoxemia [6], or pulmonary edema and cardiac arrest [5]. These unacceptable incidents are not surprising as many patients after head trauma develop hypoxemia due to ventilation/perfusion imbalance, due to pulmonary vascular regulatory dysfunction leading to increased venous admixture [3, 7]. Other causes of post-traumatic hypoxemia include lung contusion, adult respiratory distress syndrome (ARDS), pneumonia and chronic pre-existing lung disease. The lowest baseline PaO₂ value in our patients after ventilation with 100% O₂ for 15 min was 147 mmHg, while in other series there were patients with PaO₂ values of 60–70 mmHg on FIO₂ = 1.0 [3,

7]. Our results clearly show that the reduction in PaO_2 during the apnea period can be significantly minimized by leaving the patients connected to the IMV ventilator. The continuous flow of 100% O_2 , and the positive end-expiratory pressure keep the patients well oxygenated during this period. It is true that prior ventilation with 100% O_2 during 15 min did ensure adequate arterial oxygenation during the entire period of apnea; however, using this method we encountered one PaO_2 value of 54 mmHg, certainly below the acceptable range.

We therefore conclude that the population of suspected brain-death patients has a high incidence of arterial hypoxemia, and since the injured, swollen brain should not be exposed to further hypoxia, the combination of high flow of oxygen and PEEP is the method of choice for the testing of apnea in the diagnosis of brain death.

References

1. Black PM (1978) Brain death. *N Eng J Med* 299:338, 393
2. Brandfonbrener M, Kroll G, Borden C (1969) Posthyperventilation apnea and the criteria of brain damage and death. *Am Heart J* 78:573
3. Frost EAM, Arancibia CU, Shulman K (1979) Pulmonary shunt – as a prognostic indicator in head injury. *J Neurosurg* 50:768
4. Graybar GB, Smith RA (1980) Apparatus and techniques for intermittent mandatory ventilation. In: Kirby RR, Graybar GB (eds) Intermittent mandatory ventilation. *International Anesthesiology Clinics* 18:53
5. Pitts LH, Kaktis J, Caronna J, Jennet S, Hoff JT (1978) Brain death, apneic diffusion oxygenation and organ transplantation. *J Trauma* 18:180
6. Schafer JA, Caronna JJ (1978) Duration of apnea needed to confirm brain death. *Neurology* 28:661
7. Schumaker PT, Rhodes GR, Newell JC, Shah DM, Scovill WA, Powers SR (1979) Ventilation – perfusion imbalance after head trauma. *Am Rev Resp Dis* 119:33
8. Conference of Medical Royal Colleges in the United Kingdom (1976) Diagnosis of brain death. *Br Med J* 2:1187

Azriel Perel, M. D.
Department of Anesthesiology
Kiryat Hadassah University Hospital
Jerusalem
P. O. Box 12000
Israel, 91120