Apnea testing for the determination of brain death: a modified protocol

Technical note

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The absence of spontaneous respirations at a PaCO₂ of 60 mm Hg or above has traditionally been accepted as the respiratory criteria for the determination of brain death. The testing of patients for the presence or absence of apnea has been complicated because the rate of PaCO₂ elevation may vary substantially from patient to patient, and a nonlinear relationship exists between the rate of PaCO₂ increase and the duration of apnea.

In an attempt to refine the apnea test and to further elucidate the physiology of hypercapnia in humans, 11 patients who met all but the respiratory criteria for brain death were evaluated using a modification of a previously utilized apnea testing protocol. All patients were brought to a PaCO₂ of 40 mm Hg or above prior to the apnea test. Baseline PaCO₂ ranged from 40 to 45 mm Hg in six patients (Group I) and from 46 to 51 mm Hg in five patients (Group II). The mean rate of PaCO₂ increase was 5.1 ± 1.4 mm Hg/min in Group I and 6.7 ± 3.1 mm Hg/min in Group II. No problems with cardiovascular instability or hypoxia were encountered during testing in this series. This refinement of the apnea test allows for a streamlined and safe approach to brain death detection.

Key Words: brain death • apnea test • hypercapnia • acidosis • oxygenation

The determination of brain death is of concern to all physicians caring for critically ill patients. It is, of course, crucial to demonstrate that cerebral function has ceased before a patient can be considered brain dead. This is not demonstrated unless apnea is documented.1,3-5

The methodology of apnea testing differs from center to center. Controversies persist regarding the safest and most appropriate techniques. An analysis of 11 consecutive patients undergoing brain death determination studies was therefore undertaken in order to evaluate the effect of an increased baseline PaCO₂ on the duration of apnea required for the completion of the test.

Clinical Material and Methods

Patient Population

From July, 1988, to April, 1989, 11 consecutive patients who met the neurological criteria for brain death1,2 underwent a modified apnea test as the final step of their brain death determination. There were 10 males and one female, with a mean age of 39 years (range 17 to 69 years). Five were black, and six were white. The etiology of the neurological injury was trauma in three patients, hypertensive intracerebral hemorrhage in three, subarachnoid hemorrhage in two, meningitis in two, and metastatic tumor in one.

Apnea Test Procedure

With the exception of minor modifications, the technique of apnea testing utilized here is as previously reported.1 After a 15-minute preoxygenation period with 100% O₂ delivered via mechanical ventilation, the ventilation was adjusted to allow the patient's PaCO₂ to rise to 40 mm Hg or above. Then, 100% O₂ was delivered via a cannula placed through the endotracheal tube to the estimated level of the carina. After a baseline arterial blood gas determination, further samples were
drawn at 30-second intervals for the first 2 minutes, then at 1-minute intervals for the remainder of the test (12 minutes). Respiratory effort, if present during the test, was recorded (although this was not observed in any of the patients presented here). Additional data collected included core body temperature, serial blood pressure, and heart rate.

The patients were divided into two groups based on their baseline PaCO₂. Group I included six patients with a baseline PaCO₂ between 40 and 45 mm Hg; Group II included five patients with a baseline PaCO₂ above 45 mm Hg.

**Results**

The data are reported using standard deviation of the mean as the measure of variability. The mean core body temperature immediately prior to testing was 97.0° ± 1.0°F. The mean arterial blood pressure was 86 ± 12 mm Hg at the initiation of testing and 73 ± 13 mm Hg at its conclusion. The mean heart rate was 105 ± 9 beats/min at the onset and 101 ± 7 beats/min at the conclusion of the test. The mean baseline PaCO₂ was 41 ± 0.7 mm Hg in Group I and 49 ± 0.8 mm Hg in Group II.

The average rate of PaCO₂ elevation in the two groups is shown in Table I and illustrated in Fig. 1. In Group I, this rate was 5.1 ± 1.4 mm Hg/min, whereas in Group II it was 6.7 ± 3.1 mm Hg/min. The mean time required to reach a PaCO₂ of 60 mm Hg was 4.67 ± 2.66 minutes in Group I patients and 3.9 ± 3.76 minutes in Group II patients. The pH response of each of the two groups is illustrated in Fig. 2, the pO₂ response in Fig. 3. No patient became hypoxic during apnea testing (12 minutes). Mean pO₂ fell from 260 to
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| TABLE I |
| Rate of elevation of the PaCO₂ in patients during apnea testing |
|-----------------|-----------------|-----------------|
| Group* | No. of Cases | Rate of PaCO₂ Elevation (mm Hg/min) | Time to Reach PaCO₂ ≤ 60 mm Hg |
| I | 6 | 5.1 ± 1.4 | 4.67 ± 2.66 min |
| II | 5 | 6.7 ± 3.1 | 3.9 ± 3.76 min |

* Group I = patients with a baseline PaCO₂ between 40 and 45 mm Hg; Group II = patients with a baseline PaCO₂ above 45 mm Hg.

110 mm Hg in Group I and from 330 to 250 mm Hg in Group II.

No patient developed cardiovascular instability or demonstrated any respiratory effort during the test. Therefore, each patient was declared brain dead following completion of the test.

Discussion

A standardized, efficient, and safe method of apnea testing has been a goal for those physicians who make brain death determinations. The method chosen should: 1) assure attainment of an adequate respiratory drive within a "reasonable" time; and 2) achieve this without the patient experiencing hypoxia, acidosis, or cardiovascular instability. Thus, a modification of a previously reported protocol was employed in this study in order to refine the technique of apnea testing. Changes included preoxygenation with 100% inspired fraction of O₂ for a period of 15 minutes and manipulation of the ventilator settings to achieve a PaCO₂ of at least 40 mm Hg prior to the initiation of testing.

The Rate of PaCO₂ Elevation During Apnea

Arterial PaCO₂ was elevated an average of 3.7 ± 2.3 mm Hg/min in previously reported brain-dead patients whose baseline PaCO₂ was less than 40 mm Hg. It was also shown that patients with a baseline PaCO₂ of less than 30 mm Hg reached a PaCO₂ of 60 mm Hg or above after an average of 11 minutes; patients with an initial PaCO₂ of between 30 and 40 mm Hg reached a level of 60 mm Hg in 5.5 minutes. In the present study, a significant reduction in the time required for completion of apnea testing was observed. Figure 4 illustrates this by comparing data from our previous study utilizing lower PaCO₂ baseline levels with the data presented here. The time period required to reach a PaCO₂ of 60 mm Hg or above decreases as the baseline PaCO₂ is raised. In addition, the initial rate of PaCO₂ elevation is increased (increased slope of the curve, Fig. 4). It should be emphasized that observations in both the present and previous studies have shown that the rate of PaCO₂ elevation varies considerably from patient to patient, regardless of baseline PaCO₂.

Responses of pH and pO₂:

The changes in pH levels observed in the patients presented here were not pronounced. In addition, arterial oxygenation at the end of the test was greater in Group II patients than in Group I patients. These findings imply that a higher baseline pO₂ and the modifications of the apnea test applied in this study do not increase the risk to the patient or his/her organs.

References


Manuscript received July 5, 1991.
Accepted in final form November 15, 1991.
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