Letter to the Editor

To the Editor

Apnea test in the diagnosis of brain death

Guidelines for the diagnosis of brain death have been recently published by the Canadian Neurocritical Care Group in the *Journal*.[1] Apnea testing is an important component in the determination of brain death. In the recent guidelines it is suggested to insufflate 100% oxygen via a cannula inserted in the endotracheal tube to prevent hypoxemia during the apneic period. This is in agreement with the classic method of apnea testing. However, we would like to warn about serious complications of such a technique of apneic oxygenation. A few years ago we experienced a case of subcutaneous emphysema, tension pneumothorax followed by cardiac arrest during the performance of an apnea test with the classic method of inserting an oxygen catheter into the endotracheal tube. Subsequent investigation revealed that the oxygen catheter was wedged in the endotracheal tube. The 6 l/min flow of oxygen rapidly produce lung hyperinflation and major barotrauma. More recently, two very similar cases have been very well described by Bar-Joseph et al.[2] Brandsetter et al[3] have also reported a similar complication. The consequences of such complications can be catastrophic, especially when the patient is a candidate for organ donation. Moreover, they could be easily prevented by simple modification of the technique of apnea testing.

The first important step is to ensure a careful preoxygenation with 100% oxygen while maintaining normoventilation ($PaCO_2$ 40±5mmHg) during at least 10 minutes. Then the endotracheal tube can be connected either to a T-piece or to a high flow continuous positive airway pressure (CPAP) system.

If the patient required a FiO_2 less than 0.50 prior to apnea testing, a T-piece system delivering 100% oxygen will suffice to maintain an adequate arterial oxygenation throughout the apneic period. Alternatively, if prior to apnea testing the patient has hypoxic respiratory failure requiring a high FiO_2, the endotracheal tube can be connected to a high flow CPAP system of 5 to 10 cmH_2O delivering 100% oxygen. Practically, we use a PEEP valve (Vital Signs®, Totowa, N.J.) that we adapt to the distal extremity of the T-piece. A similar but more complex technique, the "bulk diffusion" technique, has also been described by Al Jumah et al.[4] With these techniques the risk of barotrauma and tension pneumothorax is minimal and probably entirely eliminated. Therefore, we suggest that the Guidelines for the Diagnosis of Brain Death should be amended to revise the technique of apnea testing.

Martin Lessard, Rachel Mallais, André Turmel
Quebec, Canada.

References

1. Canadian Neurocritical Care Group. Guidelines for the Diagnosis of Brain Death. Can J Neurol Sci 1999; 26: 64-66.
2. Bar-Joseph G, Bar-Lavie Y, Zonis Z. Tension pneumothorax during apnea testing for the determination of brain death. Anesthesiology 1998; 89: 1250-1251.
3. Brandsetter RD, Choi MY, Mallepalli VN, Klass S. An usual cause of pulmonary barotrauma during apneic oxygenation testing. Heart Lung 1994; 23: 88-89.
4. Al Jumah M, McLean DR, Al Rajeh S, Crow N. Bulk diffusion apnea test in the diagnosis of brain death. Crit Care Med 1992; 20: 1564-1567.

Response to Lessard et al.

Lessard and colleagues discuss possible risks of apnea testing using the standard protocol.[1] They draw attention to the extremely rare complication of pneumothorax related to the standard recommended therapy of oxygen delivery via a tracheal cannula inserted in the endotracheal tube. In over 20 years of performing apnea tests for brain death in adults and children, we have never encountered such a complication. The risk of a tracheal cannula becoming wedged in a small bore endotracheal tube, e.g., in a small child, must be greater than such a complication in an older child or adult. Such risks are minimized by ensuring that such catheters are of a caliber that will not become wedged in the endotracheal tube and that they are thin and flexible and do not protrude beyond the end of the endotracheal tube.

Their suggestion of using a technique involving continuous positive airway pressure (CPAP) is potentially problematic. An earlier report documented respiratory-like movements when apnea testing utilized CPAP in a patient who was apparently brain dead.[2] Rhythmic, thoracic, respiratory-like movements have been reported in brain death with earlier methods of testing and may arise from remaining cervical spinal cord function.[3]

Some centres utilize increased carbon dioxide concentration in the administered air during apnea testing. This also can also create problems. Too high a partial pressure of arterial carbon dioxide ($PaCO_2$), e.g. greater than 90 mm Hg, can depress any respiratory drive by inducing carbon dioxide narcosis.[4] Also, a very abrupt rise in $PaCO_2$ can cause a rapid rise in intracranial pressure in the patient who is not brain dead.[5]

We stress that the apnea test should be performed only after other criteria for brain death are met. It should not be used when exclusions apply or where it is deemed to be risky or the results uninterpretable.

Complications from the apnea test, such as that cited by Lessard et al., are rare. We suggest that the standard procedure, with precautions noted above, is still the best approach for the majority of cases. There may be special circumstances in which the methods suggested by Lessard et al. may be appropriate, but these too have their pitfalls. We look forward to hearing of other experiences with the apnea test in adults and children.

Shasi Seshia, Jeanne Teitelbaum and Bryan Young
for the Canadian Neurocritical Care Group
(The authors thank Dr. Frank Rutledge for helpful advice.)

References

1. Canadian Neurocritical Care Group. Guidelines for the determination of brain death. Can J Neurol Sci 1999;26:64-66.
2. Turnbull J, Rutledge F. Spontaneous movements with clinical brain death. Neurology 1985;35: 1260.
3. Ropper AH, Kennedy SK, Russell L. Apnea tesing in the diagnosis

of brain death. J Neurosurg 1981 ;55: 942-946.
4. Rudolf J, Haupt WF, Neveling M, Grond M. Potential pitfalls in apnea testing. Acta Neurochir (Wien) 1998; 140: 659-663.
5. Wijdicks EF. In search of a safe apnea test in brain death: is the procedure really more dangerous than we think. Arch Neurol 1995;52:338-339.

The Alphabetization of Journal Articles

I am a great believer in textual efficiency in scientific writing, but there are limits. Take for example, this sentence from a recent CJNS editorial: "The American Heart Association produced a CPG stating that ACS was a suitable indication for CE, provided that the perioperative M/M rate was <3%".[1] I would like to suggest to TF that it is a PIB to have to KLE in the article to FO the meaning of the SBR.

RSM

TF = Tom Feasby
PIB = pain in the butt
KLE = keep looking elsewhere
FO = find out
SBR = sentence being read
RSM = R.S. McLachlan

Reference

1. Feasby TF. Endartectomy for Asymptomatic Carotid Stenosis in the Real World. Can J Neurol Sci 2000;27:95-96.

Response to The Alphabetization of Journal Articles

I regret that RSM felt challenged by my concise style, although I noticed that he used the cryptic abbreviation CJNS himself. While he apparently found it to be a PIB to KLE, E isn't very far away in a brief editorial. I agree with Pascal, who said of a letter "I have only made this longer because I have not had the time to make it shorter". Here's to short letters, and editorials, and let's save the rain forest.

Thomas E. Feasby
Calgary, Alberta

Issues Relating to Functional Disability in Essential Tremor

Essential tremor (ET) is probably not a homogeneous condition, and subtypes of ET (i.e., those forms of ET that may differ with regard to their etiology, rate of progression, prognosis or response to treatment) probably exist.[1] Identification of such subtypes has importance in both clinical settings and research studies.

Cenk Akbostanci et al[2] reported that ET patients with synchronous activity of antagonist muscles had greater disability than did patients with alternating activity, arguing for a separation of these two into distinct clinical subgroups of ET. While these findings are important, there are a number of methodological issues that their study raises.

First, the rating scale used by the authors[3] assessed the patients' subjective complaints of tremor (i.e., self-reported disability) rather than disability as assessed using a performance-based test of function.[4] Although there is a correlation between the answers on this subjective scale and more objective measures of tremor severity (e.g., the correlation, r, between the disability score and spirography = 0.659),[3] the correlation is not perfect, so that the subjective measure can not serve as a substitute for objective measures. Therefore, it would be important to establish that the two groups actually differed in terms of an objective performance-based measure of function.

The second comment relates to the issues of depression and anxiety. Although these generally are not assessed in studies of functional impairment in tremor, their impact on reported disability is significant. In the Columbia University Assessment of Disability in ET,[4] we studied functional disability in 178 subjects using a tremor disability questionnaire, a performance-based test of function, a clinician-rated tremor score, quantitative computerized tremor analysis, and psychological assessments. While we found that the score on the tremor disability questionnaire was associated with the clinician's rating of a videotaped tremor examination ($p < 0.001$) and the performance-based test ($p < 0.001$),[4,5] it was also independently associated with depression assessed using the depression module of the Structured Clinical Interview for DSM IV ($p = 0.02$) and the Hamilton Anxiety Rating Scale score ($p = 0.017$).[5] These data suggest that depression and anxiety, independent of the severity of the tremor itself, are associated with greater reported functional disability in ET, so that these factors must be considered when assessing disability in ET.

Third, the authors reported that subjects who had synchronous activity of antagonist muscles were marginally older and had disease of longer duration than those with alternating activity. Although these differences did not reach statistical significance, this may have related to the relatively modest sample size rather than the absence of an effect. Multivariate regression analyses would help to verify the reported association between the physiological type of tremor (synchronous vs. alternating activity of antagonist muscles) and reported disability, independent of the effects of age and tremor duration.

Elan D. Louis
New York, NY, USA.

References

1. Louis ED, Ford B, Barnes LF. Clinical subtypes of essential tremor. Arch Neurol 2000;57:1194 -1198.
2. Cenk Akbostanci M, Ulkatan S, Yigit A, Aydin N, Mutluer N. Differences in disability between electrophysiological subgroups of essential tremor. Can J Neurol Sci 2000;27:60-64.
3. Bain PG, Findley LJ, Atchison P et al. Assessing tremor severity. J Neuro Neurosurg Psychiatry 1993;56:868-873.
4. Louis ED, Barnes LF, Wendt KJ, et al. Validity and test-retest reliability of a disability questionnaire for essential tremor. Mov Disord 2000;15:516-523.
5. Louis ED, Barnes LF, Albert SM, et al. Predictors of functional disability in essential tremor. Mov Disord 2000 (Submitted).