It must be understood that the clinical evaluation for loss of neurological function in brain death examinations only detects the absence of brainstem function. This comprises the loss of consciousness and the absence of brainstem reflexes, including the capacity to breathe. The clinical examination cannot distinguish the complete loss of whole brain function from the isolated loss of brainstem function. The distinction between whole brain and brainstem death can be made based on etiology of brain injury and neuroimaging. It can only be confirmed by the use of ancillary tests that show an absence of electroencephalographic activity, or preferably, the absence of intracerebral blood flow. For this reason, ancillary testing is used frequently in the United States but only rarely in the United Kingdom.

Dr. Doyle and others[1] suggest that laboratory evidence of retained hypothalamic-pituitary activity is inconsistent with the whole-brain formulation. Bernat[2] rejects laboratory evidence of cellular function, arguing that isolated cellular activity may persist in the absence of clinical signs of brain activity. While intracerebral blood flow arrests in whole brain death, small degrees of intracranial flow can persist via vessels arising extradurally. Wijdicks provides a pathophysiologic explanation for preservation of hypophyseal-pituitary axis activity in brain death, noting that perfusion to these structures arises from extracranial vessels.[3] Continued cellular activity may be a manifestation of retained blood flow to these nests of cells despite intracerebral circulatory arrest. As noted by Dr. Doyle, irrespective of these explanations, the wording used in the American Uniform Determination of Death Act ("irreversible cessation of all function of the entire brain") is subject to varying interpretation.

In our recently published Canadian consensus guidelines,[4] we attempt to address this conceptual and practical confusion by defining death determined by neurological criteria as follows: "The irreversible capacity for consciousness combined with the irreversible loss of all brainstem function including the capacity to breathe". This may occur as a consequence of intracranial hypertension and/or primary brainstem injury. We acknowledged that currently there are no adequate ancillary tests for the confirmation of brain death in instances of isolated primary brainstem injury.

I applaud Dr. Doyle's suggestion that there is a need to reformulate the definition of brain death to reflect current clinical realities and our evolving understanding. Although difficult to influence the entrenched lexicon, we advocate abandoning the term "brain death" in favour of "the neurological determination of death (NDD)".[4] As discussed in a recent editorial in the Canadian Journal of Anesthesia,[5] brain death is better understood as brain arrest - the complete loss of clinical brain function. If there is a known proximate cause accounting for the brain arrest, and an absence of reversible or confounding conditions, then NDD is the corresponding process and procedure to determine this death.

Sam D. Shemie MD
Montreal Children's Hospital, McGill University Health Centre, Montreal, Canada
E-mail: sam.shemie@muhc.mcgill.ca

References

1 *Halevy A, Brody B.* Brain death: reconciling definitions, criteria, and tests. Ann Intern Med 1993; 119: 519–25.
2 *Bernat JL.* Ethical Issues in Neurology, 2nd ed. Boston: Butterworth Heinemann; 2002: 243–81.
3 *Wijdicks EF.* Brain Death. Philadelphia: Lippincott Williams & Wilkins; 2000: 29–43.
4 *Shemie SD, Doig C, Dickens B, et al.* Severe brain injury to neurological determination of death: Canadian forum recommendations. CMAJ 2006; 174: S1–13.
5 *Shemie SD.* Brain arrest to neurological determination of death to organ utilization: the evolution of hospital-based organ donation strategies in Canada. Can J Anesth 2006; 53: 747–52.

Meningismus after metaraminol administration in a patient with Familial Mediterranean fever

To the Editor:

A 38-yr-old male was admitted to our hospital for treatment of Familial Mediterranean fever (FMF)-related severe episodic abdominal pain. Familial Mediterranean fever is a hereditary inflammatory disease characterized by self-limited recurrent attacks of fever and serositis; the recurrent attacks of fever are accompanied by severe abdominal pain, arthritis and/or chest pain along with a marked increase in acute phase reactants.[1] It was decided to implant a spinal cord stimulator (SCS) for pain control because of inadequate pain relief despite high-dose opioid therapy. Following the epidural placement and positioning of the SCS leads under local anesthesia, general anesthesia was administered for *sc* implantation of the pulse generator. The patient developed hypotension during the course of the anesthetic and this was corrected with fluids and intermittent *iv* metaraminol boluses (cumulative dose: 10 mg). The procedure was completed uneventfully and the patient awakened. On

regaining consciousness, he complained of severe head and neck pain, accompanied by photophobia, nausea and retching. This initially led us to consider a diagnosis of inadvertent dural puncture during epidural placement of the SCS leads. However, on examination, he was found to be pyrexial (38.6°C) and hypertensive (blood pressure 176/104 mmHg); nuchal rigidity and Kernig's sign were also evident. Intravenous morphine, tramadol and paracetamol were of limited analgesic benefit. Within 24 hr, however, the pain and fever abated and all neurologic symptoms resolved completely. The patient later revealed that he suffered from intermittent headaches of a similar nature, but had always considered them to be 'migraine attacks'. Case reports have shown recurrent aseptic meningitis, though rare, may occur in FMF.[2,3] Interestingly, the meningitis attacks can be precipitated by injection of metaraminol intravenously; indeed, the metaraminol provocative test has been proposed as a specific diagnostic test for FMF and benign recurrent aseptic meningitis (Mollaret's meningitis).[3] It therefore appears likely that this patient's meningismus symptoms were triggered by the administration of metaraminol, and we would suggest anesthesiologists remain vigilant to this little-known risk associated with the use of metaraminol in patients with FMF.

Sandeep Kapur MD FRCA
Hirachand Mutagi MD FRCA
Jon Raphael MSc FRCA
Russells Hall Hospital, Dudley, West Midlands, United Kingdom
E-mail: drsandeepkapur@hotmail.com
Accepted for publication May 17, 2006.

References

1 *Ozen S.* Familial mediterranean fever: revisiting an ancient disease.Eur J Pediatr 2003; 162: 449–54.
2 *Collard M, Sellal F, Hirsch E, Mutschler V, Marescaux C.* Recurrent aseptic meningitis in periodic disease or Mollaret's meningitis? (French). Rev Neurol (Paris) 1991; 147: 403–5.
3 *Barakat MH, Mustafa HT, Shakir RA.* Mollaret's meningitis. A variant of recurrent hereditary polyserositis, both provoked by metaraminol. Arch Neurol 1988; 45: 926–7.

Glidescope™ /gastric-tube guided technique: a back-up approach for ProSeal™ LMA insertion

To the Editor:

We read with interest the article by Garcia-Aguado *et al.*[1] reporting the use of a suction catheter inside the drain tube as a guide for Proseal™ laryngeal mask airway (PLMA) positioning. This technique may improve the success rate of PLMA insertion with less trauma to the mouth. For several years, we have been performing PLMA insertion with digital or introducer tool techniques. Our first-attempt success rate with a midline or lateral approach technique is > 80 %, similar to that reported by Cook *et al.*[2] We agree with the authors that priming the PLMA with a 'guide' may provide an advantage in assuming better anatomic conformation of the mouth. For example, a narrow palate or an angle < 90° between the oral and the pharyngeal axis of the posterior tongue may result in folding over the distal cuff of the PLMA, preventing its correct positioning. We also observe that the PLMA first-attempt success rate is lower for less experienced users, who often find that this 'guide' directs the distal PLMA cuff towards the oesophagus.

We generally use a 14 F gastric tube (GT) as a prime in the drain tube to facilitate positioning of the PLMA. To overcome the limitation of the "blind GT insertion" experienced by Garcia-Aguado, we perform GT positioning using direct visualization of the pharynx with the Glidescope™. The Glidescope may be less traumatic than direct laryngoscopy,[3] and we have used this device to facilitate five cases of difficult PLMA positioning (Table) where the digital (either midline and lateral approach) and introducer tool insertion techniques failed. The Glidescope/GT

TABLE Demographic data, etiology of failed insertion and PLMA Glidescope/gastric-tube insertion time

Patient	*Age*	*Sex*	*Weight (kg)*	*Anatomic features*	*Insertion time* (sec)*
1	62	Female	62	Very narrow palate	40
2	42	Female	55	Inter-incisor gap < 3 cm	55
3	35	Female	71	Hypertrophic tonsils	50
4	51	Female	59	Narrow palate	38
5	24	Female	55	Oropharyngeal axis < 90°	35

*Time requested only for Proseal™ laryngeal mask airway (PLMA) insertion with the Glidescope/gastric-tube technique.