CORRESPONDENCE

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In Reply:—The author wishes to thank Drs. Wijdicks and Doyle for their thoughts regarding my recent report.[1]

Dr. Wijdicks's 1995 review of brain death determination in adults[2] and the accompanying summary statement practice parameters of the American Academy of Neurology in determining brain death[3] would have made excellent references; however, neither article contradicts or adds substantially to the information regarding medical aspects of brain death declaration, which I directed toward anesthesiologists. Dr. Wijdicks suggests that neurologists and neurosurgeons arguably may be the physicians best qualified to determine brain death. I sympathize with Dr. Wijdicks's discomfort with the suggestion that anesthesiologists have an ethical obligation to review chart data to ascertain that brain death has been documented appropriately before undertaking the care of vital organ donors. Nevertheless, there are compelling problems with any argument that only physicians in the neurologic specialties are, or should be, qualified to determine brain death. The facts do sometimes speak for themselves. An attending neurologist or neurosurgeon was indeed involved in each of the cases that I presented—cases in which obvious errors occurred in the process of determining death. A more detailed description of confirmatory neurologic testing would have been superfluous because in each case it was clinically obvious that the patient simply did not meet brain death criteria.

Several studies have consistently shown the physician's lack of ability to accurately discuss, define, and recognize brain death. For example, a recent study[4] presented to the Society of Critical Care Medicine demonstrated that only 39% of pediatric attending physicians correctly defined brain death, and slightly more than half knew when confirmatory tests were not needed. Neurologists, neonatologists, and other subspecialists were less accurate than pediatric intensivists in correctly defining brain death, interpreting a clinical scenario, and determining whether confirmatory testing was necessary.

Although I stated that "most institutions also require that at least one of the physicians be a neurologist or a neurosurgeon," this requirement clearly does not ensure accuracy in determining brain death, and neuroscience subspecialty training is not necessary to properly educate physicians in brain death determination.

Ninety-nine percent of vital organ donors are declared dead in intensive care units, but hospital location and demographics seriously influence whether neuroscience subspecialists are involved in determining brain death. The Illinois Brain Death Study[5] indicates that primary care physicians were responsible for brain death determination in 28% of cases, whereas critical care specialists were responsible for brain death determinations in only 9% of cases. Furthermore, only 24% of institutions involved neurologists in all brain death determinations. A neurospecialist was most likely to be involved in this determination in hospitals with a neuroscience residency, and was least likely to be involved in rural hospitals or hospitals with small numbers of potentially brain dead patients. Finally, not all hospitals surveyed had protocols for declaring brain death.

The reality in the United States is that many medical specialists of differing education and abilities are involved in declaring brain death. With such physician variability, it becomes the ethical responsibility of any physician, regardless of specialty, who accepts the care of a vital organ donor to review the data by which the determination of death was made, and to question inconsistencies in test results, regardless of his or her specialty. Anesthesiologists literally may be the last physicians to have an opportunity to examine a brain dead patient, and as "the court of last resort," should be knowledgeable about brain death criteria. It is the important responsibility of every physician to be sure that no living patient is sacrificed to obtain vital organs for another patient.

Dr. Doyle correctly points out that "perfect" brain-death testing is probably not possible; not because we do not have well-defined medical criteria that prospectively and accurately predict brain death, but because the application of diagnostic tests has intrinsic errors. Yet the presence of presumably inalterable, and hopefully low, type I and type II errors in the tests should not serve as an excuse for physicians to be less vigilant correctly applying the tests or interpreting the results. Accepting that a medical test is not perfectly accurate is not equivalent to accepting improper testing conditions or the misinterpretation of test results because of a lack of knowledge.

To Dr. Doyle's question about what to do with patients with "zero prognosis," who do not meet brain death criteria, I would answer that studies have shown that physicians are notoriously inaccurate in predicting time of death for individual patients, and that the patient with zero medical prognosis is difficult to accurately identify, despite persistent physician perceptions to the contrary.[6] Predicting how a patient will want to be cared for as the time of death approaches is even more problematic.

Conflicts of interest between transplant physicians and dying patients, between patients needing expensive end-of-life care and healthcare administrators strapped for dollars, and between patients requiring vital organ replacements and dying patients with vital organs to offer are inherent to the transplant process. Without clear guidelines to manage such conflicts, we risk practices that place vulnerable patient populations in peril and that also risk frightening away potential organ donors. Dr. Doyle's altruistic desire to benefit others with the gift of his vital organs before death may make excellent personal sense, yet may represent poor public policy.

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Malignant Hyperthermia as a Cause for Postoperative Rhabdomyolysis

To the Editor:—We read with great interest the article by Dr. Uratsuji concerning a case of rhabdomyolysis after abdominal surgery in the hyperlordotic position.[1] The authors concluded that rhabdomyolysis and the increase of creatine kinase (CK), lactate dehydrogenase, and serum myoglobin were sufficiently explained by lumbar muscle damage. However, malignant hyperthermia (MH) as another possible cause was not ruled out.

First, it is important to know whether this patient was anesthetized before this incident and whether the patient's family members had anesthetic complications or a history of muscle disease. This patient had an elevated CK of 168 U/l at rest, which might be caused by subclinical myopathy. Furthermore, it is well known that MH is characterized by a hypermetabolic response to inhalational anesthetics (*e.g.*, sevoflurane) or depolarizing muscle relaxants, leading to muscle rigidity, metabolic acidosis, hypercapnia, tachycardia, and fever. However, relevant clinical parameters necessary for interpretation of this syndrome, such as temperature, end-tidal carbon dioxide concentration, arterial blood gases, heart rate, and muscle tone (*i.e.*, rigidity or masseter spasm) were not presented. With these clinical parameters, it would be possible to predict the qualitative likelihood of susceptibility to MH using the Clinical Grading Scale (CGS).[2] In this case, the raw-score rank of the CGS has a minimum of 15 points (CK elevation > 10.000 U/l; MH rank 3, which is defined as somewhat less than likely). However, one might speculate that the use of all clinical indicators of the CGS might produce a higher MH rank.[3]

The clinical course of MH is highly variable (*e.g.*, fulminant, moderate, and mild forms) and postoperative rhabdomyolysis may be the only symptom of MH. Although the probability of MH susceptibility in patients with anesthesia-induced rhabdomyolysis is only 0.07,[4] the *in vitro* contracture tests with halothane and caffeine are necessary for diagnosis of MH susceptibility.[5,6] This view is also emphasized in several case reports that present clinical courses of postoperative rhabdomyolysis after the use of volatile anesthetics.[7-9]

We recommend that the qualitative likelihood of susceptibility to MH should be assessed using the CGS in all cases with MH-like symptoms. Furthermore, all patients with clinical suspicion of MH should undergo muscle biopsy for *in vitro* contracture tests, histologic examination, and genetic screening.

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