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### American Academy of Neurology Guidelines and the Neurologic Determination of Death-Reply

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that our mention of “interventional therapies”<sup>1</sup> encompasses the broad range of both potential surgical and endovascular options; our own experience also supports the authors’ observations of beneficial outcomes after surgical revascularization. At our institution, after careful evaluation using the Vertebrobasilar Flow Evaluation and Risk of Transient Ischemic Attack and Stroke imaging protocol, each patient’s case is discussed in a multidisciplinary setting including the cerebrovascular and endovascular teams, and the best treatment option, whether microsurgical or endovascular is considered. The authors nicely describe their experience with intracranial bypass procedures and their results compare favorably with previous work.<sup>2-4</sup> We should add that extracranial revascularization procedures are also available and include vertebral-carotid transposition, bypass techniques using interposition grafts from the subclavian or common carotid arteries to the vertebral artery and endarterectomy of the vertebral artery or subclavian artery,<sup>5</sup> all of which may be considered for the microsurgical management of low-flow vertebrobasilar disease.

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## American Academy of Neurology Guidelines and the Neurologic Determination of Death

**To the Editor** Greer et al<sup>1</sup> analyzed compliance of hospital protocols on brain death (BD) determination with the 2010 American Academy of Neurology (AAN) guidelines. They posited that this neurologic standard is 100% accurate. We comment on the accuracy claim.

First, accuracy is judged by resumption of a specific set of ceased neurologic functions within a predefined short timeline. Longer waiting times negatively affect organ

donation. However, the irreversibility timeline of BD findings has not been scientifically established. The eventual outcome is terminal organ procurement or treatment withdrawal, but, although resulting in a 100% death rate, inherently confounds the reported accuracy or false-positive rate. Indeed, false-positive cases are generally reported when court orders support families’ request for prolonged life-support treatment. The McMath<sup>2</sup> and Hailu<sup>3</sup> cases are examples.

Second, for a similar severity of brain injuries, the AAN standard is 370% more likely to diagnose BD than other more stringent worldwide guidelines.<sup>4</sup> The almost 4-fold increase implicitly challenges the standard’s accuracy. A normal or minimally ischemic brainstem was reported by histopathology at autopsy in 60% of donors who were determined dead by the AAN standard.<sup>5</sup>

Third, the accepted medical standard must comply with the legal standard.<sup>1</sup> The Supreme Court of Nevada<sup>3</sup> opined in 2015: “Although ‘it is for [the] law to define the standard of death,’ courts have deferred to the medical community to determine the applicable criteria for deciding whether brain death is present” and “though courts defer to the medical community to determine the applicable criteria to measure brain functioning, it is the duty of the law to establish the applicable standard that said criteria must meet.”

The AAN standard requires confirming unresponsiveness (equated with coma) and absent motor brainstem reflexes including respiration. It excludes other residual brain functions present in BD. The Supreme Court of Nevada<sup>3</sup> has clarified this issue: “Are the AAN guidelines considered ‘accepted medical standards,’ which adequately measure all functions of a person’s entire brain, including the brain stem?... Based on the foregoing, and the record before us, we are not convinced that the AAN guidelines are considered the accepted medical standard that can be applied in a way to make Nevada’s Determination of Death Act uniform with states that have adopted it, as the [Uniform Determination of Death Act] requires.”

We posit that the AAN standard describes severe neurologic disabilities not equivalent with biologic death, but instead fall within the spectrum of disorders of consciousness. Research is needed to characterize this neurologic state and validate a reversibility timeline.

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**In Reply** Rady and Verheijde appear to have missed the point of our article<sup>1</sup> or otherwise have chosen to use it as a platform to criticize the American Academy of Neurology Practice Parameters (AANPPs). Our study was designed to look at variability of US hospital policies in comparison with the AANPPs, which are rather uniformly considered the standard document in the United States. Indeed, to our knowledge, there is no position statement from another US society, or any document from another country, that is used as an example to construct hospital policies in the United States.

Our original study in 2008 demonstrated concerning variability in hospital policies in comparison with the 1995 AANPPs,<sup>2</sup> which is what led us to update the parameters in 2010.<sup>3</sup> The goals of that update were multiple: (1) to provide an evidence-based review of the literature on brain death since the 1995 parameters, including the important finding that there had been no legitimate reports of inaccurate determination of death using those criteria; (2) to provide a minimum standard for brain death determination, which was, in fact, far more detailed and proscriptive than the 1995 parameters; and (3) to provide a comprehensive and detailed explanation for how brain death should be determined in a meticulous and highly careful manner, ensuring a conservative approach and that patients would not be determined dead if there was any concern for confounding or inaccuracy. Our hope was that US hospitals would readily change their local policies to ensure a highly stringent and accurate approach.

Rady and Verheijde have chosen to distract from the goals and findings of our study, and their letter is nothing but a straw man argument. First, there are no legitimate or objectively confirmed cases of erroneous brain death determination in the medical literature. Second, they suggest that there must be histopathological confirmation of death of all brain cells. However, according to the Uniform Determination of Death Act,<sup>4</sup> there must be irreversible cessation of function of the entire brain. Brain death is a clinical diagnosis; there is no requirement for, or practicality to, requiring a pathological confirmation. Third, the Nevada Supreme Court inexplicably ruled that the AANPPs might not be “accepted medical standards” as stipulated by the Uniform Determination of Death Act. The AANPPs from 2010 most certainly do appropriately and thoroughly measure the function of the entire brain. Ancillary tests do not measure brain function, are subject to false-positives and false-negatives,<sup>5</sup> and are not necessary if the clinical evaluation (including the exclusion of possible confounders) is done correctly. No ancillary testing to date has improved (and thereby questioned) prior methods of clinical diagnosis.<sup>6</sup>

The 2010 AANPPs remain the authoritative statement, have been heavily vetted, and provide overtly stringent instructions on brain death determination. Efforts should be made to ensure widespread hospital adoption of the more stringent standards as outlined in that work, which is the only proper conclusion of our study.<sup>1</sup>

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## Olfactory Loss—On the Road to Potential Diagnosis Criteria of Alzheimer Disease

**To the Editor** Rosebud and colleagues<sup>1</sup> reported on a longitudinal study strengthening the idea that olfactory impairment has potential to be a biomarker for diagnosing mild cognitive impairment (MCI) and Alzheimer disease (AD) or early detection for cognitively normal elderly individuals who would likely progress to MCI or AD. The underlying mechanism may be due to, at least in part, the involvement of AD neuropathological processes in both the olfactory bulb and other cerebral areas that are related to olfactory function.

One study<sup>2</sup> even pointed out that olfactory deficit, in terms of the prediction of cognitive decline in cognitively normal individuals, was superior to impairment in episodic memory, which is one of the earliest cognitive symptoms and has an excellent specificity for AD. However, olfactory impairment is not very specific to AD; it is also a common feature of other neurodegenerative diseases, including Parkinson disease (PD), dementia with Lewy bodies, PD dementia, frontotemporal dementias, corticobasal syndromes, and progressive supranuclear palsy. As for PD, the prevalence of olfactory impairment is more than 90% and appears to exceed the prevalence of other cardinal motor signs.<sup>3</sup> The sensitivity and specificity of olfactory testing in discriminating PD from non-PD is consider-