LETTER TO THE EDITOR


In the June issue, Edlow et al used a cutting edge imaging tool to demonstrate tracts in the autopsied brain of a victim of traumatic coma (1). Histologic study of the same brain showed widespread axonal damage in keeping with the long-standing understanding that closed head injury results in diffuse axonal injury throughout the brain (2). The imaging analysis showed, however, that only 1 set of pathways, that is, the “arousal” pathways connecting brainstem nuclei to the basal forebrain and to thalamic intralaminar and reticular nuclei, was completely disrupted. They proposed that traumatic coma is specifically related to this disconnection rather than to widespread axonal damage throughout the brain, as has been suggested by others studying nonhuman primates (2). I should like to point out that the proposal of Edlow et al is consistent with the findings I reported with coworkers in 1981 (3). In that study, we showed that more than 80% of patients who died as a result of head trauma had midbrain lesions. This was consistent with evoked potential data gathered within 72 hours of injury from 165 patients that showed that midbrain deficits were associated with death or poor recovery while those without midbrain deficits made good to moderate recoveries. Because our article focused on the prognostic implications of midbrain lesions in the context of widespread diffuse axonal damage, the relationship of our findings to coma may not have been appreciated. Indeed, the word “coma” was only used when describing exclusion criteria: “Patients excluded… included those whose coma was thought to be due to alcohol, drug overdose, or epilepsy.” Thus, it was implied (and true) that all of our autopsied patients were believed to be in coma from the moment they were found and remained so until death. Consistent with their diagnosis of coma was the fact that, on arrival at the hospital, 21 of the 23 patients with midbrain lesions at autopsy had impaired or absent oculocephalic reflexes and 19 had absent or diminished pupillary reflexes.

A study of pigs has also suggested that immediate coma after traumatic brain injury is dependent on axonal damage in the brainstem (4). No relationship was found between coma and the extent of axonal damage in other parts of the brain. That study, like the recent report by Edlow et al, did not recognize that we reported the essential role of midbrain injury in producing posttraumatic coma.

It seems important to alert readers to our findings because they consist of human rather than animal data and involved a set of 35 patients. Our findings offer strong support for the conclusions of Edlow et al, which are based on the extensive study of a single brain using high angular resolution diffusion imaging tractography. Our findings, like theirs, challenge the belief that posttraumatic coma is a consequence of widespread axonal damage throughout the brain (2) and point instead to the essential role of midbrain damage in producing what they describe as a subcortical disconnection syndrome related to disconnection from brainstem arousal nuclei.

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REFERENCES

Authors’ Reply:

We thank Dr. Rosenblum for bringing this important publication to our attention. As he points out, the midbrain lesions described in their study (1) add compelling evidence in support of the association of traumatic midbrain lesions and acute coma that is described in our human case (2) and in the piglet cases reported by Smith and colleagues (3). Of note, the neuroanatomic localization of midbrain lesions described in Dr. Rosenblum’s article primarily involves the dorsolateral quadrant of the midbrain. In contrast, another midbrain traumatic hemorrhage syndrome in which a hemorrhagic lesion localized to the midline, presumably affecting the raphe nuclei, was also associated with acute traumatic coma, pupillary abnormalities, and ophthalmoparesis was described by Ropper and Miller (4) in 1985.

Despite the apparent association between both dorsolateral and midline traumatic midbrain lesions and poor outcome (i.e., persistent alteration of consciousness and/or death), we and others have recently reported cases in which early magnetic resonance imaging data demonstrated hemorrhagic axonal injury involving the midbrain, yet the patients nevertheless recovered consciousness, communication, and functional independence (5, 6). For this reason, despite the evidence from detailed clinicopathologic studies of an association between traumatic midbrain lesions and death, we caution against the assumption that all such lesions are associated with poor outcome. There is a critical need for studies to determine whether a specific midbrain lesion is compatible with recovery or whether it so severely disrupts the ascending reticular activating system that consciousness cannot be restored.

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