In Reply:

The letter by Casson et al. shed light and clarification on the entity of chronic traumatic encephalopathy that was lacking in the article by Omalu et al. (1). As they note, it was originally characterized by a tetrad of findings, including abnormalities of the septum pellucidum, cerebellar scarring, degeneration of the substantia nigra, and widespread neurofibrillary tangles in the cerebral cortex and brainstem. They correctly point out that the case presented by Omalu et al. (1) had only one of these pathological findings, raising concern as to whether the individual actually met the criteria for chronic traumatic encephalopathy. Based on these criteria, it would seem not. Consequently, the authors’ assertion that the individual’s pre-mortem cognitive decline, depression, and Parkinsonian symptoms where a manifestation of chronic traumatic encephalopathy seems largely unfounded. Unfortunately, as previously noted by several of the reviewers of the original manuscript, there was no evidence presented linking the player’s NFL career to his neurocognitive decline or to the neuropathological findings. Furthermore, the player had no documented concussions during his career and he played mostly as an offensive lineman which is one of the positions associated with the lowest frequency of concussion. Ultimately, the authors’ lack of alternative explanations for the neuropathological findings, the selective and seemingly inappropriate use of the definition of chronic traumatic encephalopathy, and the “N of 1” nature of this study severely weaken it. Given this lack of hard evidence and the overly assuming nature linking the available data together, I agree that retraction or a major revision by the authors is warranted. Casson et al. are to be thanked for further educating us about the entity of chronic traumatic encephalopathy and completing the editorial process.

Daniel F. Kelly
Los Angeles, California

More important, however, is the issue of the first several paragraphs of their letter, which consist of repetitious expressions regarding how Omalu et al. incorrectly used diagnostic nomenclature. They do not dispute his findings, they simply dispute the name Omalu et al. have given to those findings. This seems to be an issue that requires brief discussion and clarification, not relentless table-pounding.

In summary, I see the Casson et al. letter as raising several valid points regarding the intrinsic limitations of the case material used in Omalu et al.’s study. However, because these limitations were noted by Omalu et al. in the published version, I do not see the point of publishing a letter reiterating them. Additionally, and perhaps more importantly, I see no value in the repetitious initial paragraphs of the letter devoted entirely to nomenclature.

Lastly, there is the issue of the “tone” of the letter. Disagreements among clinicians and scientists are important and should be published, but they need to reflect appropriate collegial respect.

Joseph Bleiberg
Neuropsychologist
Washington, D.C.

In Reply:

My review of the first submission from Omalu et al. (1) identified many of the issues noted in Casson et al.’s letter to the editor. I recommended that Omalu et al. explicitly state the authors’ lack of alternative explanations for the neuropathological findings, raising concern as to whether the individual actually met the criteria for chronic traumatic encephalopathy. Based on these criteria, it would seem not. Consequently, the authors’ assertion that the individual’s pre-mortem cognitive decline, depression, and Parkinsonian symptoms are a manifestation of chronic traumatic encephalopathy seems largely unfounded. Unfortunately, as previously noted by several of the reviewers of the original manuscript, there was no evidence presented linking the player’s NFL career to his neurocognitive decline or to the neuropathological findings. Furthermore, the player had no documented concussions during his career and he played mostly as an offensive lineman which is one of the positions associated with the lowest frequency of concussion. Ultimately, the authors’ lack of alternative explanations for the neuropathological findings, the selective and seemingly inappropriate use of the definition of chronic traumatic encephalopathy, and the “N of 1” nature of this study severely weaken it. Given this lack of hard evidence and the overly assuming nature linking the available data together, I agree that retraction or a major revision by the authors is warranted. Casson et al. are to be thanked for further educating us about the entity of chronic traumatic encephalopathy and completing the editorial process.

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Omalu et al. resubmitted a substantially revised and truncated paper, much more modest in scope, and much more cautious regarding sweeping generalizations. I thought they had been responsive to my initial review.

The above statements are regarding the last few paragraphs of the letter by Casson et al.

Daniel F. Kelly
Los Angeles, California

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