Commentary

The lesion(s) in traumatic brain injury: implications for clinical neuropsychology

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We applaud Dr. Bigler’s signaling the need for attention to the future prospects of neuropsychology in a competitive environment, and his enduring enthusiasm and leadership in encouraging neuropsychologists to involve themselves in neuroradiological imaging (Bigler, 2001). However, we have several concerns about possible misinterpretation of the data and views presented in the article entitled “The lesion(s) in traumatic brain injury: implications for clinical neuropsychology.” Our first concern is the blurring of the effects of mild traumatic brain injury (MTBI) with the effects of moderate and severe brain injury. Another concern is the pervasive tendency to suggest physiological origins of observed effects such as subjective complaints and test measures. Physiological origins are presumed far beyond the explanatory power of the available scientific literature. A third concern is a broad tendency to ignore and discount psychological explanations for the observed effects, specifically including a conspicuous failure to take into account recent literature on the profound effects of effort, response bias, and compensation-related contexts.

The title, abstract, and introduction to Dr. Bigler’s article refer to traumatic brain injury (TBI) in general. There is no language in the title, abstract or introduction to indicate that his focus would be limited to moderate or severe TBI, and his conclusion makes an ambiguous reference to damage caused by “at least mild-moderate to severe TBI” (p. 123). The introduction states, “This review focuses on the neuropathological substrates of TBI in relation to...
neuropsychological outcome” (p. 97). Then the early paragraphs of the article emphasize the
delicacy of the brain and the possibility that anything that disrupts the complex system involved,
even subtly, will produce a loss. However, in his discussion, Dr. Bigler includes illustrations
such as a high-speed collision motor vehicle accident involving a roll over and a plaintiff struck
by a car and flung over 70 ft by the impact. Then, 16 pages into the article, he mentions that
“The cases discussed in this article all had serious injury” (p. 121). However, his allusions to
MTBI are blurred with discussion of TBI in general, making the text ambiguous and probably
misleading to the reader. The most important explanatory variables for complaints following
MTBI were not mentioned, for example, effort or response bias. Furthermore, pre-existing
personality variables (Greiffenstein & Baker, 2001) and other confounding factors such as
disability and litigation status were neglected (e.g., see Binder & Rohling, 1996). Finally, the
vast literature documenting the generally mild to non-existent effects of MTBI was ignored
(e.g., Binder, 1997; Binder, Rohling, & Larrabee, 1997; Dikmen, Machamer, Winn, & Temkin,

The language used by Dr. Bigler in this article implies that the exaggerated volume of
complaints associated with MTBI is an indication of real consequences of physical injury. For
example, in noting the tendency of MTBI patients to complain more than severe TBI patients,
Dr. Bigler refers to some of his previous research published as an abstract as having “demon-
strated that patients with mild TBI actually had greater emotional dysfunction than those with
more severe head injury” (p. 111, emphasis added). In our opinion, the abstract cited does not
demonstrate such a finding. With no normal controls and no control for suboptimal effort, dis-
ability or litigation status, the abstract reports correlational data in support of the well-known
tendency for many patients with MTBI to report more somatic and psychiatric symptoms than
patients with documented severe TBI. There apparently was no attempt in the cited study to
control for self-selection bias as a source of this apparent association. In an attempt to suggest
a physiological explanation for these complaints, Dr. Bigler notes anosognosia associated with
severe injuries, implausibly characterizes MTBI patients as suffering from “keen awareness”
of deficits, and then speculates about underlying physical pathology. Unfortunately, in this
discussion he fails to mention the well known effects of response bias, suboptimal effort, comp-
ensation, litigation, or other relevant incentives that provide a more parsimonious explanation
for his findings. He did not mention that plaintiffs who neither have nor allege any TBI report
more “brain injury” related complaints than do most severely brain injured patients (e.g., see
Lees-Haley & Brown, 1993). Dr. Bigler notes, “Traumatic brain injury (TBI) represents one
of the most common disorders seen by clinical neuropsychologists . . .” We would add, for
clarification and specificity, that allegations of TBI by patients referred by lawyers are the most
common “disorder” seen by private practice neuropsychologists (e.g., see Sweet, Mober, &
Suchy, 2000). Therefore, the need for concern about confounding influences on test scores is
now well accepted. In a recent review, Iverson and Binder (2000) state that “A forensic eval-
uation that does not include careful consideration of possible negative response bias should be
considered incomplete” (p. 829).

We are concerned that the aura of objectivity surrounding neuroimaging, in combination
with advancing technology for detecting increasingly minute forms of tissue change, may
be highly misleading in a context in which there are so many competing explanations for
a patient’s complaints. This error of attribution is especially problematic because it reflects
a “little boy with a hammer” tendency to assume that patients’ complaints are produced by TBI without considering alternative explanations. For example, Dr. Bigler offers his opinion that alcohol-related accidents—involving a population fraught with “self-selection” bias problems—appear to have consequences that are more serious without qualifying the comment by pointing out that people who drink and drive are not a random or representative sample of the general population. He says, “Substance abuse at the time of injury probably exacerbates structural damage to the brain” (p. 113) based on studies controlling for age, injury severity, and head size, but does not mention the lower education levels and lower rates of seat belt use by individuals who drink and drive (e.g., see Hillary, Moelter, Schatz & Chute, 2001). Alternatively, we should also consider the possibility that people who drive while intoxicated may have pre-existing judgment deficits, lower intelligence, or different rates of pre-existing injuries associated with pre-morbid problems with attention, concentration, impulsivity or risk taking.

A recurring theme in this article is the inflation of the presumed effects of brain injury. Dr. Bigler asserts, without qualification, that “The ‘lesion’ is always larger than can be visualized on MR or CT imaging” and suggests that “Accordingly, observed structural abnormalities on MR may only represent ‘tip-of-the-iceberg’ phenomena with regards to pathology” (p. 109). He continues, “We have rather conclusively shown that mild cases of TBI, including simple concussion, typically do not result in detectable abnormalities on traditional clinical MR scans (Bigler & Snyder, 1995). However, that should not be interpreted as meaning absence of pathology . . .” (p. 109) and “… even a focal deficit in the TBI patient is certainly super-imposed on a more general pathology beyond the discrete boundaries producing the focal neurological deficit. The diffuse nature of brain injury is the common theme of this paper” (p. 100). He later says “the prototype mild-moderate to severe TBI ‘lesion’ is probably generalized, non-specific damage spread throughout the brain, but with greater involvement of the frontal and temporal regions” (p. 120). Then still later he states, “In TBI, a focal lesion will always be superimposed on the back-drop of a more global, diffuse injury” (p. 123) [sic]. In a style reminiscent of the old neuropsychological saw that absence of evidence is not evidence (when in fact it often is), Bigler argues, “What about the patient with significant brain injury, but ‘normal’ CT, MR, or SPECT scan findings? Obviously, the absence of positive imaging findings is not to be equated with the absence of brain pathology” (p. 121). But are these observed changes ecologically meaningful? Binder et al. (1997) have argued persuasively that mild TBI is associated with deficits smaller than the standard error of estimate of most neuropsychological instruments. Dr. Bigler’s position seems to entail an implicit suggestion that the patient should be presumed brain-injured until proven otherwise. Some of his language borders on suggesting a preference for relying on individual anecdotal evidence in preference to systematic controlled scientific studies: “… brain–behavior relationships will, in large part, be unique for each individual, with the exception of some dedicated motor and sensory pathways” (p. 123).

On p. 109, Dr. Bigler cites a few of the numerous recent studies of concussions in athletes as evidence for his belief that concussions produce persistent cognitive deficits. This is misleading. Some of the studies on concussions in athletes address only very brief time frames (e.g., hours, days, or a month), some authors have focused on multiple injuries rather than single instance concussions, and some of the studies of athletes report findings precisely contrary to Dr. Bigler’s thesis. For example, Echemendia, Putukian, Mackin, Julian, and Shoss (2001)
reported this:

Neuropsychological test data yielded significant differences between injured athletes and controls at 2 and 48 h following cerebral concussion; injured athletes performing significantly worse than controls. Injured athletes reported a significantly greater number of postconcussion symptoms 2 h following injury but not at the 48-h assessment. No multivariate group differences were found at 1 week, but univariate analyses suggested significant differences on a few measures. At 1 month postinjury, a statistically significant difference was found on one measure with injured athletes marginally outperforming controls” (p. 23).

One of us (MLR) calculated the effect size on similar sports data from Collins et al. (1999) and found the effect size to be too small, on an individual case basis, to be detected by the available sports test batteries.

In our opinion, the main reason for the author’s excessive exuberance about the physical basis for complaints associated with TBI arises from his failure to adequately consider base rates. For example, Dr. Bigler cites Deb, Lyons, Koutoukis, Ali, and McCarthy (1999) as his evidence that psychiatric disorder is commonplace after TBI. Although some of the specific diagnostic categories reported by Deb et al. (1999) appear higher than base rate expectations, to our knowledge there are no responsible scientists who assert that TBI patients are random, representative members of the population. And in any case, the total rate of mental disorders in the 196 TBI patients studied by Deb et al. (1999) did not exceed the base rate for the general population documented in psychiatric epidemiological research. For example, estimates of 1 year prevalence of mental disorders in the adult non-institutionalized population in the United States are about 29%—almost 3 out of 10 (approximately 19%—almost 2 in 10—if substance related disorders are excluded) (e.g., see Kessler et al., 1994; Regier, Boyd, & Burke, 1988; Robins et al., 1984). Bourdon, Rae, Locke, Narrow, and Regier (1992) suggested that the one-in-five estimate applied to a 6-month prevalence. Lifetime prevalence is, of course, higher.

Although the goal of improving sensitivity of the instruments we rely on is admirable, that goal needs to be balanced by improving overall test accuracy and interpreting results in the perspective of appropriate base rates. Just as dermatologists observe that no one is completely free of all forms of skin pathology, not one human being is entirely free of psychological symptomatology or fully functioning neuropsychologically. Every individual has an imperfect memory, variable concentration and attention, imperfect coordination, etc. Even Nobel prize-winning authors use a thesaurus and spell checkers, and have editors to check their work for errors. The most famous example of a so-called photographic memory was the man referred to by Luria as “S” in The Mind of a Mnemonist, but even S forgot things (Luria, Solotaroff, & Bruner, 1988). To our knowledge there has never been a documented case of a true photographic memory.

The language in Dr. Bigler’s article may be misconstrued by some readers to imply that all evidence of neuronal pathology is significant. He fails to separately consider mild TBI, and whether observed neuroanatomical changes simply correlate with poor neuropsychological test performance some of the time, or actually cause or meaningfully explain the pathology. We argue that making a reasonable difference in the life of an individual is what defines the significance of brain tissue pathology, not the sheer ability of experts to detect change through neuroradiological imaging or other methods.
To illustrate our point, it is useful to compare progress in neuropsychology to the history of testing in toxicology. Toxicologists have a fundamental axiom that the dose makes the poison. That is, in sufficient amounts anything can be toxic. Furthermore, a sufficiently small amount of even the most virulent toxin is perfectly harmless. Over the years, toxicologists have developed technology that detects smaller and smaller amounts of toxic substances. It has now become possible for toxicologists to detect parts per thousand, million, billion, and trillion. It has now become commonplace to detect trace amounts of various poisonous substances in randomly selected normal healthy adults. Does this mean they are suffering detectable neurotoxic cognitive injury? No. It simply means that test sensitivity technology has greatly improved.

Along these same lines, with increasing development of neuroradiological technology, we can foresee detecting increasingly finer degrees of change in brain tissue, to the point of triviality. Dr. Bigler refers ominously to injured and dead neurons in general terms. However, we should all remember that we lose neurons every day in the natural course of living. The most common cause of brain atrophy is staying alive (aging). If tests become so sensitive that they measure a loss of neuronal tissue so minute that there is no detectable or meaningful difference in the life of the individual involved, we should recognize the nominal nature of the loss. If the neuronal loss is so limited that it does not affect social, occupational, recreational, spiritual, or any other area of significance to human beings, then the loss should not be characterized in clinical language that frightens patients and their families. It is true that falling off a three-story building (360 in.) may cause brain damage. However, it does not follow that stepping off a series of 360 1 in. declines on a sloping surface causes cumulative brain injury. Yet, Dr. Bigler claims that if valid neuropsychological evaluation and testing support a finding of residual deficits, neuropsychologists should “trust that those deficits are organically based” and concludes “the neuropsychological technique remains sensitive in the detection of neurobehavioral consequences of TBI, even if no neuroimaging abnormality is detected” (p. 123). This claim will delight lawyers but it exemplifies one of the most serious flaws in the article. Neuropsychological test findings of deficits can result from a myriad of sources and it does not follow that one should trust that these deficits are organic residuals of a specific TBI with normal neuroimagery. Green, Rohling, Lees-Haley, and Allen (2001) have shown that the effects related to objectively determined symptom exaggeration are 4.5 times larger than those produced by moderate to severe brain injury.

It is common knowledge that many mild brain injury patients have positive neuroradiological imaging (e.g., see French & Dublin, 1977; Iverson et al., 2000; Jeret et al., 1993). But the majority of MTBI patients do not exhibit test deficits more than a month beyond their injury (e.g., Binder et al., 1997; Dikmen, McLean, & Temkin, 1986; Green & Iverson, 2001; Gentilini et al., 1985; Rohling, Millis, & Meyers, 2000). There are relatively few differences between the complaints of MTBI patients and controls (e.g., see Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992; Gouvier, Uddo-Crane, & Brown, 1988; Lees-Haley & Brown, 1993; Santa Maria, Pinkston, Miller, & Gouvier, 2001). The tendency to make postconcussive complaints is a better predictor of neuropsychological test performance than actual history of head injury (Hanna-Pladdy, Gouvier, & Berry, 1997; Pinkston, Gouvier, & Santa Maria, 2000; Santa Maria et al., 2001). Also, the context in which neuropsychologists receive their most referrals (medico-legal proceedings) is associated with
greater neuropsychological complaint rates regardless of head injury history (e.g., see Binder & Willis, 1991; Cicerone & Kalmar, 1995; Green & Iverson, 2001; Green, Iverson, & Allen, 1999; Greiffenstein, Baker, & Gola, 1994, 1996; Iverson, Green, & Gervais, 1999; Lees-Haley & Brown, 1993; Lees-Haley, Fox, & Courtney, 2001; Millis, 1992; Putnam & Millis, 1994; Suhr, Tranel, Wefel, & Barrash, 1997; Youngjohn, Burrows, & Erdal, 1995; Youngjohn, Davis, & Wolf, 1997).

As Miller and Donders (2001) noted, “The evaluation of persistent subjective complaints after [traumatic head injury] should consider injury severity in concert with psychological and financial/motivational factors. Great caution should be taken in attributing persistent symptomatology after mild THI to cerebral dysfunction” (2001, p. 297). Dr. Bigler dismisses a similar conclusion by Mittenberg and Strauman (2000) that most persistent PCS is psychological rather than neurological, on the speculative grounds that future research will show him to be correct. Specifically, he states: “I argue that, as greater sophistication develops in neuroimaging and neuroimaging protocols to detect structure–function relationships, this type of position will no longer be tenable.” He further rejects Mittenberg and Strauman’s conclusion on the semantic grounds that “all ‘psychological’ phenomena are rooted in CNS function” (p. 110), as if to suggest that the existence of physical substrates and correlates of behavioral phenomena somehow make behavior not real (he even puts “psychological” in quotes as if psychological phenomena are somehow not recognized). This is analogous to a chemist dismissing physiology on the grounds that what really is going on is chemistry because physiology is “rooted in” chemical interactions.

Bigler assumes that more sophisticated imaging methods will reveal biological bases for postconcussive complaints in mild head injury and also, presumably, for the impaired test scores in such patients but existing data show that this is very unlikely indeed for many cases. Although we may agree that all behavior must originate from neuronal activity of some sort, this is different from concluding that a particular behavior is causally linked to a specific accident, injury, or disease state. Rohling, Green, Allen, and Lees-Haley (2000) reported that 50% of the variance in neuropsychological test results in compensation claimants was explained by symptom validity testing, whereas a measure of brain injury severity (GCS) explained less than 5% of the variance in the same data. On an average of 36 test scores, those with mild head injuries who failed effort testing scored several times further below the normal mean than patients with severe brain injuries. In contrast, the people with mild head injuries who passed symptom validity testing scored only one-tenth of a standard deviation below the normal mean and significantly higher than do those with severe brain injuries. Thus, exaggeration had a larger effect on test scores than did severe brain injury. It is not plausible that the very severely impaired test scores in cases of mild head injury who failed symptom validity tests were valid and, therefore, structural brain changes are unlikely to explain these results. Similarly, memory complaints were found to be inversely correlated with head injury severity and uncorrelated with objective memory test scores (Green & Allen, 2000). While lack of insight might explain a lack of memory complaints in the most severe cases of TBI, it was also found that headache complaints were inversely correlated with head injury severity. It makes little sense to argue that people with the most severe brain injuries actually have more headaches but that they lack insight into their headaches. Motivational variables and symptom exaggeration offer a more plausible explanation of why people with mild head injuries complain more than those with
severe head injuries and also why those with mild head injuries are more likely to fail simple effort tests than those with severe head injuries (Green et al., 1999).

Finally, Dr. Bigler relies substantially on procedures that are experimental and not generally accepted in neuropsychology or medicine. For example, he refers to SPECT and magnetoencephalography. The Therapeutics and Technology Committee of the American Academy of Neurology has rated SPECT as an Investigational procedure with respect to mild head trauma. SPECT has not yet been established as appropriate for clinical use. The rank order of the usefulness of various procedures by the Therapeutics and Technology Committee is Established, Promising, Investigational (evidence insufficient to determine appropriateness, warrants further study. Use of this technology for a given indication in this specified patient population should be confined largely to research protocols), Doubtful, Unacceptable (Therapeutics and Technology Committee, 1996). The Society of Nuclear Brain Imaging Counsel’s position is that there is not yet adequate evidence to support the use of SPECT or PET in mild traumatic brain injury to establish cause and effect relationships (Society of Nuclear Medicine Brain Imaging Council, 1996). To our knowledge, magnetoencephalography is so new and experimental that the implications of magnetoencephalographic findings for traumatic brain injury are unknown.

While brain-imaging techniques hold great promise for understanding brain and behavior, behavior is, in itself, a subject that requires methodological sophistication. Care should be taken to avoid reductionistic thinking, within which excitement about new technology leads us to ignore more obvious explanations for human behavior (i.e., parsimony). Reductio ad absurdum is an end state that can be reached by focusing too much on physiological and structural images and paying too little attention to behavior and the factors that are known to affect behavior. As Kuncel, Hezlett, and Ones (2001) noted, “The burden of proof for a new predictor should lie with its proponent, who should demonstrate its incremental validity. This demonstration must take the form of multiple validations across several (large) samples and multiple criterion measures” (2001, p. 176). A long established tradition in psychology and medicine is to presume the most likely explanation until there is sufficient evidence to reach the less probable conclusion. For example, this is expressed in the informal adage, “When you hear hoof beats, don’t think zebras.” Along similar lines, it is statistically and empirically unsound for us to presume MTBI patients to be suffering chronic effects of brain injury until proven otherwise.

In conclusion, Dr. Bigler’s enthusiasm for neuroradiology is infectious and impressive. However, his article illustrates the danger of leaving the psychology out of neuropsychology and focusing on one side of the brain–behavior relationship. After all, we are not neurologists, we are neuropsychologists. As a result, we recognize that behavioral manifestations of neuronal activity are complex and require rigorous psychometric analyses if we are to make probabilistic statements as to the cause of any particular behavior.

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References


