

How the 1950s changed our understanding of traumatic encephalopathy and its sequelae

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At a 1954 meeting of the Canadian Neurological Society, Jean Saucier, Professor of Neurology at the University of Montreal, declared concussion “a misnomer.” The problem, Saucier observed, was that the symptoms associated with closed head injury appeared identical in concussion, contusion and

laceration. “If we are to retain the term ‘concussion,’” he argued, “we feel that it cannot be limited to the effects of the mildest blows since severe blows also concuss.”¹ Instead, Saucier called for its replacement with “the concept of diffuse head injury, benign or malignant, or still better, with that of *traumatic encephalop-*

athy,” an acute condition possible from even minor blows.¹

Traumatic encephalopathy referred to an acute pathology of the brain that likely resolved (but could leave residual weakness), but the 1950s saw the conjunction of multiple interests in the long-term consequences of head injury — and of traumatic

encephalopathy’s lingering cousin, chronic traumatic encephalopathy. Saucier’s lecture came at a time when neurologists, neurosurgeons, physiologists, psychiatrists, even engineers, were discussing the risk of closed head injuries. By then, to paraphrase historian Robert Proctor, medical writing securely showed that acute, chronic, remote and delayed sequelae, some degenerative, sometimes followed traumatic encephalopathy.² Indeed, Saucier’s concerns echoed those made by clinicians for decades previously. This essay traces the story of the crystallization of traumatic encephalopathy as a medical concept.

The genealogy of Saucier’s preferred wording for concussion — “traumatic encephalopathy” — is traceable to the 1890s. From that decade on, “traumatic dementia,” “traumatic neurasthenia,” “traumatic neuroses” and



On Sept. 23, 1952, in the 13th round of the world heavyweight title championship, Rocky Marciano landed his famous “Susie Q,” a devastating and decisive blow that knocked out Jersey Joe Walcott. Photographed by Herb Scharfman at Philadelphia’s Municipal Stadium.

“traumatic encephalitis” were all deployed to refer to the sequelae of a closed head injury. The word “trauma,” however, brought with it a complex history: the word originally meant physical injury to bodily tissues, but by 1910, the influence of Sigmund Freud, Pierre Janet and others had imbued “trauma” with environmental significances in addition to its somatic meanings.³ The word thus facilitated a rhetorical slippage between organic degradation of the brain and functional disarray of mind and tissues. It overlay common cultural understandings of degeneration as a bodily, psychic, hereditary and cultural process.⁴

“Encephalopathy,” too, had long been associated with brain disease and cerebral symptoms. By the 1920s, the symptoms of encephalitis had achieved widespread attention in the pandemic of encephalitis lethargica.⁵ In 1927, neurologists Osnato and Giliberti argued that the symptoms of that disease showed that changes to the nervous system could result in subjective brain symptoms similar to those seen in “traumatic encephalopathy.”⁶ This was further elaborated in papers by pathologist Harrison Martland in 1928 and neurologists Israel Strauss and Nathan Savitisky in 1934. Martland’s paper provided the classic description of traumatic encephalopathy in boxers;⁷ Strauss and Savitisky, meanwhile, insisted that “postconcussion syndrome” be taken as best evidence for injury to the tissues of the encephalon resulting from concussion.⁸

In the ensuing decades, these observations about closed head injury had spread widely — military research in several nations from World War I on was a major stimulus. Outside of medicine, they could be found in warnings that appeared in industrial hygiene and occupational safety literature about concussion, the risks of falling objects and banging heads. Military authorities in the United States, meanwhile, saw the risks of brain damage from repeated concussions in sparring and boxing matches, recognizing “dementia pugilistica” in 1938.⁹ These bodies of research on single and repeated exposures became increasingly connected in the 1940s.

And so, by the 1950s, the dangers of repeated concussion were medically

well understood, even if this was not always true among those groups of the public most likely to experience them. In a provocative polemic address in 1951, prominent American neurologist Frederic Gibbs deplored the public’s ignorance of the risks of repeated concussions.¹⁰ Augustus Thorndike, chief of surgery at Harvard and a founder of the field of sports medicine, declared, in light of the pathology of the punch-drunk boxer, that the risks of three concussions were too many to indicate *any* further play in sport. Awareness of this sporting context in particular can be surmised from a 1958 paper on helmet design, in which A.G. Gross further characterized subconcussive and concussive blows to the head as causes of chronic traumatic encephalopathy.¹¹

A series of examples of clinical research published in the 1950s highlights the synthesizing of not only concussion with chronic traumatic encephalopathy, but also with underlying changes in brain pathology. In 1955, Elisha Gurdjian and colleagues noted cellular changes caused by subconcussive blows,¹² a point Gurdjian and Webster echoed in their 1958 classic *Head injuries: mechanism, diagnosis and management*, in which they described postsubconcussion syndrome (a term they deployed for the sequelae of head injury that had not necessarily caused temporary blackout and memory loss) as “probably the result of functional and of organic cerebral disturbance due to a subconcussive injury.” When, in 1957, neurologist Macdonald Critchley characterized the tangle pathology of chronic traumatic encephalopathy seen in boxers,¹³ it appears that he thus only delineated an injury that boxers (with their repeated head injuries) made particularly apparent in postmortem examination.

In the English-speaking world, these descriptions set off controversies about amateur and professional boxing. Yet many of the boxing medical authorities accepted the reality of the dangers from blows to head. G.H. Graham, chief medical officer to the British Boxing Board of Control, vociferously defended the sport’s safety in Britain and declared the punch-drunk boxer a creature of the past. He

appeared to have accepted as uncontroversial the dangers of traumatic encephalopathy, recognizing (and potentially shifting blame onto) a broader arena of sports scenarios that might cause its appearance:

Irreversible brain damage caused by regular excessive punching can cause a boxer to become punch drunk, a condition known euphemistically in medical terms as Traumatic Encephalopathy. The condition can be caused by other hazards of contact sports — taking too many falls while hunting or steeple chasing or the continual use of brute force rather than skill in the rugby field or heading a football incessantly over many years. Anything which entails intermittent trauma to the head can cause it.¹⁴

By the 1960s and 1970s, concerns about single and repeated concussions would inform the design of safer automobiles, and result in epidemiological studies of head injuries and the elaboration of concussion guidelines for contact sports. For, however much Saucier’s paper had desired a shift in terminology, it had acknowledged the descriptive purchase that “concussion” maintained on the public; concussion lasted as the preferred descriptor after the introduction of the Glasgow Coma Scale in 1974 and the 1993 formal definition for mild traumatic brain injury.

In Saucier’s hands, traumatic encephalopathy offered a material view of the function of the brain. Functional disturbance meant a (potentially) reversible disruption of physiologic patterns. Traumatic injury, meanwhile, spoke to an area of diminished resistance, a location in the brain where a nascent pathological process had begun, perhaps to become an inexorable cascade into degeneration. In mid-20th-century US and beyond, Saucier and others’ conceptions of these physical and organic changes had established a presence in medicine and industry, and across sports professions. Chronic traumatic encephalopathy was a condition to be avoided — and to do so would require better safety and the reduced risk of hits to the head.

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A full bibliography is available upon request from the author.

References

1. Saucier J. Concussion: a misnomer. *Can Med Assoc J* 1955;72:816-20.
2. Proctor RN. *Golden holocaust: origins of the cigarette catastrophe and the case for abolition*. Berkeley (CA) and London (UK): University of California Press; 2012:19.
3. Hale NG Jr. *Freud and the Americans: the beginnings of psychoanalysis in the United States, 1876–1917*. Oxford (UK): Oxford University Press; 1971.
4. Young A. *The harmony of illusions: inventing post-traumatic stress disorder*. Princeton (NJ): Princeton University Press; 1997.
5. Kroker K. Configuring epidemic encephalitis as a national and international concern. In: Casper ST, Gavrus D, editors. *The History of the Brain and Mind Sciences: Technique, Technology, and Therapy*. Rochester (NY): University of Rochester Press; 2017.
6. Osnato M, Giliberti V. Postconcussion neurosis — traumatic encephalitis: a conception of postconcussion phenomena. *AMA Arch Neurol Psychiatry* 1927;18:181-214.
7. Martland HS. Punch drunk. *JAMA* 1928;91:1103-7.
8. Strauss I, Savitsky N. Head injury: neurologic and psychiatric aspects. *AMA Arch Neurol Psychiatry* 1934;31:893-955.
9. Through the editor's specs: punch drunk. *Nation's Business* February 1938;26:7.
10. Gibbs FA. The most important things. *Am J Public Health Nations Health* 1951;41:1503-8.
11. Gross AG. A new theory on the dynamics of brain concussion and brain injury. *J Neurosurg* 1958;15:548-61.
12. Gurdjian ES, Webster JE, Lissner HR. Observations on the mechanism of brain concussion, contusion, and laceration. *Surg Gynecol Obstet* 1955; 101:680-90.
13. Critchley M. Medical aspects of boxing, particularly from a neurological standpoint. *BMJ* 1957;1:357-62.
14. Graham JW. *Eight, nine, out! Fifty years as boxer's doctor*. Manchester (UK): Protel; 1975:56.