UNITED STATES DISTRICT COURT

DISTRICT OF MINNESOTA

In re NATIONAL HOCKEY LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION This Document Relates To: ALL ACTIONS.	MDL No. 14-2551 (SRN/JSM) DECLARATION OF STEPHEN T. CASPER, PH.D

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I. QUALIFICATIONS

1. My name is Dr. Stephen T. Casper. I am a tenured Associate Professor of History in Clarkson University's Department of Humanities and Social Sciences. By training, I am an expert in the history of modern medicine and science.

2. I hold a Bachelors of Science degree in neuroscience and biochemistry from the University of Minnesota, where I also briefly worked in the Department of Neurology as a Junior Scientist in an Alzheimer's Research Laboratory before beginning my doctoral training in the history of medicine and science.

3. I earned my PhD from University College London in 2006. I was awarded a fellowship covering tuition, travel, books, and living expenses for the entirety of my doctoral training, which came from an award jointly funded by the Wellcome Centre for the History of Medicine and the Association of British Neurologists. I have also held, respectively, visiting scholar and fellowship positions in the University of Cambridge's Department of History and Philosophy of Science, and at the Rockefeller Archive Center in Sleepy Hollow, NY.

4. My expertise is the history of neurology, physiology, neuroscience, and psychiatry. I am also regarded as an authority on the specialization of medicine in history and the historiography of 'the patient' in medicine.

5. I have written and published a scholarly monograph focused on the history of British neurology entitled *The Neurologists: A History of a Medical Speciality in Modern Britain, c.1789-2000*. I also co-edited with L. Stephen Jacyna the book, *The Neurological Patient in History*. I have a second edited volume forthcoming with the University of Rochester Press titled *The History of the Mind and Brain Sciences: Technique, Technology, and Therapy*. I have published peer-reviewed articles, essays, and book reviews in premier history of medicine and history of science journals, including *Bulletin of the History of Medicine*, *Medical History*, *Isis*, and *Science in Context*. I have also published in scientific and clinical journals, including *Science*, *Brain: A Journal of Neurology*, and the *British Medical Journal*.

6. My published scholarship has been recognized with numerous invited speaking opportunities at prestigious academic institutions, including Columbia University, New York University, Princeton University, and the Max Planck Institute for the History of Science. I have also presented my research at prestigious national and international academic conferences focused on the history of science, medicine, and neuroscience. My scholarship in the history of neurology has also been recognized by invitations to speak to neurologists and psychiatrists: I have, for instance, given Neurology Grand Rounds at Columbia University and The Ohio State University. I have been an invited lecturer to the Association of British Neurologists and the XII World Congress of Neurology.

7. My service record in the history of medicine is extensive. I am an active member of the American Association for the History of Medicine, where I have served in the past by invitation as a member of prize and program committees. I was recently elected to the Executive Council of that organization. For three years I was Media Reviews editor for the journal *Medical History*, and I am routinely asked to offer expert review of manuscripts that have been submitted to academic journals and book publishers.

8. At my home institution, Clarkson University, I teach courses on such topics as the history of medicine, the history of the human sciences, neuroscience and society, and European history in the nineteenth century. I have won two teaching awards for these efforts. I was also the first member of my department to receive the Clarkson University's Researcher of the Year Award, the most prestigious award granted at Clarkson University and given to one member of the faculty only each year.

9. I am charging the plaintiffs $300 per hour for all of my services, except for my research, which is being billed at a rate of $220 per hour, and non-working travel time, which is being billed at $150 per hour. I have charged the plaintiffs a total of $253,285.00 to date for my work. Within the preceding four years, I have not testified as an expert at trial or by deposition in any case. Attached as **Exhibit A** is a list of materials reviewed in preparing this declaration. A list of all publications I have

authored in the previous 10 years is included in my Curriculum Vitae attached to this declaration at **Exhibit B**.

II. SCOPE OF OPINIONS

10. I was retained by the plaintiffs in this litigation to undertake a historical analysis of developments in scientific and medical knowledge relating to (i) head trauma, concussions, and subconcussive blows; (ii) treatment of head trauma and concussions; and (iii) the association between head trauma, concussions, and subconcussive blows and prolonged or permanent symptoms and permanent neurological conditions. I was also asked to look at whether, and, if so, specifically what time, there was any reliable scientific and medical evidence that concussions and subconcussive blows posed a serious risk to individuals, including, but not limited to those playing professional contact sports, of prolonged or permanent symptoms or permanent neurological conditions. I also looked at the issue of how evolving knowledge, including evolving nomenclature, was utilized by the medical and scientific community, as well as the extent and nature of any dissent in the literature.

III. SUMMARY OF OPINIONS

A. Concussions in the Historical Record

11. The historical record presents a very clear picture of the cause, effects, and resulting associations with neurological conditions of both single and repeated concussive injuries.

12. The definition of concussion has evolved historically, but in ways that "naturally extend definitions" that were first made in the nineteenth century. The family resemblance between historically changing definitions is very strong. The historical differences in the definition of concussion are insignificant in terms of how concussions were historically managed or treated. Historical management guidelines have clearly indicated that a precautionary approach should be taken to avoid concussion.

13. The historical record indicates that since at least 1936, the terms "traumatic encephalopathy," "chronic traumatic encephalopathy," "punch drunk,"

"punchy," "goofy," "slap happy," "cutting paper dolls," or "slug nutty," and "traumatic dementia" have all referred to the same condition.

14. The historical record indicates no meaningful distinction between blows to the head suffered in one context versus another. The risk of sequelae indicated in the historical record is not dependent upon whether a blow to the head was suffered during a hockey game, football game, boxing match, car accident, physical assault, domestic battery, military exercise, combat, or any other context.

15. The historical record of science and medicine records the association between concussive and subconcussive blows to the head and the risk of negative, long term neurological effects since the nineteenth century, and since the nineteenth century medical authorities have observed that repeated blows to the head heighten risks of those effects.

16. The historical record indicates that for more than a century scientists and clinicians have formulated scientific hypotheses about concussions and found them to be in some instances true and in other instances false. The clear tendency has been towards increasingly refined explanations for the pathological consequences of concussive and subconcussive blows to the head as a cause for the resulting sequelae of those injuries, both in their immediate and also in their long-term effects.

B. Concussions in Sports

17. The historical record indicates that head injuries in hockey have been common since the 1920s, and that hockey players receive head injuries in a variety of ways.

18. The historical record clearly indicates from the 1920s to the present that a concussed patient – athlete or not – should be allowed a significant period of recovery from such an injury.

19. Since 1928, there has been a clear association in the historical record between repeated blows to the head in sports and pathological changes leading to long term, permanent, life-altering and sometimes degenerative personality changes, dementia, movement disorders, and disability.

20. As of 1936, the historical evidence in medical sources indicates that informed doctors could have warned contact sports players of the potentially dangerous and lasting effects posed by head injuries, concussions, and knockouts. As of 1952, the medical sources indicate that informed doctors concerned with recurrent injuries in athletes could have informed athletes who sustained three or more concussions that clinical experience warranted the cessation of play altogether to avoid recurrent injuries and the risks of traumatic encephalopathy.

21. By at least 1975, with an article published in *The Lancet*, the historical record indicates that doctors treating athletes had been advised that they had a duty to convince controlling bodies and participants in sports where concussion was frequent that the effects of concussive injuries were cumulative and that the casual acceptance of concussive injury was dangerous because concussion diminished intellectual performance.

C. Controversies Over the Danger and Management of Concussion Injuries

22. Historically, there have been narrow scientific debates about the precise manner that concussion damaged the brain, but such scientific debates over why concussions produced their observed effects have rarely influenced precautionary clinical management principles and decisions about its treatment.

23. Policy debates and litigation involving large corporate enterprises and governing institutions being liable to individuals with head injuries have routinely been the driver of controversies around concussions. For example, railroad companies, insurance companies, workman's compensation cases, military pensions, motor vehicle accidents, and professional sports franchises have all challenged the validity of claims to disability made by individuals with head injuries.

24. The historical medical and scientific record shows some evidence of controversies surrounding specific aspects of the management and danger of concussion injuries, but the record indicates a decades-long broad consensus across many medical specialties that concussions were dangerous injuries, that even a single concussion in

rare instances could result in irreparable damage, that symptoms of concussion could become permanent, and that repeated concussions, in particular, were liable to increase this risk of permanent side-effects or neurodegenerative disease.

25. The historical record is clear that voices of dissent that cast doubt on the danger and risk of concussive head injuries are rare, but frequently cited. This is likely due to the careful practice of researchers seeking to present both sides of the story in their own work and having only a small sample of dissenting citations to choose from. This would not, however, inherently lend additional credence to their findings.

26. Regarding challenges to liability from an individual asserting long term effects of concussion, a few individual patients with purported head injuries throughout the last century were found to be malingering (faking or overstating effects for financial gain). However, there is no evidence in the historical record that any group of similarly situated individuals (within a common industry, for example) have ever colluded en masse to malinger in order to obtain compensation.

27. There is substantial evidence in the historical record of concussed individuals receiving compensation for the effects of concussion injuries, including their permanent effects.

28. Direct and circumstantial evidence in the historical record indicates that from the 1990s to the present, there has been an intense disinformation campaign seeking to minimize the risks posed by concussion injuries. These efforts have mainly been led by doctors, engineers, and psychologists working for the National Football League, but not exclusively for that organization.

IV. METHODOLOGY

A. Historical Methods

29. This declaration was prepared using standard historical methods.[1] Keywords, subject-categories, chronology and primary sources were chosen with care and in a manner conforming to the American Historical Association's guidelines for

[1] Ludmilla Jordonova, *History in Practice* (2000).

historical practice.[2] All sources were documented with care.[3] All charts were prepared in a way permitting each data-point to be traceable back to an original reference.

B. Potential sources of bias

30. The plaintiffs' attorneys initially provided me with a short bibliography of historical sources they had identified. I did look at their bibliography, but I ignored it because it was too decontextualized to be of much use to an historian. It is included in **Exhibit C** for comparison with my own historical bibliography.

C. Libraries and archives

31. For this declaration, I relied upon books, journals, technical reports, and miscellaneous published sources available in the Countway Medical Library at Harvard University (Cambridge MA), the Wellcome Medical History Library (London, UK), the Cambridge University Library System (Cambridge, UK), the British Library, and the Clarkson University Library System. I also made extensive use of online access at home provided by those libraries to full-text searchable journals collections. I made extensive use of Clarkson University's interlibrary loan system. I also made great use of my own professional library.

D. Timespan

32. I was asked initially to look at the period from the nineteenth century through the 1980s. It was on my advice that this study began in the nineteenth century and concluded in 1980. I was subsequently asked to bring the bibliographic endpoint forward to 2005, and then to 2011. These changes in end point fall within the range of

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[2] American Historical Association, *Statement of Standards of Professional Conduction*, https://www.historians.org/jobs-and-professional-development/statements-and-standards-of-the-profession/statement-on-standards-of-professional-conduct (last visited Aug. 24, 2016).

[3] Not only is documenting sources essential, it is essential to think about the way the sources themselves have changed and the ways that documenting sources has changed. Works doing that and which inform this study include: Anthony Grafton, *The Footnote: A Curious History* (1997) (for the footnote), Adrian Johns, *The Nature of the Book: Print Knowledge in the Making* (1998) (for the book), and Marina Frasca-Spada & Nick Jardine (eds) *Books and the Sciences in History* (2000) (for books in the sciences).

my research and teaching expertise, *i.e*. modern, post-war, and contemporary medicine and science.

33. Periodization is a common practice in historical scholarship. For the purposes of this study, "modern medical history" is from 1789 to 1945, "post-war medicine" is 1945 to 1989, and "contemporary medicine" is 1989 to the present.[4]

E. General historical method

34. The majority of the historical research focused on developing a primary source chronology of the history of concussion and the sequelae of concussion from medical and scientific journals, monographs, and technical reports. This work can be identified in a tradition of historiography called intellectual history.[5] It required the identification of historically appropriate key words and subject areas across the period.[6] It also required the identification of historically appropriate primary sources. Those sources should have been reasonably accessible and reasonably retrievable to any clinician or scientist in any period with an interest in understanding, or a practice treating, head injuries. Editorials, letters to the editor, scientific and clinical reviews published in the medical and clinical literature might well have reached a wide audience as well and these were included as well. All of these primary sources were identified utilizing four methods:

- Keywords or subject areas were identified using *Index Medicus* and the *Surgeons General Catalogue of the United States*;
- Literature was identified in *Index Medicus* and the *Surgeons General's Catalogue of the United States*;
- Online full-text journals and database archives were searched using historically appropriate keywords and subject categories; and
- Citations by historical actors to other publications were used to identify additional primary sources.

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[4] Jordonova, *supra* n.1, at 114-141.

[5] A major landmark collection of essays that defined modern interpretations of the intellectual historiography of medicine was Owsei Temkin, *The double face of Janus and other essays in the history of medicine* (1977).

[6] *See* Table 1.

35. All primary sources that were read were transferred into a chronologically organized annotated bibliography from 1871 to 2011. The vast majority of primary sources were collected *before* they were read ensuring that I was blind to the content of most sources beyond the title and subject and keyword specificity.

36. It is important to note that the primary sources used in this study are a representative snapshot of the published literature on the topic *and not a complete source archive*. Undoubtedly there is literature missing, and likely for every year of this study. This fact would be true of any work of historical scholarship. That said, so far as I have identified, this study represents as comprehensive an intellectual history of the subject as has ever been undertaken by any historian or clinician for the modern, post-war, and contemporary period of medicine and science.

F. Generating keywords and subject-classifications

37. The list of keywords and subject classifications used in this study appears in Table 1 and Table 2. The sources used all appear in the bibliography. The constancy of the word concussion made it a particularly powerful keyword in this study.

38. Medical historians have long observed that medical nomenclature varies across time.[7] Thus developing an extensive list of keywords and subjects suited to past times and past places is essential. For this purpose, I first consulted general reference sources readily available to past medical practitioners and scientists. The sources I used were in no way special and they (or sources like them), would have been readily available to past clinicians and scientists. They included medical dictionaries, thesauruses, encyclopedias, and bibliographic resources published across the 19th and 20th centuries. I judged that entries in these sources would provide a clear, unprejudiced means of assessing actors' categories and definitions as they would have appeared in other published sources in the past. Often these were words that appeared in bold in these texts.

[7] It is this theme that captures the meaning of the term "framing" as it is used throughout Charles E. Rosenberg & Janet Lynne Golden, *Framing Disease: Studies in Cultural History* (1992).

39. The medical dictionaries and thesauruses reflected a broad pattern: in the nineteenth century they routinely adopted the very broad subject category: head injury. Within those entries, discussions of concussion, skull fracture, subdural hematoma, traumatic insanity, and other similar such conditions appeared with great frequency. Over the course of the pre-World War II period, dictionaries increasingly narrowed their focus. Eventually brain injuries began to appear, and that pattern continued into the contemporary period. In other words, in the nineteenth century, medical generalists and general practitioners described medical conditions in general terms. In the twentieth century, medical specialists described medical conditions in specialist terms. Some words were used with ubiquity throughout the whole period, of which the most obvious were concussion, contusion, and laceration, although the definitions of those terms doubtlessly did vary slightly across sources from different periods and different geographies. All of the dictionaries referred to in framing this study appear in the bibliography.

40. Encyclopedias published in different decades also reflected these trends. However, because encyclopedias by their nature contained longer entries than dictionaries, the keywords and subject categories identified were more extensive. They also sometimes referenced published literature in medical journals. Moreover, these sources observably spoke to matters of concern in the time that they were written. These sources provided me with a broad view of historical debates that shaped medical understanding of head injuries. All of the encyclopedias referred to in framing this study appear in the bibliography.

Table 1. Historical keywords & subject classifications in the *Surgeon's General Catalogue of the United States*

Series I 1881-1893	**Series II 1897-1913**	**Series III 1922-1931**	**Series IV 1936-1948***
Brain *concussion and compression of*	Brain *concussions etc., of*	Brain *concussions of*	Brain concussion
Concussion	*Concussion*	Concussion	Brain concussion, *diagnosis*
Contre-coup	Contre-coup	Contre-coup	Brain concussion, *pathology*
Head *Injuries* of	Head *injuries of*	Head *injuries of*	Brain concussion, *Sequelae*
Head *Injuries of, Cases of*	Head, *injuries of, Cases & Statistics of*	Head *injuries of, Complications and sequelae of*	Brain concussion, *treatment*
Head *Injuries of, Complications of*	Head, *injuries of, Complications and sequelae of*	Head *injuries of, complications and sequelae of, Mental*	
Head *Injuries of, Sequelae of*	Insanity *Traumatic*	Insanity Traumatic	
Insanity *Causes of, Traumatic*	Neuroses *Traumatic*	Neuroses Traumatic	
Railroads *accidents on, and surgery of*	Railroad, *Injuries on and their sequelae*	Neuroses Traumatic, *Diagnosis and semiology of*	
Shock	Railroad, *Injuries on, Jurisprudence of*	Neuroses Traumatic, Treatment of	
Traumatic neuroses and psychoses	Shock	Railroad Accidents on, Prevention of	* Series was incomplete
	Traumatic neuroses	Shock	
		Traumatic neuroses	

Table 1. These keywords were generated by following cross-references that appear throughout the *Catalogue* and associated with the words "head" or "concussion". A diligent researcher in these periods would have used these internal cross-references in the same way.

Table 2. Historical keywords and subject classifications found in dictionaries, encyclopedias, published literature and *Index Medicus, 1884-2000*

acceleration-deceleration injury – accident neuroses - boxer's dementia - boxer's traumatic encephalopathy – closed head injury – comotio cerebre - concussion – concutere – contusion - coup-contrecoup - chronic brain syndrome - chronic cerebral injury - chronic traumatic encephalopathy - CTE - dementia pugilistica – encephalopathy - litigation neuroses – mild traumatic brain injury - postconcussion neuroses – postconcussion syndrome - punch drunk - second impact syndrome - traumatic brain injury - traumatic progressive encephalopathy - traumatic insanity - traumatic neuroses

Table 2. These keywords were either identified at the beginning of the study in the work that produced Table 1 or they emerged over the course of the project as a result of the research process.

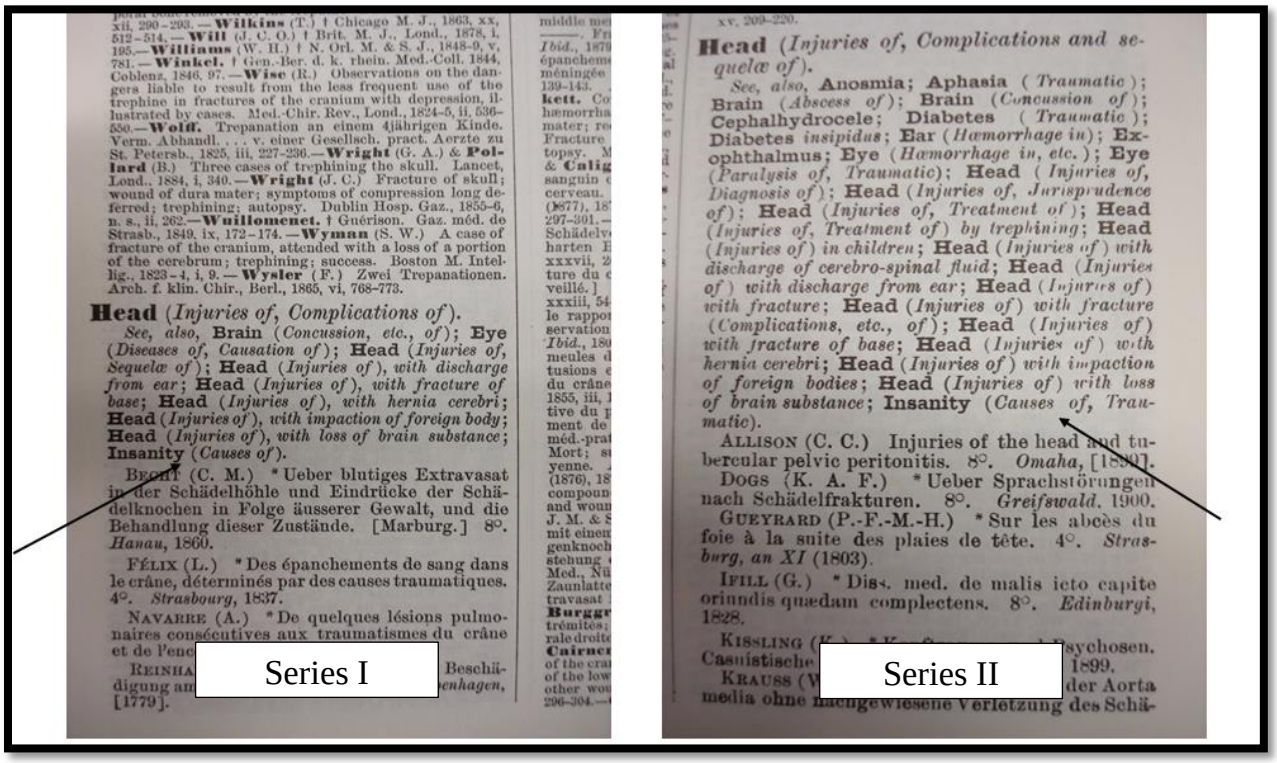

Figure 1 These images reflect typical entries that appear in the US Surgeons General Catalogue in Series I (1884) and Series II (1901). Note the transition in nomenclature (arrows) and internal cross-references.

41. By their nature, medical dictionaries and encyclopedias are ephemeral sources, produced by authoritative authors but updated routinely to reflect new trends. For this reason, these sources were treated more as a general gateway into the past than as a canonical source for keywords and subject-classifications. Having established which keywords and subject categories were clearly significant in those sources, two sources deemed more canonical and rigorous were also consulted. Both sources were

and are widely available and considered definitive medical bibliographies: *Index Medicus* and the *Surgeons General's Catalogue of the United States*.

42. *Figure 1* shows representative differences in entries in the *Surgeons General's Catalogue of the United States*. I looked closely at many such entries. Modest changes in entries were treated as substantive changes worthy of historical attention. For example, in *Figure 1*, Series I published in 1884 "Head, *Injuries of, Complications of)*" appears as a subject classification. Series II published in 1904 has a subject classification called "Head injuries, *complications and sequelae of*." Both entries contain guidance for researchers seeking more precise nomenclature in the form of cross references. Those are keywords. The consistent appearance of the term concussion, for example, suggests basic uniformity of meaning across time. Similarly, the marked change of "Insanity, *causes of*" in Series I to "Insanity, *causes of traumatic*" is also a striking change in keywords.[8]

43. Both sources provide a comprehensive list of most publications that appeared on medical topics across the late nineteenth to the mid-twentieth century. Both organized that literature by subject and keyword. *Index Medicus* is more comprehensive, and includes letters to the editor and other non-research oriented materials. The editors of the *Surgeons General Catalogue* are known to have been more measured in their judgment when recording important literature, and they listed only research articles, medical books, and medical theses in what is now the National Library of Medicine in Bethesda. *Index Medicus* was published yearly with few exceptions. *The Surgeons General Catalogue* appeared every decade. Both sources were used to identify

[8] It is important to note that some authorities expressed misgivings about the inconstancy of medical keywords as reflected in such canonical works. But historians have been able to use the inconstancy of those keywords and subject classifications to study changing definitions, emerging diseases, and changes in the social structure of medicine. On such misgivings, *see* S.V. Larkey, *The Army Medical Library Research Project at the Welch Medical Library*, 37 BULL. MED. LIB. ASS'N 2, 121-24 (1949); for a comparison of the canonical sources, *see* S.J. Greenberg, *The great contribution: Index Medicus, Index-Catalogue, and IndexCat*, 97 J.MED. LIB. ASS'N, 2, 108-13 (2009).

further keywords and subject categories (and changes in nomenclature overtime were noted as well).

G. Selection of journals

44. All journal sources used in this study appear in the bibliography. Table 3 provides a year by year picture of the number of sources from journals that were consulted. Articles, essays, editorials, reviews, lectures, and miscellaneous items, including a few newspaper articles, were included in this study.

45. It was obvious that the journals grounding this study should be sufficiently general as to have been easily accessible to past clinicians. For this purpose, four journals were selected which can be called canonical and representative: *The Lancet*, *The British Medical Journal*, *Journal of the American Medical Association*, and the *New England Journal of Medicine*, which was originally called the *Boston Medical and Surgical Journal*. All of these journals are now online and full-text searchable. Articles, editorials, published lectures, reviews, and letters to the editor in these journals were selected and read for each year for the entirety of the period from their origins in the nineteenth century to 2011. In some years, the amount of literature in these journals was so extensive it became necessary to sample. In these moments original research and reviews were prioritized over shorter notices. Also prioritized were articles with keywords in the title. All of these works are cited in my annotated bibliography of primary sources.

46. Specialist journals became vehicles for the publication of original research in the twentieth century. I deemed *Brain – A Journal of Neurology*, *American Journal of Psychiatry*, *Archives of Neurology and Psychiatry*, *Journal of Clinical Neurosurgery*, *Journal of Clinical Neuropsychology*, and the *British Journal of Sports Medicine* representative specialist outlets. These journals are also online and full-text searchable. Again the amount of literature in some years became unwieldy. In these moments original contributions and reviews were prioritized, along with articles with keywords in the title. All of these works are cited in the bibliography of primary sources.

47. The remaining literature in this study was chiefly identified through bibliographic cross-reference with the literature cited in the papers. There were some exceptions. It was observable, for example, that publications in journals devoted to such new specialties as emergency medicine began to appear in the 1970s and 1980s. Public health journals also began to reflect on head injuries in the same period. In this way, new disciplinary fields opened up requiring broader engagement with the subject. When possible, full-text searchable databases for those new specialist journals were treated as consistently as possible with the pattern of previous work.

1871	1872	1873	1874	1875	1876	1877	1878	1879	1880	1881	1882	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900	1901	1902	1903	1904	1905
A1871	A1872	A1873	A1874	A1875		A1877					A1882	A1883		A1885	A1886	A1887	A1888	A1889	A1890	A1891	A1892	A1893	A1894	A1895	A1896	A1897	A1898	A1899	A1900	A1901	A1902	A1903	A1904	A1905
	B1872											B1883		B1885	B1886			B1889	B1890	B1891	B1892	B1893	B1894				B1898	B1899	B1900	B1901	B1902	B1903	B1904	B1905
	C1872														C1886			C1889	C1890	C1891	C1892	C1893	C1894				C1898		C1900	C1901	C1902	C1903	C1904	C1905
	D1872																		D1890	D1891	D1892	D1893	D1894						D1900			D1903	D1904	D1905
																			E1890	E1891	E1892	E1893	E1894						E1900			E1903	E1904	
																			F1890	F1891	F1892	F1893										F1903		
																					G1892	G1893										G1903		
																					H1892													
																					I1892													
																					J1892													

1906	1907	1908	1909	1910	1911	1912	1913	1914	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928	1929	1930	1931	1932	1933	1934	1935	1936	1937	1938	1939	1940
A1906	A1907	A1908	A1909	A1910	A1911	A1912	A1913	A1914	A1915	A1916	A1917	A1918	A1919	A1920	A1921	A1922	A1923	A1924	A1925	A1926	A1927	A1928	A1929	A1930	A1931	A1932	A1933	A1934	A1935	A1936	A1937	A1938	A1939	A1940
B1906			B1909	B1910	B1911		B1913	B1914	B1915	B1916	B1917	B1918	B1919	B1920		B1922	B1923	B1924	B1925	B1926	B1927	B1928	B1929	B1930	B1931	B1932	B1933	B1934	B1935	B1936	B1937	B1938	B1939	B1940
C1906			C1909	C1910	C1911		C1913	C1914	C1915	C1916		C1918	C1919	C1920				C1924	C1925	C1926	C1927	C1928	C1929	C1930		C1932	C1933	C1934	C1935	C1936	C1937	C1938	C1939	C1940
D1906				D1910	D1911			D1914	D1915	D1916		D1918	D1919					D1924			D1927	D1928	D1929	D1930			D1933	D1934	D1935		D1937		D1939	D1940
									E1915	E1916		E1918	E1919					E1924				E1928		E1930			E1933	E1934	E1935		E1937		E1939	E1940
									F1915	F1916		F1918						F1924				F1928		F1930			F1933	F1934	F1935					F1940
									G1915	G1916		G1918										G1928		G1930				G1934	G1935					G1940
									H1915	H1916		H1918										H1928						H1934	H1935					
										I1916												I1928						I1934	I1935					
										J1916												J1928							J1935					
										K1916												K1928							K1935					
										L1916												L1928												

1941	1942	1943	1944	1945	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975
A1941	A1942	A1943	A1944	A1945	A1946	A1947	A1948	A1949	A1950	A1951	A1952	A1953	A1954	A1955	A1956	A1957	A1958	A1959	A1960	A1961	A1962	A1963	A1964	A1965	A1966	A1967	A1968	A1969	A1970	A1971	A1972	A1973	A1974	A1975
B1941	B1942	B1943	B1944	B1945	B1946	B1947	B1948	B1949	B1950	B1951	B1952	B1953	B1954	B1955		B1957	B1958	B1959	B1960	B1961	B1962	B1963		B1965	B1966	B1967	B1968	B1969	B1970	B1971	B1972	B1973	B1974	B1975
C1941	C1942	C1943	C1944	C1945	C1946		C1948			C1951		C1953	C1954	C1955		C1957	C1958		C1960		C1962	C1963		C1965	C1966	C1967		C1969	C1970		C1972	C1973	C1974	C1975
D1941	D1942	D1943	D1944	D1945	D1946		D1948			D1951		D1953	D1954	D1955		D1957	D1958		D1960		D1962				D1966	D1967		D1969	D1970		D1972	D1973	D1974	D1975
E1941	E1942	E1943	E1944	E1945	E1946		E1948			E1951		E1953	E1954	E1955		E1957	E1958		E1960		E1962				E1966	E1967			E1970		E1972	E1973	E1974	E1975
F1941	F1942	F1943	F1944				F1948			F1951		F1953	F1954	F1955											F1966	F1967					F1972	F1973	F1974	F1975
G1941	G1942		G1944									G1953		G1955											G1966						G1972	G1973	G1974	G1974
H1941	H1942		H1944									H1953													H1966							H1973	H1974	
I1941	I1942		I1944									I1953																					I1974	
J1941	J1942		J1944																															
K1941			K1944																															
			L1944																															

1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011
A1976	A1977	A1978	A1979	A1980	A1981	A1982	A1983	A1984	A1985	A1986	A1987	A1988	A1989	A1990	A1991	A1992	A1993	A1994	A1995	A1996	A1997	A1998	A1999	A2000	A2001	A2002	A2003	A2004	A2005	A2006	A2007	A2008	A2009	A2010	A2011
B1976	B1977	B1978	B1979	B1980	B1981	B1982	B1983	B1984	B1985	B1986	B1987	B1988	B1989	B1990	B1991	B1992	B1993	B1994	B1995	B1996	B1997	B1998	B1999	B2000	B2001	B2002	B2003	B2004	B2005	B2006	B2007		B2009	B2010	B2011
C1976	C1977	C1978	C1979	C1980	C1981	C1982	C1983	C1984	C1985	C1986	C1987	C1988	C1989	C1990	C1991	C1992	C1993	C1994	C1995	C1996	C1997	C1998	C1999	C2000	C2001	C2002	C2003	C2004	C2005	C2006	C2007		C2009	C2010	C2011
D1976		D1978	D1979	D1980	D1981	D1982	D1983	D1984	D1985	D1986	D1987	D1988	D1989	D1990	D1991		D1993		D1995	D1996	D1997	D1998	D1999	D2000	D2001	D2002	D2003	D2004	D2005	D2006	D2007		D2009	D2010	D2011
E1976				E1980	E1981	E1982	E1983	E1984	E1985	E1986		E1988	E1989	E1990	E1991		E1993		E1995	E1996	E1998	E1998	E1999	E2000	E2001	E2002	E2003	E2004	E2005	E2006	E2007		E2009	E2010	E2011
F1976				F1980	F1981	F1982	F1983	F1984		F1986		F1988	F1989	F1990	F1991		F1993		F1995	F1996	F1997	F1998	F1999	F2000	F2001	F2002	F2003		F2005	F2006			F2009	F2010	F2011
				G1980		G1982	G1983	G1984		G1986		G1988	G1989		G1991		G1993		G1995	G1996	G1997	G1998	G1999	G2000	G2001	G2002	G2003		G2005	G2006			G2009	G2010	G2011
							H1983	H1984		H1986		H1988							H1995	H1996	H1997	H1998	H1999	H2000	H2001		H2003		H2005	H2006				H2010	H2011
								I1984													I1997	I1998	I1999	I2000	I2001		I2003		I2005					I2010	I2011
																						J1998	J1999		J2001		J2003		J2005					J2010	J2011
																						K1998					K2003								K2011
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Table 3. Peer-reviewed articles, reviews, editorials, letters to the editor, and miscellaneous works, including a few newspaper articles, analyzed in this declaration appearing in scientific, medical, and technical literature published between 1871 and 2011 (n = 701). See Exhibit D for a bibliography of these works specifically.

H. Selection of other published primary sources

48. The primary sources used in this study appear in the bibliography, **Exhibit A**.

49. In the course of this work, numerous textbooks on surgery, neurology, and psychiatry were surveyed in both the Countway Library at Harvard and at the Wellcome Library for the History of Medicine in London. The advantage that these collections offered was that their open stacks included multiple editions of textbooks. The table of contents and the indexes of those sources were read with a focus on closed head injuries, concussions, contusions, and lacerations. Textbooks by-and-large reflected trends in the literature, although there tended to be some delay in the updating of these sources in new editions, as would be expected.

50. There were fewer monographs on head injury available to review, and most were published after 1970. Those identified were purchased and read. With few exceptions, there is little evidence that these sources provided new, novel interpretations of closed head injuries, concussions, and head injury sequelae at any point in time. Although sometimes useful sources, they appear to have been intended as longer reviewers for graduate students and clinicians looking to learn about research in this area. This view was echoed in the reviews of the books that were published in the scientific and clinical literature.

51. There were numerous other sources that could provide broad insight about head injury diagnoses and prognoses. Among these, the *Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death*, which was published in multiple editions across the twentieth century, seemed especially significant.

I. Selection of social and cultural sources

52. These primary sources are all cited in the bibliography of primary sources.

53. Although this study focuses mainly on published scientific and clinical literature, historical newspaper databases available at Clarkson University and Harvard University were also consulted to study head injuries and contact sports. The original

keywords were "'head injury' AND 'hockey.'" A chronology of reported head injury cases sustained in professional hockey from 1920 through to 1980 was developed from these sources. Newspaper reports after 1980 were so numerous that two refined keywords were introduced: "loss of consciousness" or "unconsciousness" was tied with "hockey." In a more haphazard fashion a search was undertaken for the use of "punch-drunk" in historical newspaper databases. The search through these sources was in no way as systematic as the study of the peer-reviewed literature. That said, the materials identified were acquired in ways appropriate to historical practice.

J. Selection of secondary literature

54. All secondary literature used in this study appears in the bibliography.

55. Historians cite historical literature that has shaped their views of the past. I have read much secondary literature in the course of this study. I also brought a deep understanding of the secondary literature on the history of neurology, physiology, psychiatry, and psychology to this project that has been shaped by my extensive experience as an historian. In addition to those subjects, I have also a deeply grounded understanding of the specialization of medicine, the historiography of the patient in modern medicine, the history of the human sciences, and a professorial understanding of modern medicine and biology cultivated in the course of writing and preparing undergraduate lectures. I endeavor throughout this declaration to show the interpretations upon which I am drawing.

K. Exclusions

56. It is known that past physicians and scientists read scientific and medical literature published in non-English sources. It is also clear that there were studies published in German, French, Italian, and Spanish which might well have added further and important information to this one. However, I have excluded all non-English sources. I do not read any of these languages well enough to be able to capture the proper contextual meaning of scientific and medical jargon. Moreover, I assume in this study that physicians and scientists in North America and Britain gravitated towards English-language sources across the whole period. There is evidence supporting this

view, since most authors in this study appear to have cited English language literature only.

57. That said, there are some discernable patterns. When authors cited literature in languages other than English, then they tended to cite mainly French and German contributions. These citations were usually to publications that appeared in the nineteenth century. There were exceptions to this rule. For example, an important, much-cited essay by Brandenburg and Hallervorden appeared in 1954.[9] This essay focused on the pathology of dementia pugilistica as it had been observed in autopsies of boxers. The authors of this article appear to suggest that the cause was repeated blows to the head [die wiederholten Koftraume zurückzuführen] and authors cited this work occasionally in medicine's post-war period.[10] For consistency, however, this study was excluded from the historical analysis.

58. I was provided upon my request with some case materials by the plantiffs' attorneys, including NHL concussion protocols and a list of physicians and psychologists who were known to have consulted with the NHL. In general, however, I have worked in isolation from NHL records.

L. Organization of the Declaration

59. At the outset, this declaration provides a basic overview of the history of historic medical changes as they related to concussion research and puts such changes in brief context of the medical and scientific sphere at the time of their development. It also considers the way that the history of concussion has previously been written about by other authors, including popular writers, historians and by doctors and physicians looking back at the history of their field.

60. After establishing these basic historical and contextual parameters in the history of concussion research and its interpretations, this declaration presents detailed

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[9] W. Brandenburg & J. Hallervorden, *Dementia pugilistica mit anatomischem Befund*, 325 VIRCHOWS ARCHIV FÜR PATHOLOGISCHE ANATOMIE UND PHYSIOLOGIE UND FÜR KLINISCHE MEDIZIN 6, 680-709 (1954).

[10] *Id.* at 706.

findings on the state of concussion knowledge and research from 1871 arranged under several thematic headings. Each of these sections address a fundamental question; does the historical record taken as a whole suggest that physicians and scientists have known that head trauma, concussions, or subconcussive blows could pose a serious risk to individuals, including workers in professional sports industries? Combined, and as explored *infra*, they provide abundant evidence that the answer to that question is yes.

V. ANALYSIS OF THE HISTORICAL RECORD

A. Introduction

61. English language medical and scientific literature published or widely available in the United States of America since 1871 shows a clear progression of knowledge, understanding and awareness about concussion and repeated head injury in the medical profession and beyond:

- At the beginning of that period, concussion of the brain was understood to be a dangerous injury of potentially grave long-term consequences, but the medical world lacked a clear account of the physiological, biomechanical, and biochemical processes that explained why it was a dangerous injury.
- Today, the medical record possesses a complex and sophisticated understanding of concussion, including its symptomology, physiology, biochemistry, pathological hallmarks in the short and longer term, as well as an established medical model for what happens to brain tissue, nerves, axons, and glial cells biomechanically during and after concussion.
- Throughout this whole period there were discussions about the dangers of concussions in sporting and other leisure activities and the ways to prevent them from occurring.

62. This declaration provides an evidence-based account of these changes over the last 145 years. It covers:

- When the medical and scientific world began to understand various aspects about concussion;
- The contexts in which these developments took place;
- The nature of consensus and dissent about the findings of various time periods; and
- The expansion and specialization of medical professions and its effect on concussion research.

- Finally, present day, when concussion is researched and advised upon by a broad range of specialists, medical associations, professional sports industries, and governmental organizations.

63. One guiding premise for this study is that it is the nature of science to be a provisional way of knowing.[11] Historians of medicine and science have long shown that scientists seek to discover new knowledge and to improve current knowledge. For this reason, it would be expected to find scientific scholarship calling attention to alternative hypotheses, hypotheses that proved wrong, contradictory data, failed predictions, or other facts that call into question the science (and indeed suspicious to find an absence of these). Scientific progress often reflects back and forth exchanges between scientists over the explanation of data, predictions, and clinical findings, which to lay people might appear to be controversies but are in fact the normal bread and butter of scientific inquiry and expert trust.[12]

64. A second guiding premise for this study is that the provisional nature of medicine, in sharp contrast with biomedical and basic science research, requires clinical judgment and decision-making.[13] This fact can create potential conflicts between scientific findings and the realities of medical practices. For example, it is a relatively challenging matter in evidence based medicine to correct statistically for high incidences of "non-events," meanwhile it is often the rare "events" that create management challenges for healthcare professionals. Given the very high incidence of concussive injuries it is likely that clinicians will encounter over the course of their career the comparatively rare case with, for example, persisting even permanent post-concussion syndrome.[14]

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[11] Thomas S. Kuhn, *The Structure of Scientific Revolutions* (1962).

[12] Steven Shapin, *Social History of Truth: Civility and Science in Seventeenth-Century England* (1994).

[13] John C. Burnham, *Healthcare in America: A History* (2015).

[14] This observation was only made comparatively late in concussion research. Dikmen and colleagues observed that there was: (a) a likely small subset of mild head injury patients with protracted even permanent mild symptoms, (b) that some but not all of

65. Historians of medicine and science have a long scholarly record of explaining and navigating these challenges.[15] They do so by considering individual contributions in the light of the longer historical record and they view that longer record in the light of the social, cultural, political, and geopolitical context. From this view, any primary source has to be seen in the context of the others. Thus, for example, the appearance in the past, present, or future of a single report saying "concussions are dangerous injuries" or, alternatively, "concussions are not dangerous injuries," while perhaps interesting, would not fundamentally transform historical understanding.

66. Historical understanding derives from reviewing a broad collection of representative sources that speak to each other across a whole period and that provide a clear explanatory view of the ways that medical and scientific knowledge has evolved, accumulated, and why. In such a record, the social, cultural, economic, policy, and legal contexts would matter, not because the science produced in those contexts would possess more or less validity, but because those contexts might well explain different anxieties and pressures with which scientists and healthcare professionals in the past (and likely in the present) contended (*see* Figure 2).

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those patients might be explained by other confounding variables like demographics and age, and (c) that those confounding variables might also explain why other researchers fail to observe those patients, hence the difficulties studying them. Essentially when the effects of one mild TBI (on formal cognitive measures for instance) are small and/or also uncommon, then other factors (such as age, education) are also related to such cognitive factors. This creates study design challenges which may simulate or mask TBI effects. S. Dikman et al., *Mild Head Injury: Facts and Artifacts*, 23 J. CLIN. EXP. NEUROPSYCHOL. 6, 729-38 (2001).

[15] The scholarship has reached a point of maturity that it is now possible to write substantial textbooks covering the nature of medicine in context from antiquity to the present. *See*, the classic compilations, L.I. Conrad et al., *The Western Medical Tradition : 800 B.C.-1800 A.D.* (2006); W.F. Bynum et al., *The western medical tradition : 1800 to 2000* (2006).

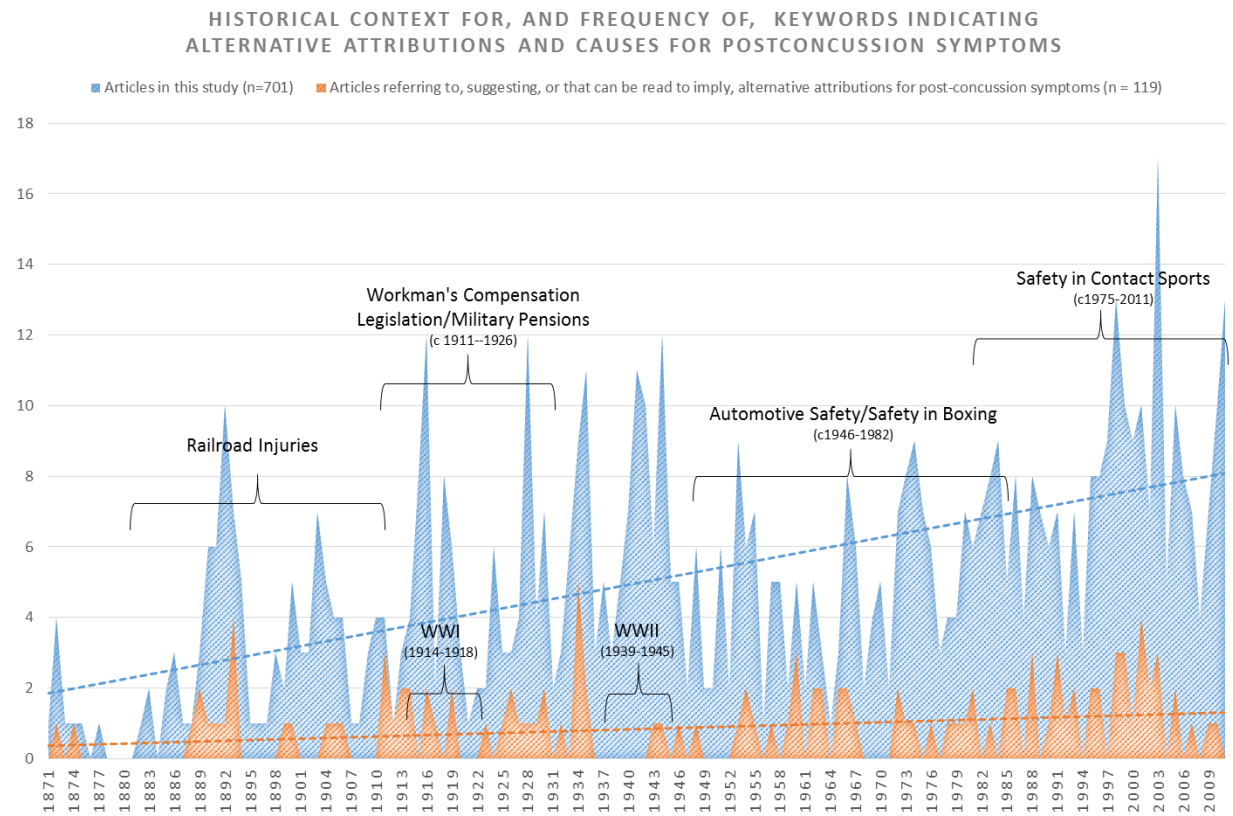

Figure 2. Over the long history of concussion research, authors have been fairly consistent in recognizing the broader context in which their work was situated. It is thus possible to detect very clearly the social, cultural, economic, policy, and litigation contexts that mattered both in the historical background and sometimes in the historical foreground of the published medical and scientific literature. Nevertheless, using the most generous metric possible, those sources that most clearly opposed established views of the delayed and long-term dangers of even a single concussions appear to have mainly coincided with a context of concern about litigation proceedings or pension payments. *See* **Exhibit D** for a bibliography of these works specifically.

67. Head injuries research during the period of this declaration spanned many controversial developments in civil society. The most obvious broad context in the historical record is the litigation and legislation context. Head injuries have been compensable injuries in many countries since the nineteenth century. In the historical record, citing the first instance in this analysis, reveals that there are examples showcasing the spectrum of litigation and legislative issues that concerned the medical profession:

- Recognition that the litigation could deform otherwise decisive medical opinion;[16]

[16] *See, e.g.*, C.H. Jones, *Clinical Remarks on a Case of Concussion Followed by Chorea and Drowsiness*, 2 BR. MED. J. 612, 319-20 (1872); *Medico-Legal and Medico-Ethical*, BR. MED. J. 1: 490-492 (1679).

- Recognition of the role of medical expertise in court proceedings and the consequences of those roles for medical professionalism and autonomy;[17]
- Litigation concerned with injuries sustained by individuals on railroads involving spinal concussion and spinal concussion sequelae;[18]
- Litigation concerning occupational injuries among coal minors, construction workers, police officers, and other groups;[19]
- Litigation involving by medical experts working for corporations or advocacy for corporate interests;[20]
- Litigation involving insurance claims for disability allowances;[21]
- Expressions of concerns about the emergence of Welfare State protections, including Worker's Compensation Legislation;[22]
- Public policy debates about granting veterans with shell shock disability pensions vs. granting veterans with known brain concussion disability pensions;[23]
- Observations about the experts working for the insurance industry;[24]

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[17] *Neurology and Psychological Medicine*, LANCET, 450-51 (1912).

[18] *See, e.g.*, B. Bramwell, *Concussion of the Spine, More Especially in its Relationship with Railway Accidents and Injuries*, 2 BR. MED. J. 1716, 1089-97 (1893). It is noteworthy upon inspection of the primary source record that these debates about railway spine injuries have comparatively little role in brain concussion research, although it is clear that arguments advanced against the legitimacy of spinal injury sequelae have a close family resemblance to later arguments advanced again the legitimacy of brain concussion sequelae.

[19] On colliers, *see The Traumatic Neuroses*, 2 BRI. MED. J. 1716, 1115-16; 1955 (1893); on construction workers *see American EEG Society - Symposium: Round Table Conference on Head Injuries*, 7 ELECTROENCEPHALOGRAPHY & CLINICAL NEUROPHYSIOLOGY 3, 495-502; on police and fire workers, *see* A. Adler, *Mental Symptoms Following Head Injury*, 53 ARCH. NEUROL. & PSYCHOL. 1, 34-43 (1945).

[20] A.C. Brush, *A Study of the Traumatic Insanities*, 53 J. AM. MED. ASS'N 14, 1081-84 (1909); H. Judd, *Domestic Correspondence: Spinal Injuries from Railroad Accidents*, JAMA, 284-85 (1889).

[21] B. Bramwell, *An Address on Malingering, Valetudinarianism, and Their Prevention*, 1 BRI. MED. J., 2729, 805-10 (1913).

[22] A.G. Gullan et al., *Discussion on Adminstrative Provisions for the Prevention of Malingering*, 2 BRI. MED. J., 2692, 221-26 (1912).

[23] J.R. De Fursac, *Traumatic and Emotional Psychoses*, 75 AMERICAN JOURNAL OF PSYCHIATRY 1 , 19-51 (1918).

[24] N. Savitsky, *Further Comments on Head Injury - The Postconcussion Syndrome*, 34 N.Y. STATE J. MED. 21, 909-13 (1934).

- Efforts by healthcare professionals to exclude patients with current or pending litigation to investigate the sequalae of head injury;[25]
- Public policy efforts to improve the safety of amateur and professional boxing;[26]
- Litigation about head injuries sustained in motor vehicle accidents and public policy efforts to make motor vehicles safer;[27]
- Reporting mechanisms to the Occupational Health and Safety Administration of the United States Department of Labor;[28]
- Litigation surrounding professional sporting industries;[29] and
- Efforts by the Veteran's Administration of the United States to address the co-morbidity of post-concussion syndrome and post-traumatic stress syndrome.[30]

68. Given the adversarial nature of the courts, it is unsurprising to find a legacy of expressions of differences between healthcare professionals and scientists brought in as expert witnesses in litigation settings.[31] In this way litigation more than any other context appears to have exerted enormous pressures on the production of scientific and medical knowledge. The majority of these cases involved single individuals versus large corporate interests or government actors. Often, too, the historical record makes clear that there was little question of the plaintiff receiving a settlement in these cases – the question was primarily of what magnitude. The exceptions to this rule appear only in the context of professional sports industries, where

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[25] P. G. Denker, *The post-concussion syndrome: prognosis and evaluation of the organic factors*, N.Y. STATE J. MED. 44, 379-84 (1944).

[26] J. L. Blonstein & Edwin Clarke, *The Medical Aspects of Amateur Boxing*, 2 BR. MED. J. 4903, 1523-25 (1954).

[27] G.V. Buehler, *Abused Claims*, NEW ENG. J. MED. 72 (1938).

[28] U.S. Department of Labor Occupational Safety and Health Administration. *OSHA Field Operations Manual.* Rockville: Government Institutions, particularly pp. IV-18, IV-21, IV-22 (1993).

[29] A premonition of what was to come first appears in R.C. Cantu, *Guidelines for Return to Contact Sports After a Cerebral Concussion*, 14 PHYSICIAN AND SPORTSMEDICINE 10 (1986).

[30] *Grappling with Traumatic Brain Injury*, 370 LANCET 9603 (2007).

[31] *See* Figure 3.

there is a recent history of class action lawsuits regarding industrial negligence in disclosing the risks of repeated head injury.

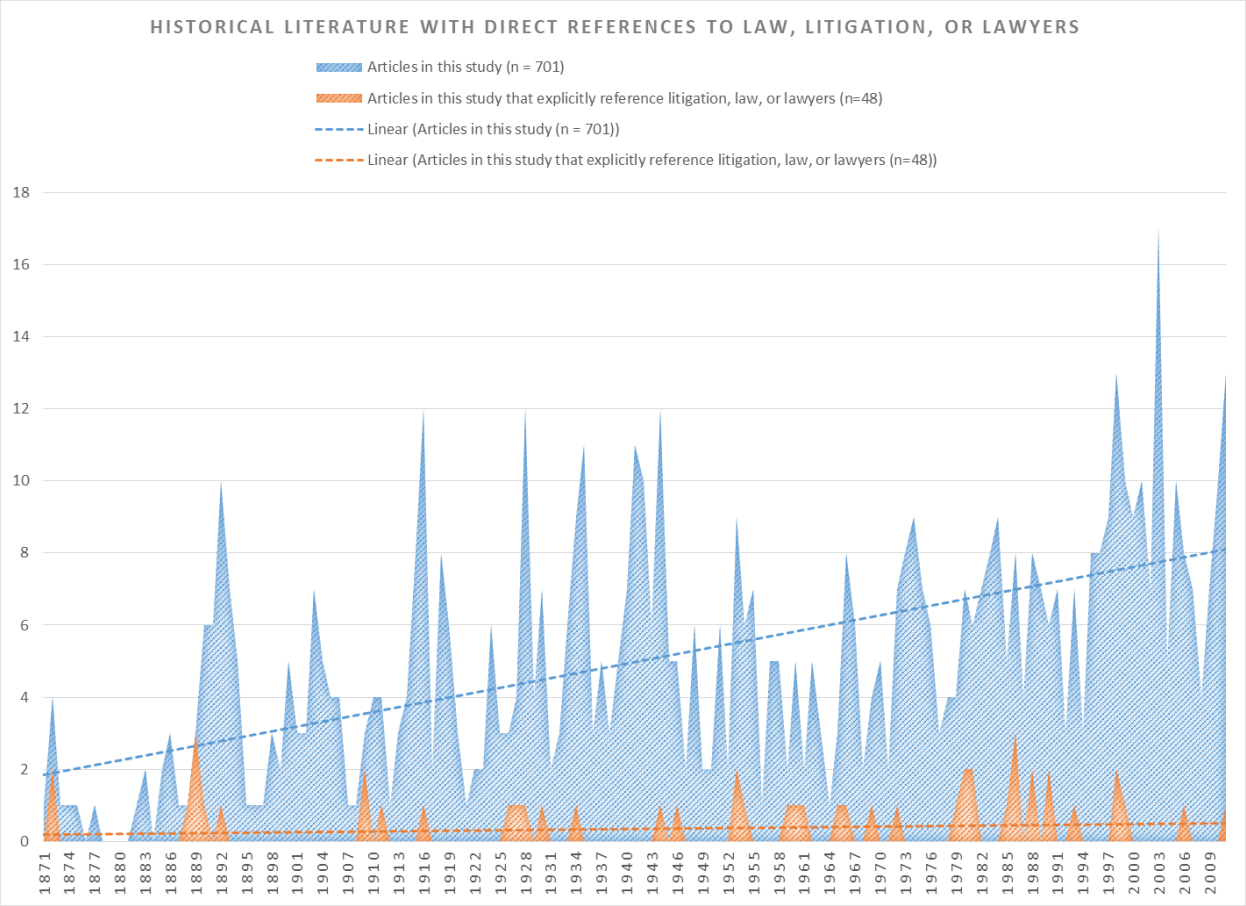

Figure 3. References in the historical literature occasionally speak directly to the litigation context. Not represented in this figure are sources that also speak more indirectly about court proceedings. Nevertheless, much can even be gleaned from this narrower set of sources. The references the late nineteenth century are mainly about litigation with railroad companies. Literature in the early 1910s through to 1916 is mainly about worker's compensation claims. Literature in the 1920s and 1930s is about insurance claims following industrial accidents. Articles from the 1960s through to the 1980s speak either about workplace accidents or motor vehicle accidents. *See* Exhibit D for a bibliography of these works specifically.

69. That final observation deserves further qualification for this declaration. It is now known from a Congressional Report that the National Football League sought to influence the production of concussion research at even the highest levels.[32] It has also

[32] U.S. House of Representatives Committee on Energy and Commerce, *The National Football League's Attempt to Influence Funding Decisions at the National Institutes of Health*, CONGRESSIONAL REPORT, May 2016. https://democrats-

been shown by investigative journalists that the NFL MTBI Committee (formed in 1994 and chaired by Dr. Eliott Pellman) engaged in deeply questionable, perhaps fraudulent, scientific practices.[33] While there can be little doubt upon reading them that all of the MTBI Committee's published articles advanced the interests of the National Football League, it is, however, difficult for a historian to determine how deficient the individual articles the MTBI committee published between 2003 and 2009 were, and whether all of the articles were equally corrupted.[34] Further questions this raises are: to what extent

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energycommerce.house.gov/sites/democrats.energycommerce.house.gov/files/Democratic%20Staff%20Report%20on%20NFL%20NIH%20Investigation%205.23.2016.pdf (last visited August 23, 2016).

[33] *See, e.g.*, A. Schwarz et al., *N.F.L.'s Flawed Concussion Research and Ties to Tobacco Industry*, N.Y. TIMES, Mar. 24, 2016. http://www.nytimes.com/2016/03/25/sports/football/nfl-concussion-research-tobacco.html?_r=0 (last August 23, 2016).

[34] *See*, specifically, the articles published by the NFL MTBI committee: P. Tagliabue, *Tackling Concussions in Sports*, 53 NEUROSURGERY 4, 796 (2003); E.J. Pellman et al., *Concussion in Professional Football: Neuropsychological Testing??? - Part 6*, 55 NEUROSURGERY 6, 1290-302 (2005); E.J. Pellman, *Background on the National Football League's Research on Concussion in Professional Football*, 53 NEUROSURGERY 4, 797-98 (2003); E.J. Pellman et al., *Concussion in Professional Football: Location and Direction of Helmet Impacts - Part 2*, 53 NEUROSURGERY 6, 1328-37 (2003); E.J. Pellman et al., *Concussion in Professional Football: Reconstruction of Game Impacts and Injuries*, 53 NEUROSURGERY 4, 799-812 (2003); E.J. Pellman et al., *Concussion in Professional Football: Repeat Injuries - Part 4*, 55 NEUROSURGERY 4, 860-876 (2004); E.J. Pellman et al., *Concussion in Professional Football: Players Returning to the Same Game -- Part 7*, 56 NEUROSURGERY 1, 79-92 (2005); E.J. Pellman et al., *Concussion in Professional Football: Injuries Involving 7 or More Days Out - Part 5*, 55 NEUROSURGERY 5, 1100-19 (2004); E.J. Pellman et al., *Concussion in Professional Football: Epidemiological Features of Game Injuries and Review of the Literature - Part 3*, 54 NEUROSURGERY 1, 81-96 (2004); D.C. Viano & E.J. Pellman, *Concussion in Professional Football: Biomechanics of the Striking Player - Part 8*, 56 NEUROSURGERY 2, 266-80 (2005); D.C. Viano et al., *Concussion in Professional Football: Animal Model of Brain Injury - Part 15*, 64 NEUROSURGERY 6, 1162-1173 (2009); D.C. Viano et al., *Concussion in Professional Football: Biomchanics of the Struch Player - Part 14*, 61 NEUROSURGERY 2, 313-28 (2007); D.C. Viano et al., *Concussion in Professional Football: Comparison with Boxing Head Impacts - Part 10*, 57 NEUROSURGERY 6, 1154-72 (2005); D.C. Viano et al., *Concussion in Professional*

individual scientists and clinicians writing those articles (and responding to them) acted in duplicitous ways; how far those studies went in influencing the judgment and scientific work of other unconnected scientists and clinicians; how broadly those reports confused efforts at genuine consensus building; and just how substantially the revelations about that work have broken down trust between experts. It is also unclear how much the physicians and scientists who assisted the National Football League in producing those scholarly reports might have influenced other professional sports industries, including the National Hockey League.[35]

70. What can be said with some historical clarity, however, is that these activities appear without precedent in the historical record of concussion research. It can also be said that the records of individuals who participated in the production of those reports, whether they acted in duplicitous ways intentionally or not, fundamentally call into question the veracity of their other scholarly contributions and communications as primary sources for the intellectual history of concussion research. This cost is a painful one, for it also implies that scientific work citing the work of those individuals must also

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Football: Brain Responses by Finite Element Analysis: Part 9, 57 NEUROSURGERY 5, 891-916 (2005).

[35] Dr. Mark Lovell, cited in several places in this declaration, for instance, published with the NFL MTBI Committee. *See* E.J. Pellman et al., *Concussion in Professional Football: Neuropsychological Testing??? - Part 6*, 55 NEUROSURGERY 6, 1290-302 (2005). It appears Dr. Lovell also helped the National Hockey League with their concussion program. Writing in 2001, Ruben Echemendia and Laura Julian stated: "At the professional level, the National Hockey League and the National Hockey Leagues Player's Association have developed a league-wide neuropsychological testing program. This program, codirected by M. Lovell and R. Echemendia, has developed a network of 28 neuropsychologists across North America that allows players to be neuropsychologically evaluated in any city where they play. To date, over 1,500 baselines have been collected and over 125 players have been evaluated post-mTBI. The data from this project are currently being analyzed. In sum, ice hockey is a high risk sport for mTBI and the prevalence of mTBI has been well-documented in this group. However, to our knowledge, there are no prospective neuropsychological studies that have been published to date in ice hockey (81)." *See* R.J. Echemendia & L.J. Julian, *Mild Traumatic Brain Injury in Sports: Neuropsychology'sNeuropsychology's Contribution to a Developing Field*, 11 NEUROPSYCHOLOGY REVIEW 2, 69-88 (2001).

be treated with a degree of concern. From a historical view, scientific and medical work as an accumulative enterprise is only as good as the trust that can exist in its experts' reports and foundational research. This is not to say that such records should be excluded from historical study – indeed they should be included precisely because they *are* a part of what was published after 1990.

71. The historical record furthermore makes clear that long before the National Football League was involved in concussion research, there was more than sufficient medical and scientific evidence that professional sports industries could have disclosed to their employees the dangers of subconcussive and concussive blows.

B. Professional Hockey is an Industry in which Head Injuries are a Common Occupational Hazard

72. Outside of the medical record, it is first useful to establish whether professional hockey has been deemed a hazardous occupation in which head injuries are common. The best proxy for this belief over time is the appearance of newspaper articles documenting and discussing NHL sports injuries. The historical record makes clear that head injuries in NHL hockey were, and are, a common event for workers in this occupation. In the history of public health, such workplace injuries are commonly referred to and examined under the umbrella of *occupational injuries*.

73. Hundreds of newspaper articles published from the 1920s to 1980 in major outlets describe NHL players who sustained concussions or were knocked unconscious in the course of their playing careers.[36] Some reports describe career ending injuries for

[36] The historical newspapers included in this study were consulted using full-text searchable and digitized collections of Proquest Historical Newspapers. The newspapers included in this database were: *Atlanta Constitution* (1868-1985), *Austin American Statesman* (1871-1975), *The Baltimore Sun* (1837-1989), *The Boston Globe* (1872-1983), *The Chicago Tribune* (1849-1991), *The Christian Science Monitor* (1908-2001), *Cincinnati Enquirer* (1841-1922), *Dayton Daily News* (1898-1922), *Detroit Free Press* (1831-1922), *Hartford Courant* (1764-1989), *Indianapolis Star* (1903-1922), *Los Angeles Times* (1881-1991), *The Louisville Courier Journal* (1830-1922), *Nashville Tennessean* (1812-1922), *The New York Times* (1851-2011), *New York Tribune/Herald Tribune* (1841-1962), *Newsday* (1940-1986), *San Francisco Chronicle* (1865-1922), *St.*

workers because of head injury.[37] One tragic case was that of Bill Masterton, who played for the Minnesota North Stars. He was killed in 1968 as a result of a severe head injury sustained in a game. Masterton's tragedy did not, it appears, result in any immediate changes to industry safety measures.

74. Newspaper authors recorded many ways that head injuries, including concussions, occurred in this occupation (*see* Figure 4).[38] Photographic evidence

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Louis Post-Dispatch (1874-1922), *Wall Street Journal* (1889-1997), *Washington Post* (1877-1998), and *Globe and Mail* and the *Toronto Star* (1894-2011).

[37] Laurel Zeisler observes of career ending injuries: "Several hockey players have had their careers end abruptly due to injuries received on the ice. Shorty Green of the New York Americans retired after a kidney injury on 27 February 1927. On 12 December 1933, Eddie Shore of the Boston Bruins tripped the Toronto Maple Leafs' Ace Bailey. Bailey's head injury was severe that he was not expected to live. He survived, but his playing career was over. Lou Fontinato of the Montreal Canadiens broke his neck after crashing into the boards on 9 March 1963. The Montreal Canadiens' Jacques Laperriere suffered a career-ending injury to his knee in a game against the Boston Bruins on 19 January 1974. The Philadelphia Flyers' goaltender, Berie Parent, suffered a career-ending injury when he was hit in the eye in a game against New York Rangers on 17 February 1979. Walt Tkaczuk of the New York Rangers retired at the age of 33 following a severe eye injury from the Kings on 2 February 1981. On 20 October 1995, only 11 seconds into his first shift for Boston University's college hockey team, 20-year old Travis Mitch Vig avoided Roy's check. The awkward impact with the boards left him paralyzed. On 8 March 2004, Steve Moore of the Colorado Avalanche had his National Hockey League career cut short because of a punch to the head by Todd Bertuzzi of the Vancouver Canucks. Moore suffered a broken neck and a concussion. As a result of the brutality, Bertuzzi was suspended for the rest of the season." *Historical Dictionary of Ice Hockey*, p. 61 (2013).

[38] Laurel Zeisler observes of head injuries: "As a full-contact sport, head injuries are the most common injury in hockey. Prior to the introduction of helmets, several players had career-ending injuries, including Ace Bailey, Donald Brashear, and Pat LaFontaine. In 1968, while playing for the Minnesota North Stars, forward Bill Masterton suffered a fatal head injury after being body checked and hitting his head on the ice. Checking from behind and checking to the head are the main causes of head injuries today. Due to the seriousness, the National Hockey League has made penalty rules to enforce this major and gross misconduct. Players also can hit the boards on their own due to their skating speed. On 16 March 1994, the Chicago Blackhawks Michel Goulet slid into the boards headfirst after losing his footing. He suffered a severe concussion and head trauma and never returned to the ice. Concussions are one reasons players miss games and repeated concussions cause players to retire early. Eric Lindros, his brother, Brett

accompanied some of these articles. From the 1920s to 2000, there are descriptions of accidental and intentional swings of hockey sticks striking NHL players' heads; collisions between players; players colliding with the boards and goal posts; and instances of pucks hitting NHL players in the head. There are also mentions of players falling forward and backward on to the ice. Prominent among all of these reports are accounts of fights between players that resulted in head injuries. In one instance, the fight was so violent it resulted in court proceedings.[39]

Sawchuk Introduces a New Sickness: Pace Faster But Hockey Goalies No Softer
Kaese, Harold
Daily Boston Globe (1928-1960); Dec 13, 1956; ProQuest Historical Newspapers: The Boston Globe
pg. 19

Sawchuk Introduces a New Sickness

Pace Faster But Hockey Goalies No Softer

By HAROLD KAESE

To all the accidents and miseries that can befall a big league hockey goalkeeper, add infectious mononucleosis.

Goalies have capitulated to broken bones, allergies and shattered nerves, but Terry Sawchuk of the Bruins is probably the first to leave the lineup because his white corpuscles are not behaving.

The current 70-game schedule seems harder on goalies than other players. Only two out of six—Glenn Hall of Detroit and Lorne Worsley of the Rangers—played all 70 games last season, and nobody made it two years ago.

DURNAN

George Hainsworth, who guarded the portal in the days of the 30 and 44-game schedules, missed only one game in his entire career. He had tonsilitis.

The pace was slower then. Hainsworth did not retire until he was 38.

Sawchuk is the third goalie to leave the lineup already this season.

Jacques Plante, the Wandering Minstrel Boy of Montreal, missed nine games because of an asthmatic condition. He chases the puck so far afield he sometimes runs into ragweed.

Worsley missed a game because his coach, Philharmonic Watson, was mad at him—'cutive anger being another affliction 's must combat.

'njury has always been the goalkeeper's 'y Worsters' rupture, the puck that hit under Tiny Thompson's nose and split it on both sides, Frank Brimsek's broken bugle, Lorne Chabot's cut eye, Chuck Rayner's broken cheekbone, Jim Franks' broken wrist . . . such accidents are almost commonplace.

Nerves Have Beaten Many Good Ones

Rarer, fortunately, was the tuberculosis with which Georges Vezina played; and the brain tumor that resulted in Chuck Gardiner's death only a few weeks after his 1 to 0 shutout against Detroit had clinched the Stanley Cup in 1934.

Many a good goalie has been beaten by his nerves. Bill Durnan, possibly the best of all, left the Montreal net during a play-off, saying, "Take me out. I can't play."

Gerry McNeil, another Montreal goalie, got the jitters in 1954 and retired temporarily.

And nearly 25 years ago, Tiny Thompson was sent to the mountains in mid-season because Art Ross said he was suffering from a "nervous condition." A couple of years later, Thompson asked for a relief goalie, even if he had to pay him out of his own salary.

Injuries to goalkeepers have resulted in some of hockey's most dramatic situations.

The Rangers clinched the Stanley Cup in 1928 with their manager, Lester Patrick, playing goal, because Chabot had suffered an eye injury in the final game.

More recently, the Detroit trainer, Lefty Wilson, finished a game for an injured Harry Lumley, without being scored upon.

Brave Charlie Sands Played Goal

Wilf Cude hurt a shoulder in 1940, and Charlie Sands bravely played the last 25 minutes in goal. He was scored on five times, and Chicago beat Montreal, 10 to 1.

recalled John Henderson (6-5) from Hershey. Henderson reached Montreal in time for the game, but his pads didn't.

The Bruins then borrowed Montreal's fourth-string goalie, Claude Pronovost, and with him beat Les Canadiens, 2 to 0, to end an 11-game losing streak.

Are goalies softer? No, but the demands made on them are greater. Goalies soft? Well, hardly. A couple of years ago Al Rollins of Chicago suffered two head injuries here in the second period.

"Don't let him play any more," the doctor advised Dick Irvin, Chicago coach.

But Rollins played, and brilliantly, as the Hawks tied the Bruins, 2 to 2. The doctor was understandably angry, but Irvin said, "Al wanted to play. He's no American baseball player, you know."

Figure 4. While it is unclear how often players return to the game following a head injury and against medical advice, circumstantial evidence suggests it did happen on occasion.

75. Newspaper reports throughout this period make frequent mention of NHL workers who were knocked unconscious during both practices and games. There are also frequent reports of dental injuries, broken bones, and facial lacerations. These facial injuries indicate that even those hits that did not result in reports of concussion,

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Lindros, and the Philadelphia Flyers' Keith Primeau are a few players who have gotten multiple concussions resulting from aggressive hockey play." *Id.* at p. 143.

[39] *Arraignment Set in Hockey Brawl*, N.Y. TIMES, Dec. 25, 1969.

were nevertheless sufficiently severe to cause bodily harm. Reports of such subconcussive blows suggest that there was significant evidence that blows to the head could not be taken by these workers with impunity. Such facts led Dr. Charles Drake, a brain surgeon and associate professor of surgery at the University of Western Ontario Medical School, to advise in 1965: "Hockey officials should devote the same interest to safety, from peewee to professional, as is done in football" because "serious brain damage can occur with little or no evidence of head injury."[40]

76. Dr. Drake's estimation of the dangers in the hockey industry were grounded in scientific and clinical publications and observations of his day. As the rest of this declaration shows, medical literature published between 1880 and 2011 contains numerous articles, editorials, reviews and other miscellaneous writings describing the risks of brain damage in all contact sports. Ice-hockey specifically was frequently mentioned in historical medical and scientific literature. One contributor writing in the pages of the *American Journal of Sports Medicine* in 1976, for example, captured the various sources of occupational risk in ice-hockey (causes of risk to which earlier figures like Drake were doubtlessly responding): "It is important to realize that for all its speed and flare, grace and beauty, hockey is still rooted in violence. Everything else being equal, the meanest team wins; and anybody close to hockey will attest to the fact that when two players drop their sticks and gloves and start swinging their fists, they are not throwing Hollywood punches. Hockey has been criticized for the occasional fighting that interrupts play, and the criticism will likely continue because the fighting is bound to."[41] The "injuries sustained by hockey players" the author concluded, "from paperweights to professionals…are varied, generally accidental, often violent, and frequently preventable. Ice hockey, a contact sport with intrinsic hazards, demands that a participant be willing to accept physical abuse. The number of injuries in the sport has increased in direct proportion to its popularity. The discussion of the mechanism and

[40] *Pro hockey players and motorists should wear helmets, doctor says*, GLOBE & MAIL, Nov. 26, 1965, at 43.

[41] G.W. Sutherland, *Fire on Ice*, 4 AM. J. SPORTS MED. 6, 264-69 (1976).

forces involved in such injuries leads to the recommendation that specific standards be established in hockey equipment to provide maximum protection and decrease substantially the risk factor."[42]

77. From 1928 through 2000, newspaper authors echoed the spirit of such commonplace medical warnings about the preventability of injuries in the hockey industry and the potential need for the enhancement and compulsory use of safety equipment. For example, in 1949 reporters noted conflicts between NHL coaches and the NHL president over whether penalties were sufficiently harsh. At the beginning of the season NHL President Clarence Campbell had directed that: "club swinging has no place in our business and penalties will get tougher if there is any more of it…a fellow doesn't have to swing a stick to prove he's tough." Yet the reporter was quick to point out that Campbell's ruling appeared to have been hot air. When NHL player Red Hamill (Black Hawks) deliberately smashed Vie Lynn (Leafs) with his stick, the penalty was not made harsher because Campbell's medical men "assured me he will be fit to play hockey almost immediately" and that "all the standard neurological tests were made and proved negative." Such a policy bothered the Leafs' managing director and coach, who were quoted in the article as having observed that, in effect, Lynn's good fortune to have not been more seriously hurt was being used to justify a do-nothing approach which hurt their team and advantaged their opponents.[43]

78. Newspaper writers also called on the NHL to make helmet wearing mandatory in the sport.[44] In 1928 Boston Bruins' Bill Carson "was badly hurt when

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[42] *Id*. at 268.

[43] *Different Opinions: Hamill Punishment Enough: Campbell Act Treated Too Lightly: Smythe*, GLOBE & MAIL, Feb. 23, 1949, at 19.

[44] Laurel Zeisler observes of helmets, "When hockey was first played in the late 1800s, ice skates, a puck, and a hockey stick were the main pieces of equipment used. Throughout the years, protective gear, including padding and hard plastic guards, were added. Throughout the years, protective gear, including padding and hard plastic guards, were added. In 1928, George Owen, a center for the Boston Bruins, wore his leather football helmet from Harvard during ice hockey games. He became the first player to regularly wear a helmet for protection. At the time, most professional players were

[Percy] Traub spilled him with a clear, hard body check" and he was "carried off unconscious" with a "serious concussion of the brain." The newspaper writer noted that Carson's example, when combined with the fact that a "head injury" had also "deprived the Chicago Hawks of Dick Irvin's services for the season," was a "convincing argument in favor of the compulsory use of fibre protection helmets by all hockey players."[45]

79. The argument for the compulsory use of helmets continued throughout the subsequent decades. Despite this, no actual changes were introduced until the NHL made helmet wearing compulsory in 1979, although newspapers did note that some players began wearing helmets before this date (*See* Figure 5). The death of Bill Masterton in 1968 reinvigorated the debate, although it was still a decade before this resulted in changes in the NHL. In October 1973, NHL President Clarence Campbell said: "It's one thing to regiment juniors, but that makes it no less objectionable to make them continue with something if they find it bothersome. We feel the player is sufficiently mature, and thoroughly cognizant of the risks of playing hockey to make his

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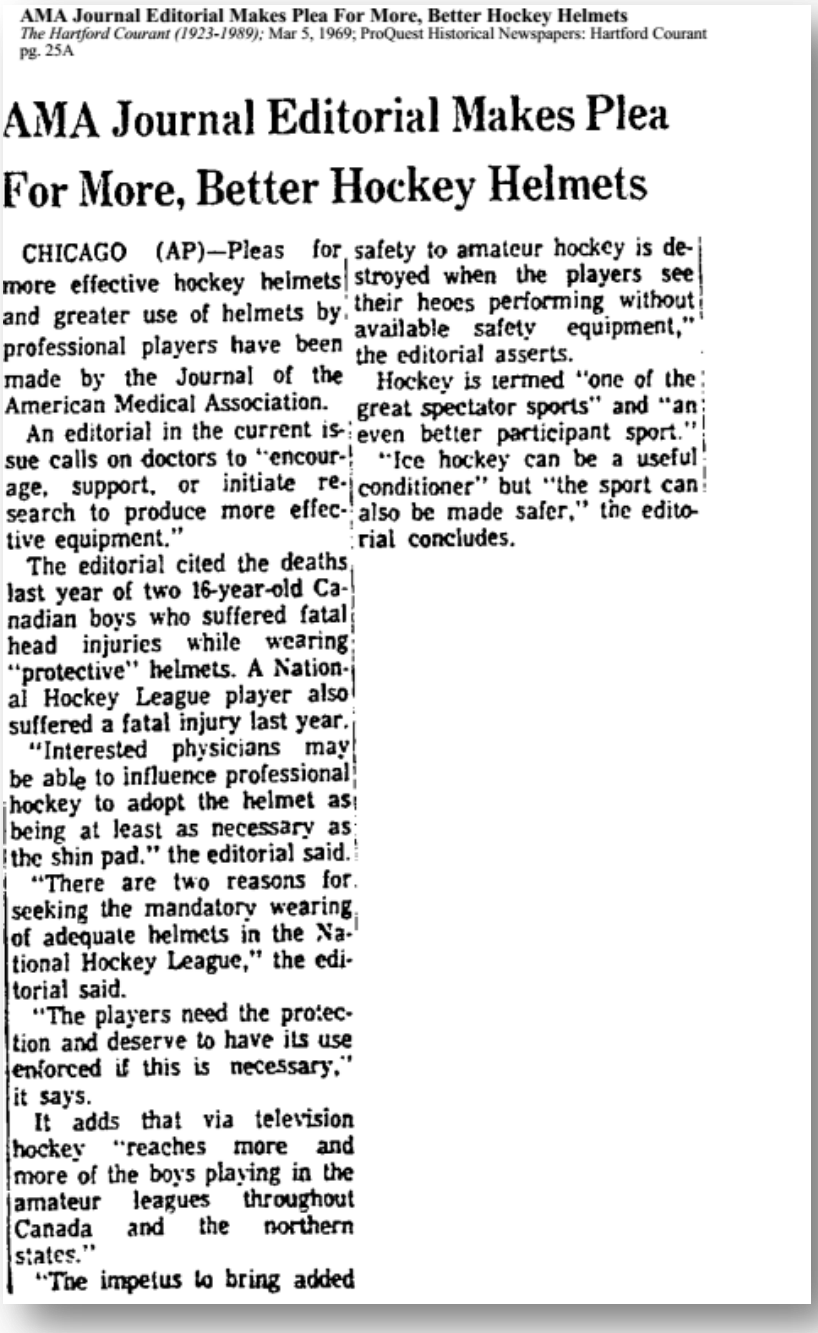
hesitant to use helmets and face masks, believing that they would cause interference and show signs of weakness, but after Ace Bailey's career ending injury caused by Eddie Shore on 12 December 1933, several Boston players, including Shore, starting wearing helmets in fear of retaliation by other NHL players. Helmet use was rare for the next 30 years, but the players who regularly wore helmets included Shore, Des Smith, Bill Mosienki, Dit Clapper, and Don Galliger. Maurice Richard and Elmer Lach briefly wore helmets. Jack Crawford wore a helm to cover baldness, and Charlie Burns and Ted Green wore them to protect the metal plates in their heads. After Bill Masterton's fatal head injury in 1968, more players, the likes of Pierre Pilote, Stan Mikita, Ken Wharram, and Dough Mohns, started wearing helmets, but, throughout the 1970s, the majority of NHL players went helmetless. In August 1979, the NHL made a rule mandating that players must wear helmets for their safety. Veterans who signed contracts prior to 1 June 1979 were able to choose whether they wanted to wear helmets. The last player to play without a helmet was Craig MacTavish, who last played during the 1996-1997 season for the St. Louis Blues. Although not mandatory, plastic visors and shields can be attached to the front of a helmet for added protection. Their popularity increased after NHL defenseman Bryan Berard suffered a potentially career-ending eye injury in 2000. Goaltenders are not required to wear facemasks, but every NHL goalie wears one for protection. *Historical Dictionary of Ice Hockey*, p. 144.

[45] *Random Notes on Current Sport*, TORONTO DAILY STAR, Feb. 6, 1928, at 10.

own choice… There are two basic things against wearing a helmet. Hockey is a game of continuous action, unlike football, and there's no hockey helmet that will stay in place if a player is jolted or hit into the fence. Secondly, many players have an acute auditory sensitivity and with a helmet they lose it. A third factor would be comfort." The President of the NHL Players Association took a different view. An internal survey of members had shown that "65 per cent of players felt helmets should be worn, and 95 per cent said they would wear them if the vote on the first question was positive. Ultimately, it was noted: "if the owners ruled that the players must protect themselves with helmets, it would be a simple matter."[46]

[46] D. Proudfoot, *Too many head injuries: Ron Ellis to Rejoin NHL helmet-wearers; expects to play Tuesday*, GLOBE & MAIL, Oct. 20. 1973, at 41.



AMA Journal Editorial Makes Plea For More, Better Hockey Helmets
The Hartford Courant (1923-1989); Mar 5, 1969; ProQuest Historical Newspapers: Hartford Courant
pg. 25A

AMA Journal Editorial Makes Plea For More, Better Hockey Helmets

CHICAGO (AP)—Pleas for more effective hockey helmets and greater use of helmets by professional players have been made by the Journal of the American Medical Association.

An editorial in the current issue calls on doctors to "encourage, support, or initiate research to produce more effective equipment."

The editorial cited the deaths last year of two 16-year-old Canadian boys who suffered fatal head injuries while wearing "protective" helmets. A National Hockey League player also suffered a fatal injury last year.

"Interested physicians may be able to influence professional hockey to adopt the helmet as being at least as necessary as the shin pad," the editorial said.

"There are two reasons for seeking the mandatory wearing of adequate helmets in the National Hockey League," the editorial said.

"The players need the protection and deserve to have its use enforced if this is necessary," it says.

It adds that via television hockey "reaches more and more of the boys playing in the amateur leagues throughout Canada and the northern states."

"The impetus to bring added safety to amateur hockey is destroyed when the players see their heoes performing without available safety equipment," the editorial asserts.

Hockey is termed "one of the great spectator sports" and "an even better participant sport."

"Ice hockey can be a useful conditioner" but "the sport can also be made safer," the editorial concludes.

Figure 5. As the above 1969 newspaper report makes clear, the American Medical Association was also urging the NHL to take employee safety seriously.

80. A digression is warranted here as well – Clarence Campbell's observation that the players were informed of the risks of head injury is contradicted by the memories of players. As Wayne Gretzky recalled, "We probably didn't know what a concussion was back in those days…. It was more a case of 'Take two aspirins and come

back in the morning. If you had a headache, maybe you practiced, maybe you didn't practice.'"[47] This stands as one of many examples where the NHL was archaic in addressing even modest efforts to improve workplace safety for their employees as recommended by scientific researchers.[48]

81. It appears to be a historical coincidence that the debate about compulsory use of helmets in hockey seems to have first appeared in newspapers in 1928. As will be discussed below, it was in that same year that chronic brain damage caused by repeated head injury in contact sports was first characterized in the medical literature by Harrison Martland, who described the pathophysiology and clinical characteristics of boxers known colloquially as "punch drunk." After 1928 there were occasional moments when newspaper articles discussing the long-term medical consequences of *boxing* appeared alongside newspaper reports of head injuries in NHL hockey, including injuries caused by fighting. Although these reports appeared on the same pages, the explicit similarities between the industries were rarely connected in the press.

82. However, there were those occasional moments when the explicit similarities between boxing and hockey were discussed. In those articles, efforts to reduce the risks of head injury in the NHL were often compared ***unfavorably***. In 1990,

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[47] M. Zeisberger, *Wayne Gretzky unsure if he ever had a concussion*, TORONTO SUN, Dec. 2, 2015, http://www.torontosun.com/2015/12/02/wayne-gretzky-unsure-if-he-ever-had-a-concussion (last visited May 17, 2016).

[48] A 1941 editorial explained that much had been written about head injuries and so everyone should know about their dangers. "Protective headgear for the fighting man went out of fashion with the transition from hand-to-hand combat to the lead and steel missile, but the reappearance of the steel helmet in the last war [World War I] undoubtedly saved many casualties. In less serious pursuits, American footballers have for long worn a padded leather helmet to lessen the incidence of concussion in that robust game, and from the speedway and dirt-track we learned the virtues of the crash helmet, especially for motor-cyclists. [Hugh] Cairns' [a British neurosurgeon] studies in this war [World War II] are a cogent argument for insisting that all motor-cyclists should adopt this simple and efficient protection. Industrialists have been slow to learn these lessons, but Miller reports that the incidence of head injuries among the South African miners has been greatly reduced by the adoption of the "hard hat," and the fashion will doubtless spread among other classes engaged in dangerous occupations," *Head Injuries*, LANCET 801-802 (1941).

Earl Gustkey, offering an analysis in the *Los Angeles Times* made the similarity between both industries' occupational hazards clear: "Sooner or later, hockey people will have to come to grips with the fact that repeated, concussive blows to the head can result in neurological impairment. If the violence gets any worse than it was Wednesday night at the Forum, the ban-boxing proponents will have a new target."[49] Observing that such a combative night suggested neurological exams should be required of all participants, Gustkey noted that in California: "all professional boxers must pass a yearly neurological exam…and boxers get hit with gloved fists. Hockey players get hit with bare fists, elbows, and, occasionally, sticks" and he observed as well that NHL referees should take lessons on "how to separate fighters in a clinch" from boxing referees.[50] Noting that boxing referees might be missing out on employment opportunities, Gustkey continued his analysis by offering an ironic but pointed commentary on the boxing style of several NHL professionals. At the same time, there is evidence that NHL professionals at times took offensive training from professional boxers. Sports writer Ross Bernstein quoted former professional heavyweight boxer Scott LeDoux, who purported to have taught hockey enforcers the art of fighting for three decades, and who, it should be noted felt that were differences between the industries:

> Back in the '80s I used to work with Brad Maxwell and a few of the guys from the Minnesota North Stars. I also worked with the New York Islanders back when they won their four Stanley Cups. I worked with guys like [Bob] Nystrom, [Clark] Gillies, and [Dave] Langevin, and I tried to teach them how to execute a punch properly by turning on it. I really tried to prepare them to fight under those circumstances, with skates on and them holding on the other guy's jersey. I had a great perspective, too, in that being from Minnesota I had played hockey growing up, so I could relate to how different it was from actual boxing. I also had a lot of experience bare-knuckle brawling in my day, and let's just say I never lost a fight. Ever. Hockey fighting is very different from boxing, where you can set people up with combinations. In hockey things happen so quickly and there is just no time. So, turning their shoulders on their punches so

[49] E. Gustkey, *ANALYSIS If they're Going to Fight, do it Right*, L.A. TIMES, Mar. 2, 1990.

[50] *Id.*

> that they punched straight was the big thing, along with leverage and balance. I always felt like the best strategy in a hockey fight was to just give the guy three straight jabs right down the chute as fast as you could before the other guy could grab onto your jersey.[51]

83. Of note in this passage is that LeDoux's understanding of the differences between hockey and boxing industries was tactical; the goals of the attacks in both sports remains as damaging the opponent. LeDoux went on in the same passage to offer his opinion that there were nevertheless real differences between the industries.[52] Tempting as it may be to downplay the similarities between the boxing and hockey industries, as LeDoux did, there has never been any study (so far as this author has identified) that has clarified the precise biomechanical, epidemiological, pathophysiological, or neurological differences between fighting in hockey and boxing.[53]

84. Newspapers also reported that hockey players sometimes sustained repeated concussions within weeks, had been known to return to the same game after a loss of consciousness, and to play in games while still symptomatic from earlier

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[51] Quoted in R. Bernstein, *The Code: The Unwritten Rules of Fighting and Retaliation in the NHL* 63-64 (2006).

[52] *Id*. at 65.

[53] The National Football League MTBI Committee, however, did publish a study analyzing the differences between concussions in professional football and professional boxing. These studies have been widely discredited. It is, however, worth noting that the authors felt that the major difference was that football involved translational impact forces whereas boxing involved rotational impact forces, and it was rotational impacts that were dangerous. One obvious inference from this study would be that hockey and boxing have more in common than hockey and football. *See* D. C. Viano *et al*., *Concussion in Professional Football: Comparison with Boxing Head Impacts – Part 10*, NEUROSURGERY 57, 6, 1154-72 (2005). It is also worth noting that there is some evidence of intense antagonism between NFL and NHL physicians entrusted with worker safety. Elliot Pellman, lead of the NFL's MTBI committee, accused the NHL of minimizing concussions that occurred in the industry. *See* P. Domowitch, *Matters of the Head Nature of Hockey Can Get in Way of Concussion Discovery*, PHILA. DAILY NEWS, June 7, 2000, at 110.

concussions.[54] As *The Toronto Star* reported in 1998, "If Mike Modano were a boxer in Ontario, he'd be under a 60-day suspension":[55]

> But Modano is an NHL player, the most important cog in the Dallas Stars' playoff drive, and he hasn't missed a game since he was reportedly knocked unconscious for about 30 seconds a week ago in Game 2 against San Jose.[56]

85. The same journalist went on to detail that the NHL had become "a human lab for the study of concussion." Quoting eminent neurologists, the journalist reported that much was still not known about concussions but that neuropsychological testing was being used to help. It was clear, too, that players were being given clean bills of health by neuropsychologists, yet still playing through serious symptoms:

> But as the predicament faced this season by Jim Johnson of the Phoenix Coyotes illustrates, those neuropsychological tests may not protect players from the effects of repeated concussions as intended. The 30-minute neuropsychological tests are designed to come up with a baseline of brain function for each individual player. It tests such aspects as memory and cognitive function. Injured players aren't to return to action until their neuropsychological tests return to their baseline and they pass a physical exertion test. Johnson, 35, blacked out when he got hit on the tip of the jaw Nov. 6 in a game against Ottawa. He suffered no memory loss, but was experiencing headaches and was seeing spots. Still, he passed his neuropsychological test administered two days later in Toronto and played that night against the Leafs. He didn't feel right and felt sluggish afterward, but played again three nights later against Tampa Bay and scored a goal. "I couldn't even see the net from the blue line," Johnson recalled yesterday in a telephone interview. "I got to the bench and I couldn't read the board advertising. "That was in the first period. I played the rest of the game. That's how foolish I was."[57]

86. The journalist also noted that there were multiple guidelines governing returning players to competition: "Dr. Charles Burke, team physician for the Pittsburgh

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[54] For returning to play after a loss of consciousness, *see id.*

[55] R. Starkman, *Out cold NHL's failture to deal with concussions courts tragedy*, TORONTO STAR, May 2, 1998, at C1.

[56] *Id.*

[57] *Id.*

Penguins and head of the NHL's concussion committee, said he does not foresee it recommending the league adopt a specific set of guidelines."[58] A neurologist involved in producing one of those guidelines made clear his opinion why professional teams ignoring guidelines was unsurprising: "You're dealing with a culture, plus there's a lot of money involved in this…. It's very hard to make headway and get past the denial everyone's got."[59]

87. In sum, newspaper articles are produced with a fundamentally different purpose than scientific and medical literature. They are journalism, and, moreover, primarily conveying news to sports fans about what was happening to their teams. For this same reason, newspaper articles provide useful insight into practices in hockey relating to concussion. As has already been seen:

- Head injuries in NHL hockey have long been a common injury;
- Repeated concussions in hockey were and are known to occur;
- Players have returned to play in the same game following a loss of consciousness;
- Players have sustained multiple, severe head injuries and yet not withdrawn for the season;
- Fighting has been a common cause of concussions in NHL hockey;
- NHL executives in the past appear to have been slow to even adopt modest changes that might improve worker safety; and
- NHL executives in the past were half-hearted in enforcing measures that bolstered worker safety.

C. Core Concepts Gleaned From the Historical Record

1. The Stable Evolution of the Concussion Definition

88. Is it fair to assume that the incidents reported in newspapers refer to the same thing that we today understand as "concussion"? The historical record as a whole indicates many reasons for thinking the answer to that question is "yes." In general the record shows that the definition of concussion has changed only modestly from the nineteenth-century. Minor changes often reflected an understandable variation between

[58] *Id.*

[59] *Id.*

the different clinicians and scientists in defining the condition. When more significant changes did occur, the historical evidence as a whole presents a compelling picture that these changes were towards clearer scientific and clinical precision. Between 1871 and 2011 there were no changes that indicated a departure in meaning so great as to warrant a less precautionary stance towards the prognosis for such brain injuries.

89. The evidence that there is a strong family resemblance in the definition of concussion across the generations can be demonstrated by comparison drawn from widely used medical encyclopedias, dictionaries and current institutional definitions. In 1890 Foster's *Illustrated Encyclopedic Medical Dictionary, Being a Dictionary of the Technical Terms* defined concussion of the brain in both its physiological and pathological manifestations:

> Concussion of the brain. Lat., *concussio* (seu commitio) *cerebri*. A jarring of the brain substance without laceration of its tissues or with only microscopic laceration. It is characterized by partial or complete loss of consciousness with feebleness of the pulse, coldness of the extremities, pallor, and dilation of the pupils, followed by vomiting, moaning, jactitation, and somnolences with warmth of the skin, a full, relaxed pulse, sometimes irregular, and contraction of the pupils; the whole usually ending in gradual recovery.[60]

90. Similarly, in 1897 the editor of *Lippincott's Medical Dictionary* defined concussion of the brain in 1897 as "a diseased state, producing alarming symptoms, caused usually by diffuse force applied to the head, and attended by concussion-lesions, viz. slight contusion, laceration, or miliary extravasation," (the words miliary extravasation meaning the distribution of tiny pathological lesions in the brain tissue).[61]

91. Nearer to the professional life of most current concussion researchers and medical practitioners was a 1976 entry in the *Penguin Medical Encyclopedia*:

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[60] F.P. Foster, *An Illustrated Encyclopedic Medical Dictionary. Being a Dictionary of the Technical Terms* vols. 1-4. vol 2 [1890] p. 1111 (New York, D. Appleton & Co. 1888).

[61] R.W. Green et al., *Lippincott's Medical Dictionary: A Complete Vocabulary of the Terms Used in Medicine and the Allied Sciences* 244 (London, J. B. Lippincott Co., 1897).

> Concussion. Shaking of the brain, as by a blow to the head or a jolt to the neck, with temporary disturbance of consciousness. In every case, concussion probably puts some of the millions of nerve cells in the brain permanently out of action, and repeated concussion, such as professional boxers may suffer (a knock-out is concussion), causes serious deterioration. Events immediately before concussion are usually not remembered (retrograde amnesia). Anyone who has just recovered from concussion still needs observation, because symptoms of more serious damage may be delayed for several hours.[62]

92. And now, in 2016, the Mayo Clinic defined concussion as:

> A concussion is a traumatic brain injury that alters the way your brain functions. Effects are usually temporary but can include headaches and problems with concentration, memory, balance and coordination. Although concussions usually are caused by a blow to the head, they can also occur when the head and upper body are violently shaken. These injuries can cause a loss of consciousness, but most concussions do not. Because of this, some people have concussions and don't realize it. Concussions are common, particularly if you play a contact sport, such as football. But every concussion injures your brain to some extent. This injury needs time and rest to heal properly. Most concussive traumatic brain injuries are mild, and people usually recover fully.[63]

93. Likewise the American Association of Neurological Surgeons in 2016:

> A concussion is an injury to the brain that results in temporary loss of normal brain function. It usually is caused by a blow to the head. In many cases, there are no external signs of head trauma. Many people assume that concussions involve a loss of consciousness, but that is not true. In many cases, a person with a concussion never loses consciousness. The formal medical definition of concussion is a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma. People with concussions often cannot remember what happened immediately before or after the injury and may act confused. A concussion can affect memory, judgment, reflexes, speech, balance and muscle coordination. Paramedics and athletic trainers who suspect a person has suffered a concussion may ask the injured person if they know their name,

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[62] P. Wingate, *The Penguin Medical Encyclopedia* 112 (1976).

[63] *Diseases and Conditions: Concussion*, MAYO CLINIC http://www.mayoclinic.org/diseases-conditions/concussion/basics/definition/CON-20019272 (last visited May 29, 2016).

> what month/year it is and where they are. Even mild concussions should not be taken lightly. Neurosurgeons and other brain-injury experts emphasize that although some concussions are less serious than others, there is no such thing as a "minor concussion." In most cases, a single concussion should not cause permanent damage. A second concussion soon after the first one, however, does not have to be very strong for its effects to be deadly or permanently disabling.[64]

94. As this comparison makes clear, it was already known in the 1890s that external violence to the head produced a range of symptoms, sometimes alarming ones, which were evidence of altered brain function. It was also known that a concussion could involve either a partial change in or a complete loss of consciousness. It was clear that it elicited emotional disturbances, and that it usually resulted in recovery but did not always necessarily. Moreover, there were reasons for claiming that the trauma resulted in small microscopic lesions to the brain, evidence of pathophysiological brain injury. Although there were, of course, modest changes from the 1890s – most clearly the injury was described in terms of mechanical (acceleration/deceleration) forces after 1946 – through to the contemporary period, it is possible to summarize in bullet points the extensive historical evidence connecting these past and present definitions and showing the general stability of the definition of brain concussion. Such evidence includes:

- Medical dictionaries have shown broad similarity in defining brain concussion since the nineteenth century to the present;
- Medical encyclopedias have shown broad similarity in defining brain concussion since the nineteenth century to the present;
- Since the nineteenth century major bibliographic reference works such as *Index Medicus* and the *US Surgeon General's Medical Catalog* have recognized and referred readers to the existence of publications on brain concussion;
- From 1871 to 2011 understandings of brain concussion have had sufficient constancy for authors researching concussion to refer back to and cite works on concussion by earlier authorities;
- Brain concussions have been discussed as risks of contact sports in the medical literature since 1886;

[64] American Associaion of Neurological Surgeons http://www.aans.org/patient%20information/conditions%20and%20treatments/concussion.aspx (last visited May 29, 2016).

- Grading systems for concussion have been described since 1894; most grading systems have recognized the need to differentiate very mild brain concussions from more severe brain concussion, including brain concussion resulting in death (Lane 1894; Clayton, Hamilton and Lewis 1973 (for football); Ommaya and Gennarelli 1974; Cantu 1986; Sports Medicine Committee of the Colorado Medical Society Guidelines 1990/1991; American Academy of Neurology 1997; Ayd 2000);
- After the mid-twentieth century, major medical journals and bodies have provided consensus definitions of closed head injuries that clearly reflect strong family resemblances but were updated to reflect new information and sometimes new nomenclature preferences (*e.g*. British Medical Journal: 1941; The American Congress of Neurological Surgeons: 1966; American Congress of Rehabilitation Medicine 1993); and
- The MTBI committee of the National Football League, despite mounting what appears to have been a disinformation campaign about brain concussions in football, considered describing them as mild traumatic brain injuries as both unproblematic and a "natural extension" of the definition going back to the 1966 definition of the American Congress of Neurological Surgeons.

95. In sum, this historical record presents a very clear picture of the cause and effects of single concussive injuries. These observations do require, of course, acknowledging that there were moments when authors formulated definitions at variance with each other or sought to shift nomenclature patterns. A case in point appeared in the 1924, such as the famous article by a pioneer in British neurosurgery, Wilfred Trotter, offered what he took to be a classic definition for concussion that might lead to persistent symptoms:

> In one type the accident is a fall on the head, as for example, from a horse. The patient develops a slight concussion of the brain; there is momentary unconsciousness which is quickly recovered from, and followed by dizziness and headache, which clear up in a comparatively few hours. I may say at once that I use the term concussion, as I think it should only be used in the strict classical sense to indicate an essentially transient state due to head injury which is of instantaneous onset, manifests widespread symptoms of a purely paralytic kind, does not as such comprise any

> evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident.[65]

96. This article was a lecture Trotter presented at the Medical Society of London, later published both in the *British Medical Journal* and in *The Lancet*. In his lecture, and in other articles that followed, Trotter made very clear that he felt such persistent symptoms resulted from structural damage, and he wondered if it might be more accurate to recognize this fact by calling such injuries "contusions." This classification went against the then conventional wisdom, and although some endeavored to use it afterwards, it rapidly fell out of fashion.[66]

97. It is worth noting that Trotter's views appear to have been inadvertently misrepresented by the authors of a landmark paper on the neuropathology of what they termed postconcussion neuroses and traumatic encephalitis. Neuropathologists Osnato and Giliberti accepted Trotter's classical definition, but they made the following statement:

> (1) It is not possible to say in a given case of concussion, "that the state is essentially transient," unless one recognizes that these words simply apply to the unconsciousness and the immediate paralytic phenomena which accompany or follow the unconsciousness. (2) It is not tenable, in the light of what will follow, to advance the proposition that concussion of the brain "does not as such comprise any evidence of structural cerebral injury."[67]

98. Trotter's published record indicates little disagreement with Osnato and Giliberti's modifications. The only question being debated at the time was whether contusion was a more accurate word for describing the causes of such persistent symptoms. Some authors, most notably Charles Symonds, endeavored to make the

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[65] W. Trotter, *Annual Oration on Certain Minor Injuries of the Brain*, 1 BR. MED. J. 3306, 935-39 (1924).

[66] *Id.*; *see also* W. Trotter, *An Address on the Management of Head Injuries*, 2 LANCET 1925, 953-56 (1925); W. Trotter, *An Address on the Evolution of the Surgery of Head Injuries*, 2 LANCET 169-171 (1930).

[67] M. Osnato & V. Giliberti, *Postconcussion Neurosis - Traumatic Encephalitis. A Conception of Postconcussion Phenomena*, 18 ARCH. NEUR. & PSYCHOL. 2, 181-214 (1927).

modification in the way they wrote about minor head injuries in that period, but all eventually gave the effort up. A careful reading of the record suggests that these experts were unhappy with the word concussion chiefly because concussion denoted in popular understanding a recoverable injury. The writings of authors like these from the period 1920s and to the mid-1930s indicate that they all were concerned by those rarer concussive injuries in which recovery did not follow.[68]

99. There are other examples of fairly narrow scientific and clinical debates likes these throughout the period 1871 to 2011. Stepping back, it is easy to see that it would have been a comparatively simple historical mistake for later medical and scientific practitioners, often highly busy individuals, to have selected one or two of the articles from this period and consequently misinterpreted them as revealing fierce debates in the past over the meaning of the word concussion. It is obvious historically that it would be possible to create a mountain out of a mole hill through inadvertent selective citation and thus conclude there has been little or no medical consensus about the definition of the word "concussion" across the period 1871 to 2011. A more thorough reading of the historical record indicates that such a view is incorrect and exaggerated.

100. Nevertheless, it appears to be the case that some scientific and clinical writers after 1990 increasingly claimed that the historical record offered little consensus or useful research on concussion that occurred in sport. They pointed to single moments in the records that may have shown controversy and concluded from these that both the definition of concussion was controversial and that there was no universal agreement about it. It is a noteworthy feature of the historical record that many of the authorities who have expressed concern after 1990 that the definition of concussion failed to achieve "universal agreement" studied what they called "sport concussion" or "sport-

[68] C. P. Symonds, *Observations on the Differential Diagnosis and Treatment of Cerebral States Consequent Upon Head Injuries*, 2 BR. MED. J. 3540: 829-832 (1928); C. P. Symonds, *The Effects of injury Upon the Brain*, 219 LANCET 5668, 820-23 (1932). For other authors, *see, e.g.*, D. Armour, *Some Considerations on Head Injuries*, 51 BRAIN 4, 427-39 (1928).

related concussion" as a trending topic in the comparatively new clinical specialty sports medicine.

101. A single example suffices for illustration: In 2001 Karen M. Johnston, Paul McCrory, Nicholas G. Mohtadi, and Willem Meeuwisse authored an essay in the *Clinical Journal of Sports Medicine* entitled "Evidence-Based Review of Sport-Related Concussion: Clinical Science". The authors began their article by declaring that there was "no universal agreement on the standard definition or nature of concussion." They observed: "Despite over 1000 years of conceptual development, the diagnosis and management of concussion remains controversial." The historical context, they asserted, "refers to a transient disturbance of neurologic function caused by the 'shaking' of the brain that accompanies low velocity brain injuries."[69] To evidence their claims, these authors pointed to writings from medical antiquity, the early modern period, and the late eighteenth century.[70] They also selected a couple of mid-twentieth century writings by Derek Denny Brown and William Ritchie Russell, a 1966 definition offered by the Ad Hoc Committee to Study Head Injury Nomenclature of the Congress of Neurological Surgeons, and the original studies detailing the Glasgow Coma Scale, a standard means of distinguishing severity of brain injury that was published in 1975. Noting that in their view the Glasgow Coma Scale could not be used to distinguish sport-related concussion, the authors stated: "Current definitions of concussion remain unsatisfactory from the sporting injury standpoint."[71]

102. It is, of course, impossible to disagree with the authors that there is "no universal agreement," since the authors' own apparent disagreement alone would suffice as evidence of their claim. However, the historical record makes abundantly clear that the changes in the definition have been towards greater scientific and clinical precision rather than a complete transformation. It seems, moreover, an unduly high standard to

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[69] K.M. Johnston et al., *Evidence-Based Review of Sport-Related Concussion: Clinical Science*, 11 CLINICAL J. SPORT MED. 3, 150-59 (2001).

[70] *Id*. at 156.

[71] *Id*. at 150-51.

insist as a measure for the progress of medicine and science that there should have been constancy of definition across a *millennium* of cultures, languages, and medical traditions – there might be no example in the history of medicine that would meet this standard. Certainly translation issues alone would make this highly problematic. Meanwhile, a more pragmatic measure that can be derived from the historical record is that for the last one-hundred forty-five years, at least, clinical observers have broadly agreed that brain concussion patients tend to recover but sometimes do not. For these reasons, the practices and research writing of healthcare professionals over this time has focused on a precautionary approach (striving to avoid harm in the absence of certain knowledge) to the management of brain concussion patients and also enjoined physicians to highly regard the risks and to help avoid the occurrence of concussion in the first place.

2. Sports Concussions are Brain Concussions

103. The fact that brain concussions are a common event in professional contact sports does not suggest that sport concussions are different injuries from brain concussions received in other contexts.[72] Above all, the sources from 1871 to 2011 demonstrate that concussion can happen in any setting, and although the physical concussive force on the brain can differ, any concussion carries risk. A review of the type and nature of medical and scientific literature on concussion since the late nineteenth century illustrates this finding in various ways: Early individual case reports or small group studies drew from a range of patients who had received concussions from

[72] It does deserve stipulating that there is circumstantial evidence that the National Hockey League, for example, appears to have used a different definition for the word "injury" than that used commonly in medicine. If this tendency was reflected more broadly throughout professional sports organizations, then it is possible that authorities studying sports concussion utilized industry standards in articulating the difference. As authors explained in 1978: "The National Hockey League, however, defines an injury as one that causes a player to miss five league games. This definition excludes all minor injuries, some fractures and head injuries, and most lacerations." D. Hayes, *Reducing Risks in Hockey: Analysis of Equipment and Injuries*, 6 PHYSICIAN & SPORTSMEDICINE 1978, 68 (1978). To be very clear, I do not know if this observation was true, nor have I found any evidence that sport concussion researchers understood injury in this way.

a wide variety of causes and a wide variety of settings. Studies utilizing a consecutive hospital case selection methodology similarly saw little need to distinguish concussion patients, save in rare instances to exclude those that might create ambiguity in the pool. Prospective study methodologies emulated this pattern, as did epidemiological surveys and regional studies of large patient populations. Even some of the pioneering studies of athletic populations were conducted precisely to inform from the specific population to the general case.

104. Thus, this large number of sources makes apparent that for the majority of the period in question, medical researchers, and doctors treating patients with concussion, ***did not*** differentiate between concussion in different contexts. They show that authors recognized that, regardless of the severity of concussive injury, behavioral sequelae could follow. They were also aware that permanent symptoms, debility, and diminished capacity for work sometimes followed concussions, although most authorities in the past acknowledged that such instances traumatic sequelae occurred only rarely. When studies focused on concussion from one particular arena or walk of life – such as professional contact sports or motor vehicle accidents – they did so typically to shed light on concussion as a whole.[73]

105. This pattern held true until the 1990s. In the 1990s the specialty of sports medicine bifurcated concussion research. Some specialists in this field implied, intentionally or otherwise, that concussion in sports was a special case and that conclusions and knowledge about brain concussion established in the wider medical sphere over the previous century were in some way unsatisfactory when it came to sports concussion. This pattern may, it should be noted, have been a natural effect of the specialization of medicine, whereby sports medicine researchers became effectively "cut off" from broader research patterns outside of their immediate and narrow interest and

[73] Epidemiological studies published in 1980 illustrate a pattern reflected throughout the historical record. See W.D. Kalsbeek *et al*., *The National Head and Spinal Cord Injury Survey: Major Findings*, J. NEUROSURGERY 53 Supplement: S19-S31 (1980); also J.F. Annegers *et al*., *The Incidence, Causes, and Secular Trends of Head Trauma in Olmsted County, Minnesota, 1935-1974*, 30 NEUR. 9, 913 (1980).

conversations. Regardless of the cause, from the 1990s two cohorts emerged. One was concerned with using concussion in sports as a laboratory for studying brain concussion in general and within the broader context of concussion overall. The other appears to have become concerned by questions of management as it related to athletic experience, and perhaps for this reason, came increasingly to assert the novelty of the sports arena context.[74]

106. These trends in medical and scientific studies showing that concussions were investigated in a broad variety of contexts and settings is explored in this section under the following chronological headings:

- Case reports, small group studies, and prospective studies of brain concussion
- Epidemiological studies of brain concussion
- Bifurcating brain concussion: concussion in sports arenas

a. Case Reports, Small Group Studies, and Prospective Studies of Brain Concussion

107. Almost all clinicians and scientists who studied closed head injuries from 1871 to 2011 relied upon concussion patients who received their injuries in a variety of settings. In the late-nineteenth century when authors described common injuries, they typically relied upon descriptions of one or a few exemplary cases. In some cases, individual patients were reported with uncommon symptoms. Yet the main function of the general case studies was to draw out the normal ways any brain concussion patient would present to their doctors. When investigators wished to make broader arguments about concussions, they also sometimes offered multiple case reports in their published writing. Multiple case reports allowed authors to illustrate different symptoms and outcomes accompanying concussive injuries. As will be described below, such single instances were usually offered to illustrate general tendencies.

[74] J. P. Kelly, as just one example, advocated for thinking of concussions in sports arenas as tantamount to a "natural head injury clinical laboratory." *See* J.P. Kelly, *Preface*, J. HEAD INJURY REHABILITATION 13, 2, p. v. (1998).

108. Individual case reports appeared commonly before the 1950s, but they became rarer after that time (although they did not vanish). At the same time, from the early-twentieth century on, clinicians and scientists found other ways to analyze broad pools of concussion patients. They designed studies using consecutive hospital admissions (*i.e*. selection of patients as they appeared in hospital) of head injury patients to create population for research. They also produced retrospective studies of hospital records of head injury patients. These studies looked back at hospital patient records and pulled the various relevant cases to form a population pool for analysis. Initially such studies relied upon descriptive statistics and modest statistical analysis. With the invention of computers, more complicated analytical studies appeared, including by the 1960s prospective studies of hospital admissions that used punch cards to increase analytical power.[75] These crucially provided clearer follow-up information about patients' conditions at given points after their initial admission and treatment (*i.e*. one month, one year, etc). The pronounced tendency of all of these methodologies was that their designers analyzed patients as they appeared and from whatever context they came and without concern about the cause of the injury.

109. Of all of these types of studies it is noteworthy that few studies made any attempt to exclude patients, a fact underscoring how the context of where head injuries occurred was understood by past clinicians to be insignificant for their research purposes. Many had by the 1930s expressed concerns, however, that compensation litigation (pending or ongoing) could aggravate and possible encourage the exaggeration of patient symptoms. One novel 1944 prospective study by P. G. Denker excluded such patients precisely because he wished to exclude any potentially confounding facts that might create uncertainty in the validity of the research findings.[76] As Denker explained:

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[75] The first study using computer analysis was A. H. Auerbach, A. E. Scheflen, R. B. Reinhart, and C. K. Scholz, *The psychophysiologic sequelae of head injuries,* 117 Amer. J. Psych. 6, 499-505 (1960)

[76] P. G. Denker, *The post-concussion syndrome: prognosis and evaluation of the organic factors*, N.Y. STATE J. MED. 44, 379-84 (1944).

> Though all neurologists can readily attest to the frequency with which cases of "postconcussion syndrome" or "post-traumatic encephalopathy" are encountered in practice, too little factual data as to the prognosis in these patients are available. As one reviews the voluminous literature on this subject, one is struck by the comparatively few studies of a follow-up type in so important a subject. There is still too much difference of opinion between capable men on the cause of these often prolonged symptoms, and it was with the hope of shedding a bit more light on this controversial matter that the present study was undertaken.[77]

Thus Denker's study is an example of one of the few scenarios in which the background context of a concussion was considered – as an exclusionary device rather than a special category of concussion.

110. In the context of this declaration, the details of Denker's findings are worth closer examination. There were 100 cases presented in Denker's resulting study involving 58 women and 42 men. In 12 cases there had been no loss of consciousness. The causes of the concussion in Denker's study were many. He said, "Practically all of the head injuries in this series resulted from the carelessness or misfortune of the patient – falls in homes, automobile accidents in which the patient had been at fault, and falls on slippery, icy pavements were the predominant causes."[78] According to Denker:

> The symptoms complained of were remarkably similar: headaches, dizziness, and a heterogeneous group of "nervous" changes, such as irritability, weakness, difficulty in concentration, insomnia, antisociability, hyperacusis, etc. These latter symptoms, if taken alone, bore a strong resemblance to the usual complaints of the neurotic and have been aptly named "nervous instability" by Symonds. Unlike the neurotic, however, in many these cases, the patient did not stress these vaguer symptoms, but complained chiefly of the headaches or dizziness so that one often had to get the more complete picture of personality change from the wife or husband or a business associate before one could properly appreciate the severity of emotional change that had occurred.[79]

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[77] *Id*. at 379.

[78] *Id*.

[79] *Id*. 380

111. Denker noted that such patients often enquired as to how long their symptoms would last. He observed that there was no clear answer to that question: "In this series, headache was complained of in 62 per cent of the cases. It was often described as "bursting," or "hammering," and varied in location, though again in contrast to the neurotic, it was only rarely referred to as a "pressure on the top of my head". Sudden changes in the position of the head would often bring on a paroxysmal attack of severe headache and dizziness, occasionally with nausea. These headaches persisted in varying duration, from less than twenty-four hours, to well over three years and in 20 per cent of the cases were still present after three years."[80]

112. In the context of this declaration, it should be noted that Denker's findings, drawn upon a study in which patients with pending litigation were excluded, nevertheless resulted in findings of great consequence for patients whose injuries were being contested in the courts. Denker made clear that his study showed that head injuries, including concussive injuries, could result in permanent symptoms:

> As a result of the above follow-up study, it would seem that more caution should be observed in rendering too optimistic a prognosis in cases of cerebral concussion. In round figures, only about 10 per cent were symptom-free in a month, and at the end of a year, about 30 per cent were still suffering from the usual triad of headache, dizzy spells, and nervous instability… 15 per cent of the patients were still suffering from symptoms after three years and in these cases the sequelae seemed permanent. Though at first glance these figures appear to make for a rather dismal prognosis, it must be borne in mind that a good many of the cases were seen only after the symptoms had already been persistent for a considerable period of time. The neurologist is not, as a rule, called in to see all cases of cerebral concussion, especially if the period of unconsciousness is minimal and the patient's progress seems to be satisfactory.[81]

113. Denker's prospective study was typical of hospital analyses of concussion patients after the Second World War. This pattern was seen in the original studies that

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[80] *Id.*

[81] *Id.* at 381. It should be noted that Denker admitted that the design of his study may have exaggerated the number of patients with persistent, permanent symptoms.

were used to validate the Glasgow Coma Scale, a measuring device that allowed doctors to quickly assess the severity of head injury (including concussive injuries).[82] The pattern was used as well by specialists in emergency departments who were seeking to improve protocols for immediate patient care.[83] Psychologists, too, used such broad categories. Perhaps the best evidence comes from major epidemiological studies of head injury published in 1980 and 1981, and discussed below, which make clear that the context of such injuries only mattered to healthcare professionals in terms of targeted prevention, rather than because of the belief that the context of the injury would affect either the symptoms or the prognosis.[84]

b. Epidemiological Studies of Brain Concussion

114. The first major epidemiological survey of head and spinal injuries in the United States was funded by the National Institute of Neurological and Communicative Disorders and Stroke and published in 1980. That study focused on the year 1974 and recorded the estimated frequency and economic costs of head and spinal injuries. The authors estimated that 422,000 cases of head injury occurred in that year, at a rate of 204 cases per 100,000 of population. Males experienced head injuries at more than twice the rate for females, and individuals between 15 and 24 were most likely to have a head injury. The authors identified leading causes of concussive head injuries: approximately 80 percent of head injuries in automobile accidents were concussions, approximately 75 percent of head injuries in falls were concussions, and approximately 65 percent of "all other" external causes of head injuries resulted in concussions.[85]

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[82] The Glasgow Coma Scale was introduced by G. Teasdale and B. Jennett, *Assessment of Coma and Impaired Consciousness*, THE LANCET 81-83 (1974).

[83] *See, e.g.*, L. E. Weeks, *Handling of nonsevere head injuries*, 8 JOURNAL OF THE AMERICAN COLLEGE OF EMERGENCY PHYSICIANS 7, 257-60 (1979).

[84] An obvious example would be safety belt legislation.

[85] W.D. Kalsbeek *et al*., *The National Head and Spinal Cord Injury Survey: Major Findings*, J. NEUROSURGERY 53 Supplement: S19-S31 (1980).

115. The pattern of that national epidemiological survey was replicated in more focused regional studies. Crucially, these added an additional evidentiary finding on the risks of concussion, namely the observation that the risks of receiving a concussion were substantially higher in populations of patients who had already received a prior concussion. In a work of historical epidemiology the incidence, causes, and trends in head trauma were examined retrospectively in Olmsted County in Minnesota between 1935 and 1974. In this study, Annegers and colleagues used Mayo Clinic records and examining head injuries found 3,587 instances of head trauma per 100,000 of population of which "3,3337 were the first known head injuries for individuals; 250 were second or later injuries."[86] This observation clarified the comparative rarity of individuals being hospitalized twice (at least in the same hospital) during a forty year period. Their survey used a grading system of concussion severity recently introduced by Ommaya and Gennarelli (*see* Figure 6). Annegers and colleagues excluded 'dings' or what in the Ommaya and Gennarelli system were called grade 1 concussions.[87] Like the national survey, Annegers *et al* also found that road accidents and falls were leading causes of head injury, but they also observed that recreational activities were represented in this population. There were, for example, thirty-one cases of ice-hockey players in a cohort of 333 recreationally caused head injuries. Assaults (excluding gunshot victims) were also found to be a leading cause of head injury, with a peak in males age 15 to 24. Importantly, these authors also established that individuals with a head injury were at a heightened risk for a second injury, at a rate "about three times greater than before, and after a second injury it increased to eight times that in the general population."[88]

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[86] J.F. Annegers *et al*., *The Incidence, Causes, and Secular Trends of Head Trauma in Olmsted County, Minnesota, 1935-1974*, 30 NEUR. 9, 913 (1980).

[87] A.K. Ommaya & T.A. Gennarelli, *Cerebral Concussion and Traumatic Unconsciousness: Correlation of Experimental and Clinical Observations on Blunt Head Injuries*, 97 BRAIN 1, 633-54 (1974).

[88] Annegers, *supra* n. 81 at 916.

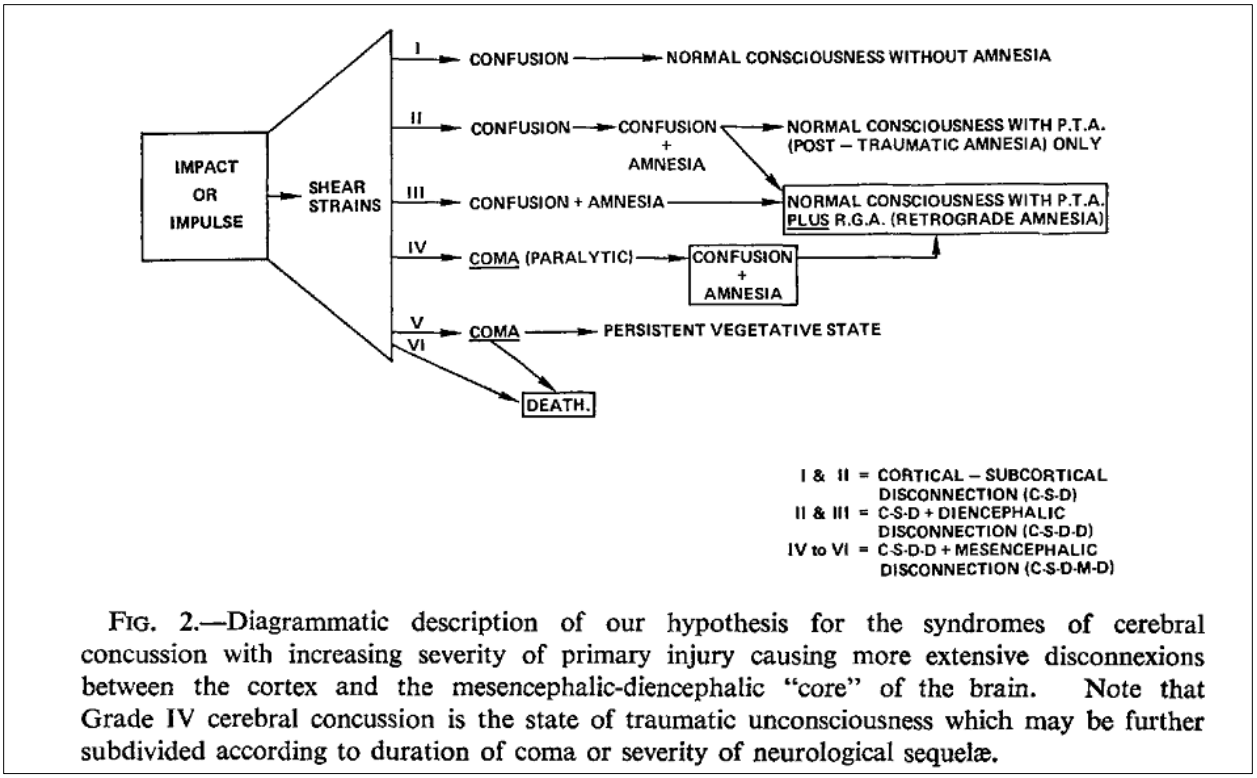

FIG. 2.—Diagrammatic description of our hypothesis for the syndromes of cerebral concussion with increasing severity of primary injury causing more extensive disconnexions between the cortex and the mesencephalic-diencephalic "core" of the brain. Note that Grade IV cerebral concussion is the state of traumatic unconsciousness which may be further subdivided according to duration of coma or severity of neurological sequelæ.

Figure 6. Ommaya and Gennarelli's 1974 grading system for cerebral concussion, which appeared in the journal *Brain*. In a later article, Ommaya clarifed that Grade 1 concussions were "dings".[89]

116. In the 1980s other epidemiologists also examined smaller communities, this time in San Diego and Chicago. Both studies closely paralleled the findings of previous and contemporaneous epidemiological studies. The author of the San Diego study reported on serious impairments resulting from many different sources of head trauma, among these: "…12 percent to assaults. Sports and recreational activities (*i.e.* organized sporting events, use of pedal cycles not in traffic, and pedestrian conveyances such as roller skates or skate boards) account for almost 10 per cent." Kraus continued: "The occurrence of brain injury from sports and recreation-related causes was six times more frequent among males than among females. Rates of injury for males were highest in the age group 10-14 years and declined thereafter."[90] Importantly, Kraus and

[89] Ommaya, *supra* n. 82; for the explanation of the system as it subsequently evolved, *see* A.K. Ommaya, *Head Injury Mechanisms and the Concept of Preventive Management: A Review and Critical Synthesis*, 12 J. NEUROTRAUMA 4, 527-46 (1995).

[90] J.F. Kraus *et al*., *The Incidence of Acute Brain Injury and Serious Impairment in a Defined Population*, 119 AM. J. EPIDEMIOLOGY 2, 192 (1984). It is worth noting that

colleagues were interested both in incidence and in outcome in their study. In their conclusion, they sought to heighten awareness of the gravity of their findings: "The incidence of brain injury in the US population, the high case fatality rate, the significant level of neurologic impairment in those discharged alive with moderate or severe levels of injury on admission, and the evidence that mild injury may have significant unfavorable sequelae, point to an extremely important public health problem."[91] Like other authors, the issue was not the cause per se but rather the ramifications of a 'do nothing approach' for a complacent population.

117. The Chicago study, conducted in 1980 and published in 1984, augmented this growing and alarming picture of the consequences of concussions and other head injuries by showing that there were dramatic differences in injury experience, depending on race and income. Inner city African Americans were found to be at a higher risk for head injury than suburban African Americans, who in turn were at a higher risk than suburban Caucasian Americans.[92] The leading cause of head injury in the inner city was person-to-person violence; the leading cause for suburban residents were automobile accidents. The injury was the same, regardless of its cause. Where the San Diego case particularly called attention to gender and age characteristics, the Chicago case called attention to the implications of race and class in the incidence of concussions.

c. Bifurcating Brain Concussion: Concussion in Sports Arenas

118. The historical record, then, makes clear that up to the 1980s, brain concussions were broadly understood to be an injury, the causes and settings of which were many. In the 1980s, these patterns could also be found in the writings of authors

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other studies of San Diego County had occurred prior to this M. Klauber *et al*., *The Epidemiology of Head Injury: A Prospective Study of an Entire Community - San Diego County, California, 1978*, 133 AM. J. EPIDEMIOLOGY 5, 500-09 (1981).

[91] *Id*. at 201.

[92] S. Whitman *et al*., *Comparative Head Trauma Experiences in Two Socioeconomically Different Chicago-Area Communities: A Population Study*, 119 AM. J. EPIDEMIOLOGY 4, 570-80 (1984).

who were concerned about concussions that occurred in amateur and professional contact sports. Although physicians and scientists had contemplated the severity of concussions in leisure activities – hunting and recreational sports – and had speculated since the nineteenth century that such concussions were usually less severe than those in other activities, only rare specialists in sports medicine appear to have insisted on differences before the publication of sports concussion management guidelines in 1990-1991, save to acknowledge that these injuries tended to recover. For example, in his pioneering stab at establishing sport guidelines for managing "cerebral concussion" in 1986, Dr. Robert C. Cantu appears to have been guided by similar general scientific and clinical research studies of concussion as those being used by authors in others areas.[93] The same was true of the literature published in the Colorado Medical Guidelines for managing concussions in contact sports in 1991.[94]

119. Yet by the mid-1990s the notion of "sports concussion" had begun to emerge as a distinct concept within the medical and scientific literature; in the hands of some authors "sports concussion" became an entity in itself, rather than a concussion that happened to have occurred in the context of a sporting arena. These studies appeared to argue implicitly that research design required making a distinction between sports concussion and non-sport concussions.

120. A keyword search of the US National Library of Medicine indexing database, PUBMED, highlights the emergence of the keyword term "sports concussion" in the second half of the 1990s (*see* Figure 7). In an editorial he published in the *British Journal of Sports Medicine*, Australian Paul McCrory, editor of that journal and an author listed in the Sports Concussion Consensus Statements, for instance, observed in 2001:

> Concussive injuries in Australian Rules football tend to be mild, with neuropsychological performance returning to levels found before injury

[93] R.C. Cantu, *Guidelines for Return to Contact Sports After a Cerebral Concussion*, 14 PHYSICIAN & SPORTSMEDICINE 10, 75-83 (1986).

[94] Sports Medicine Committee of the Colorado Medical Society. *Guidelines for the Management of Concussion in Sports*, MED. SOCIETY, June 1-4 1991.

> within a few days. Similarly, post-concussive symptoms such as headache, nausea, poor concentration, and fatigue also resolve within the first few days of injury. The classical "post-concussive syndrome" often seen after motor vehicle accidents and other forms of severe traumatic brain injury is exceedingly rare in sport.[95]

121. The supposition underlying McCrory's criticism appears to have been that there was a false equivalence between sports concussion and brain concussions suffered in most other contexts.

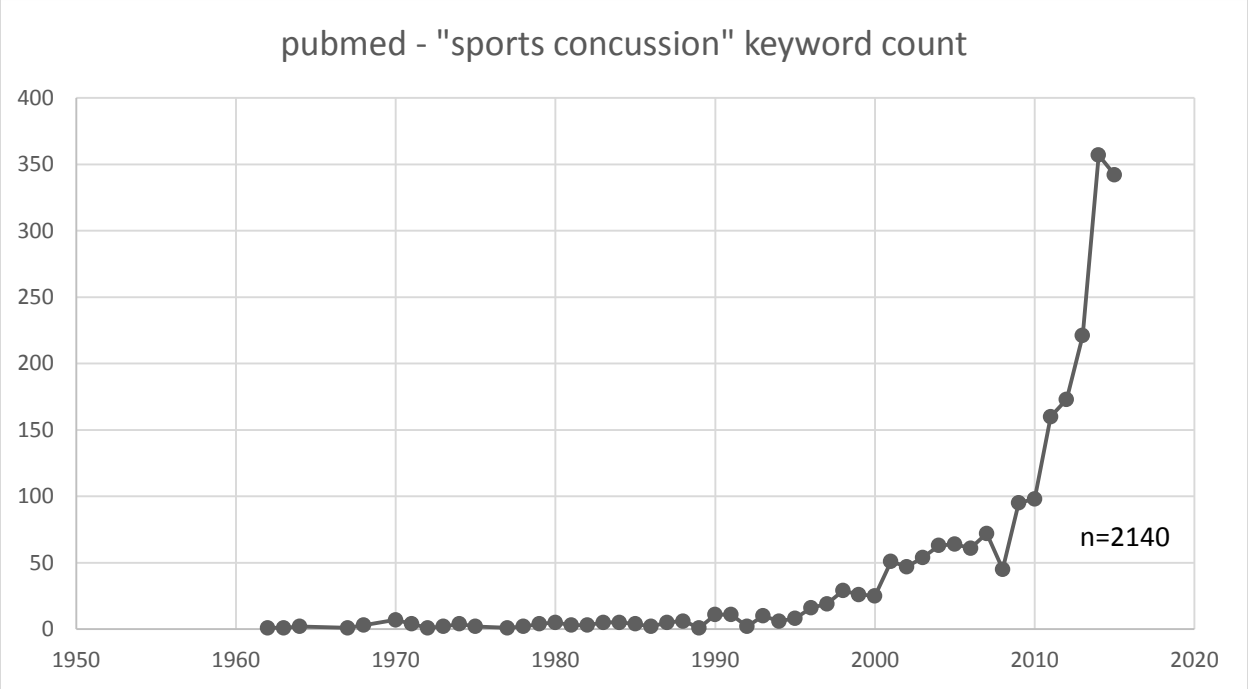

Figure 7. Pubmed generated timeline for the keyword "sports concussion."

122. Such a claim, when compared with the history of concussion research, is novel. In this sense the separation of "sports concussion" from "brain concussion" is without historical precedent. McCrory's claim appeared to be grounded in the notion that because concussions that occur in sports might be less severe than those that happen in other contexts, this separates their management and prevention concerns from similar efforts for brain concussion generally. The most noteworthy difference was that sports neuropsychologists and sports neurologists increasingly focused their attention on applied concerns within sports arenas only (sideline evaluation, return to play guidance,

[95] P. McCrory, *When to retire after concussion?* 35 BR. J. SPORTS MED. 6, 381 (2001).

baseline neuropsychological testing, etc). By increasingly focusing upon the immediate questions surrounding athletic injuries, interest and apparent awareness in medical and scientific research on concussion as a whole was, as it were, relegated to the sidelines.

123. These trends, however, continued. By a 2005 international meeting in Prague, some of the profound limits introduced by the notion of sports concussion became manifest, at least as seen in consensus proceedings published afterwards. The authors, including Paul McCrory as lead, claimed to have revised a definition of concussion from that proposed by a 1966 Ad Hoc Committee to Study Head Injury Nomenclature working within the Congress of Neurological Surgeons. The Prague group's revised definition stated: "Sports concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical features."

> Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include the following. (1) Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head. (2) Concussion typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously. (3) Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury. (4) Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. (5) Concussion is typically associated with grossly normal structural neuroimaging studies. No changes were made to the definition by the Prague Group beyond noting that in some cases post-concussive symptoms may be prolonged or persistent.[96]

124. Not only did that definition provide no clear explanation for why it differentiated sports concussion from brain concussion, it is not clear that it represented anything more than a natural extension of greater scientific precision from the 1966

[96] P. McCrory et al., *Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004*, 39 BR. J. SPORTS MED. 4 (Suppl I): i78-i86, p. i78 (2005).

definition, which defined concussion as "A clinical syndrome characterized by immediate and transient impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium etc., due to mechanical force."[97] For that matter, the Prague definition was not all that different from the slightly earlier definition of mild traumatic brain injury introduced in 1993 in the *Journal of Head Trauma and Rehabilitation*, which effectively stated the same thing:

> A patient with mild traumatic brain injury is a person who has had a traumatically induced physiologic disruption of brain function, as manifested by at least one of the following: 1. Any period of loss of consciousness, 2. Any loss of memory for events immediately before of after the accident, 3. Any alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused), and 4. Focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following: loss of consciousness of approximately 30 minutes or less; after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and posttraumatic amnesia (PTA) not great than 24 hours.[98]

125. Nonetheless, this new specificity for the definition of sport concussion in the Prague statement resulted in further claims:

> At this time, there is no existing animal or other experimental model that accurately reflects a sporting concussive injury. It is noted that, in experimental models, of more severe injury a complex cascade of biochemical, metabolic, and gene expression changes occur. Whether similar metabolic changes occur in sports concussion, however, remains speculative at this time.[99]

126. Thus, among some sports medicine figures there arose a claim that the difference between sport concussion and brain concussion was great enough to make longstanding and ongoing findings in concussion research elsewhere in medicine potentially distinguishable to sport concussion injuries.

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[97] Surgeons, Congress of Neurological, *Report of the Ad Hoc Committee to Study Head Injury Nomenclature*, CLINICAL NEUROSURGERY 12, 388 (1966).

[98] American Congress of Rehabilitation Medicine, *Definition of mild Traumatic Brain Injury*, 8 J. HEAD TRAUMA REHABILITATION 3, 86-87 (1993).

[99] McCrory *et al.*, *supra* n.91 at p. i79.

127. The apparently increasing isolation of sports concussion from brain concussion research generally was not inevitable, and several early figures studying sports concussion initially imagined that sports arenas might provide an ideal testing ground for exploring minor traumatic brain injuries.[100]

128. For example, the preface by neurologist James Kelly in a special issue of the *Journal of Head Trauma and Rehabilitation* in 1998 contrasts starkly with the aforementioned discussion. Kelly had by then become a prominent figure in the development of concussion management guidelines for athletes. In his opening remarks, Kelly acknowledged that "sports-related concussions" were not usually the kind of injuries that regular readers of the journal might expect to see in the pages of that journal, because, as he put it with rhetorically open-ended question: "Is it not true that most people who sustain concussion recover spontaneously without professional help?"[101] At the same time, Kelly argued that sports concussion could be seen as a specific case useful for informing more general study:

> It is intriguing to think of the occurrence of concussion in sports as an opportunity to study traumatic brain injury in a truly unique way. The sports arena serves as a laboratory setting for the advance of athletic injury. The opportunity to learn about traumatic brain injury in this way has not been fully explored, and the obligation of health care professional to do so for the safety of athletes is only now coming into public discussion…we usually have eyewitness reports of the concussion and its effects on the individual. We frequently have videotape images of the blow that caused the concussion, often from two or three different angles. We can test helmets and playing surfaces that were involved. None of these factors are typically available for review and analysis by professionals examining patients in the general population who sustain concussion from other more common causes. Athletes and society in general stand to benefit from improvements in neuroscience as applied to the sports setting….[102]

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[100] For a discussion, *see* S. Bender, *Historical Perspectives* (2004) in M.R. Lovell *et al*., *Traumatic Brain Injury in Sports: An International Neuropsychological Perspective* 6 (2004).

[101] J.P. Kelly, *Preface*, J. HEAD INJURY REHABILITATION 13, 2, p. v. (1998).

[102] *Id*. at v-vi.

In other words, in the study of concussion in sport, Kelly highlighted the great opportunity to study concussion that was simply not available in other settings. Athletic sports provided a controlled setting with unequalled possibilities for data collection.

129. The emergence of the category "sport concussion" thus represents a very significant departure from historical patterns of concussion research and management.[103] As we now know from the result of investigative journalism and a congressional report, from the mid-1990s to 2009, the National Football League was involved in a concerted effort to manipulate research and mislead representations of the severity of concussion experienced by NFL players, and underplay the potentially long-term risks or damaging effects of concussion and repeated exposure. An investigation of the medical and scientific literature published in leading peer-reviewed journals, such as this declaration is based on, ***cannot*** shed light on whether these types of activities occurred elsewhere in other professional sports. Yet, given the politics surrounding concussions and contact sports that emerged in the 1990s, the historical record does call to mind parallel activities in a far different corporate setting.

130. Assertions of the different nature of safety concerns accompanying "sports concussion" bares some similarity to the way that light cigarettes were marketed. Tobacco companies, according to medical historian Robert Proctor, purposefully sold the light cigarette as a 'healthy' alternative to regular cigarettes, which they knew entirely to be a medical fabrication.[104] This declaration is not intended to suggest that any authors cited above were engaged in any kind of similar deception. Equally, in the published medical and scientific literature read for the production of this declaration,

[103] There were, in earlier periods, studies of concussions sustained in such sports as boxing and football. But earlier authors and commentators did not appear to have made any discrimination between concussions and "sports concussions". Some commentators, however, did wonder about gradations of severity. See, for example, Ernest Sach's comments in the discussion at the conclusion of M. Osnato & V. Giliberti, *Postconcussion Neurosis - Traumatic Encephalitis. A Conception of Postconcussion Phenomena,* 18 ARCH. NEUR. & PSYCHOL. 2, 214 (1927).

[104] R.N. Proctor, *Golden Holocaust: Origins of the Cigarette Catastrophe and the Case for Abolition* 408-09 (2011).

there appears to have been no obvious intention to mislead behind the emergence and growth in popularity of the notion of distinct "sports concussion," as opposed to concussions experienced in other walks of life. Nevertheless, the formulation of "sports concussion" as a concept manufactured uncertainty and inconsistency in the medical record in a context where broad concerns about litigation were mounting, and informed consent a point of contestation. Into the 2000s this became even further problematic.

131. It is worth observing that these issues have particularly grave importance not only for athletes working in professional sports, but also for other victims of minor traumatic brain injury. If suggesting that what has been shown true of concussion generally across a vast territory of medical and scientific literature ***may not apply*** to "sports concussion" leaves room for the potential downplaying of the risks from concussion its professional players receive, then the same case might be applied to patients experiencing sustained effects from minor concussion obtained in other contexts. This is especially true of situations in which repeated exposure to concussion has been a concern. The risks to this general population of minor concussion victims are described with peculiar force in a 1999 article entitled "Head injury and Battered Women":

> …continual physical battering, which may not be considered severe, may put [battered women] at risk of subtle but chronic head injury. In addition, although symptoms are usually subtle, they may be sufficiently debilitating to create disruption in the person's daily life, employment status, and somatic and psychological adjustment.[105]

3. History of Nomenclature for Long-Term Consequences of Concussive and Closed Head Injuries

132. Although the word concussion enjoyed stability in terms of its definition for medical practitioners across the period from 1871 to 2011 (*see supra* Section V.C.1), the nomenclature describing the long-term behavioral consequences of closed head injuries evolved to reflect the emergence and specialization of psychiatry, psychology,

[105] K. Monahan & K.D. O'Leary, *Head injury and Battered Women, An Initial Inquiry*, 24 HEALTH & SOCIAL WORK 4, 270 (1999).

neurology, and neuroscience as fields of medical knowledge (*see* Figure 8). In the late-nineteenth century, the behavioral consequences of these injuries were typically referred to as "traumatic insanity".[106] This term reflected the many common uses of the term "insanity" in the nineteenth century, which historians of medicine have described extensively. In the first two decades of the early twentieth century, these conditions were sometimes called "traumatic neurasthenia",[107] "traumatic hysteria",[108] and "traumatic neuroses."[109] These terms reflected the broad influence of three major figures on psychiatry and neurology in that period, American George Beard, Frenchman Jean Martin Charcot, and Austrian Sigmund Freud. Beard meant the term neurasthenia to indicate the physical exhaustion of the nervous system. Charcot and Freud used the term hysteria to refer to what would now be described as psychogenic disorders. The term neuroses became a common catch-all term in the Progressive Era (1890s to 1920s), essentially updating and replacing the more derogatory term "insanity."[110]

133. By the late-1920s, some authors spoke of the behavioral consequences as occurring because of "traumatic encephalitis."[111] This term in the 1920s clearly drew upon and extended lessons from the behavioral consequences of a neurological

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[106] One of the first references in this study was T. W. Fisher, *Progress in Medicine: Report on Mental Diseases*, BOSTON MED. SURG. J. 90, 426-27 (1874).

[107] For the first appearance of this term, *see* J. S. Bury, *An Address on Trauma in Relation to Disease of the Nervous System*, 1 BR. MED. J. 2261, 997-1000 (1904)

[108] Of interest, the phrase chronic traumatic hysteria appeared in *The Traumatic Neuroses*. *Id.*

[109] The first usage of this term was in the 1890s, but it became a commonplace between 1900 and 1920. *Id.*

[110] For a discussion of terminology and these three major figures contributions, *see* A. Scull, *Madness in Civilization: A Cultural History of Insanity from the Bible to Freud, from the Madhouse to Modern Medicine* (2015).

[111] *See*, an earlier example was H P. Frost, *Traumatic Encephalitis*, 50 AM. J. INSANITY 3, 473-82 (1902); *see also* M. Osnato & V. Giliberti, *Postconcussion Neurosis - Traumatic Encephalitis. A Conception of Postconcussion Phenomena*, 18 ARCH. NEUR. & PSYCHOL. 2, 181-214 (1927); D. Armour, *Some Considerations on Head Injuries*, 51 BRAIN 4, 427-39 (1928).

pandemic of encephalitis that had begun in 1917 and continued worldwide until 1928.[112] A few authors noted that the behavioral disturbances following this illness bore a resemblance to symptoms that appeared in the wake of concussive head injuries.[113] By 1934, eminent neurologists extended those observations to describe the behavioral symptoms that sometimes followed concussions as "traumatic encephalopathy."[114] Those neurologists used this term as a general pathological explanation for the psychological condition "post-concussion syndrome," which became the preferred usage after World War II. By the 1960s, authors also spoke of "chronic traumatic encephalopathy," "post-traumatic syndrome," "chronic neuropsychological syndrome," "chronic brain syndrome," and "punch-drunk syndrome" to describe a similar constellation of behavioral symptoms and the underlying pathology in a permanent context.[115] Figure 8 shows this evolution of nomenclature schematically from the 1870s to 2011.

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[112] *See* for discussions of epidemic encephalitis: P. Foley, *The encephalitis lethargica patient as a window on the soul* (2012); L.S. Jacyna & S.T. Casper, *The Neurological Patient in History* (2012); *see also* K.K. Kenton, *Epidemic encephalitis and American neurology, 1919-1940*, 78 BULL. HIST. MED. 1, 108-47 (2004).

[113] For example, see the explicit discussion by M. Osnato & V. Giliberti, *Postconcussion Neurosis - Traumatic Encephalitis. A Conception of Postconcussion Phenomena*, 18 ARCH. NEUR. & PSYCHOL. 2, 181-214 (1927).

[114] I. Strauss & N. Savitsky, *Head Injury: Neurologic and Psychiatric Aspects*, 31 ARCH. NEUR. & PSYCHOL. 5, 893-955 (1934).

[115] E. Carroll, *Punch-Drunk*, 191 AM. J. MED. SCIS. 5, 706-12 (1936), describes on 706: "There is a clinical syndrome of frequent occurrence among boxers, to which they refer as "punch-drunk," "punchy," "goofy," "slap happy," "cutting paper dolls," or "slug nutty." Other terms might be applied, such as "traumatic dementia" or "traumatic encephalopathy," but they are not nearly so appropriate and descriptive as the epithet "punch-drunk." *See also*, Surgeons, Congress of Neurological, *Report of the Ad Hoc Committee to Study Head Injury* Nomenclature, CLINICAL NEUROSURGERY 12, 386-394 (1966). This document is a report that offers an effort to standardize head injury nomenclature by the Congress of Neurological Surgeons. The authors define several terms in a glossary-like document. "Concussion, brain: a clinical syndrome characterized by immediate and transient impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces." *Id*. 388. The authors also define: "Dementia, post-traumatic: A form of

neuropsychological disorder, post-traumatic with mental impairment." *Id*. 389. The authors also define: "Encephalopathy, traumatic: disturbance of structure and/or function of nerve cells, glia, or intracranial vessels resulting from injury." *Id*. 389. The authors describe: "Neuropsychological disorder (acute or chronic brain syndrome): disturbance of mental function due to trauma, associated with one or more of the following: 1) Psychotic manifestation. A disorder characterized by a varying degree of personality disintegration and failure to test and evaluate correctly reality in various spheres. Such persons fail in effective relationships to other people or to their work; 2) Neurotic manifestation. A personality disorder, in which such defenses as depression, conversion, dissociation, displacement, phobia formation, or repetitive thoughts and acts are utilized to handle anxiety. In contrast to psychoses these patients do not exhibit gross distortion or falsification of reality and usually do not present gross disorganization of the personality; 3) Behavioral manifestation. A disorder characterized by defects in personality structure with minimal anxiety and little or no sense of distress. (Occasionally encephalitis or head injury will produce the clinical picture, when the condition is properly diagnosed as "chronic brain disorder", with behavioral manifestation. The old term was "psychopathic personality."; 4) Psychophysiological manifestation. A disorder characterized by the visceral expression of affect, the symptoms due to a chronic and exaggerated state of the physiological expression of emotion, with the feeling repressed. These states have been termed "psychosomatic"; 5) Mental impairment. A disorder characterized by the development of intellectual defect as determined by psychological testing, by cultural, physical and emotional determinants, as well as school, vocational, and social effectiveness." *Id*. 392. The authors then defines "neuropsychological disorder, acute ("acute brain syndrome") lasting up to several months. Neuropsychological disorder, chronic ("chronic brain syndrome"): persisting months or indefinitely after injury." *Id*. 393. They define "Post-traumatic syndrome" as "a clinical complex characterized by headache, dizziness, neurasthenia, hypersensitivity to stimuli, and diminished concentration. Punch-drunk syndrome: a form of chronic neuropsychological disorder, presumably due to repeated head trauma, often associated with chronic alcoholism, characterized by emotional and/or mental impairment and/or motor deficit" *Id*. 393.

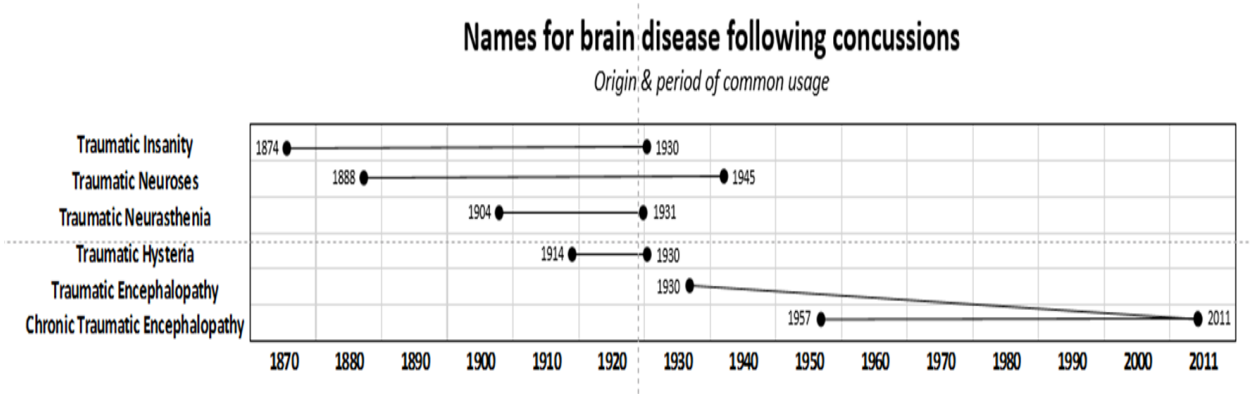

Figure 8. Depicted here are the most commonly used terms for brain disease following concussions in the period from 1871 to 2011. The earliest date for each reflects the year in which the term first appeared in the published literature consulted in this study. The last date reflects the period that best captures the end of common medical usage in this study for "traumatic insanity", "traumatic neuroses", "traumatic neuroasthenia", and "traumatic hysteria". Traumatic encephalopathy originated from the diagnosis "traumatic encephalitis" which came to be seen shortly afterwards as not sufficiently accurate. The addition of the word "chronic" to traumatic encephalopathy appears to have occurred in the postwar period. Both uses can be found after 1957, but with chronic traumatic encephalopathy becoming the preferred name as they merged together. Traumatic encephalopathy originally indicated both an acute and chronic state. The chronic state appears to garnered most attention in the post-war period. *See* Exhibit D for a bibliography.

134. The broader causes of these changes in nomenclature are complex in origin, but can be summarized thus:

- The emergence and specialization of medical and scientific fields concerned with the normal and abnormal functioning of the mind and brain;
- Neurological, psychiatric and psychological knowledge dramatically transformed in the nineteenth and twentieth-centuries;
- Historical stigma attached to mental illness often caused changing labels in an effort to depart from past expressions that had become culturally derogatory;
- Physicians, psychiatrists, psychologists, neurologists and others wrote about mental illness and disturbance through their own cultural lens; and
- All of those changes exerted influence on how mental disturbance was discussed generation by generation, particularly with the advent of Freudian theory.

D. Overview of the History of Concussive Head Blows and their Sequelae

135. The dangers of repeated concussions were first observed in 1872 by the British physician James Crighton-Brown, a later founder of *Brain: A Journal of Neurology*, who was at the time an insane asylum superintendent, and a leading figure nurturing the pathophysiological investigations of the brain. Commenting on the

dangers of repeated concussions, Crighton-Brown observed matter-of-factly (albeit in uniquely Victorian prose):

> Moral delinquency, with its preponderance of the lower feelings and propensities, is also an occasional consequence of cranial injuries; indeed there are grounds for thinking that some who come within the pale of the law as criminals, and who are incorrigible under all correction, have been plunged into vice at first, or have been confirmed in ill-doing, by that cerebral weakness which concussion, and more especially ***repeated concussion***, engenders. A career of dissipation exposes to injuries of the head — these cause a certain amount of weakness and irritability — and these again are enormously aggravated by a continuance in dissipation, so that a total loss of self-control, and an exaggerated activity of all the baser passions is soon reached….The crippling effects of a cranial injury are, in some cases, never suspected until a little excess demonstrates that the former vigour has departed, and that a condition of weakness and susceptibility has been established. If no error in regimen reveals this state of weakness and susceptibility — this change in the character of cerebral action — an unusual intellectual effort, or some little ailment may betray its presence, for the brain that has sustained an injury, and that after it bears with its wonted ease all ordinary burdens, will often break down lamentably when an extra load is imposed upon it, or when any visceral derangement supervenes. "I never knew that I was damaged," said a gentleman who had been thrown in the hunting field and had fractured his collar bone and been bewildered for a little, "until we came to balance up, and then I found that contemptible rows of figures baffled me, and that I was not the accountant that I had once been."[116]

136. As Crichton-Brown's observations suggest, already by the nineteenth century (1800-1900) the subtle effects of even a single concussion had been observed clinically to potentially produce grave changes in any patient. For this reason, it was also known that one concussion should necessitate avoidance of future concussions, for repeated concussion might ultimately result in even worse consequences. Crichton-Brown put the matter succinctly: "great caution ought to be observed ever afterwards by anyone who has experienced such an injury."[117]

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[116] J. Crighton-Browne, *Cranial Injuries and Mental Disease*, W. RIDING LUNATIC ASYUM MED. REP. 1, 97-136, 128 (1872).

[117] *Id.* at 128.

137. As the previous sections have already suggested, and as Crichton-Brown's study signals so well, knowledge of concussion was already fairly advanced by the closing decades of the nineteenth-century. Prior to the nineteenth century many of the most prominent clinical features of concussions had been observed and theorized by laymen, medical professionals, and scientists. Common symptoms such as loss of consciousness, headaches, dizziness, nausea, and memory loss had been observed in published medical and natural philosophical literature. Accompanying these studies were also reports of rarer associated permanent symptoms such as alexia, personality changes, dementia and even death without evidence of brain injury.[118]

138. Although several medical authorities had already begun to seek explanations for these symptoms as far back as the eighteenth-century, it was mainly in the nineteenth-century that the medical and scientific authorities turned their attention to systematic pathological, physiological, and clinical investigation of closed traumatic injury to the brain. Among the first investigators, historians have documented, was the military surgeon Jean-Pierre Gama who in 1830 "anticipated modern histological studies when he postulated that 'fibers as delicate as those of which the organ of mind is composed are liable to break as a result of violence to the head.'" On this hypothesis Gama began his novel experiments on a jar filled with jelly (a model head) and threads (model nerves) to study the effects of violent impacts.[119]

139. Gama's experimental work on concussion was much in keeping with the spirit of nineteenth century medicine. A medical revolution had taken place in France

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[118] Concussion research, and both common and rare symptoms in the nineteenth century, were discussed by past historians. *See* C.B. Courville, *Commotio Cerebri: Cerebral Concussion and the Postconcussion Syndrome in their Medical and Legal Aspects* (1953). Also relevant, with some caution, is M.R. Trimble, *Post-Traumatic Neurosis: From Railway Spin to the Whiplash* (1981). Other historical studies include H.S. Levin et al., *Neurobehavioral Consequences of Closed Head Injury* 1-8 (1982); H.S. Levin et al., *Mild Head Injury* 3-7 (1989).

[119] Quoted in H.S. Levin et al., *Neurobehavioral Consequences of Closed Head Injury* 6 (1982); on the experiments *see* M. Feinsod, *A Flask Full of Jelly: The First in Vitro Model of Concussive Head Injury 1830*, 50 NEUROSURGERY 2, 386-91 (2002).

and spread throughout Europe and to North America.[120] In France, the dissection of diseased and injured bodies had become a common practice by the close of the Napoleonic Wars (1797-1815). Mortuary pathology thus became medicine's gold standard of evidence. In other words, medical complaints and symptoms experienced during life were regularly being matched up with signs of lesions, abnormalities, and disease processes in the body when dead. This new system of medicine came to organize diseased or injured bodies through pathological knowledge and clinical inference. Soon it became clear that the causes of some pathological processes remained invisible in the dead body because the illness had been caused by disturbances of the normal functioning of organs, tissues, and cells, and that the cessation of chemical processes on death made these difficult to both detect and analyze. In these instances, clinicians and scientists soon learned that laboratory studies could fill in the gaps.[121]

140. Concussion, like many neurological diseases and injuries in the early-nineteenth-century, was one of those conditions with pathological features awaiting such precision. Indeed the importance of Gama's early study of concussive impacts was that it foreshadowed patterns of inquiry that would become markedly evident from the mid-nineteenth-century on in the works of physicians and scientists who studied and treated concussive injuries of the brain.[122]

141. One of the questions that surrounded concussion injury was whether it was a functional injury only or whether it also involved structural changes. Most scientists and clinicians who studied the injuries and diseases of the brain and nervous system in the nineteenth century world regard the manifestations of disease and injury in structural (*i.e*. physical) terms. Lesions, no matter how small, produced functional disorder of the structure. Functional disorder, for these physicians and scientists, implied

[120] E. H. Ackernecht, *Medicine at the Paris Hospital* (1967).

[121] *See,* for a broad discussion of these trends, W.F. Bynum et al., *The western medical tradition : 1800 to 2000* (2006); *see also* W.F. Bynum, *Science and the Practice of Medicine in the Nineteenth Century* (1994).

[122] As one observe in 1973 noted, *see* D.W.C. Northfield, *Review: Trauma, Trepan, And Trephine: Past and Present*, 3 BR. MED. J. 5871, 114 (1973).

pathophysiologically that seemingly normal tissues were not working properly. Functional disorders, one author observed in 1886, were like the "striking of a magnet with a hammer" because such an action caused the magnet's function to vanish.[123] At the same time, functional disorder sometimes indicated that the lesion had yet to be discovered. As another author put it in 1892, "most writers…regard [functional disorder] as only provisional on the assumption that some kind of structural change in nervous matter must underlie every definite nervous disorder and that such change in time will be found."[124] Speaking of concussion specifically, in 1901 Judson Bury commented that the dichotomy between organic and functional disorder was exaggerated:

> Visibility is a relative term; it depends on our eyesight and the powers of our microscopes, and, as regards nervous structures, to some extent on staining reagents, and, let it not be forgotten, on the thoroughness of our search. Hence with improved methods of investigating nerve tissue we may expect that the group of functional disorders will get smaller and smaller. If on grounds of convenience these terms be retained two points should be clearly recognized – namely (1) the term "functional" ought to mean, not the absence of morbid changes, but only the absence of detectable changes; and (2) it ought not to be taken for granted that a disorder which has no known structural changes will run a more favourable course than one in which such changes are apparent.[125]

142. In the twentieth century some authorities sought to redefine the term functional to mean what we now call psychogenic or psychosomatic disorders. Such a view was possible because of the function of the nervous system. Diseases and injuries of the brain and nervous system necessarily implied complicated investigations because the outputs of that organ system were reflexes, behaviors, and cognition. "Neurology," as one author put it, "is a book, of which psychology and psychiatry are merely those

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[123] G.L. Walton, *Cases of Functional and Organic Injury to the Central Nervous System Caused by Trauma*, 114 BOS. MED. & SURG. J. 5, 102-05 (1886).

[124] *Functional Nervous Diseases*, LANCET 436 (1892).

[125] J.S. Bury, *The Bradshaw Lecture on Prognosis in Relation to Disease of the Nervous System*, 2 BR. MED. J. 2132, 1243-50 (1901).

chapters devoted to the activities at the psychic (or symbolic) functional level of the nervous system."[126] The brain and mind were inextricably linked. The question of the relationship between mind and brain, of course, implied grander controversies of philosophical and religious significance.

143. In practical terms, however, it was self-evident neurologically and psychiatrically that pathological change of the nervous system could result in permanent changes to the patient, ranging from movement to personality disorders. These behavioral changes could be of short duration or they could be permanent. Their onset could also mark the emergence of progressively degenerating pathological processes which resulted in ever-increasing disability.[127] Other authorities in the twentieth century, influenced particularly by Sigmund Freud, dogmatically regarded many mental illnesses as having only proximal relationship to the structural anatomy of the nervous system.[128] This view, which was held by many respected physicians and scientists, was increasingly challenged by pathophysiological and biochemical investigations that ultimately demonstrated how many psychoactive substances worked upon the nervous system and even treated some mental illnesses. By the mid-1970s, there was more evidence than ever that functional changes implied structural changes, ***and vice versa***. By the 2000s, particularly as a result of psychopharmacology, the dichotomy between mental illness and brain disease had broken down almost completely.[129]

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[126] *Psychiatry and Neurology*, 72 J. MENTAL SCI. 77 (1926).

[127] The grand instance of such views of nervous injury and mental disturbance was Phineas Gage, an American railroad worker who had famously survived a penetrating wound of the frontal lobe of the brain and afterwards shown marked personality changes from the injury. Gage, however, only exemplified in spectacular fashion the general truth that injuries of the brain had behavioral consequences. L.S. Jacyna & S.T. Casper, *The Neurological Patient in History* (2012), at 9.

[128] J.C. Burnham, *After Freud Left: A Century of Psychoanalysis in America* (ed. 2012); A. Scull, *Madness in Civilization: A Cultural History of Insanity from the Bible to Freud, from the Madhouse to Modern Medicine* (2015).

[129] N. Rose & J.M. Abi-Rached, *Neuro: the new brain sciences and the management of the mind* (2013).

144. Parallel to these broad changes in the mind and brain sciences was the additional fact that medicine had broadly changed. One significant feature was the specialization of medicine.[130] The evolution of the clinical specialties and academic disciplines that dealt with the mind and brain also took place in this period, a fact that meant that new groups often competed with already established ones (and with each other) to claim ultimate authority and expertise over nervous and mental functioning.[131] The specialties and disciplines that matured in the twentieth-century included neurology, psychiatry, psychology, neuropsychiatry, neuropsychology, and neuroscience.[132]

145. A second change that took place was in dissemination of the medical notion of "chronic" disease. As medicine improved in its ability to treat through surgery and pharmaceutical interventions, the notion that an illness could be "chronic" became ever more evident.[133] In the past, before treatment options were available, many conditions were fatal. For many conditions, this was no longer the case. For example, diabetes before insulin was universally fatal, but it became a chronic condition with the advent of the medicine.[134]

146. The increasing dissemination of the concept of chronic disease can be seen in the history of the diagnostic term "chronic traumatic encephalopathy." As will be detailed in the sections that follow, the historical record shows that it was originally understood that traumatic encephalopathy was a disease condition with acute features

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[130] R. Stevens, *American Medicine and the Public Interest: A History of Specialization* (1998); also R. Stevens, *Medical Practice in Modern England: The Impact of Specialization and State Medicine* (1966).

[131] *See* for a discussion of competition between specialists and professions the classic study, A. Abbott, *The System of the Professions: An Essay on the Division of Expert Labor* (1988).

[132] For a discussion of these specialties and their interactions, *see*, *e.g.*, S.T. Casper, *The neurologists : a history of a medical speciality in modern Britain, c. 1789-2000* (2014).

[133] On the history of chronic disease, *see* G. Weisz, *Chronic disease in the twentieth century: a history* (2014).

[134] On diabetes, *see* M. Bliss, *The Discovery of Insulin* (1983).

that could become chronic ones.[135] Initially researchers implied that traumatic encephalopathy could take on a chronic form with repeated concussion exposure, and this chronic form could become fatal (as seen, for example, in boxers). Beginning in the 1960s, with the increasing promulgation of a notion of chronic disease, and mainly also because of a single publication by neurologist Henry Miller,[136] traumatic encephalopathy became merged with its acute form. Concussion, in consequence, became understood as the acute event. Post-concussion syndrome, in turn, became often described as an acute condition potentially following concussion. Chronic traumatic encephalopathy became a distinct, and (as implied by the name) chronic condition.[137] As this genealogy reveals, however, the original understanding was that there was a path leading from concussion, through acute traumatic encephalopathy, finally to chronic traumatic encephalopathy, with repeated concussion the vehicle for advancing along this medical route.

147. A final change of note that took place in medicine was the relative status of pathological studies. At the beginning of the twentieth century, pathology, histopathology, and pathophysiology were the gold standards of medicine. Robert Koch's postulates about the bacterial causes of disease, for example, would have been impossible to validate without pathological techniques. Yet over the course of the twentieth century pathological techniques relinquished their status. Replacing them was the randomized controlled experimental trial, a technique broadly powered by the emergence of computers capable of performing analysis of complex, large data sets.[138]

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[135] Strauss, *supra* n.101 at 893-955 (1934).

[136] H. Miller, *Mental Sequelae of Head Injury*, 59 PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE 3, 257-61 (1966).

[137] A.C. McKee *et al.*, *Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy Following Repetitive Head* Injury, 68 J. NEUROPATHOLOGICAL & EXPERIMENTAL NEUR. 7, 709-35 (2009).

[138] On the shift towards a new gold standard, *see* S. Timmermans & M. Berg, *The gold standard: The challenge of evidence-based medicine and standardization in health care* (2010).

Although the randomized control trial began emerging in the 1930s,[139] it would not be common until the 1970s, and it was not until the late 1980s that it became considered medicine's gold standard.[140] Prior to this, pathological demonstration of the existence of lesions in individuals, which could be correlated with occupational hazards and clinical presentation, was considered the apogee of medical knowledge. When advocates today critique pathological studies as hopelessly anecdotal, they forget that many standard neurological diagnoses today were demonstrated in precisely that way.[141] Alois Alzheimer, for example, used histopathological techniques on one individual only to characterize the dementia that ultimately bore his name. James Parkinson observed six patients in his study of the shaking palsy.[142]

148. In sum, what can be said about the history of concussive head blows and their sequelae observed over the record as a whole? Across the historical record, it is striking how common are reports about concussions and the dangers of persisting and permanent symptoms following one concussion. A corollary observation is also true; it is striking how few clinicians and scientists there were who were willing to dismiss all such cases as malingers, fakers, psychoneurotics, or defectives.

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[139] On the history of the randomized control trial, *see* H.M. Marks, *The progress of experiment: science and therapeutic reform in the United States, 1900-1990* (2000).

[140] The difficulty of conducting randomized controlled trials in concussion research was first mentioned in A.K. Ommaya, *Trauma to the nervous system*, 39ANN. R. C. SURG. ENG. 6, 317-47 (1966); in a substantially later study, the normative significance of such studies was placed very clearly into a context that stipulated that other forms of evidence still matter. *See* J.P. Kelly & J. Rosenberg, *The Development of Guidelines for the Management of Concussion in Sports*, 13 J. HEAD TRAUMA REHAB. 2, 53-65 (1998). These authors wrote: "Class I: evidence provided by one or more well-designed randomized controlled clinical trials, Class II: evidence provided by one or more well-designed clinical studies, Class III: evidence provided by expert opinion, nonrandomized historical controls, case series, or case reports" *Id.* 56.

[141] For a detail critique, *see* L.E. Bothwell et al., *Assessing the Gold Standard – Lessons from the History of RCTs*, 374 NEW ENG. J. MED. 22, 2175-81 (2016).

[142] J.F. Ballanger, *Self, Senility, and Alzheimer's Disease in Modern America: A History* (2006); T.G. Beach, *The History of Alzheimer's Disease: Three Debates*, 42 J. HIST. MED. & ALLIED SCIS. 3, 327-49 (1987).

149. Equally striking is the fact that most of the studies were restricted to individuals with a history of a single concussion. Despite epidemiological observations that individuals who received one concussion were at a heightened risk to receive a second, the literature's focus on single concussive events makes clear that individuals who are at risk of repeated exposure must by extension have a heightened risk of sequelae. The historical record, however, makes mention of only a few occupational groups at such heightened risk: coal miners, railroad company employees, soldiers, and athletic workers in professional sporting industries.[143] The general fact most obviously communicated throughout the whole historical record is that any brain concussion in any setting is potentially dangerous, and that while long-term neurocognitive impairment is far from certain following a single concussion, the medical and scientific literature throughout the period proposed and demonstrated with ever-increasing clarity that the risks of permanent impairment are also real.

E. A Series of "Firsts" Develop the Medical Community's Understanding of Concussion

150. Observations of the long-term consequences of concussive head injuries have a long history, although it is not precisely clear historically who first connected a blow to the head with subsequent symptoms. Although there are extensive accounts of the history of neurology as a discipline, writing by medical historians analyzing the longer history of concussion has been comparatively slight. Nonetheless, authorities addressing the subject have noted observations about behavioral consequences from head injury in antiquity, the medical revolution of the European Renaissance, the eighteenth century, and at the time of the dramatic transformation of medicine that took place during the Napoleonic Wars. As is outlined below, it is clear from medical and scientific literature published in the nineteenth century that it was common knowledge

[143] On colliers, *see The Traumatic Neuroses*, 2 BRI. MED. J. 1716, 1115-16; 1955 (1893); on construction workers *see American EEG Society - Symposium: Round Table Conference on Head Injuries*, 7 ELECTROENCEPHALOGRAPHY & CLINICAL NEUROPHYSIOLOGY 3, 495-502; on police and fire workers, *see* A. Adler, *Mental Symptoms Following Head Injury*, 53 ARCH. NEUROL. & PSYCHOL. 1, 34-43 (1945).

then, among medical professionals and scientists, that even a comparatively modest blow to the head could have significant long-term neurological and psychiatric effects. Moreover, as this section clearly establishes, this observation was one continually made throughout the twentieth-century and into the first decade of the twenty-first.

151. The historical record of published medical and scientific literature validating this point is vast. As such it is entirely impossible to address all of this literature in detail within the scope of this declaration. Figures 9 and 10 below provide an overview of the numbers of articles consulted for this declaration that addressed long-term consequences of concussive and closed head injuries, and their frequency over the period 1871 to 2011. Figure 8 provides the more general overview of all the publications describing acute and chronic neurological and psychiatric sequelae following a blow to the head; Figure 9 looks in more detail at publications describing ***specific*** persistent or permanent symptoms, particularly intellectual decline, personality changes, and evidence of neurological deterioration.

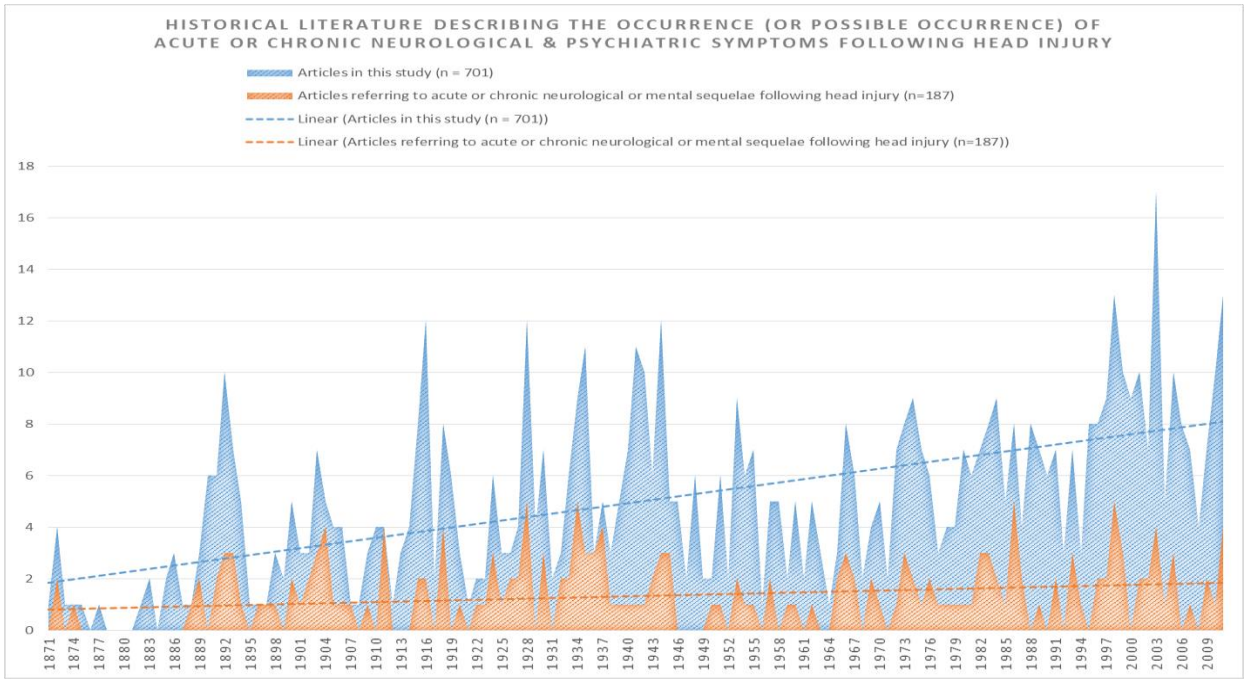

Figure 9. Historical literature describing acute and chronic neurological and psychiatric sequelae following a blow to the head. See Exhibit D for a bibliography of these works specifically.

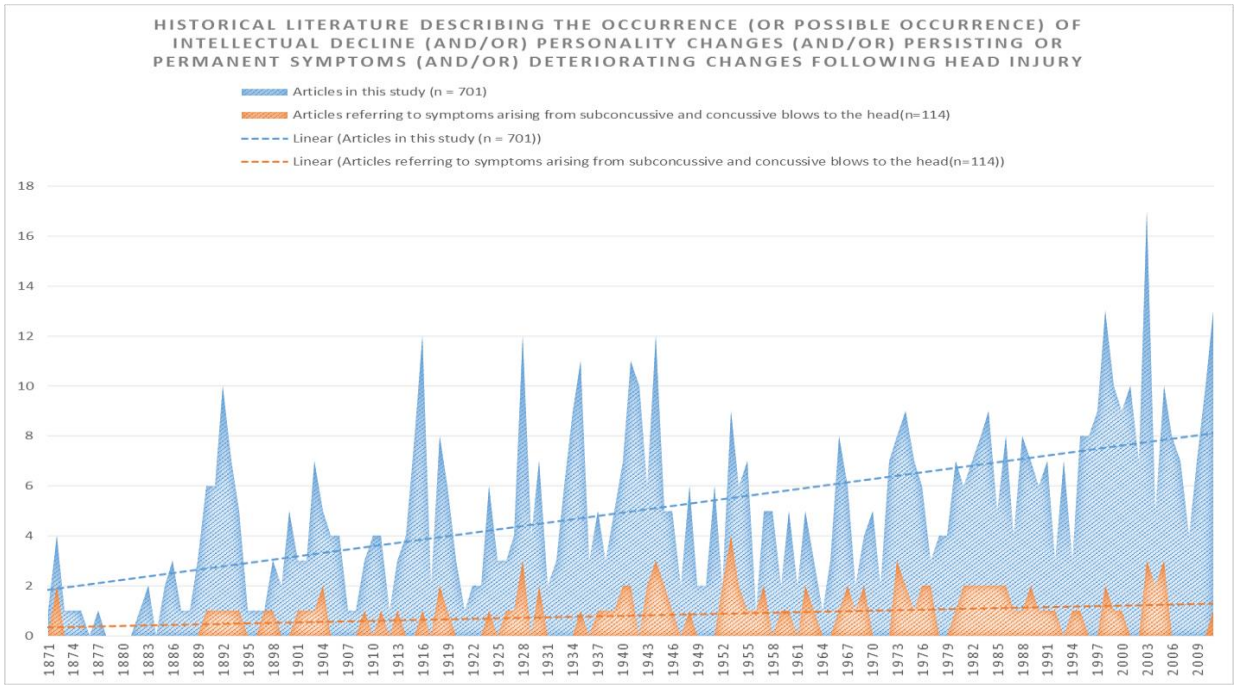

Figure 10. Historical literature describing specific symptoms which persisted or were permanent, including intellectual decline, personality changes, and evidence neurological deterioration. *See* Exhibit D for a bibliography of these works specifically.

152. To manage the volume of published medical and scientific literature highlighting the long-term neurological effects of closed head injuries, this section focuses in detail on the literature by way of an explicit look at ***firsts*** in the medical and scientific record, such as the first publication consulted for this declaration that made a new observation, discovery, or new type of investigation into the long term effects of concussive and closed injuries.[144] Accordingly, it addresses the:

- First attempt to systematically organize the nomenclature for the long-term consequences of concussion and closed head injuries
- First clinical review of the scope of existing medical knowledge about concussion of the brain
- First discussions of surgical interventions in head injury to reduce or resolve persistent sequelae
- First reports on laboratory studies providing animal models of brain concussion

[144] It is important to stress that this declaration cannot establish that each of these examples was the actual historical first. In other words, I am not claiming that these authors deserve priority. They may have been preceded by others. Rather the purpose here is to briefly summarize the diverse methodological ways evidence was gathered establishing and analyzing this population of patients.

- First analysis of the causes of insanity in an asylum
- First follow-up study of the long-term outcome of patients with trauma to the head
- First retrospective study of psychiatric symptoms following head injury
- First study comparing different groups of head injury patients using cross-disciplinary methods
- First Controlled Treatment Study Analyzing the Outcomes of Cerebral Concussion after Different Hospital Procedures
- First Prospective Cohort Neuropsychological Study of Concussion in Football.

Milestones in Concussion Research

1871 - First report of a concussion from ice-skating
1872 - First report that repeated concussions cause permanent mental changes
1875 - First report of animal models of concussion
1883 - First report of a fatality from a concussion or "traumatic encephalitis" during a sport
1886 - First call for sport teams to keep medical statistics on injuries
1889 – First discussion of the medico-legal issues surrounding closed head injuries
1890 - First description of "drunk" like symptoms following closed head injury
1891 – First mention of boxing and concussion
1898 – First attempt to analyze outcomes from head injury statistically
1903 – First paper analyzing the mechanism of the "knock-out" punch
1915 – First proposal to treat post-concussion symptoms with psychotherapy
1916 – First suggestion that a concussions leaves a residual area of vulnerability in the brain
1927 – First suggestion that subconcussive blows might lead to brain pathology
1934 – "Post-concussion syndrome" is coined
1938 – First suggestion that a concussion might lead to demyelination
1941 – First usage of EEG to study concussion in animal models
1943 – First study of the biomechanical effects of head injury
1953 – First observation of a "head injuries epidemic" resulting from car accidents
1955 – First studying showing repeated, subconcussive blows lower the concussion threshold
1959 – First observation of neurofibrillary tangles in closed head
1960 – First use of a computer for statistical analysis
1972 – First controlled trial for treatment of concussion symptoms
1975 – First study urging doctors to warn athletic authorities that concussions are cumulative
1990 – First medical society issues guidelines for concussion management in sports
1999 – First description of tau-protein in concussion occurring in sport
2001 – International Symposium on Concussion in Sport in Vienna, Austria

1. First Attempt to Systematically Organize the Nomenclature for the Long-Term Consequences of Concussion and Closed Head Injuries

153. Although many authors had observed mental symptoms following head trauma or repeated head trauma, it was William Julius Mickle who made the first systematic attempt to organize the diagnostic nomenclature surrounding what he termed "The Traumatic Factor in Mental Disease." This was published in a lengthy article in *Brain: A Journal of Neurology* in 1892. Mickle's study sought to define groups of patients who suffered a broad range of traumatic injuries to the brain, and thus injuries both more and less severe than concussion were also contemplated in his study. He wrote:

> I include all injuries, produced by external violence, affecting the nervous system so as to become a factor in the production of mental disease. Of these, the most important are direct injuries to the head, but to limit our subject to these, as some would, is narrow and unscientific, inasmuch as precisely the same mental result and the same lesion may follow cerebral disturbance of injury produced indirectly.[145]

154. Mickle was even-handed in the way he addressed his topic. He recognized that the causes of the symptoms of insanity after these injuries were diverse. He noted that the injury might only produce an already-present tendency, or excite an "insanity already about to appear."[146] He accepted, too, that such injuries might result in stress reactions to the trauma that mimicked nervous disease, but he was also clear that there was a type that was perhaps "primarily, coarse brain-damage; and either secondarily, or without such damage, organic and often progressive destructive brain-disease." These last two groups include the cases of insanity more completely and characteristically of traumatic nature."[147]

155. Mickle observed that the injuries that produced such mental disease "may be molecular perturbation of brain (concussion); or contusion, crush, rupture of its

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[145] W.J. Mickle, *The Traumatic Factor in Mental Disease*, 15 BRAIN 1, 76-102 (1892).

[146] *Id*. at 77.

[147] *Id*.

substance, or haemorrhage into its tissue or into the meningeal spaces; or vaso-motor effects of damage to brain, cord, or sympathetic nervous system."[148] He furthermore observed that traumatic insanity could emerge after some delayed period: "Insanity traumatically occasioned may come on immediately or rapidly after the injury, or months, or many years later."[149] Mickle elaborated the point further through discussion of the symptoms:

> When insanity comes on some considerable time, or long, after the injury, certain conditions are apt to mark the interval between the cessation of the immediate effects of the blow and the advent of the morbid psychosis. Such are the easy production and exaggeration of the effects of alcohol, narcotics, coition, extreme heat, mental over-work or agitation, the troubles of life and knocks of fortune. Even the excitement of physical exertion occasions an undue cerebral effect and mental commotion. A change of character is frequent; and usually is in the directions of unwonted impatience, irascibility, furious outbursts, a quarrelsome, overbearing brutality; or moody, unsocial taciturnity and suspicion, and with these, or alone, general mental failure, or gapes in memory; cerebral fatigue on the least mental exertion or strain of attention; or mental confusion – a dazed, bewildered condition; or emotional dejection and hypochondriacal notions; or an uneasy nervous state, a general *status nervosus*; bad dreams; ready addiction to alcoholic indulgence or sexual pleasures. These conditions may deepen unto the prodromal stage of the coming psychosis; or, when this is reached, we may find suicidal and homicidal impulses, or an expansive phase, and usually insomnia; or preliminary to the onset of over psychosis there may be disquietude, tremor, headache.[150]

156. Mickle then described several cases he had seen in practice. In keeping with the very wide meaning of the term insanity in the nineteenth century, Mickle lumped together, for example, both cases of severe paranoia and delusions with cases of senile dementias. Both, he declared, could be organic in origin. His case of a demented patient profiled a man, aged 71, with a history of repeated head injuries.

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[148] *Id.*

[149] *Id.* at 79.

[150] *Id.* at 78-79.

157. Mickle's paper was the first attempt made by medical professionals to *categorize* types of mental illnesses arising from traumatic injuries like concussion and to establish a nomenclature for these categories of illness. There was an intense focus on the particular forms of causation and symptomology found in these types of cases.

2. First clinical review of the scope of existing medical knowledge about concussion of the brain

158. In 1894, shortly after the publication of Mickle's specialist study, the first clinical review of medical knowledge about concussion of the brain appeared in the *Journal of the American Medical Association*. The author, L. C. Lane, was Professor of Surgery at Cooper Medical College in San Francisco. Where Mickle had ambitiously sought to bring order to psychiatric and pathological nomenclature, Lane set for himself the comparatively simpler task of reviewing the literature on brain concussion from the nineteenth-century.

159. The chief challenge with cerebral concussion through the ages, Lane observed, was that medical authorities derived the:

> . . . elements of diagnosis of any disease from three sources, to-wit: the causation, the symptoms arising, and the direct inspection of the parts affected. But in the case of cerebral concussion one or more of these sources of information is often wanting. The inspection of the injured part is wholly denied to the surgeon; the extent of the violence done can only be vaguely and imperfectly estimated; and not infrequently it is wholly unknown; as when the patient is found unconscious or unable to explain how he was injured; and finally the remaining source of knowledge, viz., the symptoms are often far from being clear and well defined.[151]

160. Lane nevertheless felt confident dividing cerebral concussion into three grades of injury, "the mild, severe, and fatal."[152] His confidence arose from the great work of French, German, and British authorities on the subject. To Lane, reports by Haworth had first described contrecoup injuries in the 1840s. Reports in 1850s by Fano, Chassaignac and Haas, Lane wrote, demonstrated that autopsy of a concussed patient

[151] L.C. Lane, *Concussion of the Brain* 23 JAMA 2, 48-52 (1894).

[152] *Id.*

would show small points of effused blood "not larger than millet seeds," although in mild cases these might be absent. To Aliquié, Lane ascribed the notion that: "from concussion, one or many functions of the head, or the parts dependent on it, may be impaired or annulled, and this disturbance may be brief or lasting. Concussion in its action resembles grave mental disease."[153] Summarizing all of these discoveries, Lane submitted:

> In the mildest grade there is a brief diminution of the blood pressure in the part; its tension is temporarily lowered. As the result of this there is mental disturbance. The cells in the cortex of the brain, on whose normal condition the evolution of thought in some unknown way depends, are deprived for a few moments of their accustomed supply of nutrient material; the result is a brief perversion of intellect; memory is lost; the power to recognize surrounding objects is weakened or lost.[154]

161. Lane commented that prognosis of cerebral concussion depended upon the degree of injury severity. Mild cases usually recovered. But severe cases could have a variable outcome:

> …not infrequently there remains some vestige or memento of the injury in the form of perverted motion, sensation or impaired mentality. From the observations of Griesinger and other alienists it has been seen that the mental character often undergoes some change in those who have been the subject of severe cerebral concussion. The memory may be weakened or altered in some way…. Besides this, the character of the individual may be slightly or greatly modified; the hitherto well-controlled temper may become vehement and rash; he has outbursts of anger, and may become the actor of lawless deeds. He becomes distrustful of those around him. These changes may be plainly manifest or they may be slight as only to be perceptible to the subject's intimate friends; and whatever character these changes assume they will probably remain permanent, inasmuch as they depend on structural change which will continue unchanged.[155]

162. Lane observed that if course and outcome were uncertain, treatment for all concussion patients was relatively straightforward. "Frequently two or three months are

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[153] *Id.*

[154] *Id.* at 50.

[155] *Id.* at 51.

required for entire recovery of the patient, so that he can resume some occupation; and for a yet longer period, limited exercise of the body and mind should be enjoined."[156]

3. First Discussions of Surgical Interventions in Head Injury to Reduce or Resolve Persistent Sequelae

163. Lane's review and Mickle's article were followed by other observation studies of mental disturbance resulting from traumas to the head. Of note among these are the writings of the British surgeon Herbert Page. Page was famous for pronouncing patients with subjective symptoms frauds and malingers. His views of head injury and brain concussion are thus worthy of consideration, precisely because he was no bleeding heart for patients purporting subjective symptoms. On the sequelae of cerebral concussion, Page was explicit:

> Concussion of the brain may be followed by all sorts of lasting disturbances, by changes in temper and disposition, by impairment in mental power and physical endurance, by incapacity for the same amount of headwork as before, and even by distinct insanity.[157]

164. Page's usual approach to surgical cases of head trauma was a therapeutically nihilistic one. Put simply, Page viewed such surgeries as so dangerous that he preferred to leave patients in the hands of fate.[158] As historian Delia Gavrus has shown, however, Page's surgical contemporaries were beginning to engage in such operations more frequently and mainly in response to emerging scientific research.[159] Page, too, was not immune to these trends. In 1893 he described reading the scientific work of Alexander Miles, discussed *infra* Section V.E.4, and contemplating a severe case of concussion with "repeated attacks of maniacal excitement" that ultimately

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[156] *Id.* at 52.

[157] H.W. Page, *A Clinical Lecture on Some Cases of Head-Injury, Including One in which There was Lesion of the Occiptal Lobe* (1901).

[158] A typical example of his refusal to operate is H.W. Page, *Clinical Lecture on a Case of Head Injury Followed by Hemiplegia and Dilated Pupil* (1892).

[159] D.Gavrus, *Men of Dreams and Men of Action: Neurologists, Neurosurgeons, and the Performance of Professional Identity, 1920-1950*, 85 BULL. HIST. MED. 1, 57-92 (2011).

necessitate sending "him to the asylum as unquestionably insane."[160] Page observed, citing an Oxford thesis on "Aspects of Traumatic Insanity," that new scientific and clinical research suggested to him that the removal of small fragments of surface lesions might at times result in therapeutic benefit.[161]

165. When in 1902 Page delivered the Presidential Address to the Neurological Society of London, which was published in the pages of *Brain: A Journal of Neurology*, it became clear that he had become convinced of the value of operations in head trauma.[162] This address appears to have been the first major address offered at a medical conference on the topic. He opened his address: "I shall use the term concussion of the brain in the widest sense, to include much that accompanies, and is a good deal more than simple concussion alone."[163] Admitting that the subject was well worn, he suggested it would nevertheless interest a highly diverse audience of clinicians:

> In the first place to the surgeon, because of the exercise of his skill, to the physician whose part it may be to interpret some of the later consequences of intra-cranial injury when the immediate effects of accident have long since passed away; to the physiologist, because of the many transient last derangements of function in all parts of the body to which concussion may rise; to the psychologist, because of the subtle derangements of intellect which may seem to have had their beginnings in sudden physical commotion of the brain, and to all alike, singly or in company, be reasons of the problems which concussion may present for solution at the beside."[164]

166. At first Page described instances of concussion that might call for surgical intervention. Reprising the theme of his career, he observed that waiting was often best. However, he then provided an alternative view:

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[160] H. W. Page, *A Clinical Lecture on a Case of So-Called Concussion of the Brain followed by Acute Mania*, LANCET 1159 (1893).

[161] *Id.*

[162] *See also* H.W. Page, *St. Mary's Hospital: Five Cases of Injury to the Head*, LANCET (1898).

[163] H.W. Page, *On Concussion of the Brain in Some of its Surgical Aspects* 1 (1902).

[164] *Id.* at 1-2.

> There is, however, yet another aspect from which the question of operative interference may be viewed. No one will deny that various kinds of mental disorder are fairly common after concussion of the brain. In my own limited experience, and out of the comparatively small number of cases which have passed through my wards, not a few have shown signs of mental aberration, not the mere transient clouding and confusion in the days immediately after the injury, but mental derangement of a more lasting kind. In medical literature there is no lack of instances of unquestionable traumatic insanity and the views of many writers are to be found therein. To name one or two only. Crighton-Browne has written that "a careful inquiry will establish the principle that cranial injuries are, at least, prolific sources of mental derangement" and that "concussion is…the more important element in the vast majority of cranial injuries in relation to subsequent mental incapacity" and "the most fruitful source of ulterior misfortune."[165]

167. Noting that it was possible that surgery might help in such cases. Page admitted that some might disagree with his views of the subject:

> It is true that Clouston with his unrivalled experience is of opinion that "accidents to the head do not loom largely in the production of the insanity of the world"; but, while recognizing his high authority, and granting that the natural tendency on the part of relatives to regard injury rather than inheritance or disease as the cause of insanity detracts from the value of statistical evidence, the record of published cases is sufficiently striking to arrest attention, even though our own experience did not infallibly tend in the same direction. How often, indeed, have we not heard that the onset of "fits", using the term in the widest sense, has followed some injury to the head, that changes in disposition and temperament, incapacity for sustained mental occupation, inability to engage in the former pursuits, liability to suffer from severe headaches, persistent sleeplessness, loss of memory, attacks of depression and melancholy, moral deterioration, and other kindred steps in nervous dissolution have seemed to have had their beginnings and to date their onset from some head-injury spoken of at the time perhaps as slight concussion of the brain? We are hardly in the position yet to attribute the various mental phenomena to definite focal lesions, to say, for example, that changes of temper is always due to injury of the frontal lobes, much though there is in support of this view, or melancholia to lesion of the parietal, but is it not possible, nevertheless, that many of the conditions which have been named may own a material cause in cortical degeneration, started not by gross lesions like depressed

[165] *Id.* at 12-13.

> bone and torn membranes and bruised cerebral tissue, but in surface hemorrhages which are comparatively harmless at the time of their effusion?"[166]

168. As Page's published comments make abundantly clear, clinicians and scientists were beginning in this period to engage in scientific research and endeavoring as well to gather statistical evidence of the medical problems. In the late-nineteenth century, these represented new and illuminating forms of objective evidence, with laboratory studies being especially significant.

4. First Reports on Laboratory Studies Providing Animal Models of Brain Concussion

169. From the 1870s on, the medical and scientific press in Britain and North America reported on scientific investigations of animal models of concussion that relied upon "rapping the head with a light hammer" or injecting fluid into a small hole in the skull.[167] The first significant study examining the physiological mechanism of concussion appearing in this historical research was published by Alexander Miles in *Brain* in 1892. Miles' study first reviewed the history of concussion studies. He adopted as his subject the question of whether a concussion could result in fatality, and, more broadly, what the physiological mechanisms of a blow to the head might be. He observed that there were several competing theories for the mechanism, ranging from simply the effect of vibrations on brain tissue, to capillary hemorrhages and anaemia of the brain.

170. Miles performed several different experiments, and through these demonstrated that a blow to the head could slow the action of the heart. He claimed from this that "symptoms of concussion are due to a profound disturbance of the circulation of the brain."[168] He observed that blows to the heads of animals left little

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[166] *Id.* at 13-14.

[167] For hammer, *see* J.J. Putnam, *Recent Progress in the Pathology of the Nervous System*, BOSTON MED. SURG. J. 128 (1876); for fluid *see The Pathology of Concussion*, BRI. MED. J. (1877).

[168] A. Miles, *On the Mechanism of Brain Injuries*, 15 BRAIN: J. NEUR. 2, 153-89 (1892).

observable pathology in the brains of the animals, even those blows that resulted in fatalities. In his conclusion of his paper, which was supplemented by much research on animals and cadavers, he argued that his work demonstrated that "concussion of the brain is the result of a temporary anaemia of that organ", that this was caused by reflex action, that a wave of cerebrospinal fluid stimulates the brain and disturbs the equilibrium of the nerve cells, and that the small hemorrhages are not the cause of the concussion symptoms but rather "an index of the force producing the injury" which left in its wake microscopic brain trauma.[169]

171. Miles' study initiated a vigorous debate about the physiology of concussion, one that would continue for decades. His work was followed by other studies that argued that the physiological mechanism of concussion was respectively mechanical shock, mechanical stress, anemia or vascular disorder, intercranial pressure increases, or congestion of the brain. As will become clear in this declaration, most of the authors of those futures studies would agree with Miles' broader contention that whatever the physiological mechanism, the pathological changes took place at the level of the nerve cell.

5. First Analysis of the Causes of Insanity in an Asylum

172. Descriptive statistical evidence on insanity resulting from trauma to the head was beginning to appear in the early years of the twentieth-century. As has already been discussed *supra* Section V.C.4, Goddall attempted the first meta-analysis of insanity and found in 18,606 published cases, 480 had mental disorder arising from head injury.[170] Few authors had, at that point, attempted such ambitious surveys of past descriptive statistics. More typically, medical writers surveyed their own institutional experiences and compared that with observations by others.[171] In the first descriptive

[169] *Id.* at 188.

[170] E. Goodall, *Four Cases of Insanity After Injury to the Head, with Observations Relative Thereto*, 152 Lancet 3928, 1540-41 (1898).

[171] For another example, *see* A. Meyer, *The Anatomical Facts and Clinical Varieties of Traumatic Insanity*, 60 AM. J. PSYCH. 3, 373-441 (1904).

survey of an asylum population, A. B. Richardson, Medical Superintendent of the Government Hospital for the Insane in Washington D.C., discussed multiple cases of insanity arising from head injuries that he had observed in his own institutional setting. Comparing his finding to those of other authorities, Richardson observed that the then-famous German psychiatrist Griesinger was aware that "injury to the head is of great importance as a cause of insanity." In making that claim, Richardson observed that Griesinger had quoted a contemporary's figures who had found: "In 21 [patients] the concussion was followed by immediate loss of consciousness, in 16 by simple mental confusion, in 12 by dull pain in the head. In 19 the mental disease began within one year, but in the majority the commencement of the insanity dated from four to ten years after the injury. In 18 cases there was dullness of hearing, in 20 great irritability and tendency to violent outbursts. In 14 suicidal tendencies were present. Weakness of memory and confusion of mind were not infrequent…"[172]

6. First Follow-Up Study of the Long-Term Outcome of Patients with Trauma to the Head

173. Statistical methods also opened another dimension for the study of the consequences of head injury. In 1904, *The Lancet* published three papers that T. Crisp English delivered as his contribution to the medically prestigious Hunterian Lecture Series, offering what may have been a proto consecutive and prospective study of head injuries. These were based upon an analysis of 300 patients with injuries of these head seen at St. George's Hospital London. English divided his patients into three cohorts: The first was fracture of the skull. The second was concussion, contusion, and laceration. The third represented a miscellaneous groups of patients who did not fit appropriately into either of the previous groups. English divided the groups into three classes: patients with no symptoms, patients with slight symptoms, and patients with

[172] A.B. Richardson, *The Symptomatology and Treatment of Traumatic Insanity*, 60 AM. J. PSYCH. 1, 19-25 (1903).

marked effects, including "mental impairment, traumatic epilepsy, and conditions which prevented the patients from working."[173] His findings can be seen in Figure 11.

	First series.	Second series.
Class 1, no effects	31	48
Class 2, slight effects	50	42
Class 3, marked effects	19	10

Figure 11. Data from T. Crisp English's consecutive and prospective study of head injury patients at St. George's Hospital in London, delivered in his Hunterian Lectures, and published in *The Lancet*. The First Series comprised cases of skull fracture. The Second Series comprised cases of concussion, contusion, and laceration without evidence of fracture.

174. Crisp added to these consecutive statistics a still finer observation: "These figures throw a more serious light on the prognosis of head injuries than is usually recognized…. My experience is that some degree of mental impairment, though rarely sufficient to be included under the title of traumatic insanity, occurs in over 10 per cent of patients."[174] Crisp demonstrated this claim through analyzing the prospective impact such injuries had on patients' future working capacity (Figure 12).

Effects on Working Capacity.

	First series.	Second series.
No marked effects	52	63
Did lighter work	13	6
Only able to do a little work	7	4
Gave up trade	8	2
Totally disabled	6	3
Total	86	78

Figure 12. Data from T. Crisp English's consecutive and prospective study of head injury patients at St. George's Hospital in London, delivered in his Hunterian Lectures, and published in *The Lancet*. The First Cohort comprised cases of skull fracture. The Second Series comprised cases of concussion, contusion, and laceration without evidence of fracture.

175. As Figures 11 and 12 show, English did not include the third series in his tables. He did, however, offer an extended comment on those cases, and one that made clear he regarded even seemingly minor concussive blows as having potentially lasting consequences:

[173] T.C. English, *Abstract of Hunterian Lecture on After-Effects of Head Injury: Lecture 1*, 485 (1904).

[174] *Id.* at 485.

> The third series, the miscellaneous cases, includes a very important group which from their nature are not found in Series 1 and 2. I refer to those very mild forms of concussion in which loss of consciousness lasts a few seconds only or may be practically absent…. Such cases are rarely admitted into hospital and the apparently trivial nature of the injury usually leads to entire neglect of treatment or to treatment of an entirely inadequate kind. Thus it comes about that grave consequences are more liable to follow the slighter forms of concussion the seriousness of which is overlooked than the more marked cases in which proper treatment is enforced.[175]

176. English's third series underscores the perhaps most salient discovery about concussions prior to the First World War. The concussions that did not seemingly require hospitalization could nevertheless deteriorate. This fact was one with which general practitioners, doctors and surgeons who visited patients in their homes, may have been acquainted, but it was a fact rarely commented on in the published literature. The first author to do so in this research wrote in the *Journal of the American Medical Association* in 1911:

> The importance of this class of mental disturbance to the general practitioner, and especially for the practicing neurologist, can hardly be overestimated, especially when we remember that according to Friedman's statistics, 60 per cent of all concussions of the brain are followed by psychic disturbances for a year or more after the injury, and that according to Ziehen, the traumatic psychoses form 3 per cent of all admission to hospitals for the insane. The general practitioner is the first one called on to attend these cases, and while he is prepared to meet the immediate effects of the head injury, he must not forget that next to the question of whether the patient is going to live or not, he is confronted with the problem of giving a prognosis as to what effects the injury will have on the patient's future life.[176]

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[175] *Id.* at 486.

[176] B. Glueck, *Traumatic Psychoses and Post-Traumatic Psychopathic States*, 56 JAMA 13, 943-48 (1911).

7. The First Retrospective Study of Psychiatric Symptoms Following Head Injury

177. English's study was followed by others like it, mainly relying upon consecutive case series of head injury patients. Although many published reports drew upon retrospective analyses of individual clinician's practices, the first formal systematic retrospective study was conducted in 1937 by Edward Mapother. Mapother's study was psychiatric in focus. He identified retrograde amnesia as the clearest strongest evidence of physiogenic causation. On the question of incidence of psychiatric symptoms following head injury – and severe enough to potentially indicate admission to an asylum – he observed that statistics were not kept on this question. Mapother found it surprising that insurance companies did not keep statistics. He was suspicious too of military statistics:

> It is like putting science in blinkers to disregard, in considering such statistics, the possible preconceptions of those who collect them and who draw the deductions. I am afraid that as a clinician I am left obstinately convinced that some undue skepticism on the part of officials tired of importunacy must here be allowed for. The figures indicate that, apart from gross damage to brain, head injury of war has no influence whatever in determining psychoses. Surely this is too good to be true. When a clinician has seen the immediacy with which, in civil cases, psychoses evolve from the early syndrome following concussion without gross damage and either clear up or persist, can he accept such conclusions?[177]

178. Mapother used his own case records, and he also surveyed retrospectively 30,000 case records from the London County Council medical institutions. In his own practice, he found an incidence of eighteen per 1000 patients where mental disturbance followed head injury. In the County Hospitals around London, he found a rate of three per 1,000 admissions. Four were found per 1000 admissions in other institutions, and seven per 1000 at the Maudsley Hospital, London's leading hospital specializing in

[177] E. Mapother, *Mental Symptoms Associated With Head Injury: The Psychiatric Aspect*, 2 BR. MED. J. 4012, 1056 (1937).

psychiatry. He concluded that in some of these cases the prognosis for no recovery was a sad reality.[178]

8. The First Study Comparing Different Groups of Head Injury Patients Using Cross-Disciplinary Methods

179. At the close of the Second World War, the first study utilizing a natural groups design, based on individual differences, was published (1945). This study was a controlled comparison of patients that relied upon observational methods to analyze inherent differences between various groups of head injury patients. Jurgen Ruesch and Karl M. Bowman, both psychiatrists in San Francisco, compared 125 cases of late-traumatic syndromes with 140 controlled cases taken from three groups: "70 cases with acute head injuries, 30 cases with brain disease (not related to head injury), [and] 40 cases with psychoneuroses with no history of head injury."[179] The authors further divided the 125 cases between those with "chronic head injuries with signs of brain damage" and those with "chronic head injuries without signs of brain damage." This study was novel in another way because it sought to bridge disciplinary differences by characterizing all patients using neurological, psychiatric, and psychological observational methods. The authors, in general, concluded that psychometric tests failed to distinguish between chronic head injury patients with and without neurological signs. They also observed that pre-traumatic personality factors were associated with persistent or prolonged symptoms, and they observed that post-traumatic personality factors also mattered greatly. In their assessment, most unresolved cases were caused by "personality problems" rather than permanent brain damage – a fact that spoke to Ruesch and Bowman's background as respected psychiatrists.[180]

180. In spite of this as many other clinicians had found, Ruesch and Bowman noted that it remained difficult to distinguish neurological cases from the psychiatric

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[178] *Id*. at 1061.

[179] J. Ruesch & K.M. Bowman, *Prolonged Posttraumatic Syndromes Following Head Injury*, 102 AM. J. PSYCHIATRY 2, 145-63, 145 (1945).

[180] *Id.* at 161.

ones. The problem again was the comparative rarity of such patients: "Psychiatrically speaking, only a few of the cases in this group could be called "traumatic neuroses" in which the head injury actually had changed the personality of the patient."[181]

9. First Controlled Treatment Study Analyzing the Outcomes of Cerebral Concussion after Different Hospital Procedures

181. Almost three decades later Finnish neurosurgeons Mikael Relander, Henry Troupp and G. AF Björkesten published the first controlled treatment study analyzing the outcomes of cerebral concussion after different hospital procedures, which appeared in the *British Medical Journal*. The authors hypothesized that hospital patients would have different outcomes with active rehabilitation efforts as opposed to passive ones. For the authors, an active rehabilitation approach meant continuity of care and efforts to make clear to patients that concussions had a very good prognosis. The authors were able to return the experimental group to work faster than the controls. They also found that, one year later, outcomes were better for the actively managed group than the routinely managed one. However, noteworthy in this context is that the authors found that of 178 patients with cerebral concussion, several in both groups were still showing either minor symptoms, some disability, or complete disability one year after receiving the injury (*see* Figure 13).[182]

TABLE VII—*Condition of 59 Patients at One-year Follow-up elicited by Questionnaire*

Treatment Given	No Symptoms	Minor Symptoms	Some Disability	Disabled	Total
Routine ..	15	7	2	1	25
Active ..	21	10	2	1	34

Figure 13. Persisting symptoms following cerebral concussion as reflected in the first controlled treatment study found in this research by Finnish neurosurgeons. The total number of patients in this study was 178.

[181] *Id.* at 160.

[182] M. Relander *et al*., *Controlled Trial of Treatment for Cerebral Concussion*, 4 BR. MED. J. 5843, 777-79 (1972).

10. First Prospective Cohort Neuropsychological Study of Concussion in a Sport

182. Of perhaps final note in this series of firsts is the first major prospective cohort study of concussions in a sport (it is important to note that there had been numerous other forms of research into concussion in sport at all levels prior to this study).[183] It was a neuropsychological study that sought to analyze the effects of concussion; it involved collegiate-level football players in multiple universities. The study was conducted by Jeffery Barth and colleagues at the University of Virginia from 1982 to 1986, and was first published in 1989.

183. Some context is important for understanding the motivations for this prospective cohort study of concussion in college football players. Barth and colleagues had been pioneering the use of a neuropsychological battery (a run of sequential psychological tests) to examine the cognitive and behavioral deficits experienced by patients with minor head injuries.[184] In an earlier prospective study undertaken by Barth and colleagues which was published in 1983, they found that of 70 minor head injury patients followed up at three months:

> …a significant percentage of mildly head-injured patients demonstrate decreased adaptive functioning. Our results have several implications for the management of minor head trauma. First, some patients with seemingly insignificant periods of unconsciousness or posttraumatic amnesia may evidence considerable postconcussive impairment. Second, older patients with lower levels of education may be differentially affected. Less educated patients may have greater difficulty understanding and coping with their cognitive sequelae. Third, in addition to a thorough neurological evaluation, a brief neuropsychological assessment focusing on attention-concentration skills, visuomotor functioning, memory abilities, and emotional status may help to identify individuals at risk for school or employment failure. Finally, in some cases simply informing

[183] J.T. Barth *et al*., *Mild Head Injury in Sports: Neuropsychological Sequelae and Recovery of Function*, MILD HEAD INJ. 257-275 (Harvey S. Levin, Howard M. Eisenberg, Arthur L. Benton eds., 1989).

[184] Barth's early work is described in K.E. Bachynski, *No Game for Boys to Play: Debating the Safety of Youth Football, 1945-2015*, COLUM. U. ACAD. COMMONS 281-282 (2016).

> patients that minor head injury has the potential to interfere with normal functioning may significantly increase individual adaptation. Mild head injury is a multifactorial disorder, and outcome is related to patient's age, educational level, specific cerebral trauma, premorbid functioning, and psychological reaction to the injury. The precise role that each of these factors plays in the recovery process and what interventions are most effective remain to be answered. Well-designed, interdisciplinary, prospective research may help to provide answers to these complex questions. In this regard, we have begun to evaluate several hundred football players to examine experimentally the effects of mild head injury on healthy, bright, young men. For now, it is important to recognize that mild head injury is an often overlooked condition that may result in neuropsychological dysfunction that requires intensive evaluation and treatment.[185]

184. By the 1980s these authors were drawing on a rich legacy of scientific investigations (described in the next section) to explain the mechanism of minor head injury. As Barth and colleagues commented in their 1983 study: "The sheer-strain hypothesis is generally supported by head injury studies finding microscopic lesions in the medulla and pons primarily in the area of the rostral pyramids. Because approximately 80% of all minor head injuries are related to vehicular accidents, falls, and sports injuries, an experimental model of sheer-strain emphasizing acceleration-deceleration and rotational forces seems especially relevant to minor head injury."[186]

185. The first major analysis resulting from their study of concussion in football players appeared in an edited monograph entitled *Mild Head Injury*, published in 1989.[187] Barth and colleagues both reviewed the literature on concussive injuries in sports and provided extensive summary of their findings from their prospective study of

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[185] J.T. Barth *et al*., *Neuropsychological Sequelae of Minor Head Injury*, 13 NEUROSURGERY 13 5, 529-33, 532 (1983).

[186] *Id.* at 531.

[187] It is curiously of note that the results of the study did not appear in a peer-reviewed journal until 1996. I trust that this observation is correct, because M.R. Lovell & M.W. Collins, *Neuropsychological Assessment of the College Football Player*, 13 J. HEAD INJ. REHAB. 2, 9-26 (1998) cite only these two papers when they describe this study as the first of its kind to show the usefulness of neuropsychological baseline testing, *id*. at 11.

2,350 football players. All had undergone baseline neuropsychological testing. Players who experienced a mild head injury, defined "as a change in or loss of consciousness lasting for less than two minutes,"[188] had been retested with a number of psychometric tests across several intervals of time (within 24 hours of the injury, five days later, 10 days later, and post-season). Of note was the fact that in the pre-season period of assessment 19% of the players reported one previous head injury, 11% reported two previous head injuries, and 11% reported three or more previous injuries.[189]

186. In general, Barth and colleagues found that a single mild head injury in college football caused noteworthy psychological symptoms which tended towards recovery, albeit "perhaps incomplete" recovery.[190] The authors also felt that football injuries tended towards mild concussions, or what were colloquially known as "dings."[191]

187. Barth and colleagues clearly saw the results of this study as validating their earlier studies that found brain damage caused the symptoms of mild head injuries they had seen in other contexts. They observed that the pattern of recovery seen in football players: "gives us rather considerable evidence that the sequelae of mild injury are unique to head injury and not a consequence of general trauma or of population reporting rates for individuals of similar age and sex." In other words, the mental symptoms following mild head injury were genuine and not a consequence of a reporting anomaly. Summarizing their results, the authors stated that:

- 10% of college football players experienced mild head injury;
- 40% would sustain at least one concussion in high school or college;
- Neuropsychological testing could measure the effects; and
- In general, this young population tended to recover within 10 days of injury.[192]

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[188] Barth, *supra* n.170 at 265.

[189] *Id.* at 268.

[190] *Id.* at 271.

[191] *Id.* at 272.

[192] *Id.*

188. The authors were also clear that there were considerable limits placed on interpretations of their study. They stated: "questions still remain regarding the full extent of recovery and compensation, the short- and long-term effects of multiple head trauma, and factors predisposing a player to the risk of head injury."[193] This fact led them to advise that a "conservative approach" should be followed when offering advice about when players should return to competition:

> …which emphasizes individual evaluation of the head injury circumstances, including severity of trauma, number of previous head injuries, length of time since last cerebral insult, premorbid and present neuropsychological function, and recovery from postconcussion symptoms.[194]

189. Why was conservative management warranted in 1989? By 1989 there had been considerable studies indicating that concussive head injuries could result in permanent neurological symptoms, debility, and degenerative neurological and psychiatric conditions. It was a very rare case that occurred from one traumatic episode, but such rare cases had been recorded through the variety of methodological techniques that were invented in the twentieth century. This fact had been shown in numerous anecdotal reports; it had also been shown as well in meta-analyses, consecutive case studies, retrospective studies, natural group comparison studies, and case-controlled prospective studies.[195]

190. As will be explained below, a rich and coherent patho-physiological explanation for this clinical phenomenon had also developed across that period. Many studies had shown that there was an association between the severity of the concussive injury and the consequences, but there was ample evidence too that even injuries presumed mild or minor might have severe consequences. Even authors who interpreted such cases as reflecting psychosocial adjustment issues, such as Jurgen Ruesch and Karl

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[193] *Id.*

[194] *Id.*

[195] *Supra* §§ V.E.6-10.

Bowman, admitted that there were a few cases in which trauma appeared to create chronic symptoms.

191. Attempting to make a clear and precise analysis of the effects of a head injury in football by studying players before, immediately after, and in later follow-ups, was made further problematic by the very fact that many of the players had *already* experienced concussion or mild head injury (as noted above, 19% of players in the study reported already having had 1 previous, a further 22% reported 2 or more prior head injuries). Thus, in contrast with their first publication on the effects of head injuries in college football, when they published their second in 1996 in the journal *Neurosurgery*, Barth and colleagues excluded all individuals who had sustained more than one head injury from their sample. This fact alone suggests that the authors considered more than one head injury a significant enough variable that it might exaggerate their findings. Even when this population of players was removed from the analysis, the data was still suggestive. As they stated:

> Whether football injuries are pathophysiologically analogous to mild head injury sustained under great acceleration-deceleration forces, such as vehicular accidents, remains to be determined, but players did evidence a combination of neurocognitive impairment, headaches, and dizziness typically observed after mild head injury of mixed causes in a more heterogeneous (age, education, occupation, cause of injury) population.[196]

192. In other words, a single mild head trauma resulted in an event that elicited brain changes unlikely to be psychosomatic in origin.[197] The fact that Barth's prospective study ultimately originated in numerous similar studies of the neuropsychology of sports concussion in the 1990s and after, thus represents a considerable irony in the history of concussive injuries. It seems obvious that Barth and colleagues were seeking to generalize their findings to make this single clinical point and not to initiate a whole field of inquiry focused on the neuropsychology of sports

[196] S.N. Macciocchi *et al*., *Neuropsychological Functioning and Recovery After Mild Head Injury in Collegiate Athletes*, 39 NEUROSURGERY 3, 510-14, 514 (1996).

[197] This point was one hinted at in Barth, *supra* n.170 at p. 271.

concussion only. As they put it in the 1996 article, most football players recovered quickly without residual symptoms.[198] And yet, it was not the fact of recovery from concussion that had concerned scientists and clinicians for over a century, it was the fact that sometimes recovery *did not* occur.

193. So, a conservative or precautionary approach to managing concussion in sport was a prudent response to the mass of research findings available by the 1990s. As Mark Lovell and Michael Collins put it in an argument for baseline neuropsychological testing of athletes in 1998:

> Given the potential for serious and permanent injury with multiple concussions, we suggest that if deficits are found on any of the neuropsychological tests, compared to the player's baseline performance, the player not be returned to the field until these scores have returned to normal.[199]

194. As shown throughout this section focused on historical firsts, medical and scientific authorities using a wide variety of methodologies routinely observed that mild head injury could result in long-term behavioral and cognitive changes ranging in severity from persisting annoyances through to complete disability and degenerative disease. The record of publications throughout the twentieth century certainly justified significant consideration of the risk that multiple concussions could result in permanent injuries even if one concussion had not. Such observations, however, became more sharply politicized after 1998. But the important, clarifying, point is that in 1998 it was an uncontroversial observation that cumulative head injuries posed a significant risk to players, a recommendation that echoed the clinical injunction first made by James Crichton-Brown in 1872 that "great caution ought to be observed ever afterwards by anyone who has experienced such an injury."[200]

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[198] *Id.*; *supra* n.181, at 514.

[199] Lovell, *supra* n. 174 at 19.

[200] J.C. Browne, *Cranial Injuries and Mental Diseases*, 128 (1872).

F. Scientific and Clinical Research Providing Evidence and Explanation for the Effects of Concussion and Brain Injury

195. The historical record provides great discussion of the nature and extent of published observations on the effects of single and multiple concussions and minor closed head injuries, and the advancement of studies of populations of patients who had experienced concussive and sub-concussive injuries – as seen both in the general population, and specifically within the sports arena. Alone these studies represent an expansive knowledge base that existed since the late-nineteenth century on the medical effects of concussion and the occurrence of long-term sequelae deriving from these injuries. And yet, these studies did not have to stand alone. In the medical and scientific literature, these came to exist alongside a diligent scientific investigation of the precise ways that observations seen in these patient studies could be scientifically explained.

196. Pathological, physiological and a host of other laboratory, or controlled, experiments initiated in new clinical specialties and across academic disciplines sought to question, hypothesize, test, clarify and confirm ***how*** these injuries involved the brain, and ***how*** this produced the neurological and psychological symptoms that had been repeatedly witnessed over a century and a half. Such questions drove the patterns of research that followed from the 1870s until 2011.

197. In the nineteenth century, formal pathological investigations and experiments were first initiated. So, too, were surgical, physiological, neurological and psychiatric investigations, and almost all of those studies relied upon pathological and physiological techniques. In the 1910s and afterwards, clinicians and scientists increasingly showed marked awareness of psychological theories, and this created conflicts between those who saw pathological investigations as necessary for studying concussive injuries, and those who ascribed the symptoms to psychological processes only.

198. By the 1940s a biomechanical model began to influence the ways scientists and clinicians conceptualized concussive blows. At the same time, many of the core neuropsychological methods, especially studies relying upon reaction time,

were mobilized to study head injuries. These techniques obtained greater significance to psychological science as Freudian theory diminished in the 1960s and thereafter. In the mid-century, new staining techniques and the refinement of previous ones enabled still better pathological investigations into the impact of concussive and sub-concussive blows upon the tissues of the brain.

199. The invention of the computer and its rapid adoption into science facilitated significant change in the quantitative analysis of data findings. After 1961 clinical and scientific investigations into concussion and closed minor head injuries and their effects upon the brain began to rely upon more sophisticated statistical analysis. By the 1970s, developments in neurobiochemistry (the study of the chemicals, neurotransmitters and other molecules that make the nervous system function) underlay a shift from the characterization of concussion not simply in pathological terms, but also in chemical terms, introducing what people came to call the concussion cascade in the 1990s. In the 1980s, the influence of genetics led to studies of the association of genetic markers with long-term neurological consequences of repeated head injury.

200. After a limited impact of new imaging and scanning technologies on concussion research up until this point, in 2011 the introduction of a new imaging technique – diffusion tensor imaging – offered the possibility of demonstrating in the living body the structural disturbance of function previously only revealed by pathological investigation of the brain after death.

201. As evidenced by this brief overview, between 1871 and 2011 scientific laboratory investigation and other forms of controlled testing and imaging of the brain had advanced apace detailed scientific knowledge of concussion and closed minor head injuries and their effects upon the brain. Indeed, analysis of the historical record makes clear that pathophysiological investigations have been central in the elaboration of knowledge about the immediate and remote effects of concussions. The exact types of observations, investigations and hypotheses are explored in much greater detail in the chronology provided below.

G. Chronology of Significant Pathophysiological Investigations into Concussion and Closed Minor Head Injuries and their Effects upon the Brain

202. A chronology of pathophysiological investigations published can be used as an example that demonstrates the multigenerational pattern of observation, hypothesis formation, evidence gathering, and prediction testing that is reflected throughout the entire record:

- In 1872 a physician observes pathological evidence of brain damage from concussion.[201]
- In 1874 a neurologist observes that there are reasons for believing concussion can result in shock.[202]
- In 1877 there are reports of experiments in continental Europe of investigators using hammers on the heads of animals to create animal models of concussion.[203]
- In 1892 a scientist using animal models of concussion hypothesizes that concussions are not caused by compression or small hemorrhages but rather are caused by the bulging of cerebrospinal fluid in the brain, and that this is the mechanism by which brain damage occurs.[204]
- In 1892 an asylum superintendent hypothesizes that the mechanism of concussion is caused by an increased volume of blood in cerebral blood vessels which results in gaps in the brain tissue that leads to chronic brain degeneration.[205]
- In 1914 a physician with interests in nervous and mental diseases hypothesizes that trauma to the nervous system can create both organic and functional changes and furthermore hypothesizes that this explains the remote effects of head injuries.[206]

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[201] P.G. Hewett, *Abstract of a clinical lecture on a case of injury of the head*, 1 BR. MED. J. 576, 39-40 (1872).

[202] J.J. Putnam, *Progress in Medicine. Report on Diseases on the Nervous System*, 91 BOS. MED. & SURG. J. 5, 111-15 (1874).

[203] *The Pathology of Concussion*, 2 BR. MED. J. 887, 930 (1877).

[204] Miles, *supra* n.154.

[205] J. Macpherson, *Vacuolation of Nerve-Cell Nuclei In the Cortex in Two Cases of Cerebral Concussion*, LANCET 1127-1129 (1892).

[206] C.M.H. Howell, *Trauma in Relation to Certain Aspects of Nervous Disease*, LANCET 302-305.

- In 1916 a physician who is also a pathologist hypothesizes that a head injury creates, what he terms, a *locus minoris resistentiae* (a point of less resistance to injury) in the brain which he predicts makes patients more vulnerable to subsequent head injuries.[207]
- In 1917 a physician who is also a pathologist observes congestion and evidence of cellular decay following concussion in a post-mortem study of the brains of two soldiers and notes that authors have predicted small microscopic hemorrhages might follow concussion.[208]
- In 1924 a pathologist finds small hemorrhages in autopsied brains of individuals with known concussions who died of other causes and hypothesizes that the injury may have been caused by bulging cerebrospinal fluid.[209]
- In 1927 two neurologists hypothesize that what they call postconcussion neuroses are symptomatic evidence of the structural pathological changes observed by pathologists and advise that these neuroses would be better termed traumatic encephalitis.[210]
- In 1928 a pathologist observing the condition termed "punch drunk" in boxers, hypothesizes that the condition will be found in other boxers because of the risks of their occupation, and hypothesizes that it is similar to post-traumatic encephalitis, multiple concussion hemorrhages, and postconcussion neuroses and psychoses.[211]
- In 1930 a neurologist hypothesizes that trauma causes traumatic encephalopathy which gives rise to what he calls postconcussion neuroses.[212]
- In 1934 two neurologists hypothesize that physical trauma causes an acute state of traumatic encephalopathy and, moreover, argued that label to be

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[207] F.W. Mott, *The Lettsomian Lectures on The Effects of High Explosives Upon the Central Nervous System: Lecture II*, 1 LANCET 12, 441-48 (1916); *see also* F.W. Mott, *The Lettsomian Lectures on The Effects of High Explosives Upon the Central Nervous System: Lecture III*, LANCET (11), 545-52 (1916).

[208] F.W. Mott, *The Microscopic Examination of the Brains of Two Men Dead of Commotio Cerebri (Shell Shock) Without Visible External Injury*, 2 BRI. MED. J. 2967, 612-15 (1917).

[209] C.S.B. Cassasa, *Multiple Traumatic Cerebral Hemorrhages*, 24 PROC. N.Y. PATHOLOGICAL SOC'Y 101, 101-06 (1924).

[210] Osnato, *supra* n.67 at 181-214 (1927).

[211] H.S. Martland, *Punch Drunk*, 91 JAMA 15, 1103-07 (1928).

[212] M. Osnato, *The Role of Trauma in Various Neuropsychiatric Conditions*, 86 AM. J. PSYCHIATRY 4, 646-60 (1930).

the appropriate generic term for what they call postconcussion syndrome.[213]

- In 1943 a physicist produces a model of concussive injuries that expresses the injuries in biomechanical terms, resulting in the now accepted view that concussions are impact acceleration and deceleration injuries, that the damaging forces are caused by rotational acceleration, and that these forces create shear strain across the brain (Figure 14).[214]

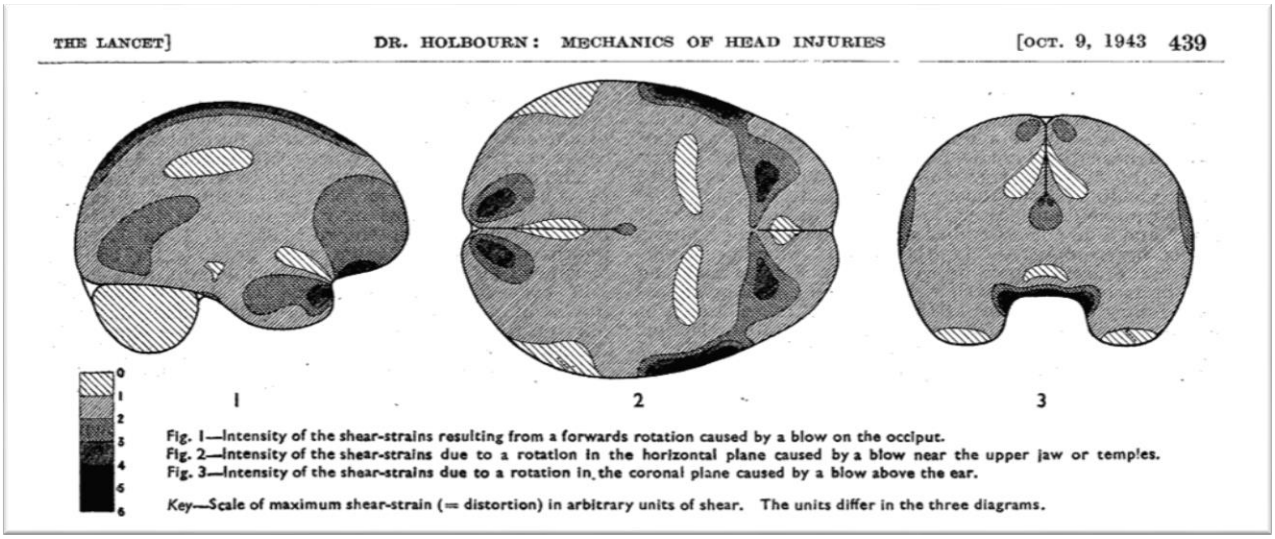

Figure 14. In 1943 A. H. S. Holbourne performed an experiment in which he created a gelatin model of the brain. The figure reproduced here is his diagram of a parasagittal section of the brain showing shear-strains produced by rotational force by a blow on the occiput. The gelatin was made in such a way that shear-strains would produce colors making the effect of the force visible.[215]

- In 1945 two authorities experimentally observed in an animal model a cumulative effect from successive concussions that also resulted in neuronal loss.[216]

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[213] Strauss, *supra* n.101 at 893-955 (1934).

[214] A.H.S. Holbourne, *Mechanics of Head Injuries*, 242 LANCET 6267, 438-41 (1943).

[215] *Id.*

[216] These authors, it should be noted described previous experiments where they have demonstrated cytological changes following concussion. "The rapid clinical recovery from symptoms of uncomplicated brain concussion has led to the suggestion that the condition may be a reversible one. Were it not for the marked changes we have seen in nerve cells after concussion there would be little reason to suspect permanent brain damage. From the cytological point of view it is inconceivable that extreme degenerative changes involving ballooning of the cell and complete loss of Nissl substance can be reversible. Nevertheless no direct proof has been offered that such altered nerve cells die and disappear after concussion. We cannot fully understand post concussional behavioral changes until we know whether structural damage is permanent." *Id*. at 201-

- In 1956 a pathologist analyzing rotational acceleration shows that shear strain forces cause nerve fiber damage. The pathologist makes clear that it is unknown whether nerve degradation produces the signs of concussion but stresses that the possibility should be borne in mind.[217]
- In 1957 a neurologist building upon further studies of repeated head trauma in boxers observes that this condition results in a tangle pathology characteristic of senility. The neurologist states clearly that this condition can be understood generally as a form of chronic traumatic encephalopathy, which is hypothesized to have resulted from multiple minor cerebral contusions, perhaps with pinpoint haemorrhages, later gliosis (reactive change of glial cells) and cortical atrophy.[218]
- In 1959 a physician reviewing the literature on boxing injuries stated that concerns about chronic traumatic encephalopathy were overblown and that it had never been proved to be a neurological syndrome peculiar to boxers or produced by boxing. Despite offering this criticism, the physician clearly connected acute cerebral trauma with chronic traumatic encephalopathy.[219]
- In 1962 a neurologist reviewing the literature on concussive injuries hypothesized that the accumulated evidence suggested that even mild concussions result in nerve cell damage which might have a negligible immediate effect but leaves the brain more susceptible as a whole to the effects of further damage.[220]

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202. In their conclusions, the authors claimed that they have found that blows to the head lead to structural alterations. The swelling of the nerve cells results in chromatolysis and the stronger the blow the more nerves involved. Importantly, "A series of blows [spread out over weeks] appeared to have a cumulative effect. After two light concussions and four severe concussions, each spaced approximately a week apart, in one animal the large interneurons of the reticular formation of the brain stem as well as the large neurons in the lateral vestibular nucleus were reduced in number to less than half found in these groups of the control animal." *Id*. at 209.

See also W.F. Windle & R.A. Groat, *Disappearance of nerve cells after concussion*, 93 THE ANATOMICAL RECORD 2, 201-09 (1945); *see also* W. F. Windle & H. W. Magoun, *Functional and Structural Changes in the Central Nervous System During and After Experimental Concussion*, TRANSACS. AM. NEUR. ASS'N 70, 117-22 (1944).

[217] S.J. Strich, *Diffuse Degeneration of the Cerebral White Matter in Severe Dementia Following Head Injury*, 19 J. NEUROL., NEUROSURG, & PSYCHOL. 3, 163-85 (1956).

[218] M. Critchley, *Medical Aspects of Boxing, Particularly from a Neurological Standpoint*, 1 BR. MED. J. 5015, 357-62 (1957).

[219] I.A. McCown, *Boxing Injuries*, 98 AM. J. SURG. 3, 509-16 (1959).

[220] C. Symonds, *Concussion and Its Sequelae*, 279 LANCET 7219, 1-5 (1962).

- In 1965 a neurologist at a professional meeting said that postconcussion syndrome cannot be evidence of brain damage and is different from chronic traumatic encephalopathy. The neurologist offered as evidence for this claim his broad experience of thousands of patients with pending or current litigation whom he implies are either malingerers or psychological cases.[221]
- In 1965 a neurologist at the same professional meeting as the above points out in criticism of the former talk that there would be considerable selection bias in drawing any lessons about head injury from a pool patients with pending or current litigation.[222]
- In 1968 pathologists showed that permanent damage can be inflicted on the brain from a concussion and concluded that such injuries, if repeated, would result in progressive, cumulative loss of tissue and of nervous function.[223]
- In 1973 a pathologist using autopsied brains from boxers found chronic traumatic encephalopathy in those new samples and noted the presence of tangle formations. He predicted that while not every punch need visibly alter the structure of the brain, one or many of them to the head would eventually leave a mark, which would initiate the early stages of the degenerative nervous disease and become exaggerated with repeated injuries.[224]
- In 1974 neurosurgeons hypothesize that shearing takes place in head injuries, including concussion, and they produce a flow chart of the mechanics of head injury and the areas where sufficient experimental research has been done to formally test. They also identify areas where further research is still needed (Figure 15). They hypothesize that the biological response to cerebral concussion is post-traumatic sequelae, but note further observation work needed to be done to clarify whether primary lesions lead to the formation of secondary lesions, initiating degenerative changes.[225]

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[221] H. Miller, *Mental Sequelae of Head Injury*, PROC. ROYAL SOC'Y MED. 59: 257-61 (1965).

[222] W.R. Russell, *Comment*, PROC. ROYAL SOC'Y MED. 262 (1966).

[223] D.R. Oppenheimer, *Microscopic Lesions in the Brain Following Head Injury*, J. NEUROL., 31 NEUROSURG., & PSYCHOL. 4, 299-306 (1968).

[224] J.A. Corsellis *et al*., *The aftermath of boxing*, 3 PSYCHOL. MED. 3, 270-303 (1973).

[225] Ommaya, *supra* n.82.

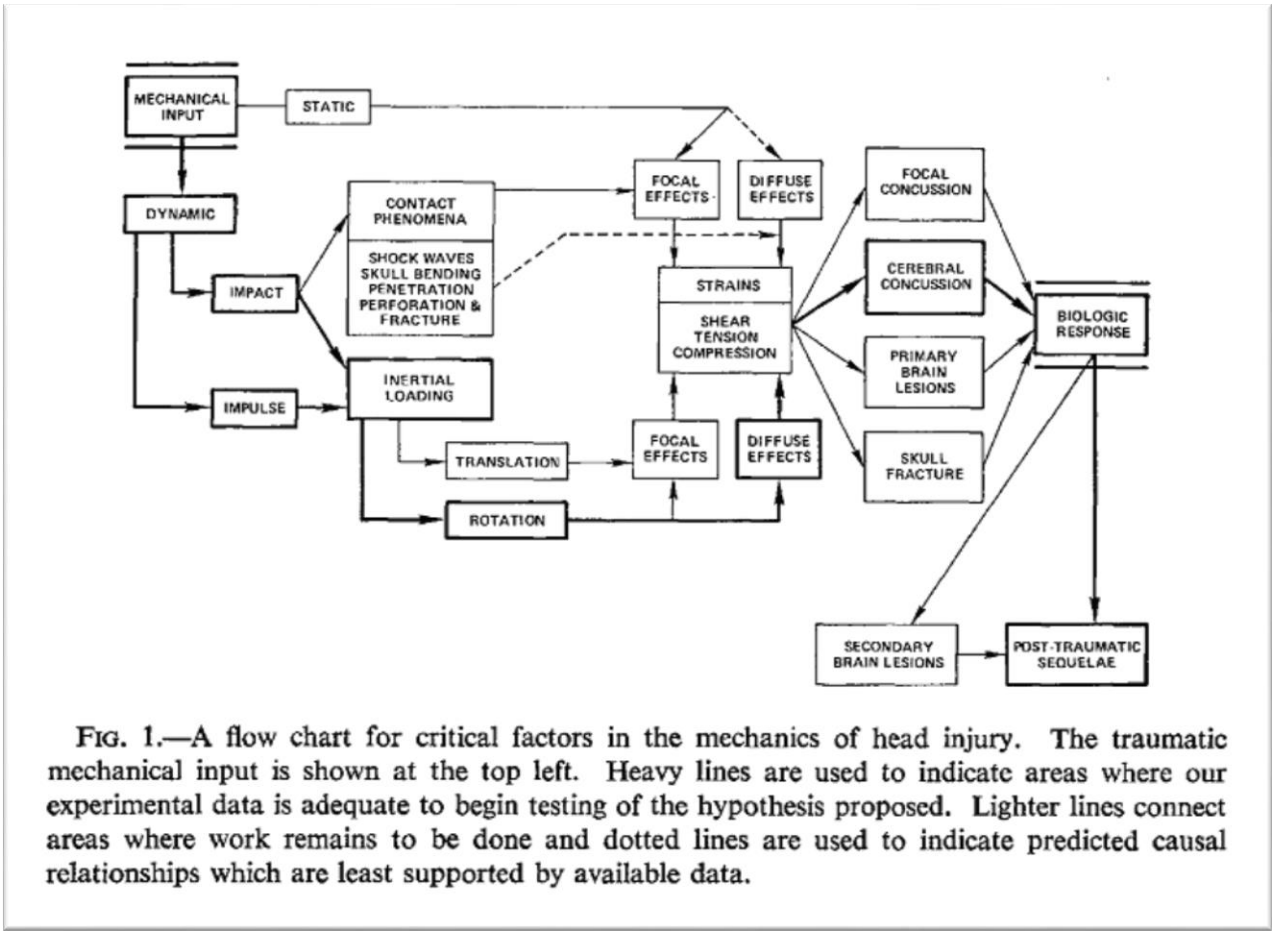

FIG. 1.—A flow chart for critical factors in the mechanics of head injury. The traumatic mechanical input is shown at the top left. Heavy lines are used to indicate areas where our experimental data is adequate to begin testing of the hypothesis proposed. Lighter lines connect areas where work remains to be done and dotted lines are used to indicate predicted causal relationships which are least supported by available data.

Figure 15. A 1974 flow chart and caption showing hypotheses that had been tested or needed to be tested. The diagram provides evidence that extensive scientific investigation had by then taken place.

- In 1975 two neuropsychologists demonstrate intellectual dysfunction as a result of cumulative concussions; they speculate that pathophysiology may explain these changes, but say that even though it remains unclear, athletes should be notified that it appears the effects of concussion are cumulative.[226]
- In 1981 authors of a prospective study of 538 hospital admissions with head injury find noteworthy persisting difficulties and say that these observations lend wait to the observation that the effects of concussion, however slight, might not be completely reversible.[227]
- In 1983 a neurosurgeon at a conference on car accidents argues that the evidence gathered from many studies fit, a) the biomechanical model of

[226] D. Gronwall & P. Wrightson, *Cumulative Effect of Concussion*, 306 LANCET 7943, 995-97 (1975).

[227] R.W. Rimel *et al*., *Disability Caused by Minor Head Injury*, 9 NEUROSURG. 3, 221-28 (1981).

concussion, b) the connection between concussion and more severe injuries, c) the fact that neuropsychological sequelae follow concussions and in cumulative ways, d) neuropathological evidence, and e) population data.[228]

- In 1986 a neurosurgeon reviewed the trends of concussion research and hypothesized that the reversal of neural dysfunction normally seen following concussion is due to the unexcitability of axonal membranes following primary mechanical strains. He observed that it was unknown why some dysfunctions persisted but suggested it may be due to permanent structural changes in axons or neural networks.[229]
- In 1989 a pathologist described the pathology of repeated head trauma using boxers as an example; confirms the presence of tangles but absence of amyloid plaques, and observed that current advanced imagining techniques cannot show the incipient disintegration of living fiber pathways or groups of neurons.[230]
- In 1996 and 1999 a group of pathologists validated previous findings about chronic traumatic encephalopathy and noted in the later study that the presence of tau fragments is evidence that nerve cells are breaking down.[231]
- In 2002 a pathologist observed the pathological hallmarks of chronic traumatic encephalopathy in the autopsied brain of a retired National Football League player. The study was published in 2005. It was followed by another case in 2006.[232]
- In 2005 sports medicine researchers surveyed retired professional football players and expressed concern "that the onset of dementia-related

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[228] A.K. Ommaya, *The Head: Kinematics and Brain Injury Mechanisms,* THE BIOMECHANICS OF IMPACT TRAUMA, 120 (1984).

[229] T.A. Gennarelli, *Mechanisms and pathophysiology of cerebral concussion*, 1 J. HEAD TRAUMA REHAB. 2, 23-29 (1986).

[230] J.A. Corsellis, *Boxing and the Brain*, 298 BR. MED. J. 6666, 105-09 (1989).

[231] J.F. Geddes *et al*., *Neurofibrillary Tangles, but not Alzheimer-type Pathology, in a Young Boxer*, 22 NEUROPATHOLOGY & APPLIED NEUROBIOLOGY 1, 12-16 (1996); J. F. Geddes *et al*., *Neuronal cytoskeletal changes are an early consequence of repetitive head injury*, 98 ACTA NEUROPATHOLOGY 2, 171-78 (1999).

[232] B.I. Omalu *et al*., *Chronic Traumatic Encephalopathy in a National Football League Player*, 57 NEUROSURG. 1, 128-34 (2005); B.I. Omalu *et al*., *Chronic Traumatic Encephalopathy in a National Football League Player: Part II*, 58 NEUROSURG. 5, 1086-93 (2006).

syndromes may be initiated by repetitive cerebral concussions in professional football players."[233]

- In 2009 a research group reviewed the symptoms of 47 previously identified cases of chronic traumatic encephalopathy and compared them with three new cases.[234]
- In 2011 radiologists introduced a new imaging technique called diffusion tensor imaging which produces evidence, as previously predicted, of declining white matter integrity following post-concussion syndrome.[235]

203. Two further, later studies came to attention as this declaration was nearing completion, deserve mention:

- In 2014 radiologists demonstrate alterations in white matter microstructure in ice hockey players using diffusion tensor imaging;[236] and
- In 2016 a research group demonstrated that a patient predicted to have chronic traumatic encephalopathy based on clinical presentation did not, providing strong evidence that CTE researchers can both falsify as well as validate clinical observations, an essential component of good science.[237]

204. As this chronology shows, the historical record from 1871 through 2011 contains ample evidence of a long-standing, multigenerational research program focused on the pathophysiological investigation of concussive and minor closed head injury. It is essential to acknowledge that the evidence gathered by scientists and clinicians in their investigations was studied and refined by subsequent generations of investigators, a

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[233] K.M. Guskiewicz *et al*., *Association between Recurrent Concussion and Late-Life Cognitive Impairment in Retired Professional Football Players*, 57 NEUROSURG. 4, 719-26, 719 (2005).

[234] A.C. McKee *et al*., *Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy Following Repetitive Head Injury*, 68 J. NEUROPATHOLOGICAL & EXPERIMENTAL NEUROL. 7, 709-35 (2009).

[235] M. Smits *et al*., *Microstructural brain injury in post-concussion syndrome after minor head injury*, 53 NEURORADIOLOGY 8, 553-63 (2011).

[236] T. Sasaki *et al*., *Hockey Concussion Education Project, Part 3. White Matter Microstructure in Ice Hockey Players with a History of Concussion: A Diffusion Tensor Imaging Study*, 120 J. NEUROSURG. 4, 882-90 (2014).

[237] J. Branch, *Autopsy Shows that the NHL's Todd Ewen Did Not Have CTE*, N. Y. TIMES, Feb. 10, 2016, http://www.nytimes.com/2016/02/11/sports/hockey/autopsy-shows-the-nhls-todd-ewen-did-not-have-cte.html?_r=1.

fact often indisputable because of the visible patterns of citation that reference the work of earlier figures. It is noteworthy, too, that with each generation the pathophysiological evidence became increasingly refined and explanatory.

205. For most of the generations of clinicians represented in this declaration, the burden of persuasion tended to fall on the shoulders of pathologists and physiologists, a reality that only began to dramatically change with the introduction of the randomized clinical trial in the postwar period. In the early 1930s the evidence of traumatic encephalopathy in a patient was the presence of subjective symptoms following a blow to the head. When these symptoms persisted, as they did in an occupational group exposed to dramatically higher risks for head injury than normal population (boxers), clinicians drew a line between an acute, potentially reversible phase, and a chronic phase which might not deteriorate but could with further exposure to head injury. The pathological hallmarks of the acute phase were harder to demonstrate, precisely because it was a rare individual who came to autopsy following a known concussion. As this chronology shows, however, when those rare cases did make it into autopsy, investigators reported seeing traces of physical brain damage (initially identified as microscopic contusions, then as neuronal loss, then senile tangles, and by the 1990s, as tau protein). This association between a pathological observation and clinical observations alone was insufficient evidence that the pathology was pathognomic.

206. Investigations after the 1930s thus sought, in physiological and biomechanical studies, explanations for the pathological and clinical observations that had already been made. By the 1980s this research was sufficiently developed that a neurosurgeon could hypothesize that simply stretching – not destroying – a neuron could result in dysfunctions that would be experienced by patients as the acute symptoms of concussion. Anything worse might result in the cell loss observed by pathologists from the 1930s on. By the 2010s, the new technology of diffusion tensor imaging appeared to validate the prediction of white matter dysfunction following mild concussion. In sum, the tendency in the research record here has been progressive advancement of the

science towards a clearer understanding of why even a single concussion can be a dangerous injury, and certainly why multiple concussions provide compounding risk of permanent impairment.

H. Head Injury Risk in Amateur and Professional Contact Sports

207. Given everything described above in this declaration, it is perhaps unsurprising that parallel to these was the emergence of medical queries regarding the safety of sporting activities in both amateur and professional contexts, the production of scientific and clinical investigations into the specific effects of concussion in sporting arenas, and, ultimately, medical guidelines for the assessment and management of concussion in sports.

208. In British and North American medical journals in the earliest years covered by this declaration, it was the dangers that football (both British and American variants) posed that drew specific attention. These early editorials and clinical articles importantly show that there was already some suspicion on the part of medical authorities that the blows received to the head in contact sports were of greater danger than popular understanding might have made them out to be. But the landmark studies were published in the 1920s and 1930s. These studies collectively demonstrated that a history of repeated blows to the head and multiple knockouts resulted in degenerative brain pathology. The earliest study focused solely on boxers who were described colloquially as "punch drunk." Studies subsequently published in the 1930s and beyond began to extend the lessons learned with regard to boxing to all contact sports. As a consequence, medical authorities in the 1930s, 1950s, and 1970s suggested in explicit terms that athletes should be apprised of the risks engendered by blows to the head sustained in contact sports. These concerns culminated in the publication of evidenced-based guidelines for the management of concussion in sports.

209. The development of medical and scientific literature on the risk of head injury in sports specifically is thus addressed in the rest of this section under the following headings:

- Early medical voices expressing concern about the risks of contact sports;

- Landmark studies of the 1920s and 1930s demonstrating degenerative brain pathology in sports players;
- Contact sports and the emergence of sports medicine, 1940 to 1990; and
- Medical guidelines for the management of concussion in sports, 1970s to 1990s.

1. Early Medical Voices Expressing Concern about the Risks of Contact Sports

210. In an 1886 editorial in the *British Medical Journal* an anonymous author complained that it was only fatalities or such serious injuries as "concussion of the brain" that were reported in the newspapers. But beyond such lay reports, the author noted, there was little actual medical data available to judge the dangers of sports.[238] In a subsequent editorial describing another "concussion of the brain" sustained during English football, it was observed:

> A great many habitual players appear to be unaware of the number of more or less serious accidents which occur weekly throughout the winter season; and it is a fact, whatever its explanation that it is exceedingly difficult to obtain complete statistics. If the Football Association would take seriously in hand the duty of collecting these statistics, it would do a very useful work, and the result would probably compel it to take steps to put an end to the existing scandal.[239]

211. In 1894 *The Lancet* also reflected on the safety of English football and noted yet again such severe injuries as brain concussions,[240] with the result that the editors of *The Lancet* ultimately called for rule changes to make the Association Football safer.[241] These calls were followed by other similar ones in the same year seeking to regulate high school boxing, with the dangers of brain concussions being raised as a commonplace example.[242]

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[238] *The Foot-Ball Accident Season*, 2 BR. MED. J. 1348, 831 (1886).

[239] *Id.* at 1115.

[240] *The Perils of Football*, LANCET 764-767 (1894).

[241] *Id*

[242] Authorities, for example, sought to regulate high school boxing in Britain in 1898. "At the least, concussion of the brain must ensue when a vigorous and dexterous youth

212. After the turn of the twentieth century, the medical and scientific literature reflects that such debates occurred in the United States as well. In a 1905 editorial *The Lancet* observing the debate in the United States from afar, it was commented that the violent nature of American-style football had resulted in the President of Harvard questioning the spirit of the game and "President Roosevelt" intervening to introduce reforms to the sport.[243] In 1906 in the course of that civic debate, Nicols and Smith published in the *Boston Medical and Surgical Journal* the first prospective survey of football injuries in a season at Harvard. This survey had asked players to report injuries, "no matter how trivial."[244] In the section of their report dealing with head injuries, Nicols and Smith observed that "cases of concussion were frequent . . . but two games were played during the entire season in which a case of concussion of the brain did not occur."[245] They commented:

> The mental state of the players who had concussion was variable, some being highly excitable and hysterical, others merely confused, and in a few cases, knocked completely unconscious. In every case there was a certain loss of memory, both previous and subsequent to the injury. The loss of memory previous to the injury varied from a few minutes to a week….Concussion was treated by the players in general as a trivial injury and rather regarded as a joke. The real seriousness of the injury is not certain. Our own experience with the after effects of the cases is not sufficient for us to draw any definite conclusions, but from conversation with various neurologists, we have obtained very various opinions in regard to the possibility of serious after effects.[246]

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lands his fist against the chin of his opponent. The force may be simultaneously transmitted to both condyles, so that the base of the brain receives a shock to the right and to the left. In fact, the "incapacitation" at which the striker aims is due to concussion. This injury is bad enough, but worse may be inflicted by the blow. Clearly schoolmasters cannot tolerate the concussion of their boys' brains even by a recognized blow in boxing." *The Knock-Out Blow*, 2 BR. MED. J. 1, 1946-1947 (1894).

[243] *The Question of Football*, LANCET 1422-1423 (1905).

[244] E.H. Nichols & H.B. Smith, *The Physical Aspect of American Football*, 154 BOS. MED. & SURG. J. CLIV 1, 1-8 (1906).

[245] *Id.* at 3.

[246] *Id.*

213. If Nicols and Smith were uncertain, the editors of the *BMJ* were less sanguine: "In every game except two, one or more players suffered from concussion of the brain, the cases being of every grade of severity, from mere hysterical irresponsibility or confusion to complete coma."[247] American football, for the editors, was more violent than "prize-fighting, cock-fighting, and bull-fighting."[248]

214. While early editorials and clinical articles had thus already evinced concern about the dangers of concussion and minor closed head injury in professional and amateur sports, it was in the 1920s and 1930s that landmark studies were published that made the risks explicit.

2. Landmark Studies of the 1920s and 1930s Demonstrating Degenerative Brain Pathology in Sports Players

215. The punch-drunk fighter had been a figure of public imagination long before he was a figure of medical knowledge. An example quoted in the medical literature in 1934 was drawn from the memoir of Gene Tunney, the world heavyweight champion boxer of 1926, who had an illustrious boxing career of seventy-seven bouts with only one recorded professional defeat.[249] Tunney reflected upon the events that had resulted in his retirement in 1928:

> One day while boxing with a sparring partner, Frank Muskie, we bumped heads. The part of my skull which is the thinnest, near the temple, struck the toughest part of his, the top. I was terribly dazed. As I straightened up a long, hard right swing landed on my jaw. Without going down or staggering I lost all consciousness of what I was doing and instinctively proceeded to knock Muskie out. Another sparring partner, Eddie Eagen, entered the ring; we boxed three rounds. I have no recollection of this, nor have I any recollection of anything that occurred until the next morning when I was awaked in my little cabin by the water's edge, wondering who I was and what I was doing there. As I lay in this awful state of retuning consciousness I became greatly frightened. Gradually my name came to me. That I was a pugilist soon followed, then the thought of being

[247] *American Football*, 1 BR. MED. J. 2354, 344-45 (1906).

[248] *Id.*

[249] *Gene Tunney*, ENCYCLOPAEDIA BRITANNICA, http://www.britannica.com/biography/Gene-Tunney (last visited Mar. 30, 2016).

> champion – impossible – unbelievable. I must have had a long dream. Gradually came the realization that I had not been dreaming. I rose and asked guarded questions. I wanted to know all about the events of the day before. For 3 days I could not remember the names of my most intimate acquaintances. I had to stop training. I did not leave my cabin except to eat or take a short walk. On these occasions all seemed queer. I was unable to orient myself. The sensation I had was as though hot water had been poured through a hole in my skull and flowed down over the brain to my eyes, leaving a hot film. There were three newspaper men at camp reporting my activities. They had to be deceived. This story was too sensational to permit it to get out. After returning to normal, I decided that any sport in which such accidents could occur was dangerous. I realized I had a concussion. The first seed of retirement was sown then. The possibility of becoming punch drunk haunted me for weeks.[250]

216. As Tunney's reminiscence shows clearly enough the consequences of repeated blows to the head were recognized in boxing circles. As another medical authority observed in 1936, lay people called the condition by many names, including: "'punchy,' 'goofy,' 'slap happy,' 'cutting paper dolls,' or 'slug nutty.'"[251]

217. In 1928 the first medical report characterizing both the pathology and prognosis of the condition appeared. The author, Harrison Martland, an American pathologist, claimed to have been drawn to study the condition by lay accounts of the infirmities of boxers. Through autopsy of deceased boxers' brains, as well as extensive personal interaction with pugilists and the boxing world, Martland's seminal report showed an association between blows to the head in a contact sport, multiple punctate hemorrhages, and chronic brain symptomology. Thus, "Punch Drunk" provided an opportune case study through which to test and further develop earlier pathology reports by his contemporaries who had shown some of the same findings in their studies. Martland, in particular, discussed the pathological and neurological observations of Casassa and Osnato and Giliberti.[252]

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[250] Strauss, *supra* n.101 at 893-955 (1934).

[251] E. Carroll, *Punch-Drunk*, 191 AM. J. MED. SCIS. 5, 706-12 (1936).

[252] Throughout his article, Martland discussed the work and findings of Casassa, Osnato, Giliberti, and his own previous work with Beling. *See* Martland, *supra* n.197.

218. Martland opened his study by observing that a tremendous blow during a boxing fight might appear to initiate the condition. He wrote, "I know of one fight that was stopped by the referee because he thought one of the fighters intoxicated."[253] But, punch-drunkenness, Martland clarified, was rarely the result of a single blow. Rather it was an occupational disease that resulted from repetitive blows to the head. The symptoms, he wrote:

> …usually appear in the extremities. There may be only an occasional and very slight flopping of one foot or leg in walking, noticeable only at intervals; or a slight unsteadiness in gait or uncertainty in equilibrium. These may not seriously interfere with fighting. In fact, many who have only these early symptoms fight extremely well, and the slight staggering may be noticed only as they walk in their corners…. Many cases remain mild in nature and do not progress beyond this point. In others a very distinct dragging of the leg may develop and with this there is a general slowing down in muscular movements, a peculiar mental attitude characterized by hesitancy in speech, tremors of the hands, and nodding movements of the head…. Later on, in severe cases, there may develop a peculiar tilting of the head, a marked dragging of one or both legs, a staggering, propulsive gait with the facial characteristics of parkinsonian syndrome, or a backward swaying of the body, tremors, vertigo and deafness. Finally, marked mental deterioration may set in necessitating commitment to an asylum.[254]

219. Martland claimed that the occurrence of these symptoms appeared about 50 percent of the time in fighters, either in a mild or severe form. This fact, he stated "seems to be good evidence that some special brain injury due to their occupation exists."[255] For Martland, it was plainly obvious that the punch drunk symptoms were "due to single or repeated blows on the head or jaw" and that the brain pathology of the punch drunk boxer resulting from these repeated blows to the head was "multiple concussion hemorrhages in the deeper portions of the cerebrum."[256] He postulated that "these hemorrhages are later replaced by a gliosis or degenerative progressive lesion in

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[253] Martland, *supra* n.197 at 1103.

[254] *Id.*

[255] *Id.*

[256] *Id.*

the areas involved…."[257] He was also clear that he regarded the relationship between mental disturbance following a concussion to be related to the extreme pathology embodied by the boxer. In his conclusion, he wrote "Punch drunk bears the same relation to multiple concussion hemorrhages as do many of the postconcussion neuroses and psychoses that follow blows or falls on the head."[258]

220. In this way, Martland's study refined his contemporaries' knowledge of the dangers of brain concussions. The significance of his study was that it clarified the hazards faced by those rare individuals in occupations where repeated concussions represented a real predictable risk. Indeed, Martland's prognosis was that repeated acute brain injuries, if not stopped, would result in grave, permanent, and likely degenerating neurological disease.

221. For all its great originality and importance, it is worth noting that symptoms of seeming drunkenness following head injury had been observed anecdotally decades before Martland published his paper, and Martland's study had also been partially anticipated in past medical literature by his contemporaries in pathology. In 1890, for example, Battle had described two patients who, following head injuries, showed symptoms of intoxication. These cases resulted ultimately in death.[259] In 1894, an editorial in the *Journal of the American Medical Association* observed that cases of concussion could be confused with drunkness by ambulance physicians, druggists, and officers of the law.[260] "Drunk or Dying?" asked an editorial in *The Lancet* in 1900, warning that it was easy to conflate the symptoms of cranial head injury with drunkenness.[261] Martland's study had also been anticipated in observations made in

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[257] *Id.*

[258] *Id.* at 1107.

[259] W.H. Battle, *Three Lectures on Some Points Relating to Injuries to the Head: Lecture III*, 2 BR. MED. J. 1542, 141-47 (1890).

[260] *Outdoor cases of coma*, JAMA, 243-44 (1894).

[261] The word "drunk" was not used in this study as a keyword. I am confident that were a historical search conducted of the medical literature cited throughout this declaration

James Gray Duncanson's study of the "knock-out blow" in boxing. As Duncanson observed of the upper-cut blow, the recipient "staggers around as if groping in the dark or like a drunken man"[262] and the consequences of such a devastating blow, he stated, could result in a "man being left dull and apathetic, never to gain his old nerve and courage."[263]

222. These early studies demonstrate the many ways that Martland's research did not exist in a vacuum. Careful attention to Martland's paper makes clear that he was asserting a position held by many of his contemporaries about the pathophysiological mechanism of concussion, particularly in the way in which he sought to augment the pathological studies of his contemporaries (discussed *infra* Section V.G). All of those authors were seeking to clarify the pathology of concussion by showing structural lesions, evidenced by visible multiple punctate hemorrhages that followed blows to the head. Martland, like other pathologists, was clear that it was in such residual pathological details that the causes of the observable mental disturbances following concussion were to be found.

223. Martland's study was seen as a vindication of this position. As an anonymous author wrote in the *Journal of the American Medical Association* in 1929, Martland's study hailed "a new era in our knowledge of this subject [that] is bound to begin with the accurate investigation of the brains thus affected, of the early and late changes, and especially of their topographic distribution. These there should be added reliable details about the nature of the violence concerned in producing the pathologic changes in the tissues."[264]

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specifically searching the intersection of "drunk" and "head", then numerous reports of these kinds would be found.

[262] J.G. Duncanson, *The Knock-Out Blow On The Point Of The Jaw*, 1 BR. MED. J. 2205, 782-83, 782 (1903).

[263] *Id.* at 783.

[264] Martland, *supra* n.197 at 314.

224. Martland's study was followed by others in the 1930s. In a 1934 paper, entitled "Traumatic Encephalopathy ('Punch Drunk') of Professional Pugilists," Harry L. Parker of The Mayo Clinic in Rochester, Minnesota, described three punch drunk boxers. Importantly, Parker clarified sharply that the punch-drunk boxer should be understood to have developed chronic symptoms through repeated exposure to a usually acute condition. As he stated in the opening of his paper:

> The result, immediate or remote, of repeated injuries to the brain of a professional boxer forms a study all in itself, and contrasts with the more usual sequelae seen when patients have received only one injury in the course of industrial or other pursuits. The problem, moreover, forms one phase of the extremely complicated and highly controversial subject of head injury and its consequence. For purposes of description, the injuries received by pugilists in activities of their profession may be divided into those received during an actual bout, serious enough to cause death immediately or a few hours later, and those which more by their repetition than by their severity lead to slower development of disability during the fighter's career. It is with this latter group that this paper is concerned."[265]

225. Parker, like Martland, also recognized that the condition could be mild in some, while:

> in the more severe types the legs drag in walking, and tremors, dysarthria, deafness, physical slowing-up and even mental deterioration which requires commitment to an institution may supervene.… Lastly, a progressive neurological syndrome may appear, putting an end to all fighting, and leading finally to mental or physical helplessness. It is well understood, moreover, that pugilists are not immune to the ravages of syphilis, alcohol, or even epidemic encephalitis, and certainly the incidence of these diseases is no lower among pugilists than in the normal population. The point that Martland urged, however, was that the high frequency with which professional pugilists develop crippling disease of the central nervous system of one sort or another suggests a result of the repeated injuries to the brain that they received while carrying on the activities of their profession.[266]

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[265] H.L. Parker, *Traumatic Encephalopathy ("Punch Drunk") of Professional Pugilists*, 15 J. NEUROL. & PSYCHOPATHOLOGY 57, 20-28, 20 (1934).

[266] *Id.* at 20-21.

226. Parker then presented three cases. In one, Parker illustrated that the condition progressed as a consequence of fights; symptoms began, worsened through to a final injury, and then continued degenerating after retirement. Parker saw this as an almost unbroken chain of causality. In another case, Parker demonstrated that symptoms began in the course of the boxing career but stopped deteriorating further when the individual retired. He concluded:

> It is assumed that these patients' difficulties resulted from repeated injuries to the brain received during their pugilistic careers. If probabilities are to be discussed, the first patient was without reasonable doubt injured during his last fight and his subsequent difficulties resulted from it. In the case of the third patient, his difficulties appeared during his fighting career but stopped progressing as soon as he left the ring. The probability here, that his disease was connected with his occupation, is good.[267]

227. In the same year that Parker's publication appeared, Israel Strauss and Nathan Savitsky, two New York neurologists reviewed the general evidence about head injury. These authorities were among the most prominent neurologists in America at the time. In their study, they drew attention to the way Sigmund Freud and other psychoanalysts had greatly refined understanding of psychological dysfunction by calling attention to unconscious processes at work in the creating of anxiety, depression, and other mental disturbances. Yet they cautioned that in cases where evidence of acute head injury could be shown, it was essential to understand that an actual injury to the brain had occurred and that mental disturbances accordingly had been caused by the blow to the head. Importantly, it appears to have been these authors who first coined the term "post-concussion syndrome" to describe the various mental abnormalities that followed immediately after a concussion and usually resolved. Like Parker, Strauss and Savitsky also described this condition as "traumatic encephalopathy." It is important to quote from their conclusion to see that these authorities implied a spectrum from concussive injuries through post-traumatic syndrome, while suggesting that traumatic encephalopathy could refer to other, related conditions:

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[267] *Id.* at 28.

> 1. We have limited our consideration to cases of actual physical injury to the head.
> 2. Clinically, the organic sequelae of head injury should be differentiated from the terror and anxiety reactions following a threat to bodily integrity. The term "traumatic neurosis" may be confined to the latter type of reaction.
> 3. In our opinion, the subjective posttraumatic syndrome, characterized by headache, dizziness, inordinate fatigue on effort, intolerance to intoxicants and vasomotor instability, is organic and is dependent on a disturbance in intracranial equilibrium due directly to the blow on the head. We suggest the term "postconcussion syndrome" for this symptom complex.
> 4. "Traumatic encephalopathy" may be used as a generic term if it is understood that it includes cases in which physiologic disturbances of the cerebral mechanisms are present although organic lesions are not demonstrable.[268]

228. The inability to demonstrate the presence of lesions was implied precisely because the patient survived the blow to the head. In effect, Strauss and Savitsky were articulating the fact that there were two diagnostic possibilities. One was that the head injury was coincidental and that the symptoms described by the patient were psychosomatic. The other possibility, appearing to them the more commonsense explanation, was that a blow to the head actually did something tangible to the brain. The "something," in this sense, was traumatic encephalopathy revealed by the presence of postconcussion syndrome. In other words, these authors were envisioning a spectrum of injury. The concussion patient normally recovered because the brain was a resilient organ that could heal from a bout of acute traumatic encephalopathy. However, repeated episodes of acute traumatic encephalopathy could ultimately result in permanent, chronic traumatic encephalopathy. In this the example of a punch drunk boxer might be an extreme example, but he was also a clinically predictable casualty of repeated head injury.

229. Yet another publication in the 1930s (in 1936), echoed these investigations. Entitled "Punch-Drunk", the article appeared in the *American Journal of*

[268] Strauss, *supra* n.101 at 954.

Medical Science. Here, the author Edward Carroll adopted an almost anthropological interest in what he, like Parker, also called "traumatic encephalopathy": [269]

> I determined to study "punch-drunk" in its natural habitat, the boxing world. Accordingly, I frequented training quarters, helped examine fighters, and made friends with all sorts of "pugs." I observed declining veterans of the ring, talked with their trainers, and drank beer with their managers. I interviewed sports writers, referees and boxing commission physicians. I sought out reputed "punch-drunks" and studied them to the fullest extent that their feeling of exuberant good health would permit of a medical examination. My knowledge of the subject, then, is based upon the information given me by laymen whose business is a keen study of boxers, and the incomplete observation, over a period of 2 years, of many cases of "punch-drunk."[270]

230. Reflecting on the incidence of the condition, Carroll observed that it was not boxers alone who showed evidence of the disease:

> …competent followers of the [boxing] estimate that about 5% of men who remain in the professional ranks for a period of 5 years or more will become definitely punch-drunk. About 60% of fighters remaining in the ring for this period will develop mental and emotional changes which are obvious to people who knew them previously. A few deteriorate to the extent that institutionalization is necessary. Punch-drunk is said to occur among professional football players also.[271]

231. This fact led Carroll to reflect on the duty of his profession:

> It is probable that no head blow is taken with impunity, and that each knock-out causes definite and irreparable damage. If such trauma is repeated for a long enough period, it is inevitable that nerve cell insufficiency will develop ultimately, and the individual will become punch-drunk. The cognizance and investigation of this condition by the medical profession would be a contribution to the neurologic and psychiatric study of traumatic disorders. But a higher end would be the education of layman to the remote dangers incident to repeated minor head traumas. The occurrence of this type of degenerative brain change must be recognized and publicized rather than disregarded and discounted. ***It is especially important that athletes entering into competitions in which***

[269] Edward Carroll, *Punch-Drunk*, AM. J. MED. SCIS. 191 (5):706-712, 706 (1936).

[270] *Id.* at 706.

[271] *Id.* at 709.

> ***head injuries are frequent and knock-outs are common should realize that they are exposing themselves not only to immediate injury, but also to remote and more sinister effects.***[272]

232. It is essential to stress that last sentence. Medical writers had been calling attention to the dangers of concussions in sports since the 1880s. By the mid-1930s, reputable medical professionals were urging their field to educate lay people about the dangers of blows to the head in contact sports. Still more importantly the notion that athletes entering into competitions should receive full disclosure about the "remote and more sinister effects" of concussion had been clearly articulated in the medical literature.

233. By the beginning of the Second World War, North American authors were beginning to describe effects of repeated concussions as "traumatic encephalopathy." They were furthermore in consensus that the symptoms of that traumatic encephalopathy could be mild or severe, static or degenerating. Martland and Parker had described the condition as a hazard of an occupation. Strauss and Savitsky had coined the term postconcussion syndrome to describe an acute episode of traumatic encephalopathy. Carroll had suggested that the condition might also effect professional football players and had moreover indicated that ***any athlete*** who participated in contact sports at any level should realize the dangers to which they were exposed. He had also suggested that a morally higher purpose for the medical profession in this instance was heightening public awareness. All of these authorities thought brain damage occurred as a consequence of blows to the head.

3. Contact Sports and the Emergence of Sports Medicine, 1940 to 1990

234. The implications of traumatic encephalopathy found routes into other areas of scientific research. By 1940, high-altitude physiologists, for example, were contemplating the cumulative effects of oxygen deprivation in pilots and wondering whether such anoxia effects represented a model for understanding the pathophysiology

[272] *Id.* at 711 (emphasis added).

of punch-drunkenness.[273] A new trend, too, would become evident in this period as well. As the implications of traumatic encephalopathy sank in on both sides of the Atlantic, defenders of boxing emerged and with them a tendency to blame the victim rather than the cause.[274]

235. A 1941 editorial in *The Lancet* foreshadowed arguments to come in the 1950s and 1960s:

> There is, however, that specific problem of boxing, "punchdrunkenness" - the permanent damage due to repeated cerebral injury. This problem is more complicated than one would think …. There is no evidence to show, and some reason for disbelieving, that punchdrunkenness can be produced by one or two knockout blows. It is much more likely that continuous hammering, too frequent fights, fighting against heavier or otherwise superior opponents and continued fighting in a "groggy" state are responsible for traumatic encephalopathy. There is also evidence that a considerable proportion of punchdrunk boxers are defectives to begin with. The incidence of punchdrunkenness varies with the social status of the boxer; amateurs are rarely affected, good professionals and paid sparring partners are most liable. The social and economic pressure on these poor "chopping blocks" which makes them go on fighting in spite of all warnings is the main cause of punchdrunkenness.[275]

236. This dehumanizing and inherently political argument was echoed again and again by boxing apologists in the 1950s and 1960s, and it rang of class and race prejudice. It implied both that sufferers of traumatic encephalopathy were biologically defective in some way and that it was their lack of economic opportunity that made them carry on despite their own supposed awareness of inherited defects. The celebration of amateurism against professionalism rang of another class war that had long been ongoing in boxing and in other such contact sports as football and ice hockey – namely

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[273] W.W. Scott, *Physiology of Concussion*, 43 ARCH. NEUROL. & PSYCHOL. 2, 270-83 (1940); Martland, *supra* n. 197.

[274] An early letter writer defending boxing in the medical press was E. F. St. John Lyburn, *Medical Aspects of Boxing*, 238 LANCET 6165, 500-01 (1941).

[275] C.E.W. Lambert, *Punch with care*, 237 LANCET 6148, 759 (1941).

the view that sports and sportsmen should be unsullied by the corrupting influence of money.

237. But the word "defective" in 1941 also conjured another scientific landscape that dominated in wartime North America and Europe.[276] It was a word in the United States hearkening back to the infamous 1927 Supreme Court decision, *Buck v. Bell*, 274 U.S. 200 (1927) – it was an expression of eugenic commitment.[277] It is difficult to avoid seeing the continuation of those arguments even up to the studies of the association between traumatic encephalopathy and the APO E4 allele that would become a favorite device of distraction from the central issues surrounding the dangers of repeated head injuries, following the late 1990s.[278] These recurring arguments would find new iterations with each passing generation and were already established in opinion pieces published in the medical literature in 1941.

238. Carroll's 1936 injunction to the medical profession to educate lay people and athletes about the dangers of blows to the head was followed by other similar precautionary calls throughout the post-War period. Among the earliest figures was Augustus Thorndike, physician at Harvard University and a founder of the medical subspecialty of sports medicine. Thorndike had written extensively on athletic injuries.[279] His perhaps most famous scholarly contribution, however, was a 1952 review essay published in the *New England Journal of Medicine* which was entitled

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[276] D. Pick, *Faces of Degeneration: A European Disorder, c. 1848 – c. 1918*, CAMBRIDGE U. PRESS (1989).

[277] A. Bashford & P. Levine, *The Oxford handbook of the history of eugenics*, OXFORD U. PRESS (2010); M.M. Sokal, *Psychological testing and American society, 1890-1930*, RUTGERS U. PRESS (1987); S.F. Weiss, *The Nazi Symbiosis: Human Genetics and Politics in the Third Reich*, U. CHI. (2010).

[278] B.D. Jordan *et al*., *Apolipoprotein E e4 Associated With Chronic Traumatic Brain Injury in Boxing*, 278 JAMA 2, 136-40 (1997).

[279] A. Thorndike, *Athletic injuries: prevention, diagnosis and treatment*, LEA & FEBIGER, 1956; also *see* his: *Trauma Incident to Sports and Recreation*, 219 NEW ENG. J. MED. 13, 457-65 (1938) and *Prevention of Injury in Athletics*, 162 JAMA 12, 1126-32 (1956).

"Serious Recurrent Injuries of Athletes: Contraindications to Further Competitive Participation." In this essay, Thorndike argued that figures with responsibility for safety in organized college athletics had become increasingly aware of their objective duties to young college students. Observing that it was important to understand which recurrent injuries might lead to the "possibility or probability of a permanent disability to mind, body or limb," Thorndike stated that while it was a matter of opinion about how much physicians should allow repeated concussions, he thought it clear that "[p]atients with cerebral concussion that has recurred more than three times or with more than momentary loss of consciousness at any one time should not be exposed to further body-contact trauma. The college health authorities are conscious of the pathology of the 'punch-drunk' boxer."[280]

239. Thorndike added in his conclusions: "Body-contact sports should not be permitted for any student athlete who has suffered removal of the spleen or a kidney, or who has suffered three cerebral concussions of moderate degree, or one concussion, resulting in the diagnosis of laceration of the brain, or loss of an eye."[281]

240. Thorndike's article was treated by other authors as sage advice in the decades that followed.[282] It was only after 1990 that the article became a favorite punching bag of authors writing on various topics pertaining to the dangers of concussions in sports. Paul McCrory, a lead author on the various Consensus Statements on Sport Concussion, for example, pronounced Thorndike's approach anecdotal in 2001, even as he thoroughly misquoted him:

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[280] A. Thorndike, *Serious Recurrent Injuries of Athletes: Contraindications to Further Competitive Participation*, 247 NEW ENG. J. MED. 15, 554-56, 554 (1952).

[281] *Id.* at 556.

[282] *See, e.g.*, G. McLatchie & B. Jennett, *Head Injury in Sport*, 308 BR. MED. J. 6944, 1620-24 (1994). Also see the same recommendation in J.A. Bullard, *The Dimensions of Responsibility of the Team Physician*, 9 BR. J. SPORTS MED. 3, 117-21 (1976), a paper that discusses the responsibilities of the team physicians, with reference to a university team doctor. The author outlined what is required: the physician should identify completely disqualifying conditions, and included in this is "History of three or more concussions with loss of consciousness." *Id.* 117.

> This anecdotal approach was originally proposed by Quigley in 1945 and subsequently adopted by Thorndike, who suggested that if any athlete suffered "three concussions, which involved loss of consciousness for any period of time, the athlete should be removed from contact sports for the remainder of the season."[283]

241. It is easy to suspect why Thorndike's article presented a clear challenge to investigators who studied brain concussions in sports arenas and thus why McCrory, who was appointed editor of the *British Journal of Sports Medicine* in 2001, might have wished to creatively misread it in that year. Describing Thorndike's approach as anecdotal ignored precisely all of the extensive research that had been published in the preceding fifty years on both the dangers of a single concussion and also the graver dangers of repeated concussions, a fact revealed by Thorndike's emphasis on the ***pathology*** of the punch-drunk boxer. As this declaration has made clear, Thorndike, and his contemporaries, could draw upon an extensive scientific literature that treated that pathology as an extreme specific example of a more general case of injuries to the brain of athletes. In effect, the accusers were guilty of the thing for which they accused Thorndike. By failing to place Thorndike's article in historical context, they in turn cherry-picked him out of his historical context to accord with designs in their own. It is unclear whether those designs were duplicitous, but the opinion certainly relied on very bad history.

242. Thorndike's views were profoundly mainstream across science and clinical research. Safety helmet design provides a clear case in point. In a 1958 theoretical essay seeking to develop criteria for measuring the performance of helmets, A. G. Gross stated emphatically that the consensus position was that "the primary function of the helmet is to provide protection from brain concussion in case of accidental impact to the head."[284] Describing the physical and mechanical principles at stake in measuring the safety standards of helmets Gross stated:

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[283] McCrory, *supra* n.90 at 380.

[284] A.G. Gross, *A New Theory on the Dynamics of Brain Concusion and Brain Injury*, 15 J. NEUROSURG. 5, 548-61, 548 (1958).

> Research in the field of brain concussion has, in the past, accordingly been conducted mainly by those trained in the field of medicine. It must be recognized, however, that research dealing with impact to the head involves engineering dynamics, a highly specialized field that is normally foreign to medical research.[285]

243. Gross observed that supposed mechanism of brain concussion was focal violence, which collapsed cavities in the brain and produce the petechial hemorrhagic processes observed by pathologists.[286] For Gross the point about punch drunk boxers was clear enough:

> The well-known "punch drunk" effect suffered frequently by boxers who have taken too many hard blows to the head indicates that the damage to the brain from these successive blows is cumulative in nature. The sectioned brain of a punch-drunk fighter shows small areas of damage dispersed throughout the brain. Such progressive damage may well be caused by minute cavities produced by sub-concussive blows.[287]

244. Gross's article on helmet design makes clear that research on concussions was sufficiently developed in the 1950s to compel authorities across many disciplines to seek good helmet design as an effort to prevent the injuries. That Gross's work was pitched at an intersection of biophysical, biomechanical, and pathological principles also reveals that the broader lessons observable in the historical record up to that point could be absorbed by scientific and clinical investigators in a variety of fields. Whether helmets could actually prevent concussions was another matter (largely the answer is no), but the understanding of the short-term and long-term pathological and clinical consequences of repeated sub-concussive and concussive blows was by 1958 decades old and the desire to prevent such injuries manifestly wide.[288]

245. Arguments showing how mainstream Gross's and Thorndike's research findings were appeared throughout the 1960s and 1970s. In 1962 British neurologist

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[285] *Id.* at 548.

[286] *Id.* at 552-59.

[287] *Id.* at 559.

[288] *See, e.g.*, *Head Injuries*, LANCET 801-02 (1941).

Charles Symonds reviewed the state of the literature from his biographical point of view which spanned the period when those authors wrote. Symonds recalled: "In 1940 I suggested that 'the instantaneous loss of cerebral function so frequently observed after head injury is due to sudden, direct damage, by stretching or compression, to the nerve cells or fibres of the brain.'"[289] How this might happen, of course, was another matter.

246. Symonds stated that Holbourne, in 1943, had suggested that shear strains and rotational forces probably caused both stretching and compression, and that Pundenz and Shelden subsequently showed this fact in monkeys in 1946. Studies by Denny-Brown and Ritchie Russell published in 1941 also gave solid supporting evidence that "the paralytic phenomena of concussion were due to a direct physical injury to the neurones."[290]

247. Throughout the twentieth-century, many authors had sought histological evidence of damage in experimental concussion. According to Symonds' writing in 1962, "using the Marchi method" in 1913 Jakob had "found as the most constant abnormality diffuse degeneration of nerve fibres."[291] Subsequent authors had:

> found that the amount of damage to the nerve-cells was in proportion both to the number and strength of the blows inflicted. For example, two blows which were subconcussive, as judged by preservation of corneal reflexes, produced as much chromatolysis as a single light concussive blow. These workers justly remark that previous negative reports of histological investigation after experimental concussion do not mean that there were no changes, but simply that the nature of the examination was such that no changes were revealed.

248. Turning to the sequelae of concussion injury, Symonds echoed the findings of many authors throughout the twentieth century:

> The post-traumatic amnesia may be regarded as a symptom of generalized cerebral injury. The other symptoms that are constant enough in a case of moderate or severe concussion are the sequelae – meaning that they continue after the conclusion of the post-traumatic amnesia. These should

[289] Symonds, *supra* n.206.

[290] *Id.* at 2.

[291] *Id.*

> be considered as a consequence of the initial generalized injury. They are, as we all know, mainly of the subjective kind and they have often been described and analysed. Prominent among them are anxiety irritability, difficulty in sustaining mental concentration, impaired memory and excessive liability to fatigue. These are symptoms of a psychological kind. This does not mean that they have not physical basis, but it does mean that they are related to all those qualities which constitute the patient's personality, and to his whole attitude of mind. Prominent in this will be his attitude towards his illness, its causes and prospects, and the question of compensation.[292]

249. Symonds, moreover, directed attention to the false efforts to imply that structure and function could be separated when it came to neurological and psychiatric symptoms:

> To inquire in such a case whether the symptoms under consideration are functional or organic, psychogenic or physiogenic, is fruitless, for they must always be both. The practical question is not whether there is physical damage, but whether it is still reversible. It is, I think, questionable whether the effects of concussion, however slight, are ever completely reversible. So far as symptoms are concerned, the patient makes a rapid and complete recovery from a single slight concussion, but after repeated episodes there is a gradual appearance of permanent sequelae. The punch-drunk syndrome provides a good example. In these patients who gradually develop the mental symptoms and physical signs of diffuse organic cerebral damage, concussion may never have been severe, but it has been repeated; and in addition there have been frequent subconcussive blows.[293]

250. Symonds further pointed out:

> Other evidence is to be found of cumulative concussive injury in patients who have survived more than one episode of moderately severe degree. I have observed several such cases in which there has been apparently completely recovery after the first episode, but after the second there has been permanent intellectual impairment and personality disorder out of all proportion to the duration of the post-traumatic amnesia on the second occasion.[294]

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[292] *Id.* at 3.

[293] *Id.* at 3-4.

[294] *Id.* at 4.

251. For Symonds it was also self-evident that the brain damage involved neuronal loss:

> We may therefore surmise that in the patient who has been concussed and recovered, some fraction of his reserve neurons has been lost; and, if the process is repeated, it will only be a question of the number and severity of the injuries before the reserves are exhausted and permanent symptoms appear. The earliest symptoms to be expected from such a diffuse cerebral loss would be of the kind most difficult to measure – subjective difficulty over intellectual problems, and slight personality changes.[295]

252. Symonds observed, too, that all of this was likely due to diffuse damage to white matter (the axons of nerve cells in the brain), which as biomechanical theory showed, would be uneven in its distribution.

253. Turning to treatment, Symonds observed that in the past people recommended rest. "The principle is sound; but in practice we observe that many patients after concussion, even of considerable severity, if they are young rapidly become symptom-free: and that there are others whose symptoms persist indefinitely but in disabling degree. Our object should be not only to give the injured brain a long enough rest but to get it working again as soon as possible."[296] Summing up Symonds concluded:

> In the most severe degree of concussion there is widespread irreparable damage. In the slightest degree there may be rapid and complete recovery of cerebral function; but this does not necessarily exclude the possibility that a small number of neurons may have perished – a number so small as to be negligible at the time, but leaving the brain more susceptible as a whole to the effects of further damage of the same kind.[297]

254. Symonds' account of the head injury research that had occurred in his lifetime is validated by the historical record. From the time that Symonds had published his first paper on head injury, 1928, to the point nearing the end of his career in 1962 when he wrote *Concussion and Its Sequelae*, the arc of the collected findings from

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[295] *Id.*

[296] *Id.*

[297] *Id.* at 5.

investigators producing pathological, physiological, psychological, and clinical scholarship all directed towards these judgments.

255. This legacy of research would be augmented by further studies in the 1970s. Although minor concussions in other contexts has received extensive attention, up to the 1970s no author had specifically made "dings" or "bell-ringers" (minor closed head injuries occurring ***in sports***) a separate focus apart from head injuries suffered in other contexts.[298] Yarnell and Lynch, however, offered something of a corrective by pointing out that "ding" injuries in college football could have disturbing amnesic cognitive effects:

> A professional football player turned author, Dave Meggyesy, has defined being "dinged" as "getting hit in the head so hard that your memory is affected, although you can still walk around and sometimes even continue playing. You don't feel pain, and the only way other players or the coaches know you've been dinged is when they realize you can't remember the plays.[299]

256. The report included a case worth quoting at length:

> An 18 year old left-handed flanker back was gang tackled after catching a pass on a "91 curl pattern." He got up and started to run to the sidelines, pointing to his head, but was told to stay in the game. On the subsequent play he was confused regarding the signals and had to ask for specific instructions. He then left the game. On immediate testing he had intact orientation and recent memory for the play and impact. Seven minutes

[298] For example, the topic had come up in a way in a 1928 discussion of traumatic encephalitis as a conception of the postconcussion phenomena. Neurosurgeon Ernest Sachs has asked in a question and answer session if it would be better to call such concussions "contusions and lacerations." In his response, Ostato stated: "A great many people have concussion of the brain and apparently recover completely. This is obvious if one considers the number of young men who are "knocked out" during football and other games, and in accidents that occur in industry. The majority recovery quite completely, so far as one can determine" Osnato added, however, "In most patients who persistently complain and have some objective symptoms, it seems fair to say that there is an organic structural basis…In these cases there is no question that there has been some pathologic change similar to what we have shown in this case, with later the laying down of diffuse areas of gliosis and perhaps even cellular changes." Osnato, *supra* n.67.

[299] P.R. Yarnell & S. Lynch, *The "Ding": Amnestic States in Football Trauma*, NEUROL. 23: 196-197, 196 (1973).

> later he began to have difficulty remembering the plays, and by 11 minutes after injury he was confused about any of the game events, including his finger dislocation of 90 minutes earlier. He appeared bewildered and continually repeated, "I can't remember, I can't remember; this has never happened to me before." However he was able to do simple arithmetic, reverse spelling, and follow commands. He showed no focal neurologic deficit on screening examination that included ocular, speech and facial muscle function and gait, strength and coordination testing. The player remained confused for at least 30 minutes postinjury. He was not able to recall his locker combination to obtain his clothes. Beginning at this time, however, his memory started to return, and he could recall being admitted to the Student Health Service. By two and one-half hours postinjury, he was bright and lucid. His memory then included the play and the impact on which he had gotten "his bell rung" and had wanted to leave the game and the following play with confusion regarding the signals; but he had only a vague, patchy recollection of the one-half hour of bewilderment on the sidelines.[300]

257. Yarnell and Lynch's short report was hardly a definitive statement on the topic, but the condition that they saw in their case study was nevertheless one pointing to significant brain disturbance. This report foreshadowed the emerging guidelines that warned that "dings" and "bell ringers" were concussions.[301]

4. Medical Guidelines for the Management of Concussion in Sports, 1970s to 1990s

258. Prior to the 1970s, reputable medical authorities had long called for caution and warned of the potential risks of concussions and minor closed head injuries obtained in sporting incidents. They had urged others in their field to educate and warn the lay public of these risks. In the medical and scientific literature consulted for this declaration, the first publication of particular medical guidelines for concussion in sports and management advice appeared in 1973, in an article on pre-season examinations in

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[300] *Id.* at 196.

[301] T.A. Gennarelli, *Mechanisms and pathophysiology of cerebral concussion*, 1 J. HEAD TRAUMA REHAB. 2, 23-29, 24 (1986); *see also* Sports Medicine Committee of the Colorado Medical Society, *Guidelines for the Management of Concussion in Sports*, COLO. MED. SOC'Y (June 1991); SubCommittee, Report of the Quality Standards, *Practice Parameter: The Management of Concussion in Sports (Summary Statement)*, 48 NEUROL. 3, 581-85 (1997).

football. In this article, the authors, Clayton, Hamlin and Lewis, offered explicit recommendations that were to cover all groups of players, from pee wees to professionals. To assess players and the risks they faced from head injury, Clayton *et al.* provided the following grading scale for concussion:

> Cerebral Concussion Acute 1st Degree (mild) Symptoms: No loss of consciousness; variable symptoms of temporary memory impairment, mental confusion, unsteadiness, tinnitus and/or dizziness. Signs: Perhaps none; or appearance of brief period of mental confusion. Acute 2nd Degree (moderate) Symptoms: Transitory unconsciousness (up to 5 minutes) with retrograde amnesia; variable symptoms of mental confusion, tinnitus and headaches. Signs: Appearance of transitory unconsciousness state and subsequent mental confusion. Acute 3rd degree (severe) Symptoms: Unconsciousness for prolonged interval (more than 5 minutes) with prolonged period of retrograde amnesia; variable symptoms, but of greater duration than those experienced in mild or moderate types; possible convulsions. *Signs:* Appearance of prolonged unconscious state and subsequent mental confusion. [302]

259. In a concise and practical management-oriented statement that echoed earlier cautions by Thorndike and others, Clayton and colleagues were very clear that players should ***not be allowed to return to play*** if they had three severe concussions or other neurological contraindications. They stated:

> It is our conclusion that a player who has suffered three or more 2nd or 3rd degree cerebral concussions has reached a point of "diminishing returns." That is, he has reached the point where each subsequent concussion increases the possibility of permanent damage to the individual. For this reason, after his third 2nd or 3rd degree concussion he should be excluded from competition.[303]

260. Later neuropsychological studies provided further evidence of the substance of those guidelines. In a neuropsychological study of twenty young adults published in *The Lancet*, Gronwall and Wrightson found significant cumulative effects from concussion:

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[302] M.L. Clayton, *et al*., *Football: The pre-season examination*, 1 J. SPORTS MED. 4, 19-24, 20 (1973).

[303] *Id*.

> Twenty young adults were studied after a second concussion. The rate at which they were able to process information was reduced more than in controls who had been concussed only once, and they took longer to recover than the controls. The effects of concussion seem to be cumulative, and this has important implications for sports where concussion injury is common.[304]

261. Gronwall and Wrightson were well-grounded in contemporary medical and scientific trends in concussion research. They acknowledged, for example, that some authorities persisted in ascribing the post-traumatic syndrome to psychosomatic disorder. Their findings, however, led them to offer several hypotheses, ranging from recoverable injury to the brain to permanent damage. In either scenario, the result remained: "The capacity to process information rapidly is reduced immediately after concussion. We have shown that the reduction is significantly greater and lasts significantly longer when the patient has been concussed before."[305] Ultimately, they were sufficiently worried by their findings to abruptly conclude:

> Whatever the mechanism for this fall-off in intellectual performance, ***doctors do have a duty to convince the controlling bodies and participants in sports*** where concussion is frequent that the effects are cumulative and that the acceptance of concussion injury, though gallant, may be very dangerous.[306]

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[304] Gronwall, *supra* n.211. These authors published numerous articles showing similar effects. *See* D. Gronwall & P. Wrightson, *Delayed Recovery of Intellectual Function After Minor Head Injury*, 304 LANCET 7881, 605-09 (1974); D. Gronwall & P. Wrightson, *Duration of Post-Traumatic Amnesia After Mild Head Injury*, 2 J. CLINICAL NEUROPSYCHOL. 1, 51-60 (1980); D. Gronwall & P. Wrightson, *Memory and Information Processing Capacity After Closed Head Injury*, 44 J. NEUROL., NEUROSURGERY, & PSYCHIATRY 10, 889-95 (1981).

[305] Gronwall, *supra* n.211.

[306] *Id.* at 997 (emphasis added). Gronwall and Wrightson appear to have been quoting A. H. Roberts, *Brain Damage in Boxers*, 46 (1969). This quote also appeared in an article published a year later on concussions and horse jockeys. The authors of this report were less adamant than other authors that it was obvious that concussive blows might lead to traumatic encephalopathy in sports other than boxing. The authors provided five cases in jockeys that they believed warranted suspicion. Their article concluded: "Our recent experience indicates that National Hunt Jockeys are exposed to frequent and often severe unrecorded concussive head injury, and that this can result in

262. Such extensive discussions in the medical literature clarify why by the 1970s medical textbooks of neurology were describing the dangers of repeated concussions. For example, *Grinker's Neurology* published in 1976, advised medical students:

> The clinical picture of cerebral concussion is, therefore, not a simple one. The usual patient loses consciousness briefly, soon recovers and thereafter is without symptoms. It seems likely, however, that although the patient appears to make a complete recovery from one such episode, he cannot hope to do so from repeated ones. The repeated traumatization of nerve cells, such as Windle and his associates have shown, is likely to produce a lasting deficit sooner or later. It is probable that this fact accounts for the change seen in prizefighters who become "punchdrunk." Definite changes take place in the brains of some fighters.

263. Perhaps unsurprisingly, such strident language in *The Lancet* did result in a thoughtful, editorial response (published in the same year) that observed that at the time there was no clear evidence that sports other than boxing caused an irreversible "traumatic encephalopathy." The anonymous author went on to claim that there was evidence that boxing was much more dangerous than other sports. The author added, however:

> The possibility of lasting damage [in other sports] seems to have been investigated only in an unpublished inquiry by J. A. N. Corsellis. Two years ago 165 British neurologists co-operated in replying to a questionary in which Corsellis asked whether they had ever encountered a condition resembling the punch-drunk state either in boxers or in sportsmen of any category. Professional soccer was mentioned five times with comments on two: one man was a "[soccer] centre half much given to heading and able to do so even if the ball were blasted at him from about six yards".

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brain damage and temporal lobe-epilepsy and the other features recognized as post-traumatic encephalopathy. There appears to be inadequate supervision and restriction of head-injured jockeys, and the possibilities of permanent disability and suspension early in their career are not matched by adequate provisions for compensation. To quote Roberts: "doctors have a duty to convince the controlling bodies and participants in sports where concussion is frequent that the effects are cumulative and that the acceptance of concussion injury, though gallant, may be very dangerous". *Id*. 983. *See* J.B. Foster *et al*., *Brain Damage in National Hunt Jockeys*, 307 LANCET 7967, 981-83 (1976).

> Another had played until he was forty, developed fits, and was thought to belong to the punch-drunk group. Two amateur rugby football players were suspected: one was a young school teacher who had developed a tremor and an impaired memory. Some sharp general comments were made about the risks of chronic brain damage in rugby which ranged from "of course not" from a Welsh neurologist to "I am very suspicious of some top class rugby players but have seen none professionally." Another neurologist remarked that "Rugger seems to have become a depraved and brutal sport even in schools; the players punch each other freely and of course with bare fists so they may well damage each others brains as much as boxers do. I have, however, seen no cases." Professional wrestling was twice incriminated but without details. One parachute jumper was mentioned who had been credited with 550 jumps. The sport most often mentioned, other than boxing, was horse-riding. About a dozen jockeys were believed to have developed a traumatic encephalopathy; all had been steeplechasers. A few comments may be quoted: "I have seen some incredibly damaged professional steeplechasers with a history of uncountable concussions and not one very serious head injury. One is a dependent wreck with no recent memory". A medical report on another stated "this young man is beginning to show an early punch-drunk syndrome and this can almost certainly be attributed to his multiple head injuries."[307]

264. By 1980 these trends appear to have congealed into a consistent view of concussions in contact sports. That year, physicians at the Institute of Neurological Sciences in Glasgow engaged in a retrospective study of serious head injuries, advised avoidance of cumulative damage from concussions in sports:

> Injuries producing even a few minutes' post-traumatic amnesia may cause some microscopic structural brain damage and impaired psychological function for two to three weeks. The effects of repeated minor injury are cumulative; the occurrence of permanent damage (traumatic encephalopathy) in boxing and in jockeys has led to the introduction of statutory medical cover in these sports. More recently, it has been suggested that a similar encephalopathy may occur in other sports, including Association football, rugby football, and wrestling. Boxers must wait four weeks after loss of consciousness before fighting again to allow recovery from the injury. Similar rules should probably be applied to all sports, although a short period of post-traumatic amnesia (as distinct from

[307] I.D. Adams & J. Potter, *Brain Damage in Sport*, 307 LANCET 7959, 401-02, 402 (1976).

> loss of consciousness) should not necessarily prevent a player from returning to a non-combative sport. If the postconcussional syndrome is present – namely persistent headaches, postural dizziness, irritability, and a failure of concentration – the player should not return to the sport until all symptoms have resolved. It is understandable that both trainers and players may be reluctant to follow such policies, but if further damage should occur before recovery is complete then cumulative effects may be more serious. Thorndike suggested that any player who has sustained three or more injuries with loss of consciousness of post-traumatic amnesia should be banned from further contact sports. Murphey and Simmons advised that if head injury was severe enough to produce coma, or if a player has had a craniotomy, then further contact sports should be discouraged.[308]

265. The Colorado Medical Society Guidelines on Sports Concussion brought all of these conclusions together in a forceful and short document published in 1990. In the revised edition published in 1991, the authors explained that the sports guidelines "were developed by the CMS Committee on School Health and Sports Medicine in 1990 out of a growing concern that many team physicians in attendance at contact sports events felt uncomfortable when asked to determine whether an athlete could return to the contest following a concussion."[309] The guidelines were endorsed by several leading medical organizations: the American Academy of Pediatrics; the American Academy of Sports Physicians; the American College of Surgeons – Committee on Trauma; the Colorado Society of Clinical Neurologists; and the University of Colorado – Department of Neurology, University of Colorado – Division of Neurosurgery.

266. As the authors described circumstances in their 1991 edition:

> Concussion is the most common consequence of head injury in contact sports. It is estimated that more than 250,000 such injuries occur every year in football alone. Head and neck injuries are the most frequent catastrophic sports problem. While these injuries can occur in any athletic activity, they are most common in football, boxing, horseback riding, swimming, diving, cycling, ice hockey, gymnastics, martial arts, sky diving, rugby, and motorized vehicle racing. Current information

[308] K.W. Lindsay *et al*., *Serious Head Injury in Sport*, 281 BR. MED. J. 6243, 789-91 (1980).

[309] Sports Medicine Committee of the Colorado Medical Society, *Guidelines for the Management of Concussion in Sports*, COLORADO MEDICAL SOCIETY 1 (June 1991).

> regarding head injuries must bring new respect for what is frequently dismissed as an athlete having suffered a "ding" injury. Several observations call into question the very concept of "minor" head injury: 1) Brain injury in sports can result from any rotational (angular) or translational (linear) force applied to the head. Frequently, both forces act in combination. Rotational forces more commonly cause loss of consciousness associated with deep shearing injuries of nerve fibers (diffuse axonal injury or DAI). Translational forces are less likely to cause unconsciousness but more commonly lead to skull fractures, intracranial hematomas, and cerebral contusions, 2) Central nervous system axons are more vulnerable to the shearing forces of mild head injury than the surrounding glia and vascular tissues, 3) It has long been recognized, although underappreciated, that confusion and amnesia can result from concussion even without loss of consciousness, 4) Current neuroimaging techniques of magnetic resonance imaging (MRI) and computerized tomography (CT) frequently detect intracranial lesions following mild head trauma, even without loss of consciousness. MRI has proven to be more sensitive than CT in detecting intracranial pathology, especially the nonhemorrhagic lesions of DAI prevalent in closed head injury, 5) Information processing ability can be reduced following concussion. Twenty-five percent of athletes with three minor head injuries, 3% of those with four minor head injuries, and 40% of those with five minor head injuries showed persistent abnormalities on neuropsychological testing at 6 months after injury, 6) Repeated concussions appear to impart cumulative damage, resulting in increasing severity and duration with each incident, 7) In football, the chance of having a second concussion is four times greater than the chance of sustaining a first concussion, 8) Amnesia following mild head injury frequently takes several minutes to appear, suggesting that some neuropathological process evolves slowly over time after the mechanical blow, 9) The Second Impact Syndrome, although rare, can result in catastrophic brain swelling which may occur following a second minor head injury in individuals who are still symptomatic from a prior concussion.[310]

267. Thus, the Colorado Medical Society's guidelines for the management of concussion in sports were an acknowledgement of the advanced state of concussion research after a century of medical and scientific research. By the time the Colorado Medical Society issued its guidelines for concussion in sports, the accumulated evidence from scientific and clinical studies suggested clearly:

[310] *Id*. at 2.

- That the definition of concussion was clearly understood by medical professionals;
- That the causes and effects of concussions were known;
- That a loss of consciousness was not necessary for a concussion to have occurred;
- That it was widely understood that most concussive injuries appeared to recover but sometimes did not;
- That it was widely understood that repeated concussions heightened the risk of irreversible brain injury;
- That the nature of that irreversible brain injury was cellular degradation, axonal stretching and shearing, and in extreme cases, secondary brain degeneration;
- That chronic traumatic encephalopathy was a known risk of recurrent concussion;
- That there were clear reasons for suspicion that sports other than boxing placed individuals at risk from chronic traumatic encephalopathy; and
- That contact sports were common settings in which closed head injury occurred and thus reasonable targets for preventative measures.

VI. CONCLUSION

268. This declaration has closely examined medical and scientific literature published between 1871 and 2011 in prominent scholarly journals in medicine and the sciences as pertaining to observations, investigations, and recommendations about the risks of concussion and minor closed head injury and repetitive exposure to these traumas.

269. The literature analyzed has included published peer-reviewed articles, reviews, lecture transcripts, editorials, and letters to the editor, as well as a smaller number of medical and scientific monographs, conference proceedings, technical papers, and newspaper publications.

270. The major medical and scientific journals surveyed in the research for this declaration were selected not only for their prestige (and thus representative of research that was peer-approved at the highest levels) or particular relevance to the questions at hand, but also for their accessibility to medical and scientific professionals – including those advising and overseeing sports teams and the sporting industries – over the last century and a half. Today we are used to being able to search the internet and large

indexing databases (PubMed, for example), to find medical and scientific literature relevant to our studies at hand. This, clearly, has not been possible through most of the period in question in this study. The journal contents frequently cited in this declaration were, nonetheless, still fairly readily accessible to medical and scientific professionals. They were available in medical society, university, and national library systems, and were easily found through printed card indexes and index catalogues (for instance, the index-catalogue of the library of the Surgeon-General's Office) and later computer catalogues of holdings. It was (and still is) also fairly standard practice to join the medical or scientific societies that addressed one's specialty or special interest topics, and thus acquire a subscription of the society journal delivered by mail throughout the year (most society journals now carry the option of access to current and archived journal content online). Acquaintance with these ways to access both historic and the latest medical and scientific literature was as widespread among physicians, scientists and clinical researchers through this period as understanding how to use major internet search databases is now.

271. As measured by these published contributions in the medical and scientific literature, this declaration has specifically sought to answer several overlapping questions:

1.) For how long have healthcare professionals been aware of the dangers of sub-concussive and concussive blows to the head?
2.) For how long have they observed that those blows could result in persistent, permanent, or deteriorating long-term symptoms?
3.) For how long have they been aware that repeated blows were dangerous?
4.) Finally, for how long have healthcare professionals expressed concern about leisure, occupational, and professional sporting activities that place individuals at risk from such injuries?

272. The overarching conclusions derived from this research are clearly outlined in the summary of opinions at the beginning of this declaration. In short, however, the evidence from the extensive medical and scientific literature consulted for the creation of this declaration makes definitively clear that the answer to each of the above questions is, ***for a long time***.

273. This is not to say that there has been a complete absence of competing theories, voices of dissent, or challenges to the prevailing medical consensus that had been established over decades of medical and scientific work. As reflected upon at the outset of this declaration – that is normal science. That science (and medicine) is by its nature embedded in a broader context as well, be it the specialization of medicine and emergence of new disciplines, national politics and historical change, or large sporting institutions (such as the NFL) and financial interest.

274. That these broader contexts at times introduced inconsistencies or alternate narratives into the medical and scientific record on the effects and long-term sequelae of concussion and minor closed head injury has been carefully noted in this declaration. And yet, even while these digressions occurred, the medical and scientific literature as a whole continued to demonstrate a clear progression of understanding the symptoms and long-term sequelae of concussion and minor closed head injuries and to evince concern about the dangers this posed to individuals at heightened risk of receiving this trauma.

275. Since this conclusion is an accurate reflection of the history of concussive science and medicine, then a mystery that remains is why there has not been greater public awareness about the dangers of brain concussion. Why, in short, hasn't everyone known? The answers to this question are complex and in some sense fall beyond the scope of this research. It is, however, clear that these facts have not found their way into public consciousness, even though they have, or should have, found their way into the consciousness of any inquiring medical professional. One renowned American neurologist, with more than a trace of condescension, put matters succinctly in 1951:

> The general public is as ignorant about the functions of the brain as was the cave man. Children shown a picture of a man being hit on the head with a sledge hammer laugh. They are encouraged to addle their own brains with repeated concussion in such sports as football and boxing. Boxing commissioners wonder out loud why pugilists die in the ring. Our modern literature is full of references to people being revived by a procedure which is almost pure magic – pouring water on the face. Newspaper articles weep over boys and girls or men and women who have

> lost a leg or an arm, perhaps both legs and both arms, but they say nothing of those who are really maimed, those with brain injuries.[311]

276. More than fifty years later, in 2007 the Centers for Disease Control began mounting a public health campaign, *Head's Up*, intended to educate coaches, athletic trainers, and healthcare professionals about the dangers of brain injury with presumably the intent of trickling that health information down to athletes, parents, and other interested parties.[312]

277. Against such a medical backdrop, however, is a wholly different cultural context; big hits, head punches, knock-outs, and other forms of violent head trauma are the stock-in-trade of movies, TV and sports. While on some level common sense must make clear to audiences that such theatrical conceits are mere fantasies, it is probably also true that especially in the context of sports, audiences engage in a suspension of disbelief because of the incredible prowess and talent of the athletes. It is tempting to pretend that while mere mortals cannot receive a blow to the head with impunity, perhaps athletes "can" receive such punishment and be unaffected now and later. Indeed, it is hard to think of an area of culture and society more susceptible to such a suspension of disbelief than the sports industry, where, fueled by incredible athletic talent, hero worship forms a decisive part of the lucrative business model.

278. It is a rare breed of fan who suddenly apprehends his or her own complicity in the making of this reality, as one fan, quoted at the end of a medical paper, did: "I stood talking to Muhammad Ali, embarrassed by his inarticulateness, and deeply ashamed, as it was not his own superb body that had done this terrible thing to his superb mind. I had done it too, as part of the crowd urging him on, applauding the blood. I have not watched a boxing match since then."[313]

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[311] F. Gibbs, *The Most Important Thing*, 41 AM. J. PUB. HEALTH 12, 1503-08, 1506 (1951).

[312] *Notice to Readers: Heads Up! Tool for Diagnosing and Managing Brain Injury*, 298 JAMA 8, 858 (2007).

[313] Corsellis, *supra* n.216 at 109.

279. Into this complex discussion arises a final question: what, culturally, is our medicine for? The narrow answer is, of course, to make us well. Yet there are many settings in which clinicians in particular find themselves in deeply conflicted roles. In wars, for example, clinicians heal in order to return soldiers to combat. In professional sports, clinicians heal to return players to the game. These are compromising positions for a profession that prides itself on its ethical code of conduct. In war clinicians serve States' interests, and in professional sports arenas, clinicians serve multiple parties, not least the fans, the managers, the coaches, the owners, and, of course, individual athletes too – indeed the whole economic edifice that has sprung up around professional sports. Despite these competing interests, these clinicians remain duty-bound to practice medicine in a manner that prioritizes their patients' interests. Unfortunately, there appears to be a troubling disparity between the care that the NFL and NHL clinicians offered their athlete patients, and the care demanded of them by their duty as doctors.

280. Further, medical doctors are also duty-bound to apprise themselves of modern medical knowledge on a continuous basis; this applies with equal force to the doctors working in the NHL. The American Medical Association's ("AMA") Principles of Medical Ethics have compelled this requirement of its members for 60 years, stating in 1957 that "Physicians should strive continually to improve medical knowledge and skill, and should make available to their patients and colleagues the benefits of their professional attainments."[314] When updated in 1987, the AMA reiterated that "[a] physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated." Most recently, in 2001: "A physician shall continue to study, apply, and advance scientific knowledge, maintain a commitment to medical education, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated." Put simply, ignorance is not an excuse.

[314] F.A. Riddick Jr., *The code of medical ethics of the American Medical Association*, 5 OCHSNER J. 2, 6-10 (2003).

281. Regardless, if those clinicians serving the NHL have taken the modern Hippocratic Oath, then their oath entails vowing:

> ***I will respect the hard-won scientific gains of those physicians in whose steps I walk, and gladly share such knowledge as is mine with those who are to follow***.

282. In the opinion of this medical historian, the record of those hard-won gains in medicine could not be clearer – concussions and repeated concussions are dangerous. They can be life-changing and lead to permanent illnesses, and this was known by the medical community for well over half a century. As one prominent physician observed in 1872: "great caution ought to be observed ever afterwards by anyone who has experienced such an injury."[315]

[315] Crighton-Browne, s*upra* n.103.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: 12-2-2016

Stephen T. Casper, Ph.D

EXHIBIT “A”

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Walton, John. 2003. "50 years in neurology, a retrospect." *Practical Neurology* 3: 55.

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Weiss, Sheila Faith. 2010. *The Nazi Symbiosis: Human Genetics and Politics in the Third Reich.* Chicago and London: Univeristy of Chicago.

Weisz, George. 2014. *Chronic disease in the twentieth century : a history.* Baltimore, Maryland: Johns Hopkins University Press.

2006. *Divide and Conquer: A Comparative History of Specialization.* Oxford: Oxford University Press.

White, Benjamin. 1984. *Stanley Cobb: A Builder of the Modern Neurosciences.* Boston : Francis Countway Library of Medicine.

Young, Allan. 1995. *The harmony of illusions : inventing post-traumatic stress disorder.* Princeton, N.J.: Princeton University Press.

Zeisler, Laurel. 2013. *Historical dictionary of ice hockey.* Lanham, MD: Scarecrow Press, Inc.

EXHIBIT “B”

Curriculum Vitae

Dr. Stephen T. Casper

26 September 2016

Clarkson University,
Department of Humanities & Social Sciences
8 Clarkson Avenue, Potsdam, NY, 13699 USA,
Phone: (315) 268-3882
Email: scasper@clarkson.edu
Twitter: @TheNeuroTimes

EDUCATION

2006	PhD, University College London, 2006, History of Medicine
2002	BSc, University of Minnesota, 2002, Neuroscience; Biochemistry

ACADEMIC APPOINTMENTS

2014-present	Associate Professor, Humanities & Social Sciences, Clarkson University
2015	Visiting Scholar, Department of History and Philosophy of Science, Cambridge University
2010-2014	Assistant Professor, Humanities & Social Sciences, Clarkson University
2007-2010	Visiting Assistant Professor, Humanities & Social Sciences, Clarkson University
2007	Scholar-in-Residence, Rockefeller Archive Center, Sleepy Hollow, NY

PUBLICATIONS

Books

Casper, S. (2014). *The Neurologists: A History of a Medical Specialty in Modern Britain, c. 1789-2000*. Manchester: Manchester University Press

Edited Books

Casper S. and Gavrus G. (in press), eds. *The History of the Brain and Mind Sciences: Technique, Technology and Therapy*. Rochester NY: University of Rochester Press.

Casper, S., Jacyna, L. S. (2012), eds. *The Neurological Patient in History*. Rochester NY: University of Rochester Press

Journal Articles

Casper, S (2016). "The Currency of Consciousness: Neurology, Specialization, and the Global Practices of Medicine" *Canadian Bulletin of Medical History*

Casper, S (2016). "Inexplicable Patients: Charles Martell and the Patients of Ward 4 at the Massachusetts General Hospital" *Canadian Medical Association Journal*

Casper, S., Welsh, R. (2015). "British Romantic Generalism in the Age of Specialism, 1870–1990." *Social History of Medicine*, 1-21. DOI: 10.1093/shm/hkv103

Casper, S. (2015). "Of means and ends: Mind and Brain Science in the Twentieth Century." *Science in Context* 28(1), pp. 1-7.

Casper, S. and Schilling, R. (2015). "Of Psychometric Means: Starke R. Hathaway and the Popularization of the Minnesota Multiphasic Personality Inventory." *Science in Context 28*(1), pp. 77-98.

Casper, S (2014). "An Integrative Legacy: History and Neuroscience." *Isis*, Focus *105*(1), pp. 123-132.

Casper, S. (2011). "One Hundred Members of the Association of British Neurologists, A Collective Biography, 1933-1960." *Journal of the History of Neuroscience, 20*(4), pp. 338-356.

Casper, S. (2010). "'In Consequence of Enemy Action': British Medical Students in North American Medical Schools, 1939-1945." *British Medical Journal,* 341(c7041), pp. 1336-1338.

Casper, S. (2010). "Trust, Protocol, Gender, and Power in Interwar British Biomedical Research: Kathleen Chevassut and the 'Germ' of Multiple Sclerosis." *Journal of the History of Medicine and Allied Sciences*, vol. 66(2), pp. 180-215.

Casper, S. (2008). "Conjunctures in Anglo-American Neurology: Lewis H. Weed and Johns Hopkins Neurology, 1917-1942." *Bulletin of the History of Medicine,* vol. 82, pp. 646-671.

Casper, S. (2008). “The Origins of the Anglo-American Research Alliance and the Incidence of Civilian Neuroses in Second World War Britain.” *Medical History,* vol. 52, pp. 327-347.

Edited Special Issue Journals

Casper, S. ed. (2015), “Of Means and Ends in the Mind and Brain Sciences” *Science in Context 28*(1), volume pp.1-170.

Essays and Commentaries

Casper, S. (2014). “Chickens and Eggs. A Commentary on Chris Renwick’s: ‘Completing the Circle of the Social Sciences?: William Beveridge and Social Biology at London School of Economics in the 1930s.” *Philosophy of the Social Sciences* 44(4), pp. 506-514.

Casper, S., Espinosa, M. (2012). “Introducing the new Media Reviews section.” *Medical History* 56(4), pp. 615.

Casper, S. (2007). “‘Then why not an Association of British Neurologists?’" British Neurologists and the Founding of an Elite Medical Society.” *Advances in Clinical Neuroscience and Rehabilitation*, pp. 2-3.

Casper, S. (2006). “The Association of British Neurologists, 1930-1965.” *Wellcome History*. vol. 31, pp. 9-10.

Casper, S. (2005). “British Neurology, 1920-1960: A Neurologist’s Labours.” *World Neurology*. vol. 20, p. 12.

Book Chapters

Casper, S., Gavrus, D. (in press). *Introduction.* In Stephen T Casper and Delia Gavrus (eds), *The History of the Brain and Mind Sciences: Technique, Technology, and Therapy*. Rochester, NY: University of Rochester Press.

Casper, S (in press). “Dualist Techniques for Materialist Imaginaries: Matter and Mind in the 1951 Festival of Britain.” In Stephen T Casper and Delia Gavrus (eds), *The History of the Brain and Mind Sciences: Technique, Technology, and Therapy*. Rochester, NY: University of Rochester Press.

Casper, S., (2016). “*The Political* without Guarantees: Neuroscientific Cultural Imaginaries and Neuroscientific Futures.” In Lorenzo Servitje and Kari Nixon (eds), *Endemic: Essays in Contagion Theory*, London: Palgrave MacMillan.

Casper, S., Jacyna, L. S. (2012). *Introduction* (pp. 1-20). In L. Stephen Jacyna and Stephen T. Casper (eds.), *The Neurological Patient in History,* Rochester NY: Rochester University Press.

Casper, S. (2012). "The Patient's Pitch: The Neurologist, the Tuning Fork, and Textbook Knowledge." In L. Stephen Jacyna and Stephen T. Casper (eds.), *The Neurological Patient in History,* Rochester NY: Rochester University Press, pp. 21-43.

Book Reviews and Essay Reviews

Casper, S. (2016). "Worlds of ScienceCraft: New Horizons in Sociology, Philosophy, and Science Studies." *Contemporary Sociology: A Journal of Reviews*. 45(2), pp. 228-229.

Casper, S. (2015). Reviews: "American Lobotomy: A Rhetorical History." *Literature & History. 24*(2), pp. 119-120.

Casper, S. (2015). "Nicolas Langlitz, Neuropsychedelia: The Revival of Hallucinogen Research Since the Decade of the Brain." *Social Studies of Science.* DOI: 10.1177/0306312715598398

Casper, S, (2015), "Ivan Pavlov Revealed" *Science. 347(6229)*, p.1425.

Casper, S. (2015), "Nikolas Rose and Joelle Abi-Rached, Neuro: The New Brain Sciences and the Management of the Mind," *Journal of the History of Behavioral Sciences.* 51(1), pp. 95-98.

Casper S. (2014) "Gabriel Finkelstein, Emil du Bois-Reymond: Neuroscience, Self, and Society in Nineteenth-Century Germany." *Metascience* 24(1), 85*.*

Casper, S. (2013). "James Kennaway, Bad Vibrations: The History of the Idea of Music as a Cause of Disease." *Social History of Medicine* 26(3), 590-591.

Casper, S. (2013). "West Riding Lunatic Asylum and Brain Science." Dissertation Reviews. http://dissertationreviews.org/archives/4528.

Casper, S. (2013). "Brown-Séquard: An Improbable Genius Who Transformed Medicine." *Journal of the History of Medicine and Allied Sciences.* 68(3): pp. 488-490.

Casper, S. (2012). "International Relations in Psychiatry: Britain, Germany, and the United States to World War II." *Medical History.* 56(3), pp. 405-406*.*

Casper, S. (2012). "Neurosurgery at Washington University: A Century of Excellence." Journal of the History of Medicine and Allied Sciences 64(4), pp. 666-667.

Casper, S. (2012). “The Origins of Autism Research.” Dissertation Reviews. http://dissertationreviews.org/archives/813

Casper, S. (2011). “The Cure Within: A History of Mind-Body Medicine.*” Medical History.* 55(1), pp. 133-134.

Casper, S. (2011). “Smallpox: The Death of a Disease.” *Journal of the History of Medicine and Allied Sciences.*66(2), pp. 276-277.

Casper, S. (2011). “The Collectors of Lost Souls: Turning Kuru Scientists into Whitemen;The Social Construction of Disease: From Scrapie to Prion.” *Journal of the History of the Neurosciences* 20(1), pp. 160-169.

Casper, S. (2010). “Hysteria: The Biography.” *Social History of Medicine* 23(3), pp. 692.

Casper, S. (2010). “The Evolutionary Epidemiology of Mania and Depression: A Theoretical and Empirical Interpretation of Mood Disorders.” *Medical History.* 54(2), pp. 283-284.

Casper, S. (2010). “A Revisionist History of American Neurology.” *Brain* 133(2), pp. 638-642*.*

Casper, S. (2009). “Brain, Mind, and Medicine: Essays in Eighteenth Century Neuroscience.” *History of Psychiatry.* 20(1), pp. 111-114.

Casper, S. (2009). “On Deep History and the Brain.” *Medical History.* 53(2), pp. 318-319.

Casper, S. (2007). “Harvey Cushing: A Life in Surgery.” *Medical History* 51(3), pp. 424-425*.*

AWARDS AND HONORS

2014	John W. Graham Faculty Research Award, Clarkson University (awarded to one faculty member each year at the time of tenure and promotion).
2012	RESPECT, Accommodative Services, Clarkson University (an award for disability advocacy)
2011	Commendable Leadership Award, PHALANX. (Clarkson’s University highest honor for service to the university as deemed by students)
2010	CUSA Teaching Award, Clarkson University Student Association
2009	Outstanding Young Teacher Award, Clarkson University

2006 Roy Porter Travel Prize, Wellcome Trust Centre for the History of Medicine

GRANTS

2012 Center for Canadian Studies, Clarkson University (Principal). Canadian and American Conference on Mind and Brain Sciences. (January 15, 2012 - May 6, 2012). $2,000.00.

2011 National Endowment for the Humanities (Principal). Islamic Science: Summer Seminar. SUNY, Potsdam. (June 1, 2011 - July 15, 2011). $3,000.00.

2010 New York Council for the Humanities (Co-Principal). Mini Grant for War, Disability & Society. (April 2010 - August 2010). $1,500.00

2010 Center for Canadian Studies, Clarkson University (Principal). Small Research Grant. (May 2010 - June 2010). $750.00

2008 National Endowment for the Humanities (Principal). Art, Labor, and Working Class Culture: Summer Seminar. SUNY, Potsdam. $3,000.00. (June 1, 2008 - July 15, 2008). $3,000.00

2008 Bakken Library (Principal). “Neurology and Electricity.” Travel grant. (May 1, 2008 - May 14, 2008). $500.00

2007 Rockefeller Foundation Archive Center Fellowship. “Transnational Liaisons in Interwar and Postwar Anglo-American Physiology.” (May 1, 2007 - July 30, 2007). $10,000.00

2003-2006 Association of British Neurologists. PhD Studentship. (August 2003 - August 2006). $42,000.00

2003-2006 Wellcome Trust Centre for the History of Medicine. PhD Studentship. (August 2003 - August 2006). $42,000.00

2005 Rockefeller Foundation Archive Center Fellowship. "Neurology in Britain," Travel grant. (June 2005 - July 2005). $1,750.00

INVITED PRESENTATIONS

2016 “Locked-in Patients/Locked-out Doctors: Histories of Persistent States of Being There.” ICREA Conference Personhood and the Locked-In Syndrome, Autonomous University of Barcelona, Spain. (November, 18, 2016)

2015 "Dualist Techniques For Materialist Imaginaries: Matter and Mind in the 1951 Festival of Britain." Centre for the History of Science, Medicine, and Technology, Manchester University, UK. (November 24, 2015)

2015 "Making Neurology Global: The First International Neurological Congress in Berne, Switzerland." World Congress of Neurology, Santiago, Chile. (November 4, 2015)

2014 James V. Warren Memorial Lecture. "Gentleman Generalists: British Neurologists Confront Medical Specialization, 1880-2000." Ohio State University, Columbus, Ohio. (March 18, 2014)

2014 Neurology Grand Rounds. "The Neurologists: A History of a Medical Specialty in Britain, c. 1789-2000." Ohio State University, Columbus, Ohio. (March 18, 2014)

2014 "Comment on Max Stadler's Physiology of the Nervous System." Soul Catchers: A Conference on the Material History of the Brain Sciences. Princeton University, Princeton, NJ. (February 8, 2014)

2013 "Why ask: 'What is history?'" State University of New York-Potsdam, Potsdam, NY. Plenary lecture to honors graduates in history. (April 19, 2013)

2013 "The Neurology of Everything: How the Retrospective Reconstruction of British Neurology led to the 'Decade of the Brain.'" Johns Hopkins University, Baltimore, MD. (March 7, 2013)

2011 "Psychometric Means: Neuropsychiatry and the Ends of Psychometric Testing." UCL Centre for the History of Medicine, London, UK. (December 5, 2011)

2011 "A Naturalizing Instinct: A History of Brain Science and Politics in Postmodernity." Neuro-Reality-Check, Max-Planck Institute for the History of Science, Berlin, Germany. (December 2, 2011)

2011 "The Neurological Patient in History," History of Health Sciences Lecture, August C. Long Health Sciences Library, Columbia University. (October 13, 2011)

2011 "How Physicians became Neurologists: The Case of Britain, 1800-2000." Neurology Grand Rounds, University of Calgary Medical School, Calgary, Canada. (June 16, 2011)

2010 "The Currency of Consciousness: Neurology and the Global Practices of Medicine." McGill University, Department of Social Studies of Medicine, Montreal, Quebec, Canada. (October 2010)

2010 Reynolds Lecture, "Globalizing Medical Histories." University of Alabama, Birmingham, Birmingham, AL. (May 2010)

2010 "The Specialization of British Neurology." University of Alabama, Department of Neurology, Birmingham, AL. (May 2010)

2010 Grand Rounds. "How Physicians Became Neurologists." Columbia University Medical School, Neurological Institute of New York, New York, NY. (February 2010)

2010 "Legitimizing Us; Pathologizing Them: The Recent History of Neuroauthoriatrianism." Neuroscience and Human Nature. Wellcome Trust Centre for the History of Medicine at University College London, London, UK. (February 2010)

2009 "Gossip or Evidence: The Strange Case of Kathleen Chevassut and the Germ of Multiple Sclerosis." Gallatin School History of Science Series, Gallatin School, New York University, New York, NY. (October 2009)

2007 "Opening Up Medicine: Inclusivity and British Physicians in the 20th Century." Western Michigan University, Kalamazoo, MI. (April 2007)

2007 "The Neurologists: A Global History of a Medical Specialty." University of Birmingham, Birmingham, UK. (March 2007)

2006 "People, Places, and Prosopopeia: A Prosopography of Interwar British Neurologists." Imperial College London, London, UK. (October 2006)

2006 "The Clatter of Discordant Voices: Crises in Medicine in 1916." National Army Museum, London, UK. (August 2006)

CONFERENCE PRESENTATIONS

2015 "Time, Deep History, and the Evolutionary Neurology of Nervous Disease." International Society for the History and Philosophy of Social Science and Biology, Montreal, Canada. (July 9, 2015)

2013 "The Spirit of 'Generalism' in British Neurology: Specialization, State Medicine, and the Making of an Integrative Specialty, 1860-1990." American Association for the History of Medicine, Atlanta, Georgia. (May 18, 2013)

2013 "Open Access and the Future of Scholarly Journals: A Conversation with Journal Editors." American Association for the History of Medicine, Atlanta, Georgia. (May 17, 2013)

2012 "An Integrative Legacy: History & Neuroscience." History of Science Society, San Diego, California. (November 18, 2012)

2011 "Into Mind & Brain: Defining Work in the Integrative Sciences, 1800-1950." Body and Mind Workshop, Cheiron/International Society for the History of Neuroscience, Banff, Canada. (June 20, 2011)

2011 "'A career largely concerned with investigation': E.A. Carmichael, the British State, and Clinical Neurology Research." International Society for the History of Neuroscience, Calgary, Canada. (June 19, 2011)

2010 "Whither Neuroscience? What the Recent History of 'Contagious Shooting' (1982-2006) Says About the Value of the History of the Neurosciences." International Society for the History of Neuroscience, International Society for the History of Neuroscience, Paris, France. (June 2010)

2010 "'The last line of the defense': British Medical Students in North American Medical Schools, 1939-1945." American Association for the History of Medicine, Rochester, MN. (April 2010)

2009 "Making Patients Perform." International Society for the History of Neuroscience, Charleston, SC. (June 2009)

2008 "Neurology and the Practices of Medicine." Workshop on Global Histories of Medicine, Wellcome Trust Centre for the History of Medicine, University College London, London, UK (May 2008)

2008 "Finding Cultural Commonalities in Neutral Territory: The Neurologists and the 1931 First International Neurological Congress, Berne." American Association for the History of Medicine, Rochester, NY. (April 2008)

2008 "Medicine and Science from the Perspective of Global Historians." Annual Meeting of the American Association for the History of Medicine; Cleveland, Ohio. (April 2008)

2007 "How to get kicked out of The Founders of Neurology." International Society for the History of Neuroscience, Los Angeles, CA. (June 2007)

2006 "The Specialization of British Neurology, 1920-1940." History of British Neurosciences, Royal Society of Medicine, London, UK. (December 2006)

2006 "The Foundation of the Association of British Neurologists." Association of British Neurologists, Blackpool, UK. (October 2006)

2006 "Ideologies Young and Old: Comparative Thoughts on Walter Russell Brain's Early Pacifism and Later Professional Philosophy." Scientists and Social Commitment, British Society for the History of Science, London, UK. (September 2006)

2006 "Neurology at Johns Hopkins." Annual Meeting of the American Association for the History of Medicine, Halifax, Canada. (April 2006)

2005 "The Production of 'Fairly Reliable Data': The Question of Civilian Neuroses, 1941-42." International Congress of History of Science, Beijing, China. (August 2005)

TEACHING

Courses at Clarkson University

The Clarkson Seminar
Honors Project Course
Introduction to Global History
History of the Human Sciences
Medicine in Modern Europe and North America
Time and the Body in Medicine
European History: 1789-1914
Science, Technology and the Modern World
Honors Advanced Project Course
Neuroscience and Society

2003-2006 Teaching Assistant, University College London

History of the Human Sciences
History of Biology
Framing Disease
Madness and Literature

Direction of Independent Undergraduate Honors Theses

2012-2013 "Science Fiction and 'The Futurists' in American Culture." Undergraduate honors thesis in History.

2011-2012 "The History of the Minnesota Multiphasic Personality Inventory." Undergraduate honors thesis in Psychology, Biomolecular Science.

2009-2010 "Framing 'Schizophrenia.'" Undergraduate honors thesis in Psychology.

2009-2010 "The History of Male Anorexia." Undergraduate honors thesis in History.

PROFESSIONAL SERVICE

2008-present	Research Awards Committee, International Society for the History of Neuroscience
2015	Workshop Organizer, *Phrenology, Craniology and Anthropometry in Global and Historical Perspective*. Clarkson University, Potsdam, NY. (Aug 17, 2015 – Aug 18, 2015)
2015	Reviewer, book manuscript, Southern Illinois University Press
2015	Conference Organizer, Phrenology, Craniology, and Anthropometry in Global and Historical Perspective, Clarkson University (August 14-15, 2015)
2015	Panel Organizer, International Society for the History and Philosophy of Social Science and Biology, Montreal, Canada (July 4, 2015)
2015	Reviewer, book manuscript, Oxford University Press
2014	Reviewer, journal article, *Bulletin of the History of Medicine*, May 1, 2014 - July 15, 2014
2014	Reviewer, journal article, *Bulletin of the History of Medicine*, March 1, 2014 - June 1, 2014
2014	Reviewer, book manuscript, University of Rochester Press, February 1, 2014 - May 1, 2014
2012	Panel Organizer, History of Science Society, San Diego, CA (November 15, 2012 - November 18, 2012)
2012	Reviewer, journal article, *Brain: A Journal of Neurology*
2012	Reviewer, journal article, *Medical History*

2012	Workshop Organizer, *Technique in the Mind and Brain Sciences* Clarkson University, Potsdam, NY. (May 5, 2012 - May 6, 2012)
2011	Session Chair, International Society for the History of Neuroscience, Calgary, Alberta. (June 19, 2011 - June 23, 2011)
2011	Reviewer, grant proposal, Wellcome Trust, London
2010	Reviewer, journal article, *Social History of Medicine*
2009-2010	Reviewer, journal article, *Bulletin of the History of Medicine*
2009-2010	Reviewer, journal article, *Medical History*
2010	Reviewer, book manuscript, Continuum Books
2010	Session Chair, History of Science Society, Montreal
2009-2010	Ad Hoc Reviewer, Oxford University Press
2010	Session Chair, Society for the Social History of Medicine, Durham, NC
2009	Session Chair, History of Science Society, Phoenix, AZ

DEPARTMENTAL AND UNIVERSITY SERVICE

Departmental Service

Committee Chair, Departmental Curriculum Committee (September 1, 2012 - present)

Committee Member, Search Committee for Sociology, Anthropology and Geography (September 1, 2013 – January 1, 2014)

Committee Member, Search Committee for Political Science/Sociology (September 2, 2012 - February 28, 2013)

Committee Member, Humanities and Social Science Annual Conference (September 2008 - May 2011)

Committee Chair, Formation Committee for Curriculum Development Committee (September 2009 - April 2010)

Administrative Service

Founder and Committee Chair, David A Walsh Arts & Sciences Seminar Series (June 2010 – May 2013)

University Service

Faculty Senate, Clarkson University (August 2014 – present)

Committee Member, Health Science Committee (May 1, 2011 - present)

Debate Judge, National Society of Black Engineers (November 8, 2009; April 11, 2010; October 30, 2011; November 11, 2012)

Committee Member, Search Committee for Assistant Dean of Students (August 1, 2012 - September 17, 2012)

Faculty Advisor, Phi Delta Epsilon (September 1, 2011 - September 1, 2012)

Committee Member, Physician Assistant Student Recruitment (June 15, 2011 - September 30, 2011)

Committee Member, Search Committee for Founding Director of Physician Assistants Program (October 2009 - February 2010)

PROFESSIONAL MEMBERSHIPS

2005-present Member, American Association of History of Medicine

2016-present: Executive Committee, American Association of the History of Medicine

2013-2014: Program Committee, American Association of the History of Medicine

2011-2012: Program Committee, American Association of the History of Medicine

2010-2011: Research Awards Committee, American Association for the History of Medicine

2005-present Member, History of Science Society

EXHIBIT “C”

Bibliography provided to Stephen Casper by Stuart Davidson on July 7, 2015

Concussion/Brain Injury and Disease Articles

Year	Article
1. 1927	Osnato, M., "Postconcussion Neurosis – Traumatic Encephalitis – A Conception of Post-Concussion Phenomena," Arch. NeurPsych. 1927, 18:181-214
2. 1928	Martland, H.S., "Punch Drunk," Journal of the American Medical Ass'n. 91(15); 1103-1107
3. 1934	Parker, H., "Traumatic Encephalopathy ('Punch Drunk') of Prof. Pugilists, J. Neuro. Psychopathol. 1934; 15:20-28
4. 1937	Millspaugh, J.A., "Dementia Pugilistica," U.S. Naval Medicine Bulletin, pp. 297-303
5. 1949	Critchley, M., "Punch-Drunk Syndromes: the Chronic Traumatic Encephalopathy of Boxers, Hommage Clovis Vincent," Paris: Maloin 1949
6. 1952	Burne, E.W.; Silverman, A.J., "Electroencephalographic Changes in Professional Boxers," Journal of the Am. Med. Assn. 1952; 149(17): 1522-1525
7. 1954	Brandenburg, W. and Hallervorden, J., "Dementia Pugililitistica with Anatomical Fidings," Virchows Arch. 1954; 325(6): 680-709
8. 1956	Strich, S., "Diffuse Recognition of the Cerebral White Matter in Severe Dementia Following Head Injury,: Journal of Neurology, Neurosurgery and Psychiatry 1956 Aug.; 19(3): 163-185
9. 1957	Critchley, M., "Medical Aspects of Boxing, Particularly from a Neurological Standpoint," Br. Med. J. 1957; 1: 357-362
10.1959	Cornellis, J., et al., "Observations on the Pathology of Insidious Dementia Following Head Injury," Journal of Mental Science 1959; 105:714-20
11. 1966	Miller, H., "Mental After-Effects of Head Injury," Proc. R. Soc. Med. 1966; 59: 257-61

12. 1966 Fisher, C.M., "Concussion Amnesia," Neurology 1966 Sep.; 11(5): 173-88

13. 1969 John Johnson, M.D., M.R.C.P.E., D.P.M., "Organic Psychosyndrome due to Boxing," The Brit. Journal of Psychiatry 1969; 115:45-53

14. 1969 Roberts, Anthony Herbers, "Brain Damage in Boxers: A Study of the Prevalence of Traumatic Encephalopathy Among Ex-Professional Boxers," London: Pittman Med. & Scientific Pub. Co., Ltd., 1969

15. 1970 Yarnell P.R., Lynch S., "Retrograde Memory Immediately After Concussion," The Lancet 1970; 295(7652): 863-864

16. 1973 Cornellis, J.A., Bruton, C.J., Freeman-Browne, D., "The Aftermath of Boxing," Psychol. Med. 1973 Aug.; 3(3): 270-303

17. 1974 Ommaya, A.K., Gennarelli, T.A., "Cerebral Concussion and Traumatic Unconsciousness: Correlation of Experimental and Clinical Observations of Blunt Head Injuries," Brain 1974; 97: 633-654

18. 1974 Harvey, P.K., Davis, J.N., "Traumatic Encephalopathy in a Young Boxer," Lancet 1974 Oct. 19; 2(7886): 928-9

19. 1974 Gronwall, D., Wrightson, P., "Delayed Recovery of Intellectual Function After Minor Head Injury," Lancet 1974 Sep. 14; 2(7881): 605-9

20. 1975 Gronwall, D., Wrightson, P., "Cumulative Effect of Concussion," Lancet 1975; 306(7943): 995-997

21. 1978 Corsellis, J.A.N., "Posttraumatic Dementia," Aging 1978; 7: 125-133

22. 1981 Tysvaer, A., Storli, O., "Association Football Injuries to the Brain: a Preliminary Report," British Journal of Sports Medicine 1981; 15: 163-166

23. 1991 Mortimer, J.A., Van Duijn, C.M., Chandra, V., et al., Eurodem Risk Factors Research Group, "Head Trauma as a Risk Factor for Alzheimer's Disease: a Collaborative Re-Analysis of Care-Control Studies," Int. J. Epidemiol. 1991; 20: 528-535

24. 1995 Mendez, M.F., "The Neuropsychotic Aspects of Boxing," Int'l J. Psychiatry Med. 1995; 25(3): 249-62

25. 1999 Gedder, J.F., Vowles, G.H., Nicoll, J.A., Revesz, T., "Neuronal Cytoskeletal Changes Are an Early Consequence of Repetitive Head Injury," Acta Neuropathol. 1999; 98: 171-178

26. 2005 Guskiewicz, K. M., Marshall, S.W., Bailes, J. McCrea, M., Cantu, R.C., Randolph, C., Jordan, B.D., "Association between Recurrent Concussion and Late-Life Cognitive Impairment in Retired Professional Football Players," Neurosurgery 2005, Oct.; 57(4): 719-726

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EXHIBIT “D”

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