Boxing

Boxing and the brain

P McCrory

Revisiting chronic traumatic encephalopathy

Chronic traumatic brain injury or chronic traumatic encephalopathy (CTE) is considered by some authorities to be the most serious health problem in modern day boxing.[1] The condition is often referred to by a number of names in the medical and non-medical literature including dementia pugilistica and "punch drunk" syndrome.

Whilst there exists great controversy regarding the ethics of boxing, one of the key medical issues is the risk of a boxer developing CTE either during or after his boxing career. Recent evidence suggests that exposure to boxing alone is insufficient to cause this condition.

It is believed that CTE represents the cumulative long term neurological consequences of repetitive concussive and sub concussive blows to the head.[1–4] CTE is more common in professional rather than amateur boxers, however, CTE has been documented in other sports such as American Football, ice hockey, rugby, horse racing, and soccer.[5–7]

CTE is clinically characterised by a combination of speech and gait disturbance, pyramidal tract dysfunction, memory impairment, extrapyramidal features, behavior or personality changes, and psychiatric disease.[1–3 8] In the early stages of this condition, the symptoms are transient and reversible, however, in the later stages they are progressive. The neurology of CTE includes characteristic neuropathological features of cerebral atrophy, septal fenestration, cerebellar tonsillar scarring, cavum septum pellucidum, loss of pigmented cells, and prominent neurofibrillary tangles.[7]

It is salient to review the original paper discussing the neuropathology of CTE. Although individual case reports had been published of boxers with chronic dementing illnesses, the seminal paper discussing the association of neuropathological findings in boxers was published by the English pathologist, John Corsellis.[7] He studied the brains of 15 retired boxers and retrospectively studied their fight histories. While a number of characteristic changes were noted in these brains, it is the boxers' histories that deserve specific note. Of the fighters studied, their exposure to boxing ranged between 300 and 700 bouts in the course of their careers. This was in addition to sparring and other fight training that would have occurred.

The issue then that needs consideration is that in this day and age we would seldom see a fighter with such a record. Even the top professionals report fight careers of 30–50 fights before retirement, an order of magnitude less than that described in Corsellis' landmark study.

Recent research in boxers has also suggested that CTE in boxers may be associated with a particular genetic predisposition. The apolipoprotein E ϵ-4 gene (ApoE), a susceptibility gene for late onset familial and sporadic Alzheimer's disease, may be associated with an increased risk of CTE in boxers.[1 6 9]

In a non-boxing population, ApoE polymorphism was significantly associated with death and adverse outcomes following acute traumatic brain injury as seen in a neurosurgical unit.[10] In a recent prospective study, ApoE genotypes were tested for their ability to predict days of unconsciousness and functional outcome after six months.[11] There was a strong association demonstrated between the ApoE allele and poor clinical outcome.

Furthermore, ApoE deficient (knock-out) mice have been shown to have memory deficits, neurochemical changes, and diminished recovery from closed head injury when compared to controls.[12] It is suggested that ApoE plays an important role in both neuronal repair and antioxidant activity resulting in ApoE knockout mice exhibiting an impaired ability to recover from closed head injury.

How then does this help the debate on the risks of boxing? Firstly we need to reconsider the original evidence on exposure as a risk factor for CTE. The simplistic assumption based on epidemiological data from previous studies that CTE is a manifestation of the length of a boxer's career and hence exposure to punches needs to be readdressed.

Similarly the development in understanding of the genetic risk that a boxer may carry developing CTE means that this area may need to be re-examined in light of current day research. This issue also raises a number of ethical issues, if a boxer is found to be homozygous for the ApoE ϵ-4 phenotype should his boxing career be curtailed? At the very least, informed consent, and genetic counseling should be undertaken.

Whilst one may argue the ethics and morality of boxing, it behooves us as scientists and clinicians to at least place the medical arguments regarding risk of injury on a scientific footing.

Br J Sports Med 2002;**36**:2

REFERENCES

1 **Jordan B**, Relkin N, Ravdin L. Apolipoprotein E epsilon 4 associated with chronic traumatic brain injury in boxing. *JAMA* 1997;**278**:136–40.
2 **Jordan B**. Genetic susceptibility to brain injury in sports: A role for genetic testing in athletes? *Phys Sportsmed* 1998;**26**:25–6.
3 **Jordan B**, ed. *Medical aspects of boxing*. Boca Raton: CRC Press, 1993.
4 **Nicholl J**, Coleman P, Williams B. The epidemiology of sports and exercise related injury in the United Kingdom. *Br J Sports Med* 1995;**29**:232–8.
5 **NH & MRC**. Boxing Injuries. Canberra: Australian Government Publishing Service, 1994.
6 **Unterharnscheidt FJ**. About boxing: review of historical and medical aspects. *Tex Rep Biol Med* 1970;**28**:421–95.
7 **Corsellis JA**, Bruton CJ, Freeman-Browne D. The aftermath of boxing. *Psychol Med* 1973;**3**:270–303.
8 **Unterharnscheidt FJ**. Head injury after boxing. *Scand J Rehab Med* 1972;**4**:77–84.
9 **Corder E**, Saunders A, Strittmatter W. Gene dose of Apolipoprotein E type 4 allele and the risk of late onset Alzheimer's disease in families. *Science* 1993;**261**:921–3.
10 **Teasdale G**, Nicol J, Murray G. Association of Apolipoprotein E polymorphism with outcome after head injury. *Lancet* 1997;**350**:1069–71.
11 **Friedman G**, Froom P, Sazbon L, *et al*. Apolipoprotein E-epsilon 4 genotype predicts a poor outcome in survivors of traumatic brain injury. *Neurology* 1999;**52**:244–9.
12 **Lomnitski L**, Kohen R, Chen Y, *et al*. Reduced levels of antioxidants in brains of apolipoprotein E-deficient mice following closed head injury. *Pharmacology, Biochemistry and Behaviour* 1997;**56**:669–73.

Author's affiliation
P McCrory, Editor

Br J Sports Med: first published as 10.1136/bjsm.36.1.3 on 1 February 2002. Downloaded from http://bjsm.bmj.com/ on March 3, 2022 by guest. Protected by copyright.