Abstract

Concussion is a physical injury to the brain by shockwave and acceleration. The shockwave passes through the brain from an impact, and is reflected from internal boundaries including the skull. The skull itself vibrates, inflicting a second set of shock waves on the brain. These many reflected waves reinforce or extinguish each other throughout the brain, making diffusely scattered nodes of destruction everywhere.

Simultaneously, in those first few milliseconds, acceleration distorts the brain, the inertia of its semi-solid parts drags on long connecting tracts, pulling them apart. These two stresses cause diffuse and widely scattered cell death throughout the brain, confirmed by finite element calculations, radiology and pathology.

These scattered deaths do not matter in mild concussion, because the cells are readily replaced. The brain has great reserves, it matures by discarding unused pathways, it uses parallel pathway processing, and also new neurones do grow and can be incorporated.

The management of mild concussion and the significance of repeated concussion is considered.

ABBREVIATIONS


INTRODUCTION

The torch of concussion studies has passed from Neurology to Sports and Military Medicine. The terms, concussion and mild traumatic brain injury, are inter changeable. Concussion is a disturbance of brain function from external physical force; a mild, variable mixture of shockwave and acceleration, two physical events, inflicting mild traumatic brain injury.

Computer simulations of head trauma

Humans have different shaped brains to any other species; are unavailable for experiments, so the best experiments are add they finite element calculations on computers. Computer simulations have displayed the physics of the milliseconds after impact [1-5].

Impact shockwave

Impact sends a shockwave through the skull and brain. The shockwave passes relatively slowly through the semi-solid, semi-fluid brain, over 4-5 milliseconds, and this passage attenuates it by 40-60% [6]. By contrast, the water (CSF) in the ventricles and subarachnoid spaces transmits shockwaves very quickly, with little cushioning.

Consequently, reflected waves cross each other, reflected and refracted from intracranial boundaries, where densities and elasticities change. These criss-crossing and overlapping waves, where pressures cancel or re-in force each other, create nodes of very high or low pressure. The most damaging overlapping nodes are negative, because more than -750mm Hg causes momentary micro-bubbles, separating and dissecting tissues and blood vessels [7].

Criss-crossing, wide spread, overlapping waves cause scattered nodes of destruction, all over the brain; predicted mathematically [8], detected by MRI [9], and confirmed...
pathologically [10-13] (Figure 1).

**Skull vibration shockwaves**

A second set of shockwaves comes from the skull. Impact makes the whole skull ring like a bell for the first millisecond or so, creating waves damaging the cortex [14].

At the side opposite the impact, the ringing skull momentarily enlarges, creating damaging negative pressures, “contre coup” lesions [15,16]. Contre coup lesions are not, as used to be thought, from the brain being thrown kinetically against the hard skull.

**Distortion of the brain by inertia, during acceleration**

Acceleration happens more slowly, up to 12 milliseconds, inertia distorts and pulls on parts of the semi-fluid, semi-solid brain. Acceleration includes deceleration, from hitting something, such as the head hitting on a wooden floor, when it decelerates it over about 4 milliseconds [4]. Lateral acceleration is more important in minor concussions, it damages the corpus callosum causing more intellectual difficulty. This was observed clinically [17] and on MRI after players had been wearing instrumented helmets [18].

By contrast, anteroposterior acceleration (or deceleration) is more important in severe injury because it stretches and damages the long cortico-spinal tracts, causing spasticity and slow movement [19]. In monkeys, the milder accelerations, producing less than 5 minutes of unconsciousness, did not cause white matter lesions of the long tracts, but more severe decelerations did [20].

**Blast injury**

Concussion from an explosion has the above two mechanisms of injury, also an additional effect, blast compress the lungs and abdomen, so a powerful reflux of venous blood into the brain is added to the damage from shockwave and acceleration. Multiple small scattered haemorrhages are seen, but what difference these make is uncertain. In some car accidents simultaneous injury to the chest and abdomen creates a similar picture.

**WHAT CONCUSSION DOES TO NERVE CELLS**

Stretching a nerve cell more than 5-10% disrupts its internal protein cyto-architecture [21]; experimental studies and surgical experience on peripheral nerves shows that 10% is about the limit of stretch [22]. Shockwaves and traction disrupts energy production in the cell, so the sodium pump no longer maintains the cell wall. After this, some cells die, most repair themselves [23].

Vagnozzi et al [24] did MRI spectrography on forty professional sportsmen with minor concussions, so minor that only half of them had been unconscious. They showed a block to energy production in the tissues, which restored itself before the end of six weeks, when all had resumed playing.

More severe concussions rupture cells, so they spill their cytoplasm into the surrounding inter-cellular fluid, changing the electrolyte composition and pH, and impairing the function of surrounding undamaged cells. Thus concussion is at its worst around the first 48 hours, then begins to improve [23,25,26] (Table 1).

**Acute symptoms of concussion**

<table>
<thead>
<tr>
<th>Acute symptoms of concussion</th>
<th>Continuing symptoms after</th>
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<tr>
<td>Slowed thinking</td>
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<tr>
<td>Memory loss</td>
<td>Sensitivity to light and noise</td>
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<tr>
<td>Loss of consciousness</td>
<td>Impaired recall, and poor formation of new memories</td>
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<tr>
<td>Ataxia</td>
<td>Ataxia and dizziness.</td>
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**Figure 2** The strange course of the fornix; from the hippocampus to the mammillary bodies, wrapped around the third ventricle. Evolution explains this, in fishes it was a straight tract, but the enormous development of cortex; turning the cerebral hemispheres into a shape (like the shape of the lateral ventricle) has separated the mammillary bodies from the hippocampus.
mammillary bodies lying in the wall of the ventricle, are very susceptible to concussion [28-30], so brief memory gaps are hallmarks of concussive wave in the ventricle.

**Loss of consciousness:** Synapses and tracts of the brain stem reticular formation are pulled apart, by deceleration (or acceleration). Mild concussions only pull enough to functionally disrupt the synapses, spilling some neurotransmitters, so consciousness returns within a few minutes. Prolonged unconsciousness is from axons being pulled apart [20].

**Ataxia and dizziness:** The fourth ventricle, by its shape, concentrates the effect of ventricular shockwaves on the deepest part of the cerebellum, the flocculo-nodular lobe around the ventricle, disturbing balance [31]. The skull shockwave may inflect a contre coup on the occiput and cerebellum. A frontal blow causes millisecond expansion of the occiput and a damaging negative pressure in the cerebellum, again concentrated on the deepest part, the flocculo-nodular lobe.

Above the tentorium, the occipital lobe too, is damaged by the contre coup expansion, particularly the gyrus rectus, a higher order visual centre. This may be the cause of "seeing stars" with concussion and the sensitivity to bright lights during recovery.

The shockwave or vibration in the skull may damage labrynth directly, another cause of dizziness [32]. A temporary syndrome, of vertigo with certain movements of the head, may be seen.

**Continuing symptoms in recovery from concussion**

**Fatigue:** Fatigue is a real problem after concussion; the mechanism is probably not enough spare, alternate, pathways in the damaged brain [33].

**Light and noise sensitivity:** Just as photophobia may continue for some days, so probably sensitivity to noise is due to cortical injury to the lateral temporal lobe auditory gyr from the rebounding temporal skull. Perhaps concussion has damaged the synapses or neurotransmitters of a feedback suppressor circuit. Other symptoms continue, gradually improving after injury, as cells repair their biochemistry and new pathways are located and used.

**Children:** Children differ from adults, by the very easy pliability of the skull in infancy to the more elastic skull of youths [34], making epidural haematoma as common in youngsters (the very elastic skull springs away from the dura, a relatively sluggish bag of water and brain). The very pliable skull of toddlers enables the springy skull to absorb most of the energy of impact in falls as they learn to walk. This pliability makes subdural haematoma common, before the age of five, because the skull springs away from the slower, jelly-like brain, tearing vessels crossing the subdural space.

Because children have greater reserves of brain function, and the elastic skull absorbs much of the impact, most children's head injuries are of little consequence. However, under four, severe brain injuries are very damaging, distorting future growth of the brain [35].

Children under five sometime show a strange phenomenon with moderate concussion; for 48 hours or so they are drowsy, withdrawn, vomiting, pale and the heart rate slow; it is very worrying, but usually benign. Possibly, neurotransmitters and other metabolites, from stretched synapses, flood the brain in those first 48 hours.

**The brain’s reserve:** The brain has great reserve of spare capacity, largely unused till the last few years of life. Minor concussions seem to have little effect because of this large reserve.

**The brain matures by discarding neurones.** From the age of 10 to 25 or 30, the brain matures by discarding unused neurone circuits. From 30 onwards the loss of cells is much slower, but relentless, till by the end of life few spares are left [36]. Consequently, the older the patient, the slower the recovery from concussion, but most concussions happen under 30, in those years of greatest reserve.

**The brain prefers parallel circuits.** The brain prefers using parallel circuits for identical actions, repeated movements use a closely related group, not the same neurones. This prevents exhaustion of the cell's energy supplies and neurotransmitters [33]. Repeated stimulation of the same group, as in epilepsy, does exhaust cells, so recovery takes longer. Closely related pathways can easily be pressed into service, after the spotty damage of concussion. Calculation shows that interrupted stations in a network do not prevent that net operating, but it works more slowly after concussion, as in the internet [37].

**New cells form in the brain.** It used to be thought that no new neurones formed after infancy. In fact, just below the lining of the ventricles is a layer, the sub-ependymal zone, where new cells form [38]. The hippocampus lies in the lining of the temporal horn of the ventricle, probably new cells assist the laying down of new memories. New neurones may develop in other regions of the brain also, the essential point is, if they are used, they survive [39].

**What reduces the reserve?**

Brain reserves vary, from person to person, and with age.

**Growth and age:** The elderly recover less readily because more neurones have been discarded with aging.

**Children:** Children have large reserves, though serious damage before the fourth year prevents later normal development of the brain [35]. The skull is much more elastic, so absorbing much of the energy of impact.

**Genetics:** Apolipoprotein ε4 gene accounts for much of the poor recovery from severe brain injury [40] but in minor brain injuries (“ordinary concussions”) the adverse gene has little influence, because the reserve is so large anyway [41,42]. About one in five westerners are heterozygous for this gene.

**Past disuse or abuse of the brain:** Brain shrinkage, from illness or malnutritin, alcoholism or drugs, reduces the reserve. Past depletion of the reserve, from injury or disease, hinders recovery.

**Previous personality, risk taking, education and social problems:** Those with traumatic brain injuries are not an
“average” sample of the population; the risk takers, the incautious, the impaired, the addicted and intoxicated suffer brain injury, disproportionately often. Brain reserves are an educational and social quantity, low intelligence predisposes to concussion [43,44].

**Prolonged post concussion symptoms, management**

Obviously, there is no boundary between minor and major concussion, but there comes a time when the complaints seem disproportionate to the injury. Continuing complaints of symptoms are the signals of distress.

How many patients suffer prolonged symptoms depends on the medical system. Specialised clinics collect the distressed, making it impossible to know how many of the originally injured suffer prolonged symptoms. Some clinics search for symptoms with repeated questionnaires, prompting worry about the normal stresses of life and the possibility of permanent “brain damage”.

Firstly, not everyone complains of the symptoms of concussion. Women are three times more likely to complain than men [45] suggesting that everyone has symptoms, but men must grin and bear it, while women are allowed to ask for help. Secondly, the symptoms prolonging the illness are not specific to concussion. After either concussion, or trauma without concussion, an equal number, 43%, complained of similar symptoms.

No pathology specifically related to prolonged post-concussion symptoms has been described.

**Post-concussion symptoms**

The core symptoms of brain and skull injury have been described above, demonstrably due to brain or skull pathology. At some time, after recovery was expected, the symptoms merge into post-concussion symptoms.

Multiplicity and disability are characteristic of post-concussion symptoms, rather than the specificity. This is neither a complete, nor an exhaustive, list: their relative frequencies will depend on the interests and nature of the clinic (Table 2).

Are post-concussion symptoms specific to concussion? The common symptoms of daily life.

Unexplained symptoms are normal in general practice, especially among those “concerned” by personality. In general practice and the community [46,47], at least a third to a half of symptoms remain “medically unexplained” [46,47]. A year later, of these unexplained symptoms, said to have been “lifetime”, only 39% were recalled [49]. Fatigue too, is a common symptom, without concussion [47]. These unexplained symptoms often overlap, reappearing in other contexts for which distress is substantial and medical treatment often not very effective.

In Lithuania [50], suing for injury was uncommon, so symptoms were not prolonged in hope of gain. After 3 months headache was just as common in the concussed as in the control group. Dizziness, momentary forgetfulness and slight concentration difficulty was slightly more frequent among the concussed, but this was attributed to the administration of repeated questionnaires. The concussed underestimated the frequency of headaches, dizziness, momentary forgetfulness and slight concentration difficulty, before the accident, compared to the community [50]. Similar loss of memory for past headache with post concussion symptoms was found [45]. Others, too, have found a similar frequency of post concussive symptoms among the unconcussed [51-53].

**Which syndromes overlap with post-concussion states?**

The symptoms of prolonged post-concussion states overlap with other syndromes, of obscure pathology and often unsuccessful treatment [47,54]. These distressing syndromes lack a well-defined pathology and have overlapping symptoms.

Among them are post-traumatic stress disorder (PTSD), depression/anxiety, pain syndromes, chronic fatigue syndromes, temporomandibular disorders, fibromyalgia, irritable bowel, non-ulcer dyspepsia, and interstitial cystitis [54,55]. These symptoms associate with risks of pain syndromes, substance abuse and suicide. Multiplicity of symptoms is characteristic [55,56], they respond unpredictably to treatment, including alternative medicine.

Headline symptoms draw attention to an organ or event. around this central core of specific symptoms is a back-up of nonspecific “somatiform” symptoms, the signals of distress. These symptoms are significant, yet lack a demonstrable physiological or pathological basis, though they prompt the search for cure [57].

**Post-traumatic stress disorder.** The associated symptoms in post-concussion syndrome and post-traumatic stress disorder are very similar [58-61]. The symptoms with chronic pain are very similar [62]. In Iraq, post-concussion syndrome symptoms were just as common in those who had seen combat, as in those who had suffered a mild traumatic brain injury [63].

**The predictors of prolonged post-concussion states**

The predictors are personal and social. The personal predictors are education, marriage and previous intelligence [50]. The concussed are not an “average” sample of the population; the risk takers, the incautious, the impaired, the addicted and intoxicated suffer brain injury, appear disproportionately often.

**Aetiology of prolonged post concussion symptoms**

**Expectations as aetiology:** The expectations of symptoms following head injury causes selective attention and worry about benign symptoms after injury [64]; patients underestimate the frequency of such symptoms in their own past.

**Questionnaire and checklists as aetiology:** In concussion

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<th>Table 2: Prolonged symptoms after concussion.</th>
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<tr>
<td>slow thinking and difficulty concentrating</td>
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<td>dizziness</td>
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<td>difficulty sleeping</td>
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<td>irritability and sensitivity to loud</td>
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<td>noise or strong light</td>
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clinics the Rivermead Questionnaire is often a routine, when the patient enters the door. Searching for symptoms with surveys suggests the symptom is significant, so spoils the statistics, and confirms the suspicions of the anxious. The differing estimates of frequency of post-concussional and post-traumatic disorders in soldiers, illustrate this [58].

Reconstructions by family, friends and others, general attitudes: Family and well-wishers are notorious sources of all sorts of anxieties and worries. Widespread scepticism of some stories may provoke still louder complaint, but once in a concussion clinic, staff may confirm the worst fears of the insecure, by diligent questioning.

Chronic traumatic encephalopathy

Chronic Traumatic Encephalopathy may appear at two stages in life, during a sporting career or after 50, with approaching old age. During youth it is commonest, at least in New Zealand, in jockeys, occasional other sports, and professional boxers. Professional boxers risk developing CTE, but they know the risk and have decided to take it. Amateur boxers are less likely to suffer the early onset type, because their careers are usually relatively short, and knockdowns uncommon. How many will later develop CTE is not yet known.

Later onset Chronic Traumatic Encephalopathy appears after 50 with the premature onset of dementia. Recovery from repeated concussions depletes the cerebral reserves, so when the gradual process of Alzheimer’s begins, symptoms appear earlier, because there is no reserve. Alzheimer’s and Chronic Traumatic Encephalopathy are probably different diseases, even though both deposit amyloid and Tau proteins in the brain [65]. Other diseases and lifestyle choices may also contribute, chronic hypertensive small vessel disease, or diabetic vasculopathy, smoking, alcohol and drugs, may also deplete the reserve.

Without these, the prognosis may not be too bad. An authoritative group [66] studied retired University athletes thirty to forty years after their athletic career. Refined psychological tests distinguished the concussed from the unconcussed (mainly ice hockey players) but significantly, on interview, the researchers could not distinguish the concussed from the unconcussed, socially or professionally.

While CTE obviously develops after multiple recognised concussions, it is not clear whether many small impacts, such as heading the ball in soccer, without concussive symptoms, can cause CTE [67]. It is possible, but unproven.

Instrumented helmets (Head Impact Telemetry) [68] have already confirmed that rotational concussions are more serious than others (rotational concussions cause inertial damage to the corpus callosum, by slew ing the two hemispheres in opposite directions) [27].

Occasional deaths from boxing (amateur or other) have happened, perhaps once every 5-10 years in a New Zealand population of 4 million. Usually there is some special explanation of the death, such as a blood clotting defect. Boxing is entrenched under the New Zealand Bill of Rights; it is a pleasurable activity (or profitable, for prostitution is lawful in New Zealand) undertaken by mutual consent between adults of the same sex

How many concussions is too many? Three concussions is judged to be the limit, but this must be set against the severity of the concussion and the importance of the sporting career.

A few accept more than three concussions [69,70], but most suggest a limit of three [71-76].

CONCLUSION

Concussion is a physical injury to the brain, temporarily damaging many cells, most of which repair themselves and resume normal function.

The reflections of shockwaves and the distortions of acceleration will cause scattered cell death everywhere in the brain. The brain uses parallel processing pathways and incorporates new neurones if they are used, it has many more cells than it uses during most of life and so lost cells are replaced.

Because of these large reserves a few concussions make little difference. Repeated concussion however is an insidious brain-wasting disease, of the same order of risk as untreated hypertension, diabetes, smoking or heavy drinking. It is an individual choice.

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REFERENCES


