Professorial Fellow Indigenous Research
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He waka eke noa
Diagnosis (and general treatment comments) in community care of young people with Traumatic Brain Injury
Cultural observation

• He tapu te upoko
Clinical observation

Neuropsychiatric assessment and treatment

Whānau have responded positively when I asked and wrote about their cultural beliefs and used these to inform my recommendations
Tama was a 5 year old tamatāne when I first met him. He had sustained a severe traumatic brain injury at the age of three months.

Cared for by several whānau members including his grandmother who had passed away resulting in him moving house.

Whānau were concerned about his irritability, and angry outbursts.

Given his positive response to Kuia and Koroua, karakia, waiata and kapahaka I recommended these elements be included in his daily routine so they would reliably and predictably occur.

Monitoring found good progress.
Traumatic Brain Injury

Brain injury caused by an external force
Mild (70-90%), moderate (5-10%), severe (5%)

**Mild**: LOC less than 30 minutes, PTA less than one day, GCS 13-15 (if abnormality on CT “complex”)

**Moderate**: LOC more than 30 minutes less than 24 hours, PTA more than 1 less than 7 days, GCS 9-12

**Severe**: LOC more than 24 hours, PTA more than 7 days, GCS 8 or less

Multiple possible effects

Delayed effects

Non-accidental TBI most common in under 2 yr olds

Falls, motor vehicle accidents, assault, sport

Increased rates of psychiatric illness (Elder 2012)
TBI aetiology as a function of age

Infants: NATBI (males and females equal incidence), 95% of severe TBI in children, SBS
Preschool: falls
Primary school: pedestrian vs vehicle accidents
10-14 yrs: sports and bicycles
15 yrs plus: MVA, violence
Convention to describe primary and secondary damage

Primary brain damage: within the first few milliseconds of the injury; fracture, contusion (blunt injury), contre-coup

Diffuse axonal injury; acceleration and sudden deceleration, and rotation causes stretching and tearing of long white matter tracts

This damage causes a cascade of cellular changes
Secondary brain damage

Occurs as a result of later effects of physiological processes stemming from the primary injury

Intra-cranial: hematoma, brain swelling, infection, SAH, hydrocephalus, seizures, raised ICP resulting in ischemic injury

Extra-cranial: respiratory failure, cardiovascular compromise with hypoxia
Effects

- Motor
- Cognitive
- Behavioural
- Emotional
- Language and communication
- Perceptual
- Social
- Cultural
- Psychiatric
TBI and psychiatric disorder


TBI increases risk of psychiatric disturbance, though not of a specific symptom cluster (Rutter et al 1970)

Pattern of psychiatric disorder similar to that seen in the general population (Bijur and Haslum 1995)
TBI and psychiatric disorder

- Risk of developing schizophrenia in TBI population 2-3% vs 0.8-1% in non-TBI population (Wilcox and Nasrallah 1987)
- Depression most common Axis I disorder, 30% current, Axis II disorders also common (Koponen et al. 2002)
- Increased rates of psychiatric disorder correlate with more severe injury, greater cognitive impairments and complicating psychosocial difficulties (Breslau, Rutter, Seidel 1985)
Pre and post TBI psychiatric disorder in community study

N=100 moderate-v. severe TBI, most MVA
0.5-5.5 yrs post injury recruited from hosp data base
Pre-injury rates of psychiatric disorder 52%; AOD (41%) and depression (17%) anxiety (13%)
Post injury rates 65% had at least one diagnosis, depression 46% in the first 5 yrs, 72% of the depression was new
Trend for psychiatric disorders to rise between one and four years post injury

? Insight? Lack of progress? Lack of supports over time
(Whelan-Goodinson et al 2009)
New test for school of hard knocks

By Dana Johannsen
5:30 AM Thursday Sep 13, 2012

If a massive cultural shift is what is needed to tackle sport's concussion crisis, then it's not just up to coaches and administrators to make that happen.

The sobering research coming out of the US that has found repeated head knocks in NFL players may cause lifelong neurological problems - highlighted in the Herald's special report this week - should make us all reflect on our own attitudes to head injuries in sport.

Contact sport is here to stay but attitudes towards injuries needs to change. Photo / Getty Images
Playing future of repeatedly concussed players must be taken out of their hands in the interests of the game and its reputation.

The names of Leon MacDonald and Steve Devine will for ever be linked. Not only were they All Blacks contemporaries but their rugby careers will always be associated with struggles with head injuries. Both played on long after both multiple concussions and common sense suggested they should have retired.

They were able to do this even though another All Black, Nicky Allen, died in 1984 after suffering a catastrophic head injury.
Concussion

Immediate and transient disruption of neural function (but can lead to chronic impairment)

DSMIV-TR (Appendix B Criteria sets and Axes proposed for further study)

DSM5 NCD due to TBI
Post-concussion symptoms

Cognitive: poor concentration, forgetfulness, slowed thinking, indecision, impaired learning

Emotional: depression, anxiety, irritability, reduced tolerance for frustration, low self-confidence

Somatic: fatigue, dizziness, blurred vision, tinnitus, sensitivity to light and noise, sleep problems, poor coordination

Personality: apathy or lack of spontaneity, social or sexual inappropriateness
NZ TBI incidence

- Historically data collection has been of poor quality
- Recent HRC funded study 2010-2011 data
- NZHIS, Coroner, ACC data Waikato
- Most robust data to date
- Approx Non-Maori rate 300/100,000/yr
- Approx Māori rate 900/100,000/yr
- Youth, the very young
- Peak incidence of moderate and severe TBI age 15-19
- Males

Authors emphasise these data are a likely underestimate

NZ TBI prevalence

Christchurch Health and Development Study
30% of the cohort had seen a GP for TBI by the age of 25 (38.5% men and 24% women)

NZ TBI incidence (2010-2011)
Māori                                    non-Māori
Young age at time of injury

Challenge of maturation as well as recovery
Skills developing at the time of injury maybe more likely to be compromised
Return to a previous level of functioning is not the same as return to the same trajectory of development
Myth of childhood resilience
“good” and “bad” plasticity, enriched environments = good, TBI induced alterations in brain development may inhibit this experience dependent “good” plasticity
Hidden injury; NATBI
FAS/FAE
Multiple injuries over time
Latency of manifestation of deficits
Two stage recovery
Pediatric TBI: Two Stages of Recovery

(Chapman 2007)
TBI in infancy and childhood

Children post TBI less adept at reading emotional expression as conveyed by faces, voices and eyes compared with non-injured controls. Therefore increased risk of misinterpretation of social cues, others intentions, motivation (Tonks 2007).
Child and adolescent TBI and personality change

“Personality change” can occur after C and A TBI and stroke (Max 2012)

Functionally impairing emotionality, aggression, markedly impaired social judgment, apathy or paranoia

Persistence of symptoms linked to severity of injury and frontal lobe injuries

Increased risk of learning difficulties, social and emotional difficulties, violence
Long term outcomes of mild TBI age 0-5

Mild TBI
70-90% of all TBI
Christchurch Health and Development Study (McKinley 2002, 2008, 2013a)
Cohort of 1265
Inpatient for less than 2 days (diagnosis mTBI)
Compared to outpatient group
Diagnosis of concussion excluded
Follow up at age 14-16

mTBI group
4 times more likely to have ADHD
6 times more likely to have Conduct disorder
3 times more likely to have Substance Abuse Disorder
Anxiety and Mood disorders no significant difference
Diagnoses present other challenges
Follow up at age 16-25

Self report:
Alcohol dependence: 3 times control group
Drug dependence: 3 times control group
5 times more likely to be arrested
More than 3 times property offences
More than 3 times violent offences
Similar findings

Compared to audit groups of those with mild and a group with moderate/severe TBI when 0-16 yrs of age, minimum of 5 years post injury (McKinley 2013)

18 years at time of study

Control group with other injury

Comparing IQ, age at injury: no significant difference (mTBI a little younger)
Findings

Offending behaviour: mTBI double controls, mod/severe group 3 times greater
Substance abuse mTBI 2.75 times, moderate/severe 5 times greater
Anxiety mTBI almost 6 times greater, mod/severe almost 5 times greater
Depression mTBI group not significant, mod/severe group 3 times greater
Why does Māori TBI matter?

Māori overrepresented:
BIONIC study: 31% of the study Māori (425), more than one per day (Feigin 2012)
Non-accidental TBI (NATBI) 32.5-38.5/100,000 vs 14.7-19.6/100,000 (Kelly, Farrant 2008),
Head injury age adjusted hospital based incidence 2003/2004 458/100,000 Māori vs 342/100,000 non-Māori (Barker-Collo et al 2009)
Risk of TBI is increased in families facing a higher number of adverse events (McKinley et al 2009)
Māori rates of violent TBI in youth three time those of non-Māori (Feigin et al 2012)
Māori and neuropsychological testing

Cultural bias in some neuropsychological tests
(Ogden, McFarlane-Nathan 1997, Ogden, Cooper, Dudley 2003)

Māori likely perform poorly on tests that rely on formal education or had “westernised content”

Average scores on Digit span, Complex Figure Test recall and Selective Reminding Test

Lowest score on vocabulary

Better scores on Block Design

No standardised norms for Māori have been generated

M. Dudley current post doc research
Culture and TBI

Spanish-speaking Hispanics were found to be 15 times more likely to have a chronic disability than English speaking Hispanics with a similar injury (Plata, et al., 2007).
Differences in initial management of TBI and in referral to post-Emergency Department Hospital care (Selaissie, et al., 2004; Shafti & Gentilello, 2008).
African Americans had a poorer life satisfaction than other groups in a retrospective study of 3,368 people, one-year post moderate to severe TBI (Arango-Lasprilla 2009).

Access to rehabilitation was 15% lower in Hispanics compared to non-Hispanic whites in almost 60,000 patients with severe TBI, after accounting for insurance status (Shafti, Plata, Diaz-Arrastia, Bransky, et al., 2007).
A statistically significant diminished quality of care was reported in minorities compared to ‘caucasians’ as measured by fewer minutes in the rehabilitation treatment modalities of physiotherapy, occupational therapy and speech language therapy (Burnett, et al., 2003).
Despite “whites” having higher functional status, Hispanics had higher rates of discharge home after adjusting for demographic factors, health related factors and functional status at the time of discharge (Chang, et al., 2008).
Māori specific TBI research?

Ratima, M et al Te Anga Whakamana 1995, NZ J of Disability Studies
Harwood, M 2010; descriptive paper, rights focus
Elder 2012, 2013a and 2013b
Impact of TBI within whānau

- Financial (Nabors 2002)
- Role change (Gan and Sullivan 2002)
- Social isolation (Jordan and Linden 2013)
- Poor mental health (Kreutzer 2009)
- High levels of parental stress (Hawley 2003)
- Siblings behavioural disturbance (Swift 2003)
- Family coping is well recognised as the major determinant of TBI recovery (Taylor 2002, Braga 2005, Laatsch 2007, Woods 2011)
TBI and School

40% teachers believed no lasting effect (Linden 2013)

60% believed one ABI did not increase risk of second (Linden 2013) risk is three x greater

Impulsivity, ‘mood swings’, hyperactivity, socially disinhibition leads to disadvantage, labelled “trouble makers” (Bennington and Wishart 1999)

Health care system fails to provide educators with information, school/uni vital part of recovery
Evidence for interventions in C and A TBI rehabilitation?

Attention and memory remediation (van’t Hooft 2005, 2007)

Memory training (Wilson 2001, 2009)

Processes that involve the whole family (Braga 2005, Laatsch 2007)


Improving P-C relationship (Andersen 2012)

Cultural competency (Hasnian et al 2011)
Include exploring TBI part of your approach

Ask about TBI history
Sports
Falls
Violence
Current circumstances

Ethnicity, iwi affiliations
First language
Accommodation
Household members
Occupation
Hobbies, sports, interests
Schedule of considerations

Details of the event(s)
Injuries to head, jaw, neck, thorax, abdomen, orthopedic
Retrograde amnesia, PTA, GCS at ED/ in amb
CT/MRI, surgery
Emotional response to event at the time, now?
Post concussion sx

Poor concentration
Poor memory: examples
Headaches: throbbing/ constant, phonophobia, photophobia, visual changes assoc, eye signs
Vertigo or dizziness
Fatigue describe pattern
Hyperacusis, hearing loss, tinnitus
Visual disturbance
Altered taste or olfaction
Irritability describe pattern, ABC
Expressive language
Word finding
Dysarthria
Posttraumatic seizures, Temporal lobe aura, blackouts, details of seizure frequency, investigations, medications, levels
Other sensory changes
Other motor changes
Balance and coordination
Temperature dyscontrol
Depressive symptoms

- Low mood
- Diurnal variation
- Anhedonia
- Loss of interest
- Loss or increase in appetite
- Weight loss or gain
- Loss of libido
- Insomnia or hypersomnia describe
Depressive symptoms

- Unrefreshing sleep
- Agitation or retardation
- Fatigue or loss of energy
- Worthlessness
- Hopelessness
- Guilt
- Suicidal ideation
- Plan and intent
Anxiety Sx

- Intrusive daytime recollections
- Nightmares relating to the accident(s)
- Nightmares of intrusive recollections or other events
- Phobic avoidance of driving, reminders of incident, describe
- Hyperarousal, hypervigilence, exaggerated startle
- Simple phobias, obsessions, compulsions, rituals, panic attacks, gen anx, social phobia
Psychotic symptoms

Ideas or delusions of reference
Ideas or delusions of persecution
Nihilistic delusions
Somatic delusions
Passivity delusions
A.h, v.h. other hallucinations
Substance abuse

Alcohol, units per week, intoxication, blackouts, impaired psychosocial functioning, forensic, DIC, treatment programmes
Cannabis; frequency and quantity
Other substance use frequency and quantity
Other psychiatric symptoms

- Manic episodes
- Body dysmorphic disorder
- Distorted body image
- Dietary restriction
- Binge eating
Forensic history
Prescribed medications
Past psychiatric history
Medical History: past TBI, meningitis, seizures, neurotoxin exposure (organic solvents), other NIDDM, HT, Asthma, renal disease, hepatitis etc, investigation results
Family History
Psychiatric, neurological
Developmental history

Birth
Early milestones
Ed: primary, secondary, tertiary, occupational history
Relationship history, peers as a child, intimate relationships, long term relationships
Premorbid personality
Māori cultural assessment: ngā pou e whitu

1. Wairua is fundamental and attended to as a priority,
2. Whānau are the functional unit of healing,
3. Whānau experience the clinical world as an alien culture,
4. Mātauranga Māori has a wealth of resources specific to Traumatic Brain injury in Māori,
5. Māori Identity is about connection,
6. Places have an important healing role because they define identity, and
7. Other trauma is remembered within whakapapa when Traumatic Brain Injury discussion is invited.
Theory of wairua injury in TBI

Proposes that TBI also causes an injury to wairua
A culturally defined injury
Wairua here is understood as a uniquely Māori profound sense of connection with all elements of the universe
Injury to wairua leads to a cascade of culturally determined responses
Whānau have latent cultural resources, held within whakapapa which are relevant to TBI healing
When TBI occurs these are activated (Elder 2013a)
Te Waka Kuaka

A cultural assessment of the needs of whānau where a young Māori has a suspected or confirmed TBI

Like the kuaka in flight the tool organises many ideas and values (from ngā pou e whitu) to form a navigational path for whānau.

Will be validated and modified mid 2016 (HRC funded post-doc)

Based on Ngā pou e whitu and Theory of Wairua injury in TBI
Examination

R/ L handed
MSE
Tailored physical exam
Formulation including cultural needs assessment findings
Diagnosis
Treatment recommendations
Treatment

Needs to be coordinated
Challenges in the home/community
Multimodal, flexible, culturally responsive
Environmental aspects
Education
Symptom targeting
Eliminate iatrogenic contributors
Te Waka Oranga

Bringing the recovery destination to you
A way of working that makes the interface of the two worlds of knowledge clear
Mātauranga is an equal partner with clinical knowledge
Te Waka Oranga is developmentally flexible
The clinical team must enter a Māori space
The leadership is Māori
The process follows that of a waka preparing for a journey, on the journey and arriving at a destination
Four navigational tools, wā, wāhi, tangata, wairua practices
First: Hoe tahi; learning to work together
Second: Te Haerenga; the journey and the arrival
Te Waka Oranga

Who is on board for this journey?
What skills and emotions do they bring?
Where are we wanting to arrive?
How will we know when we have got there?
What will we do when unexpected things happen along the way? (Elder 2013b)
Application

More than 50% of Māori are connected to marae *(TPK 2010, Stats NZ and MO C and H 2003)*

70% Māori scored at least somewhat important to be involved in cultural activities *(Te Kupenga Stats NZ 2014)*

For those Māori most significantly disconnected from their Māori identity this theory and praxis may have limited application

Possible missing data or bias; methods to counter these include the range of types and locations of marae, role of Rōpū Kaitiaki
General principles of neuropharmacology

Different phases of recovery
Acute/subacute/chronic
What works early on may not work later
Symptom driven treatments
Clearly documented and communicated targets and time limits for review
General principles

- Differences in brain injured sensitivities
- Do no harm
- Polypharmacy may be necessary and can be safe and effective if done correctly and regularly reviewed
- Choose agents that multitask
Conclusions

- The importance of a holistic diagnosis process in possible or known TBI
- Always ask about TBI
- Cultural approach to understanding the meaning for Māori of TBI
- Measure will be available in 2016
- Te Waka Oranga
- Different from current pediatric TBI rehabilitation paradigm where whānau are assumed to have no salient knowledge about the TBI and professionals knowledge is privileged (Braga 2005, Woods 2011)
- Neuropsychiatry of TBI is an holistic practice
Ehara taku toa i te toa takitahi, engari he toa takatini kē

Mine is not the strength of one, but the strength of many

http://hdl.handle.net/10179/4065
Rukuhia te Mātauranga ki tōna hōhonutanga me tona whānuitanga.

Pursue knowledge to the greatest depths and its broadest horizons.