

## THEME SIX PLENARY

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# Effects of Gender and Age in Neurotrauma

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### **TRAUMATIC BRAIN INJURY EARLY IN LIFE: EVIDENCE FOR INJURY-INDUCED DEVELOPMENTAL DISABILITY**

*DA Hovda, CC Giza, ML Prins, SM Lee, GS Griesbach,  
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It is well accepted that sustaining brain injury during cerebral development results in outcomes which would not be predicted had the insult occurred in adulthood. This acceptance comes from both experimental animal and clinical observations focusing on all types of injury. Although a significant number of clinical neuropsychological and outcome studies have reported significant deficits in children who sustain traumatic brain injury, the extent of basic science work remains at best modest. From the basic science studies conducted it now appears that the pathobiological events characteristic of adult head injury result in unique age-dependent consequences during development. This result is thought to be primarily due to the dynamic changes (anatomical, neurochemical and molecular) to the young central nervous system across time, interacting with the cascade of alterations imposed by the insult. This is particularly exemplified when studying injury-induced neuroplasticity, which has been well accepted to be more robust early in life. This degree of plasticity has been thought to contribute to an enhancement of recovery of function during certain stages of cerebral maturation. In recent traumatic brain injury studies, this enhancement of plasticity appears to be jeopardized. Young rats (postnatal day 17–21), although demonstrating little cell death following lateral fluid percussion injury, lose their potential for experience-dependent neuroplasticity. While this impairment partially recovers with time, it can last for days and may pose a threat to critical periods during development. The injury-induced mechanisms for this loss in plasticity potential and how it may contribute to an injury-induced developmental disability will be discussed.

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### **IS THERE A ROLE FOR HORMONAL THERAPY AFTER TRAUMATIC BRAIN INJURY?**

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The impact of sex hormones on neurological recovery following central nervous system (CNS) injury has become a fertile field of research over the last decade. Many studies have reported robust differences in outcome measures between males and females following injury. This has led to investigations of hormonal treatment strategies targeting both primary and secondary injury cascades. Specifically, the action of estrogen and estrogen receptor activation on apoptosis, excitotoxicity, cerebral blood flow, adhesion molecules, nitric oxide, immediate early genes, growth factors, and beta-amyloid have been assessed. In the area of progesterone treatment, studies have focused on membrane stabilization, cerebral edema, excitotoxicity, reduction of the glial scar, and lipid peroxidation. Because of the positive effects of hormonal treatment on outcome, a new question has been raised as to whether or not previous experimental therapeutic interventions are gender dependent. Suzuki and colleagues (2003) have recently reported a lack of hypothermic protection in female animals compared to male and ovariectomized females after TBI. The next step in the area of hormonal research will be to determine if either estrogen or progesterone can be used as a therapeutic intervention in the clinical setting. A few studies have reported either a lack of effect or an increase in health risks following hormone replacement therapy. However, these studies have been predominantly in post-menopausal women. It is unknown whether estrogen or progesterone given to

a young to middle aged adult male or female after CNS injury would provide neuroprotection. Hormones have the ability to affect multiple injury cascades and therefore can have a “cocktail” treatment effect when given after CNS injury. Future research clarifying specific pathomechanisms that hormones target should provide new avenues for therapeutic intervention.

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**EFFECTS OF AGE AND GENDER ON LONG-TERM OUTCOME FOLLOWING TBI IN HUMANS**

*J Ponsford (Melbourne, Australia)*

Numerous factors have been examined in terms of their relationship with outcome following traumatic brain injury (TBI). Animal studies have identified hormonal influences on responses to injury and recovery, creating a potential gender effect. Age has also been identified in both animal and human studies as a factor which has a significant influence on outcome, but, like gender, this operates in a complex fashion. The influence of both variables is potentially modified by environmental and developmental factors. This paper will examine long-term outcome following traumatic brain injury (TBI) in humans across the lifespan. The relationship of a number of demographic and injury severity variables with outcome will be examined in a large cohort of individuals with severe TBI. After PTA duration, age at injury represents the strongest outcome predictor. Those who are older show worse outcomes overall, particularly in the domains of mobility and occupation. On the other hand those who are younger are more likely to report being affected from a cognitive, psychosocial and emotional point of view. The impact of the ageing process on head-injured individuals has yet to be documented. There is reason to believe that these individuals may be more susceptible to neurodegeneration. Available evidence in relation to this hypothesis will be discussed.

**SESSION 6.1:** Gender

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**GENDER DIFFERENCES IN DIFFUSE VS. FOCAL TBI IN MICE**

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Recent studies in our laboratory have evaluated the time course of post-traumatic cytoskeletal damage (spectrin degradation) and neurodegeneration (de Olmos silver staining) after diffuse or focal (controlled cortical impact, CCI) in mice. In the diffuse model, male mice showed a peak of calpain-mediated cytoskeletal breakdown in cortex, hippocampus and striatum and accompanying neurodegeneration at 3 days post-injury. The magnitude of the damage waned after 1 week although a low level of ongoing degeneration was seen out to 60 days. In females, the magnitude of neurodegeneration was less than in males, and the peak did not occur until 14 days after injury. A significant increase in spectrin degradation was only seen in the striatum. In males subjected to focal (CCI) TBI, a peak increase in spectrin degradation was observed in the ipsilateral hippocampus at 24 hrs post-injury and the peak in neurodegeneration was observed at 48 hrs (24 hrs earlier than in the diffuse model). In females, no increase was observed in hippocampal cytoskeletal breakdown at 24 hrs, but the peak of neurodegeneration still occurred at 48 hrs post-injury and was of the same magnitude as that observed in male mice. These results show that the time course of damage and the manifestation of gender differences in post-TBI damage are influenced by the type of model. The disparity between the two models in regards to the influence of gender may relate to differences in the biomechanics of the two injuries and the relative role of degenerative mechanisms that are influenced by estrogen.

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**REDUCTION OF APOPTOSIS AND NEUROPROTECTION MEDIATED BY ESTROGEN THERAPY FOLLOWING CORTICAL CONTUSION IN RATS**

*JF Soustiel, E Palzur, O Nevo, E Vlodaysky (Haifa, Israel)*

Traumatic brain injuries (TBI) represent a leading cause of death and functional disability in western countries, affecting mostly young patients. Despite intense and sustained efforts deployed for the development of new therapeutic strategies, no clinical benefit

has been shown by any of the investigated compounds. Increasing attention has been drawn during the past two decades to the neuroprotective effects of estrogens although most of the available data are involved with ischemic brain injury. The purpose of the present study was to investigate the potential neuroprotective value of estrogens in traumatic brain injury as a therapeutic modality. For this purpose, a contusion was created in the parietal cortex by dynamic cortical deformation in 2 groups of 10 Sprague-Dawley male rats. Following the injury, treated animals received conjugated estrogens for three days, using a subcutaneously implanted osmotic pump. Animals were then sacrificed and TUNEL, anti-active Caspase 3, bcl-2 and bax labeling were performed in paraffin-embedded brain sections, allowing for comparative and quantitative analysis. In estrogen-treated animals, there was a marked and significant reduction of apoptosis in comparison with non-treated animals. This reduction rate was closely similar using the two apoptosis labeling methods and close to 50%. Optical analysis of histological slides prepared by anti-bcl-2 labeling showed a significant increase in bcl-2 expression in estrogens-treated animals compared to non treated animals. On the contrary, bax expression was not influenced by hormonal treatment and no difference could be noticed between the two groups. These results support the potential therapeutic value of estrogens in TBI and further clarify their mode of action.

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**INVESTIGATING GENDER DIFFERENCES IN OUTCOME FOLLOWING SEVERE TRAUMATIC BRAIN INJURY**

*J Lee, J Lim, LK Keow, WH Bee, YT Tsai, I Ng (Singapore)*

*Objective:* The objective of this study was to investigate if there are possible gender differences in relation to outcome following closed severe Traumatic Brain Injury (TBI) in a predominantly Asian population.

*Methods:* A retrospective study was conducted using our prospectively maintained severe TBI database. Four hundred and eighty patients with severe head injury admitted into our neurosurgical intensive care were studied. All patients were managed according to the "Guidelines to the management of severe traumatic brain injury." A dichotomised Glasgow Outcome Score was used to measure the outcome of patients 6 months post injury.

*Results:* There were 100 females and 380 males. The median age of female patients was significantly higher than male patients. There was a slight but not significantly higher overall case fatality rate and proportion of patients who made a poor recovery (at 6 months post injury) in females. Crude and adjusted odds ratios revealed that only increasing age, presence of pupil abnormality and a lower GCS score were significantly predictive of death and poor outcome.

*Conclusions:* Both odd ratios indicate that mortality and poor outcome did not differ significantly between men and women.

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**THE INFLUENCE OF AGE AND GENDER ON THE AXONAL CHANGES WITHIN SPINAL CORD WHITE MATTER AFTER ACUTE TRAUMATIC CERVICAL SPINAL CORD INJURY: A HISTOPATHOLOGICAL AND IMMUNOHISTOCHEMICAL STUDY IN HUMANS**

*JC Furlan, MG Fehlings (Toronto, Canada)*

Given the potential effect of age and gender on outcome after acute spinal cord injury (SCI), we examined the influence of these variables on axonal preservation within white matter tracts after SCI. Alternate sections of postmortem spinal cord tissue from uninjured and injured individuals (below injury) were stained for myelin (LFB) and axonal preservation (NF200). The extent of demyelination/degeneration and the number of axons within the corticospinal tracts (CST), dorsal columns (DC) and lateral funiculus (LF) were quantitated using Image-Pro system. Data were analyzed using Student's t-test, Mann-Whitney Rank Sum Test, Fisher's Exact Test, multiple linear regression. There were 7 SCI (2F, 5 M; ages 31–82 years; mean, 60) and 5 control cases (2F, 3 M; ages 30–73 years; mean, 51.4). Both injured and uninjured patient groups were comparable regarding age and gender. In control cases, the number of axons within the CST, LF, and DC was not correlated with age/gender. There was no significant difference in the extent of demyelination/degeneration between young (< 65 years) and elderly individuals or between men and women after severe (motor complete) SCI. There were no age-related differences regarding the number of preserved axons within CST, DC and LF after SCI. Females showed fewer preserved axons within the DC, CSF, and LF than males after severe SCI. Age and gender do not appear to affect the extent of demyelination/degeneration after SCI. However, fe-

male gender was associated with a reduced number of axons within the ascending and descending tracts after SCI suggesting a potentially greater susceptibility to neurotrauma.

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#### EFFECTS OF GENDER ON CEREBRAL BLOOD FLOW AND METABOLISM FOLLOWING HUMAN TRAUMATIC BRAIN INJURY

*TC Glenn, J Phan, J Lee, PM Vespa, DF Kelly, DA Hovda, NA Martin (Los Angeles, USA)*

**Introduction:** Human traumatic brain injury (TBI) has been associated with alterations in cerebral metabolic rates for glucose, lactate, and oxygen. The purpose of this study is to determine the effects of gender on post-traumatic cerebral blood flow and metabolism.

**Methods:** Sixty five patients with severe TBI (GCS < 9) (male:female = 46:19, mean age = 35.9 ± 17) and 34 normal volunteers (male:female = 23:11, mean age = 32.9 ± 8.8) were consented for 133Xenon cerebral blood flow studies (CBF, ml/100 g/min) and arterial-jugular venous (AVD) sampling for glucose, lactate, and oxygen and cerebral metabolic rates (CMR, mg/100 g/min) calculated during the initial 6 days post-injury. Statistical analysis utilized ANOVA with significance defined as  $p < 0.05$ .

**Results:** Normal male and female volunteers showed similar CMRs with no statistical differences. However, CBF showed a statistical trend ( $p = 0.09$ ) with greater flow in females ( $51.6 \pm 10.6$ ) compared to males ( $45.2 \pm 10.0$ ). Following trauma, overall CMRs were depressed compared to normals. Gender did not affect CMRs but CBF was statistically greater in females than in males; females  $44.1 \pm 17.6$ , males  $37.0 \pm 10.8$ . Using historical limits of 60 and 20 ml/100g/min for hyperemia and ischemia, respectively, TBI females had higher frequencies for both extremes; females 14% hyperemia and 4% ischemia, while males had 4% hyperemic and < 1% ischemic studies.

**Conclusions:** Cerebral metabolic rates for glucose, lactate, and oxygen were not significantly different between males and females. The physiologic differences in CBF between normal males and females were maintained following TBI. However, females showed a greater incidence of extreme CBF values than males. Thus gender-specific therapies for CBF maintenance may need consideration.

### SESSION 6.2: Age and Sporting Injuries

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#### LATERAL FLUID PERCUSSION IN THE DEVELOPING RAT PRODUCES A STATE OF PRE-CONDITIONING

*GG Gurkoff, D Shin, CC Giza, R Sankar, DA Hovda (Los Angeles, USA)*

In adult rats, traumatic brain injury (TBI) produces a state of cellular vulnerability that is revealed by secondary insults. It is hypothesized that a similar TBI-induced vulnerability exists in the developing brain. To investigate this we incorporated a model of lithium-pilocarpine (LiPc)-induced status epilepticus in conjunction with lateral fluid percussion injury (LFP). P19 rat pups ( $N = 34$ ), under isoflurane anesthesia, received either sham or severe LFP (loss of consciousness > 120 s) followed by saline or LiPc administration at 1, 6, or 24 hours post-injury. Using eosin fluorescence as a marker we characterized the average number of dying hippocampal neurons per section by combining counts from the hilus, CA3 and CA1 bilaterally. At 24 hours post-secondary insult we observed a statistically significant effect across groups ( $p = 0.035$ ) on the average number ( $\pm$  SD) of dying hippocampal neurons per section (sham-LiPc =  $112.4 \pm 121.0$ , LFP-sham =  $7.2 \pm 9.7$ , LFP-LiPc (1 hr) =  $31.3 \pm 6.8$ , LFP-LiPc (6 hr) =  $33.0 \pm 14.3$ , LFP-LiPc (24 hr) =  $78.7 \pm 29.6$ ). These results indicate that when LFP precedes LiPc acutely, that there is a decrease in cell death compared to status alone. Therefore, in the immature rat, LFP appears to provide a window of neuroprotection, as opposed to vulnerability, in relation to post-traumatic status epilepticus. This finding contributes to the literature describing the unique response to TBI when it occurs early in life.

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#### ATHLETES AND CONCUSSION

*P McCrory (Melbourne, Australia)*

There has been a large amount of research characterizing the epidemiological, clinical, and biomechanical aspects of sports-related concussion over the past twenty years [1,2]. A number of key differences exist

in the sports medicine approach. The term concussion is used in this setting to encompass the historical rather than neurosurgical definition reflecting a low velocity injury with transient symptoms. Sports injuries provide a useful human model of TBI that enables characterization of biomechanical causes and clinical features [3]. An international consensus has developed in regard to the management of these injuries [4]. As the vast majority of concussive injuries in sport are minor, the early return to play or activity has centered on the rapid assessment of individual clinical and cognitive recovery utilising computerised neuropsychological screening batteries [5].

## References

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### **PATHOPHYSIOLOGY OF REPETITIVE HEAD INJURY IN SPORTS: PREVENTION AGAINST CATASTROPHIC BRAIN DAMAGES.**

*T Mori, Y Katayama, T Kawamata (Tokyo, Japan)*

**Introduction:** The most common head injury in sports is concussion and repeated concussions occurring within a short period occasionally lead to fatal outcome. In the present study we investigated severe brain damages due to repetitive head injury in sports and discussed the pathophysiology of sports related repetitive injury.

**Methods:** We reviewed the literature of severe sports related repeated head injury described in detail. Total 16 cases included our cases were analyzed as age, gender, the kind of sports, symptoms before second injury and pathology of brain CT scan.

**Results:** The majority of these cases involved male adolescents or young adults (16 to 23 years old) who sustained a second head injury before symptoms from

the first head injury resolved. Ten of 16 cases did not have a loss of consciousness at the insult. Seven cases were confirmed brain CT scan after second injury and all 7 cases showed brain swelling associated with a thin subdural hematoma.

**Conclusions:** Second impact syndrome is thought to occur because of a loss of autoregulation of the cerebral blood flow, which leads to vascular engorgement, increased intracranial pressure, and eventual herniation. The present study suggests that the existence of subdural hematoma is one of the major causes of brain swelling after sports related repetitive head injury. It is important that coaches, as well as players and trainers, understand the medical issues involved in concussion. Physical evaluation is needed to decide when athlete can return to competition especially contact sports such as boxing and American football.

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### **MILD CONCUSSIVE HEAD INJURY RESULTS IN INCREASED BRAIN SUBSTANCE P IMMUNOREACTIVITY**

*J Donkin, I Cernak, K Rodgers, R Vink (Adelaide, Australia; Washington, USA)*

Diffuse cerebral swelling is a major risk factor in sports related head injury. While many suggest that the condition is part of a second impact syndrome as a consequence of recurrent concussion, few experimental studies have examined the possible mechanisms associated with this predisposition toward profound oedema development. In the present study, we used a newly developed model of mild concussive head injury in rats to characterise the release of the neuroinflammatory peptide, substance P. Rats were subjected to mild concussive head injury using the Cernak model of impact acceleration traumatic brain injury and assessed for functional outcome over the ensuing 3 weeks posttrauma. Animals displayed no motor or cognitive deficits at the level of injuries used in the present study. H&E staining of brains at 1 and 3 d after injury showed areas of dark cell change in the hippocampus but no significant changes in the cortex. Axonal injury as determined using amyloid precursor protein was not detected. In contrast, increased substance P immunoreactivity was present in perivascular axons and some pyramidal neurones and astrocytes when compared to sham animals. Our results demonstrate that even in the absence of detectable changes in motor or cognitive function, mild concussive injury results in an increase in substance

P immunoreactivity. Given the role of substance P in neurogenic inflammation, our findings suggest that concussive brain injury predisposes an individual to diffuse brain swelling, which may have implications in the management of sports related concussion.

### SESSION 6.3: Neuropsychological Outcome

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#### **EVENT RELATED POTENTIALS (ERPs) AND IQs IN THE SEVERELY HEAD-INJURED PATIENTS. MEANING OF OMISSION AND FAILED IDENTIFICATION OF P300 PEAKS.**

*Y Naito, H Ando, K Seki, T Yamada, T Kamishima, H Zenjiro, M Yamaguchi (Osaka, Japan)*

Prolonged-latency on event-related potentials (ERPs) has been reported on severely head-injured patients. However we frequently experience failed identification of P300 peak, and exact latency was not measurable accordingly. Although the patients cooperate odd-ball task, they sometimes omit the response possibly due to mental impairment. We, therefore, studied the relationship between intelligence and ERPs performances after traumatic brain injury (TBI).

*Method:* Forty patients (mean: 48.3 years) were compared with the control group (mean: 50.9 years) on P300 (latency, number of omission) and IQs of WAIS-R. These patients suffered from severe TBI (DAI or focal contusion) and reached their plateau level of cognitive function.

*Results:* Prolonged-latency, increased number of omission, and decreased IQs were observed on TBI group. In the control group, no omission was recorded on 39 individuals but one, while many omissions were observed in TBI. Then, TBI group was divided into 2 subgroups. The mild TBI showed 0–2 omissions, and the severe one 3 or more. In the mild group P300-peak was easily identified on 25 cases out of 26. However 6 patients in 14 showed P300-peak in the severe group ( $p = 0.0002$ ).

Mean total IQs were 90.1, and 70.8, in the mild and severe TBI, respectively ( $P = 0.03$ ). Difference of P300-latency was insignificant between severe and mild groups.

*Conclusion:* The severe TBI group with many omissions on ERPs showed significantly lower IQs when compared with the mild one. Failed identification of P300 peak also indicates mental impairment in TBI

groups. While measurable latency only may give less information.

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#### **DELINEATING COMMUNICATION IMPAIRMENTS ASSOCIATED WITH MILD TRAUMATIC BRAIN INJURY (mTBI)?: A CASE REPORT**

*B-M Whelan and B E Murdoch (Brisbane, Australia)*

To date, research investigating the behavioural effects of TBI has largely focused upon moderate to severe injuries associated with extended periods of lost consciousness. It has been acknowledged, however, that an absence of frank neurological disturbance subsequent to head injury does not guarantee an unmarred recovery. Indeed, mTBI associated with Glasgow Coma hospital admission scores of 13–15, has been linked to persistent physical, cognitive and affective disturbances. The cognitive sequelae of this syndrome represent the most frequently studied symptoms, including impaired information processing speed, memory, attention, and executive control. In relation to language, however, the impact of mTBI remains largely undetermined. The majority of investigations to date have been neuropsychologically based, lacking in-depth linguistic analysis. Evidence of story recall and verbal fluency deficits in the presence of intact general language skills following mTBI, however, suggest the implication of higher-level linguistic processes. The aim of the present research was to more accurately define the neurolinguistic pathology associated with mTBI, via the application of a comprehensive language assessment battery. A detailed language profile was compiled for a 37 year old female, subsequent to mild head injury. Performance was compared to a normative cohort of 10 non-neurologically impaired subjects, matched as closely as possible with respect to age, sex and educational level. The results obtained are discussed within the context of potential neuronal fall out mechanisms associated with mTBI, and implications for higher-level language functions, hypothetically supported via the frontal lobes. Clinical directives in relation to the assessment and rehabilitation of mTBI-based language impairments are also offered.

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**THE ROLE OF THE BASAL FOREBRAIN AND ASSOCIATED STRUCTURES IN THE COGNITIVE SEQUELAE OF HEAD INJURY***CH Salmond, DA Chatfield, DK Menon, JD Pickard, BJ Sahakian (Cambridge, UK)*

*Introduction:* Traumatic brain injury is the most common cause of death and disability in young people and survivors often suffer from chronic cognitive deficits. From animal, post mortem and cognitive studies, there is now increased evidence that abnormalities in the cholinergic system may be underlying these deficits (see Arciniegas 2003, *Current Psychiatric Reports* 5: 391–399). This study investigated this hypothesis in a group of survivors of moderate-severe head injury ( $n = 31$ ).

*Methods:* Patients completed a comprehensive neuropsychological assessment using the CANTAB battery ([www.camcog.com](http://www.camcog.com)) and an MRI scan. Neuropsychological results were analysed using Mann Whitney U Test (as the distribution of the data violated assumptions of normality) and whole brain structural analysis was carried out using voxel-based morphometry.

*Results:* Compared to a group of controls (matched on age, sex and premorbid IQ), the patients showed deficits in sustained attention, paired associate learning and reaction time, but comparative preservation of spatial working memory. Voxel-based morphometry revealed reduced grey matter density in the head injured group in the basal forebrain, the hippocampal formation and regions of the neocortex.

*Conclusions:* These cognitive and structural results are consistent with cholinergic dysfunction. These preliminary findings suggest that cholinergic enhancers may be an effective treatment of cognitive deficits post head injury.

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**LINKING LINGUISTIC RECOVERY AND MILD TRAUMATIC BRAIN INJURY (TBI) IN CHILDHOOD***K M. Docking and B E. Murdoch (Brisbane, Australia)*

Limited research has highlighted the existence of linguistic deficits subsequent to TBI in children, a find-

ing that runs contrary to the traditional view indicating children make a rapid and full recovery from brain trauma compared to adults. However, recent attention has turned toward the underlying pathophysiology of recovery in the paediatric population following mild TBI. The potential impact of a mild TBI upon subsequent linguistic development and recovery mechanisms in children is largely unknown despite indications that more long-term neurolinguistic difficulties may occur, resulting in abnormal developmental sequence, delayed emergence of skills, or shortfall in mastery levels. While mild insults to the developing brain have traditionally been deemed unremarkable, it is considered that those aspects of linguistic development that are ongoing at the time of insult are at greater risk for specific impairment, such as pragmatic skills or conceptual reasoning, while those that had matured prior to injury (such as basic general expressive or receptive skills) may remain intact. The present paper will discuss the underlying recovery mechanisms of the developing paediatric population in light of key linguistic considerations supported by case data and will highlight implications for clinical management.

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**ASSESSMENT OF TONGUE FUNCTION FOR SPEECH IN INDIVIDUALS WITH TRAUMATIC BRAIN INJURY: ARE MEASURES OF TONGUE STRENGTH, ENDURANCE, AND FINE PRESSURE CONTROL RELEVANT?***JV Goozée, BE Murdoch, MS Kuruvilla (Brisbane, Australia)*

Speech disturbances are common and often persistent following severe traumatic brain injury (TBI). Reductions in tongue strength, endurance, rate of repetitive movement and fine pressure control have previously been found to be exhibited by individuals following TBI and posited to underlie their speech disturbances. Empirical data to support this claim is, however, sparse, with the issue remaining contentious. The present study aimed to investigate the relationship between physiological nonspeech tongue function measures and tongue kinematics during speech in a group of 12 patients with dysarthria (neuromotor speech disturbance) following severe TBI and a group of age and sex matched control participants ( $n = 12$ ). A tongue pressure transducer instrument was used to investigate the nonspeech parameters: tongue strength, endurance, rate of repetitive movement and fine pressure control.

Speech kinematic measures including maximum velocity, maximum acceleration, distance and duration of tongue movements exhibited during the production of two consonants, /t/ and /k/, were recorded using an electromagnetic articulograph (AG100 EMA, Carstens Medizintechnik) system. The lack of significant correlations between the nonspeech and speech parameters questioned the relevance of the nonspeech tasks in the assessment of disorders of speech motor control and

performance. As a caveat, however, the appropriateness of the kinematic parameters, which were limited to the movement of the tongue up to the hard palate during consonant productions, also needs to be reviewed. The findings will be discussed in relation to the motor control system underlying tongue function. Clinically, the findings have implications for the types of tasks used in the treatment of neurogenic speech disorders.