BRAIN INJURY IN CHILDREN AND ADOLESCENTS

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EPIDEMIOLOGICAL FEATURES AND PREVENTION OF TBI IN DEVELOPMENTAL AGE

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Background

Injuries are a major preventable public health problem in the United States. They affect children disproportionately. Among children 1 to 19 years of age, injuries cause more deaths than all diseases combined, they are a major cause of use of emergency medical resources, of hospitalization, of visits to the Emergency Department, and of disability.[1] Traumatic Brain Injury (TBI) is a frequent and often devastating injury in the pediatric population. In 1993 a total of 20,945 children and youths died from injury[2]: approximately one-half of these traumatic childhood deaths are due to head injury. Although most of the nearly 5 millions/year pediatric head injuries are minor,[3-5] estimates are that 200,000 children are hospitalized each year with head trauma, and 15,000 require prolonged hospitalization.[6] Among children with severe head injuries, about 50% have major neurologic sequelae, and 2 % to 5% are disabled for life.[4]

Objectives

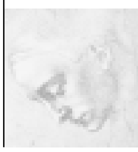
To study the variations by age group in patient characteristics, injury characteristics, nature and severity of injury, utilization of resources, and outcomes at discharge from acute care in a population of children newborn to 19 years of age hospitalized for TBI over a 10-year period. Trends over time are examined.

Design/Methods

Descriptive statistics by age groups of TBI cases reported to the National Pediatric Trauma Registry (NPTR) 1989-1998. Age groups, in years, were defined as: <1, 1-4, 5-9, 10-14,15-19. TBI cases were identified by the presence of one or more of the following ICD-9-CM codes: 800.xx-804.xx, 850.xx-854.xx. Variables analyzed include sex, circumstances, external causes of injury, nature and severity of injury, admission to the intensive care unit (ICU), surgical intervention, hospital length of stay (LOS), in hospital death rate, and disability at discharge. The injury severity was measured by the Injury Severity Score (ISS). The disability at discharge was assessed by child's level of performance in 10 functional activities: vision, hearing, speech, self-feeding, bathing, dressing, walking, bowel/bladder control, cognition, and behavior. The source of the data was the NPTR, a voluntary national pediatric trauma databank that includes hospitalizations from all injuries, except burns, poisoning, and near drowning.

Results

During the 10-year period, 79,958 cases of pediatric trauma were recorded in the NPTR and a total of 35,954 (45%) cases sustained a TBI. The proportion of children with TBI varied by age. The highest proportion of TBI was seen in the youngest children: 74.5% among the infants, 51.5% in children 1 to 4 years of age. Among the



other age groups, TBI accounted for ~ 40% of all injuries. The proportion of children with TBI decreased over the 10-year period from ~48% to ~42%.

Five groups of children with TBI were analyzed: children younger than 1 year (G1, n=2594), aged 1 to 4 years (G2, n=10368), 5 to 9 years (G3, n=9495), 10 to 14 years (G4, n-8289), and 15 to 19 years (G5, n=5208).

As usually seen in pediatric trauma, boys outnumbered girls in increasing proportion from G1 to G5 (57.6% to 67.7%). The type of injury causing TBI was mainly blunt across groups, with a small proportion of penetrating trauma that increased from 0.7% in G1 to 4.1% in G5. While the majority of injuries were unintentional across groups, more than 1 in 5 children in G1 were victim of child abuse and more than 1 in 10 children in G5 were victim of an assault. Self-inflicted injuries were uncommon in all but the oldest group.

The external cause of injury varied significantly with age, according with the child's developmental level and degree of independence. The 3 most frequent causes of trauma were as follows:
G1: fall (55.8%), motor vehicle occupant (13.7%), and beating (10.6%)
G2: fall (48.9%), motor vehicle occupant (20.0%), and pedestrian (12.1%)
G3: fall (24.4%), pedestrian (24.0%), and motor vehicle occupant (22.5%)
G4: bicycle (21.4%), motor vehicle occupant (20.8%), and pedestrian (17.5%)
G5: motor vehicle occupant (49.3%), pedestrian (8.8%), and beating (8.4%)

All groups sustained TBI without or with significant injuries to other body regions (multiple). The proportion of children with multiple trauma increased significantly with age, from 35.7% in G1 to 78.8%, more than twice as much, in G5. Different patterns emerged among the groups when TBI was classified as :a) isolated skull fracture(s), b) intracranial injury with or without skull fracture(s), and c) concussion. Isolated skull fracture(s) decreased with age from 42.5% in G1 to 10.3% in G5, while concussion increased from 16.5% in G1 to 62.2% in G5. Intracranial injury with or without cranial bones involvement was most prevalent in G1 (41.0%), then leveled off to ~28% in all the other groups.

The proportion of children with severe ISS (20-75) increased two folds with age from 11.7% in G1 to 22.6% in G5, reflecting the increasing occurrence of multiple trauma. Utilization of resources also increased with age. Rate of admission to the ICU varied from 33.8% in G1 to 43.6% in G5. Surgical interventions, not necessarily TBI related, increased from 11.4 to 29.2% in G5, and LOS varied from a mean of 4.7 days (median 2 days) in G1 to a mean of 7.0 days (median 3 days) in G5.
The in hospital death rate was ~5.5% in all groups with exception of G2 and G5 with a rate of ~7%. The proportion of children that left the hospital with any degree of disability increased with age from 8% in G1 to 33.5% in G5.

Conclusions

The yearly comprehensive cost in 1994 US dollars for unintentional injuries alone was estimated to be $347 billion, inclusive of $17 billion in medical care cost, $72 billion in future work lost, and $257 billion in lost quality of life7. These figures clearly demonstrate the possible economic benefits of prevention programs, not to mention the potential reduction in physical, emotional, and social distress to the victims and their families.

However, this study shows the complexity of developing effective prevention programs that need to address some many different causes and levels of maturity among the target population. This study also shows that, despite advances in restraining devices and in safety education, motor vehicles continue to be a major threat to the children's well being.

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PRIMARY AND SECONDARY INJURY: OCCURRENCE IN PEDIATRIC HEAD TRAUMA

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The general schema for the pathology of head injury has been of primary followed by secondary damage. The secondary damage was described in autopsy material and assumed to be a common component of injury even in those surviving their trauma. With the use of CAT scanning and more recently MRI scanning information generated can be used to question the basis of this assumption. In addition the differences between the pathology of pediatric head trauma and that of adult trauma are better delineated and more clearly based on the age of the child and the mechanism of trauma. The pathophysiology of closed head injury changes over time after trauma. The acute effects of trauma at a cellular level remain poorly defined. Recent evidence suggest that cell membrane distortion results in genetic activity within the nucleus. Exactly which genes are switched on remains to be clarified but this may be one of the mechanisms triggering apoptosis following trauma. Apoptosis has been proposed to explain the changes that are unique to the infant brain. These changes are described on CAT scan and include; diffuse low density of the brain, rapid loss of cerebral volume and early calcification within the cortex. On the MRI scan the early low density brain appears to be due to intracellular swelling as a result of ischemia. While the most likely reason for this injury is a combination of hypoxia and ischemia, occurring during the period between the injury and the onset of medical attention, the effects of the acute trauma on this process is unknown. The two major mechanisms that produce traumatic injury are impact and acceleration/deceleration. In children under two years and especially under one year impact injuries are usually the result of low falls and frequently result in skull fracture without intracranial injury. In older children the skull is more resistant to fracture and local impact injuries can produce any result from: nothing to fractures, linear or depressed, intracranial hemorrhage or focal contusion under the impact, coup or opposite from the impact, contre-coup. The most life threatening of these injuries are epidural hematomas resulting from either hemorrhage from the fractured bone, tear of meningeal arteries or venous sinuses. These are surgically treatable lesions and while the occurrence rate is only about 5% of cases of head trauma they are one of the major reasons for obtaining early neuroradiological imaging after trauma. When identified early the outcome is almost always good. When identified after cerebral herniation has occurred they can be fatal despite surgical evacuation. Focal contusions in children rarely require surgery since significant healing can occur and surgical treatment runs the risk of removing potentially functional tissue. The aggressive resection of contused lobes of the brain is discouraged in children for the same reason. Such lesions are rarely seen on early scans. Acceleration/deceleration injury is the usual cause of severe diffuse brain injury in children. In infants this occurs either in high speed automobile accidents or following child abuse injuries which usually include an impact. In toddlers and older children the causes are: falls, pedestrian - automobile injuries, bicycle injuries and

once the children are teenagers motor vehicle injuries and assaults. The mechanism is complex swirling motion of the brain within the calvarium leading to: subdural hematomas, cerebral contusions, intracerebral hematomas and diffuse shearing injuries. While early CAT scans did not demonstrate a high incidence of focal cortical contusions follow up MRI have demonstrated that the most frequent lesions in the surviving children are frontal and temporal tip contusions, similar to those described in adults. Diffuse cerebral swelling occurs more frequently in children than in adults after trauma. The cause of this swelling was initially felt to be the result of vascular congestion and relative hyperemia. More recent MRI studies suggest that the swelling is the result if intracellular edema. This is an entity whose pathophysiology is not yet clearly defined. Nor is it clear whether this should be classified as a primary or secondary injury. Secondary injury does occurs as a result of ischemia or hypoxia and possibly from toxic chemicals released after the trauma such as excitatory neurotransmitters and free radicals. The cause of the hypoxia and ischemia immediately after trauma is due to suppressed ventilation or apnea and shock from associated multiple trauma or spinal cord injury. This can only be impacted by earlier emergency care in the field or **preferably avoidance of the trauma**. After arrival at the hospital the major cause of ischemia is raised intracranial pressure and resultant cerebral herniation with large vessel occlusion or decreased cerebral perfusion globally as a result of severe decreases in cerebral perfusion pressure. In this later setting the injury produces diffuse cerebral ischemia primarily involving the watershed areas then the cortex in general, then the deep areas of the basal ganglia as a result of perforator ischemia from posterior cerebral and posterior communicator compression. This is a result of bilateral transtentorial herniation. In children subdural hematomas contributing to the raised ICP are much less common than in adults although thin subdurals are common. Other systemic injuries contribute to the secondary brain injury and include ongoing systemic shock, pulmonary contusions and clotting disorders.

Death is most often the result of raised intracranial pressure and primary cerebral death. In children who survive their severe injuries the most common findings do not include evidence of focal ischemia or watershed infarcts but are contusions of the frontal and temporal lobes, diffuse loss of frontal cortical mantle and evidence of shearing injuries in only a small percentage. It appears that the injuries from which children can recover are not only of lesser severity but of a different pathology with fewer areas of shearing injury and little or no evidence of secondary ischemia. If these assumptions can be supported by further research the implications for therapy and trials of drugs are immense. The inference is that efforts to modulate the primary injury at a molecular level may impact on the level of recovery but is unlikely to alter the mortality. Thus studies of drugs might better focus on those injured patients who are likely to survive rather than all severe head injuries. In children this would include GCS of 5 and up. Any such studies might also include children but not adults with a GCS of 4. In addition, study of possible beneficial drugs should be directed at moderate head injuries, GCS 9-13, since many of these children have measurable cognitive deficits that might be amenable to modification by an appropriate drug.

MANAGEMENT DURING THE ACUTE PHASE: GUIDELINES IN DEVELOPMENTAL AGE

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Introduction

A review of the literature on pediatric critical care management of children with severe brain injuries, demonstrates that strong evidences of the benefit of a precise therapy are lacking. However, elucidation of important pathological mechanisms leading to secondary brain lesions, better understanding of the consequences of therapeutic agents for brain physiology, and development of multimodal monitoring have led to changes in standard practice. Guidelines could be drawn outcome studies demonstrating that children with TBI experiencing hypoxia, hypercapnia, hypotension, hydroelectrolytic disturbances resulting in osmotic edema and hyperglycemia develop secondary brain injuries and have increased mortality and worse outcome. Methodological limitations preclude formal conclusions, but the frequency and impact of early hypotension and hypoxia on mortality and morbidity are pointed out specifically when they are still present upon admission. General critical care management should prevent secondary brain lesions of systemic origin, and must be regarded as a continuum that starts with initiation of critical care at the scene, uses multimodal monitoring for adaptation of therapeutics to brain hemodynamics and metabolism, and ends with neurological rehabilitation.

Critical care management at the scene

During the first minutes, hypoventilation and hypotension, that are major systemic factors of aggravation, are accessible to emergency maneuvers. The need for early control of the airway is not in question, but intubating a child remains an unresolved problem for many emergency medical systems. The fear of complications and failed attempt has led to a general policy of "scoop and run" transport to the next available hospital, with 50-75% of the children not being intubated until they reach the hospital. An adequate protocol for facilitating intubation should be used and include a balanced anesthesia including with hypnotic and a potent opioid in order to blunt the rise of intracranial pressure related to laryngoscopy and tube insertion. With controlled ventilation secured during transport, less than 8% of the children will be hypoxemic.

The first source of hypotension is acute blood loss from associated lesions, more than 50% of the severely head-injured children being multiple trauma patients. Whatever the cause of hypotension, it must be corrected as soon as possible, since it has major noxious consequences for cerebral perfusion pressure. There are strong evidences that decreasing plasma osmolality by using large amount of hypotonic fluids such as lactated Ringer's will increase cerebral water content and edema. On the other hand, decreasing oncotic plasma pressure without changing osmolality has no influence on cerebral edema. The rule, then, is to use isotonic, glucose-free fluids for vascular loading. Hypertonic saline solutions have the advantages of efficiently increasing arterial pressure with a small volume resuscitation and decreasing intracranial hypertension in the same way as mannitol. When vascular loading is insufficient to maintain arterial pressure, vasopressors must be added rapidly.

Critical care management

Developments in brain monitoring have led to a better understanding of hemodynamics in brain swelling and diffuse edema that are observed in more than 60% of comatose children. However, it is difficult to conclude from the existing literature what is the most frequent hemodynamic profile in children. It is probably more a dynamic evolution, and different profiles are probably present in different areas of a single brain at the same moment. Hyperventilation, the major stimulus of vasoreactivity, must be used cautiously to control cerebral hemodynamics, variations, and not variations in ICP. A close monitoring is there required.

Variations in cerebral blood flow must be adapted to cerebral metabolic demand. Uncoupling between cerebral hemodynamics has harmful consequences for the injured brain. The goals of metabolic therapy are to decrease the cerebral rate of oxygen and cerebral metabolism, and to adapt CBF to the metabolic demand. ICP and CPP monitoring is the routine basis, but cannot predict adaptation between hemodynamic profile and cerebral metabolic status. Jugular bulb oxygen saturation and cerebral extraction of oxygen can be measured continuously, and allows a precise adaptation of ventilation when standard management is insufficient to control CPP. Transcranial Doppler monitoring allows bedside noninvasive monitoring and bilateral measurement of cerebral blood flow velocity, but its drawback is its questionable efficiency in assessing coupling with metabolic needs.

More than ground breaking progress, an evolution toward a standard treatment protocol based on physiological evidence has emerged in the last few years. General anesthesia or deep sedation with restricted use of muscle relaxants, tracheal intubation and controlled normoventilation, and hemodynamic stabilization are the first mandatory steps in treatment. Further treatment cannot be standardized for every patient, but must rely on multimodal monitoring of brain function.

Mannitol is still in use for cerebral edema. Bolus infusion of 7.2% saline has a beneficial effect on increased intracranial pressure equipotent to that of 20% mannitol. Moreover, continuous infusion of hypertonic saline could be efficient by way of an increase in plasmatic osmolality to control ICP in head-injured children. Restricted fluid administration for brain edema has evolved. On the one hand, fluid loading with at least isotonic solutions is critical for cerebral perfusion pressure maintenance, does not increase cerebral edema and can be used liberally when necessary. On the other hand, basal fluid maintenance can easily be restricted to strict compensation of urine output with normal saline in order to maintain a normal or slightly increased plasma osmolality.

Thiopental decreases cerebral metabolic rate for oxygen by cerebral vasoconstriction. When the decrease in CBF induced by thiopental is superior to the decrease in cerebral metabolic rate for oxygen, oligemic ischemia can result. It must be used cautiously and can be best adapted with the aid of close monitoring of $SJvO_2$.

General anesthesia and heavy sedation, but not muscle relaxants alone, decrease the cerebral metabolic rate of oxygen. None of the usually used hypnotic or opioids has a deleterious effect on cerebral blood flow and intracranial hypertension, provided that mean arterial pressure remains constant. Careful titration and adaptation of dosage to the hemodynamic status of the child is aessential.

Posttraumatic seizures are more likely to occur during the early phase and are not so frequent in children with severe head trauma. The first reason for this low incidence is probably that early critical care management prevents efficiently the occurrence of seizures. The second reason is that most of these children will benefit from sedation with hypnotics that enhance GABA receptor activity implicated in the genesis of seizures. Because of the low incidence and the limited risk of late recurrence of seizures preventive treatment is not justified.

Hypothermia has been regarded as a protective situation for the brain at risk of ischemia for many years. It decreases both components of cerebral metabolic demand: those related to electrical cortical activity and those related to basal metabolism. These results must be confirmed by larger series, and to our knowledge, nothing has yet been done on this subject in children.

It can be concluded that adequate management of severe pediatric trauma requires sophisticated monitoring capabilities. Further technical developments for a better approach to cerebral blood flow and metabolism are expected. The data obtained by this multimodal monitoring help to adapt therapy derived from a standardized protocol on an individual basis. Surgical and critical care management must be closely integrated in a precise therapeutic strategy to avoid therapeutic errors resulting in an increased incidence of secondary brain lesions. Hypothermia and recent therapies aimed at the preservation of cerebral microcirculation need further evaluation before clinical application in children.

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MAGNETIC RESONANCE IMAGING IN RELATION TO FUNCTIONAL OUTCOME OF PEDIATRIC TBI

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To characterize the late neuropathological findings of pediatric TBI and assess the depth of brain lesion in relation to acute severity and long term outcome, we performed magnetic resonance imaging (MRI) at least 3 months postinjury in a prospective sample of 169 children who sustained TBI and 82 additional children who had sustained a TBI at least 3 years previously. Lesion volume was measured by planimetry and each child was classified according to their deepest lesion: no lesion, cortical, subcortical, and deep gray/brain stem. Outcome as measured by the Glasgow Outcome Scale and the Vineland Adaptive Behavior Scale which were performed at time of MRI in the retrospective group and at 3 years postinjury in the prospective sample. Focal brain lesions were present in 55.4% of the total sample. Depth of lesion was directly related to severity of acute impairment of consciousness and inversely related to outcome. A rostrocaudal gradient of hemispheric lesion frequency was observed, whereas posterior lesions of the corpus callosum were also common. These findings extend support for the Ommaya-Gennarelli model to pediatric TBI, indicating that depth of brain lesion is related to functional outcome. The relative frequency of focal brain lesions is higher than that of previous studies using computed tomography. Future studies could explore whether depth of lesion is sensitive to neuroprotective agents.

ASSESSMENT AND REHABILITATION PLANNING

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Multidisciplinary Assessment

Traumatic brain injury (TBI) is the most common cause of acquired disability in developmental age. The clinical assessment, the rehabilitative program and the evaluations of sequelae after TBI require the use and the co-ordination of multiple competencies. Head injury evaluation in children is complicated owing to contemporary development of cognitive and psychological functions and personality organisation. The assessment and the process of rehabilitation must be multidisciplinary, whereby specific problems are addressed by specialists with the necessary skills. The multidisciplinary team is not enough, each specialist should not work in isolation but within the team to ensure that the child and his family are treated comprehensively. Multidisciplinary approach permits a comprehensive assessment, facilitates the identification of main rehabilitative goals and makes the management of rehabilitative program easier. This approach promotes functional recovery from disability, facilitates the community re-entry and reduces long term economic impact of this pathology.

The examination is divided into different parts with collection of clinical history and assessment of clinical features, neurophysiological recording, neuroradiological imaging, sensorial and perceptual evaluation, cognitive and psychological examination. The evaluation must be tailored to the severity and complexity of the deficits. By no means does every child need an extensive multidisciplinary evaluation. The frequent association in post traumatic children of a sensorial impairment, mainly of visual function, can determine sensorial deprivation or, more often, of input analysis alteration.

The elaboration of distorted sensorial inputs can lead him to make mistakes in the understanding of cognitive rules and concepts. Therefore the sensorial impairment can contribute to a worse prognosis also in cognitive development. Neurophysiological and neuroradiological assessment give us important prognostic factors like alteration of SEP and brain lesion severity. The backbone in the evaluation of cognitive disorders is an assessment of the deficit, first clinically, then with appropriate neuropsychologic tests that provide a quantitative score. A battery of psychometric tests must be selected considering the opportunity to compare single items score at different ages, to verify the cognitive outcome, to study the relationship between tests and specific brain areas and to verify patient's higher order executive processes. We must define the possible behavioral troubles, family and social relationships functioning too. Mood, emotional and relational states are assessed by standardized tests, free game observation and clinical interview.

Rehabilitation Planning

The assessment is followed by the identification of main rehabilitative goals and than the young patients begin the training in different rehabilitative sectors. The rehabilitation of patients in subacute phase doesn't appear so different in developmental and adult ages. Nevertheless we believe that it is fundamental that one of the parents, usually the mother, stays with the child. All members of team, therapists, nurses and teachers must have a long experience of work with children. Moreover the rooms, the rehabilitative areas and the different parts of the building must be furnished in an appropriate way for children. Initially the child is treated in bed but, when the basic clinical features are stabilized, the patient is carried out of room and the rehabilitation continues in the specific treatment rooms. We must pay attention with treatments and stimulation to not overload the child, because usually his/her attention and information processing abilities are reduced in their span and time duration. Therefore we must treat him more often every day for short periods. In the successive phases, when the patient’s abilities are recovering, main aim of rehabilitation techniques is the achievement of the maximum possible recovery, compatible with lesions severity, and of a general well-being to enable the best possible re-entry into school and social environment. Between rehabilitative treatments the child can be put in small groups of peers with similar disability level. The groups are managed by an expert in education, under the supervision of a psychologist with behavioral-cognitive competencies. The insertion in this stimulating situation facilitates the recovery, the creative and integrated use of functions, reduce behavioral and relationships troubles and increase self-esteem of child. Even after less serious injuries family support and counseling, as well as medical care, become very important. The team must address the changing needs of the child throughout the period of rehabilitation, and this can only be achieved if the individual team members can communicate and work effectively as a unit. After the initial intensive rehabilitative treatment the children are discharged and they re-enter into community and schools. The reintroduction to school should be well planned, with whatever ancillary support if necessary. Usually, all patients need a follow-up for all developmental period, with periodic re-assessment of clinical features and outcome and up-dating of rehabilitative planning. Different intensive rehabilitative periods are planned if the team members, after the follow-up, believe that they could be useful. The same rehabilitative periods are organized if the services near the child’s home cannot provide all facilities. In conclusion, the problems of TBI children change over time and clinical phases. In ICU the patient needs of medical care, than rehabilitation takes the first place. But, over years, the post traumatic patient mainly needs of an existential project. The management of head injured patients requires a coordinate and multidisciplinary team work.

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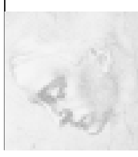

POST-TRAUMATIC VISUAL IMPAIRMENTS IN CHILDREN

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Introduction

Traumatic brain injury (TBI) is the main cause of mortality and disability in developmental age. Visual disorders secondary to a cranio-encephalic trauma are common, often multiple, of varying severity, they are associated with complex clinical pictures and are often difficult to diagnose. Given the complex distribution of the visual system in the CNS, it is very likely that a brain injury can damage the visual system in one or more of its components. While the main characteristics of visual disorders secondary to head trauma in adults have been reported in the literature, research on children is scarce. In adults, the most frequent post-traumatic visual disorders are strabismus (especially exotropia), convergence deficit, accommodation dysfunction, absence of stereopsis, reduction of visus, visual field impairments and fundus alterations. The present investigation focused on neuro-ophalmological alterations in a group of brain-injured children, investigating possible age-trauma correlations, clinical parameters in the acute phase, neuroimaging data and outcome, with the aim to identify possible risk factors responsible for the development of visual deficits and their effects on rehabilitation of these patients.

Materials and methods

Fifty-six post-traumatic children were investigated, all under 12 years of age, who suffered a closed head trauma at least 6 months earlier, with a score of 6 on the GCS motor scale. They had been referred to our Institute for clinical assessment and rehabilitation. We collected clinical data about pre-trauma visual skills, age at trauma onset, etiology and severity of trauma (Glasgow Coma Scale) as well as possible cranial fractures, endocranial hypertension, sites of the cerebral lesions at the neuroradiological investigations (CT scan in acute phase, MR at follow-up) and duration of coma. The outcome was scored on the Glasgow Outcome Scale (G.O.S.). 0All the patients received a complete neuro-ophthalmological assessment protocol. All data collected were statistically analyzed.

Results

Thirty-seven subjects out of 56 were males (66%) and 19 females. None of the subjects had presented a significant visual impairment before trauma. Mean age of the patients at trauma onset was 6.8±3.3 (range between 0.5 and 12.1). The average time lapse between injury and examination was 11.8 months (median: 3.2; range: 0.4-107.6). Mean G.C.S. score in the acute phase was 6.1±2.5. Mean duration of loss of consciousness was 4.4± 4.2 weeks. In the acute phase twenty-six patients presented a cranial fracture. In 19 children endocranial hypertension persisted for more than 4 days. The neurological examination was normal in 4 patients, 34 patients displayed hemiparesis, 8 patients exhibited diparesis and 10 patients presented tetraparesis. The most frequent sites of cerebral lesions at the neuroradiological investigation, both in the acute phase and at follow-up at least six months after trauma, were the frontal areas, followed by temporal and parietal areas. Focal atrophy was present in 67.9% of the cases; cortical and/or subcortical atrophy were less frequent. About 37% of the patients had a visus ranging between 14/20 and 2/20, 18% had a visus lower than 2/20. As expected, the GCS score was lower in children with visus <2/20. Duration of coma was longer in the group with visus <2/20. Ocular fundus examination highlighted atrophy in 5 patients and partial atrophy in 7 patients; the VEP investigation confirmed the presence of total or partial optic nerve atrophy in 83.5% of the patients. Fundus alterations appeared to be correlated with duration of coma. The visual field examination could only be made on 51 children as it is difficult to perform it with very young children and more severely impaired patients. 44.6% reported visual field deficits. Duration of coma was greater in the group with altered visual field. Both temporal and occipital lesions were associated with visual field deficits. 42.9% of children suffered strabismus: exotropia in 10 cases (17.9%) and esotropia in 14 cases (25%). Among the acute phase parameters only duration of coma was different in children with strabismus (4 weeks) as compared to children without strabismus (2 weeks). Examination of convergence highlighted an alteration in 28 patients (50%); 12 patients (21.4%) displayed nystagmus. Stereopsis could be assessed in only 37 cases owing to the difficulty to test it in very young children and in severe patients, and was altered in 8 patients (21.6%). Stereopsis alterations were correlated with the presence of occipital lesions. Nystagmus was present in 19 children exhibiting an average coma duration of 5.5 weeks, whereas in the group of subjects without nystagmus coma lasted 2 weeks. The assessment of eye movements showed fixation abnormalities in 41.1% of the cases; the same patients also exhibited saccadic and pursuit movement abnormalities, and duration of coma was longer. The average GOS score was 2.4. There was no significant correlation either between GOS score and age at trauma or GOS score and the months between the trauma and the assessment. GOS score was correlated with visus, fundus, visual field, convergence and fixation. We divided our cases in two groups: children under 4 years of age and over 4 years and analysed the occurrence of neuro-ophthalmological disorders in both groups. There were no differences in the average GCS scores between the two age groups at trauma. A greater frequency of visus, fundus and oculo-motor alterations was present in the younger age group, whereas a convergence deficit prevailed in the older age group. None of the differences between the two age groups was statistically significant.

Discussion

Data reported in the literature with regard to adults indicate a more than 50% incidence of visual disorders in traumatic brain injury. Our study confirms that also in developmental age there is a high percentage of visual disorders, of their complexity and variability. Our patients had suffered a severe cranial trauma. In this population, non-refractive visus reduction is the most frequently detected deficit (55.4% with visus <14/20). It is associated with coma severity, its duration and with severity of the neurological state. The same number of children with altered visus presented frontal lobe lesions. The greater visus impairment was associated with total or partial optic nerve atrophy. The statistical analysis showed that patients with optic nerve lesions exhibited the most severe neurological conditions, with the lowest GCS scores and the longest duration of coma. Visual reduction was significantly associated with strabismus and deficits in fixation, pursuit and saccadic movements. It must be noted that in our cases these oculo-motor deficits are closely correlated, probably owing to the employment of a common subsystem. Furthermore, the statistical analysis showed that they were associated with duration of coma, whereas there were no significant correlations with other clinical parameters in the acute phase. A convergence oculo-motor deficit is present in 50% of our cases, which is higher than in adults. The analysis - in the acute phase - of the clinical parameters related to the development of visual deficits highlights that duration of coma is the main risk factor. In the framework of a multidisciplinary clinical assessment of brain-injured children, early and accurate diagnosis and treatment of visual disorders appear to be of fundamental importance, also for correct rehabilitative planning.

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OUTCOME AND RE-ENTRY INTO SOCIETY

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Objectives: What is the natural course and long-term outcome in adolescents/young adults who had serious TBI (concussion excluded) in childhood or adolescense? Are there early identifiable prognostic factors?

Series: In the years 1987-1991 altogether 165 survivors (mean annual incidence 12/100.000) of traumatic brain injury (TBI) in the agegroup 0-17 years in the western health care region (pop 1.75 mill) of Sweden were identified. At the time of the injury their mean age was 9.35 (SD 4.96) years. The traceable individuals (132) were invited to a follow-up investigation 10 years after injury.

Methods: The entire investigation consisted of:
- a mailed questionnaire regarding 21 post-concussion symptoms, education, employment and general living conditions
- a quality of life assessment instrument (D15)
- clinical neurological examination
- a detailed motor and sensory function assessment (EB-test)
- a neuropsychological test battery (WISC-III, WAIS-R, Rey Complex Figure Test, Rey Auditory Verbal Learning Test, D2-attention test, finger tapping)
- Child Behavioral Check List (CBCL) for children, parents and teachers

Results: The questionnaire and the D15 QoL instrument were returned by 86 (65 per cent) of the traceable individuals. In this group the mean duration of unconsciuosness of those 53 who had been unconscious was 4.2 days (SD 6.8 days); 95% CI 2.3-6.1. The corresponding figure for GCS was 7.97 (SD 4.92); 95% CI 6.9-9.0 and for GOS at discharge from acute care was 4.86 (SD 0.35); 95% CI 4.79-4.94.These figures did not differ significantly from the corresponding ones for the entire group of 165 injured individuals surviving the initial phase.
At the time of the follow-up study in mean 10 years after the injury 16 individuals attended compulsory school, which was appriopriate for their age. Among these, two individuals were in special education groups whereas another five had additional support in their ordinary classes. There were 13 high-school students, of which two

had additional support. Among those 20 who continued their education after high-school there were only two studying at university. The other 18 individuals were continuing their education at trade schools or completing their high-school grades. Gainfully employed were 36, of which one was in a sheltered workplace. In the entire group one single person was unemployed.

The reporting of subjective complaints revealed eleven individuals without any such complaints. There were twelve with one complaint, 21 with 2-4 complaints and 42 with five or more complaints. The most frequently reported complaints were problems of concentration, headache, mental lability and visual problems. All these were present in 40 per cent of the individuals.

The health related 15 dimension QoL survey identified 22 individuals (26 per cent) perceiving their QoL as entirely uncompromized. Visual problems were the most common ones, as being reported in 34 per cent. Almost equally common was anxiety (32 per cent), followed by depression (29 per cent) and medical complaints (27 per cent).

The group of 86 was subcategorized so that those who were working full-time and did not receive any additional support while studying formed the first subgroup numbering 72 persons. The second group of 12 persons was composed of those on a partial sick-leave and/or requiring additional support while studying. The third group consisted of the remaining two persons of which one was unemployed and the other one was on a prolonged sick-leave. In mean 3.96 subjective complaints were reported in the first group as compared to 9.8 in the second group and 12.5 in the third group. The QoL revealed in mean 1.49 subnormal scorings in group one, 6.5 in group two and 10.5 in group three.

The mean GCS in the first group was 8.36 (SD 4.93) as compared to 5.25(SD 4.21) in the second group and 3.0 in the third group. These differences were not significant. Correspondingly, the duration of unconsciousness was distributed so that the first group had 2.49 (SD 3.26) days and the second one 10.83 (SD 11.43), $p < 0.001$ (Anova). GOS at discharge from acute care was distributed in the groups so that the first group 4.98 (SD 0.25), and the second group 4.17 (SD 0.52).

Concerning rehabilitation after the acute stage it was found that in the first group 3 out of 72 had received rehabilitation whereas 1/12 and 1/2 respectively in the second and the third group had received such support.

Conclusions: Although 35 per cent of the series were lost from follow-up it is apparent that the participants were representative for the original study population which was collected population based. The follow-up results therefore can be judged as representative for this cohort representing serious TBI (concussion excluded).

On a general level these individuals appear to be relatively well adjusted in society, as there were only two without a regular daily activity. The fact that there were only two

university students arises a suspicion that their ability to conduct higher level education has been seriously impaired, as a much higher proportion of university students would have been expected. Another concern is the high level of reported complaints representing typical post-TBI symptoms. Interestingly, the questionnaire data was verified by the independent QoL instrument.
It seems mandatory to follow this group systemtically also in the future, as the pattern of reported complaints clearly constitutes a major risk for early failure to cope with the demands of work.

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SPEECH AND LANGUAGE DISORDERS IN CHILDREN AND ADOLESCENTS WITH TBI

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Traumatic Brain Injury (TBI) is the most frequent cause of acquired speech and language disorders in children and adolescents. Unfortunately, prognosis *quod vitam* of these disorders is often still underestimated by practitioners as well as many neurologists and pediatricians. In contrast, communication and language disorders in developmental age which are caused by TBI severely impair the neuropsychological development of the individuals and pose limits to their communication skills in a period when a high level of socialization is required, thus markedly impairing their school performance. Even if they are less evident than sensorimotor deficits, the effects of communication and language disorders on the children’s psychological, relational and school development are equally disabling and therefore deserve careful clinical consideration in diagnosis, rehabilitation and formulation of life prognosis.

The best known acquired language deficits following TBI are aphasias. From the 2nd year of life lesions to the language-dominant hemisphere (generally, the left cerebral hemisphere) cause aphasias. In children and adolescents all types of aphasia were described (Broca’s aphasia, global aphasia, transcortical motor and sensory aphasia; Wernicke’s aphasia; conduction aphasia). In children with TBI vs. adults with TBI, however, non-fluent aphasias are more frequent. In addition, there is generally a better language recovery in children and adolescents with TBI vs. adults with TBI. With regard to aphasia caused by TBI in children and adolescents some important aspects need to be stressed: 1) Patient are to receive a systematic assessment of their residual language skills with batteries of standardized tests. This assessment must be repeated over time to monitor language recovery and the effects of rehabilitation of language functions. 2) Language deficits must be correlated as far as possible with the site of the cerebral lesion. 3) With regard to lesions to the language areas, neurolinguistic studies suggest that there will <u>never</u> be a full language recovery, and subtle language deficits - which seem to have resolved at routine measures - cause severe learning and academic difficulties.

As already stated, one of the most relevant aspects, from a neuropsychological point of view, is the correlation of communication and language deficits with the site of the lesion. It is known that right hemisphere lesions occurring during the first year of life cause language disorders which are more relevant than those brought about by left hemisphere lesions (Locke 1999). It is also known that lesions to subcortical structures such as the thalamus (Left), the basal ganglia (Left) and the cerebellum (Right cerebellar hemisphere) not only determine articulation disorders (dysarthrias) but also language disorders (Fabbro 1997; 2000). A verbal communication deficit which is often found following TBI is mutism. This disorder can be either transient (weeks, months) or permanent. It can be associated with aphasia (global aphasia, non-fluent aphasias) or can occur in individuals with relatively preserved comprehension and intact reading and writing skills. Mutism and/or a reduction in verbal initiative can depend on lesions to the anterior cingulate cortex, lesions to some structures of the basal ganglia (transient mutism, cf. Von Jürgens and Von Carmon 1982) and midbrain periaqueductal gray lesions (cf. Esposito et al. 1999).

As brain injuries determine multiple lesions to the encephalon and very often bilateral lesions of the frontal lobe, very often children and adolescents show pragmatic disorders of verbal communication. Pragmatics is concerned with how speakers use language to effect successful communication. In children and adolescents with TBI impaired comprehension and production of metaphors, anaphors, jokes and speech acts were reported. Recently, in bilingual subjects with TBI violations in language choice were observed, which is a typical pragmatic disorder (Fabbro et al. 2000). Detection and rehabilitation of pragmatic disorders in children and adolescents with TBI are important clinical targets as these communication aspects play a crucial role in the psychosocial development of these patients (Turkstra 2000).

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FRONTAL LOBE DAMAGE IN CHILDREN

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Objectives:
1. Review cases of frontal lobe damage in childhood.
2. Describe assessment approaches unique to this group.
3. Discuss treatment planning and intervention approaches.

The prefrontal cortex matures slowly throughout development which continues into adulthood. It comprises nearly 30% of all cortex and is thought to provide important neural mediation of higher level executive functions such as planning and completing goals over long periods of time. In addition, prefrontal cortex subserves critical processes of social behavior and emotional maturity, including empathy, theory of mind, and continuing psychological adjustment.

Prefrontal cortex damage in children may be caused by localized disease processes and by more diffuse diseases such as traumatic brain injury. The reported cases with localized prefrontal damage only are still quite few with longitudinal follow-up studies ongoing. Recent reports have differentiated between a right dorsolateral prefrontal syndrome and a polar, mesial and orbital prefrontal syndrome in childhood cases. Early right dorsolateral prefrontal damage is characterized by prominent spatial and attentional impairments in the executive domain (i.e., working memory, planning, problem-solving, divided attention). These impairments improve over time, can be aided by medication and instruction, and children may compensate well by late adolescence. By comparison, early damage to polar, mesial and orbital prefrontal cortex is associated with profound social and emotional impairments that become more problematic in adolescence and adulthood. Those impairments are more difficult to remediate and manage, and may lead to disability in adult independent functioning. Cognitive functioning is generally adequate but self-regulation, achievements and social interactions are maladaptive. Models are beginning to emerge that describe these developmental processes and their alterations with prefrontal cortical damage. Interventions are possible but still limited at this time.

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NEUROPSYCHOLOGICAL RECOVERY IN CHILDREN

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Injury to the developing brain has long-standing consequences. The authors will utilize data and case material from their experiences with the Acute Brain Injury Program at the Kluge Children's Rehabilitation Center, an acute rehabilitation program for children (and families) from a rural region. Topics will be: the effects of injury on the developing brain; medical and rehabilitation management; family-centered care; and functional outcomes.

The paper will review the effects of injury on the developing brain. The timing, mechanism and location of injuries will be discussed as relevant information in understanding the child's new developmental trajectory.

The achievement of both physiological homeostasis and a progressive neurocognitive intervention (elementary to complex skills) are essential for the restoration of the child's age appropriate daily routine/activities of daily living.

Family is primary to the well being of the child/adolescent with acute brain injury. The concept of family-centered care will be discussed both as a critical element for successful community re-entry and child outcomes, but also as a challenge to the health care providers. Experiences with the utilization of non-traditional therapies by inpatient families will be explored.

Functional outcome data at inpatient discharge and community follow-up will be presented. Return to the home community and school participation will be evaluated as markers of rehabilitation effectiveness. We will address the need to re-instate the child upon a developmental trajectory and to measure quality of life as outcome measures for rehabilitation and recovery.

SCHOOL REINTEGRATION FOR YOUTH WITH ABI: REVIEW OF ISSUES AND RECOMMENDATIONS FOR CHANGE

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Objectives:
1. Describe the relationship between behaviors after TBI and learning difficulties.
2. Outline suggestions for research priorities as complied by several special interest groups in the USA.
3. Propose methods for developing teacher trainings.
4. Describe systems change that can make a difference in the education of youth.

Reintegration to school after experiencing a TBI continues to be a challenging situation for many youth and for the professionals who serve them. We have considerable anecdotal information about the successes and challenges of school reintegration. There are numerous models in existence that suggest how this re-entry to school should occur. Recently, there has been a proliferation of texts, articles, and materials developed that contain many ideas about how education for youth should occur. However, there has been no identification of priorities for research, education of professionals, or teaching of students after TBI to determine what really works for this population.

This session will emphasize the following:

1. The transitional challenges facing youth after sustaining a TBI.
2. The problems that affect learning after TBI.
3. The conclusions regarding school reintegration that were identified by several special task forces who addressed issues affecting children and adolescents after TBI. These issues include finding alternative methods for training teachers about TBI; mandating professional training for all professionals who serve this population; developing technical assistance programs to provide instructional assistance, and disseminating training information that presently exist in a more efficient manner.
4. The themes regarding educating youth after TBI developed by a concensus paper of 10 experts working in the field. These themes include the need for both pre-service and in-service training for teachers and documentation of teaching strategies that work. Priorities for research in reintegration areas will be outlined.
5. The recommendation for systems change. If we are to develop plans for school reintegration that meet the unique needs of this population and demonstrate that they work, then systems that already exist must be involved in the development of

alternative plans for this population. Suggestions for how to affect systems change will be made.

Handouts outlining the above suggestions will be provided and audience discussion regarding ideas for change will be encouraged

Recommended Readings:

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PSYCHIATRIC AND BEHAVIORAL DISORDERS AFTER SERIOUS HEAD INJURY IN DEVELOPMENTAL AGE

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Objectives:

1. Outline the behavioral components of brain injury and possible therapeutic approaches.
2. Interpret the limits of published findings regarding psychiatric outcome with an emphasis of study design.

This presentation reviews psychiatric outcome in children and adolescents who have suffered traumatic brain injury (TBI). The studies reviewed consist of prospective and retrospective studies that have used standardized psychiatric interviews to elicit diagnoses.

New psychopathology recorded approximately two years following severe TBI has been noted in 54% to 63% of children, and following mild/moderate TBI in 10% to 21% of children, and following orthopedic injury in 4% to 14% of children. Predictors of "novel" psychiatric disorders include severity of injury, pre-injury psychiatric disorders, pre-injury family function, family psychiatric history, socioeconomic status/preinjury intellectual function, and pre-injury adaptive function. The most common "novel" disorder after severe TBI is Personality change due to head injury (PC) or its approximations in other diagnostic nomenclatures.

The Neuropsychiatric Rating Schedule (NPRS) can be used to establish a diagnosis of PC. Approximately 40% of consecutively hospitalized severe TBI subjects had ongoing persistent PC an average of two years post-injury. An additional approximately 20% had a history of a remitted and more transient PC. PC occurred in 5% of mild/moderate TBI but was always transient. The labile, aggressive, and disinhibited subtypes are common whereas the apathetic and paranoid subtypes are uncommon.

In severe TBI subjects, persistent PC was significantly associated with severity of injury, particularly impaired consciousness > 100 hours, adaptive and intellectual functioning decrements, and concurrent diagnosis of secondary attention deficit hyperactivity disorder, but was not significantly related to any psychosocial adversity variables.

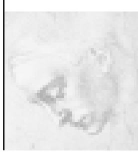
Consecutively admitted children with severe TBI who developed ADHD secondary to the TBI (S-ADHD) were significantly different to the severe TBI children who did not develop S-ADHD in terms of adaptive and intellectual function. However, they were not significantly different in terms of injury severity (within the constricted range of severe TBI), family function at the time of assessment, socioeconomic status, family stressors, family psychiatric history, gender, or lesion area. When the full range severity from mild to severe TBI is included there is a significant association with severity of injury. An overlapping study of attention deficit hyperactivity (ADH) symptoms found a similar relationship with severity and that overall ADH symptoms were associated with poorer preinjury family functioning. A referred sample of children dominated by severe TBI children had similar findings and the S-ADHD children had greater "premorbid" psychosocial adversity. S-ADHD may be associated statistically with lesions of the right putamen or thalamic lesions.

Oppositional defiant disorder (ODD) symptomatology in the first year after TBI was related to preinjury family function, social class, and preinjury ODD symptomatology. Increased severity of TBI predicted ODD symptomatology two years after injury. Change (from before TBI) in ODD symptomatology at 6, 12, and 24 months after TBI was influenced by socioeconomic status. Only at two years after injury was severity of injury a predictor of change in ODD symptomatology. The influence of psychosocial factors appears greater than severity of injury in accounting for ODD symptomatology and change in such symptomatology in the first but not the second year after TBI in children and adolescents. This appears related to persistence of new ODD symptomatology after more serious TBI. Findings from a referred brain injury clinic sample were that subjects who developed ODD/Conduct Disorder following TBI, when compared to subjects without a lifetime history of the disorder, had significantly more impaired family functioning, showed a trend toward a greater family history of alcohol dependence/abuse and suffered a milder TBI.

Only 2/46 (4%) subjects with at least one follow-up assessment developed PTSD. However, the frequency with which subjects experienced at least one PTSD symptom ranged from 68% in the first three months to 12% at two years in assessed children. The presence of an internalizing disorder at time of injury, followed by greater injury severity were the most consistent predictors of PTSD symptomatology. It is apparent that PTSD and subsyndromal posttraumatic stress disturbances occur despite neurogenic amnesia.

Four of 50 subjects (8%) from a prospective study of consecutive children hospitalized following traumatic brain injury developed mania or hypomania. The phenomenology regarding the overlapping diagnoses of mania, attention-deficit hyperactivity disorder, organic personality syndrome and the "frontal lobe syndrome" are important considerations in differential diagnosis. Severity of injury, lesion location, and family history of major mood disorder may be implicated in the etiology of secondary mania. Lengthy episodes and similar frequency of irritability and elation may be characteristic.

Findings are sensitive to referral biases and clinicians and legal professionals should beware. Speciality brain injury clinic consecutive subjects have occurrence of "novel" psychiatric disorder as high as 76%. "Novel" psychiatric disorder was correlated significantly with family psychiatric history and family function, but not with severity of injury, preinjury psychiatric status, intellectual/educational functioning, or socioeconomic status.



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MILD CLOSED HEAD INJURY IN CHILDREN AND ADOLESCENTS: INTERNATIONAL RESEARCH

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4. ***D. Fong***, ISHIP from Hong Kong, China

Objectives:

1. The main objetive of our multicenter study is to evaluate different clinical predictors and the value of CT scanning in a pediatric age with Mild Closed Head Injury

Methodology: The ISHIP group includes children between 0 and 12 years of age with history of head injury. All patients examined includes with Mild Head Injuries: Loss of Consciousness, amnesia for the event, and neurological Score (GCS or PGCS) 13-15.The management of procedure included a routine early CTscan within 6 hours after injury at the emergency department and discharge after normal CTscan. Admission only at the following aspects: Abnormal CT, fracture with high risk site, social situation, (active issues), clinical deterioration. Finally all patients were examined at the follow-up 3 months after injury. We present our first two years with 4,391 patients evaluated in 15 countries participated to the ISHIP group.

Results: of the total 4,391 patients enrolled in the study, 61,7% were boys and 38,3% girls. The age distribution was a follows: 41.4% 0-2 years; 31.3% 3-6 years; 14% 7-9 and 13.3% 10-12 years. Most (99,8%) of these patients completed the follow-up. LOC was registered in 22,7% and PTA 11%. The total sample included 1,392 children (31.7%) who required hospitalization. Skull fracture were identified in 14.4% of cases (n. 541 of the 3,759), and 2,266 CTscans wereevaluated (51.6% of the total sample) and 29.4% (n. 666) were abnormal. Of the total sample 119 patients (2.6%) suffered neurosurgical intervention, with a mortality rate of 0.07%.

Conclusions: At the moment we concluded that the physical and neurologic examination are inadequate predictors of intracranial injury, and the CT scan is more sensitive than physical and neurologic examinations for diagnosis of ICI. Liberal use of CT scans in children 6 years of age and younger with HI is because they may present without symptoms. (1-2)

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THE IMPACT OF BRAIN INJURY WITHIN THE FAMILY

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Objectives:
1. To evaluate the relationship between family resources and behavioural functioning in children up to 2 years following TBI.
2. To identify early predictorscof 3-month, 1-year and 2-year behavioural outcome for children following TBI.
3. To identify concurrent indicators (family and injury indices) of behavioural outcome in children at 1 and 2-year following TBI.

The present study investigated the interaction between family resources and behavioural functioning in a sample of children up to 2 years following traumatic brain injury (TBI). The study considered (a) early prediction of 3-month, 1-year, and 2-year behavioural outcome for the child, and (b) concurrent indicators of behavioural outcome. Behavioural outcome was assessed by postinjury change in a measure of behavioural problems as reported by parents. Measures used to predict this behavioural outcome were selected from injury severity, preinjury family resources, and early postinjury family environment. Similar measures taken at 1 and 2 years postinjury were evaluated as potential indicators of ongoing behaviorual problems for the child.

In our sample of 51 children post-TBI, we found that children with severe injuries, but not those with mild or moderate injuries, were reported by parents to have a greater incidence of behaviour problems following TBI than prior to injury. Regression analyses indicated that severity of injury was a major determinant of 2-year behavioural outcome, but the preinjury family structure and early emotional response of the parent were both predictive of child behavioural outcome at 3-month and 1-year follow-up. Furthermore, by 2-year follow-up concurrent assessment of family environment was found to be related to behavioural dysfunction of the child.

COGNITIVE THERAPY WITH IMPAIRED SELF AWARENESS FOLLOWING ACQUIRED BRAIN INJURY

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Many brain injured people who require post-acute social rehabilitation present changes in personality which alter their behaviour and affect their capacity for meaningful social relationships. This is particularly evident after traumatic brain injury because of the vulnerability of the frontal lobes and their role in self-awareness (Stuss and Benson1986). Whilst usually only too apparent to relatives and friends, these changes often appear to remain beyond the client's own awareness (Oddy et al 1985. Prigatano and Altman 1990). With clients not acknowledging there is a problem they understandably see no need to alter their behaviour or their perception of life. Thus attempts at engagement in rehabilitation often lead to confrontation, resistance and rejection. This paper discusses how cognitive therapy can help clients with impaired awareness engage in the rehabilitation process, and also how it may facilitate enduring behaviour change. Case examples are presented.

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POST-TRAUMATIC EPILEPSY: EVIDENCE-BASED APPROACHES

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Every person unfortunate enough to develop epilepsy is likely to recall a blow to the head and question whether this may be the cause of their epilepsy. Epidemiological studies, however, would not suggest that brain injury contributes greatly to the overall burden of epilepsy, head trauma being identified as the cause of seizures in 3% of patients identified in the National General Practitioners Survey of Epilepsy[1]. Even the application of sophisticated MR imaging to patients presenting with their first seizures would not suggest that these figures are an under-estimate[2].

Seizures and epilepsy are an important issue for the management of traumatic brain injury. For no other cause of epilepsy are risk factors for the development of seizures and epilepsy so well defined and so clearly predictable. A recent large community survey of a head-injured population[3] has once again emphasised that the risk for both early post-traumatic seizures and late post-traumatic epilepsy can be determined by an assessment of the severity of brain injury, supporting the earlier work undertaken by Jennett[4]. Annegers et al. have shown that, while mild (loss of consciousness or amnesia of less than 30 minutes) and moderate head injuries (loss of consciousness or amnesia of between 30 minutes and 24 hours or skull fracture) may carry a small increased risk for the development of epilepsy in subsequent years, more severe head injuries (loss of consciousness or amnesia in excess of 24hrs, subdural haematoma or brain contusion) are accompanied by a considerably increased risk (standardised incidence ratio 17.0 (95% CI 12.3,23.6) for the development of epilepsy that extends for over ten years following an injury. Within the neurosurgical population studied by Jennett, three factors had the greatest influence on the risk of post-traumatic epilepsy. These were the presence of a compound depressed fracture, the presence of intracranial haemorrhage and the occurrence of early (within the first post-traumatic week) acute symptomatic seizures. In those few subjects with all three risk factors, the likelihood of post-traumatic epilepsy could be as high as 50-80%.

The significance of early seizures has been further elucidated. Jennett recognised that seizures occurring immediately on impact do not seem to carry any subsequent excess risk of epilepsy and indeed characterise relatively mild concussive injuries. McCrory et al[5] studied such immediate "concussive convulsions" in Australian sportsmen and came to the same conclusions. They questioned whether these events are seizures rather than a form of acute temporary decerebration. Annegers et al. have further clarified the importance of early, as opposed to immediate, seizures in predicting late post-traumatic epilepsy by showing, in multivariate analyses, that early seizures have no independent effect on the risk of late post-traumatic epilepsy, but merely act as a marker of injuries of sufficient severity to cause late epilepsy.

If patients at high risk of both early (acute symptomatic) seizures and late post-traumatic epilepsy can be identified, the question then arises as to whether there are preventive measures that could be taken to limit seizure occurrence and thereby benefit patient outcomes. This brings into focus the potential for using drugs that might independently have anti-seizure or antiepileptogenic effects or conceivably have both effects. These issues have been most completely explored in the kindling model of epilepsy[6]. This model shows that there may be a dichotomy between drugs which can prevent the occurrence of seizures in fully kindled animals and drugs that are truly antiepileptogenic, and prevent the development of kindling. In more practical terms, what is the evidence that antiepileptic drugs may:

- Reduce early seizures?
- By reducing early seizures, reduce morbidity and mortality of head injury?
- Reduce the incidence of late post-traumatic epilepsy?

The systematic review available on the Cochrane Library[7] identified 10 Randomized controlled trials (RCTs) including data from 2036 patients. Four unpublished studies with another 630 patients could not be included. There was consistent evidence that the early treatment with antiepileptic drugs (AEDs), (most commonly phenytoin, but also carbamazepine and phenobarbitone) resulted in a relative risk for early seizures of 0.34 (95% CI 0.21-0.54). Treatment of 100 patients would result in 10 patients, who would otherwise have had seizures, avoiding them during the first week. However, the studies did not provide evidence that this benefit was accompanied by any reduction in mortality, though the confidence intervals were quite wide (RR 1.15, 95% CI 0.89,1.51). There is even greater uncertainty from these studies concerning the effect of antiepileptic drug treatment on a combined outcome of death or significant neurological disability (RR 1.49, 95% CI 1.06-2.08 for carbamazepine and RR 0.96, 95% CI 0.72-1.26 for phenytoin). Furthermore, there is no evidence that a reduction in early seizures is accompanied by a reduction in late post-traumatic epilepsy (RR 1.28, 95% CI 0.90-1.81). The administration of antiepileptic drugs in the acute phase was also associated with an incidence of acute idiosyncratic skin reactions (RR 1.57, 95% CI 0.57-39.8).

Haltiner et al.[8] have undertaken further analyses on the largest and methodologically most sound of the RCTs included in this systematic review. They particularly explored the relationship between the effect of phenytoin (the AED in this study) and its effects on early seizure reduction and mortality. They found more deaths among patients who had early seizures than in those who remained seizure-free (RR 2.0, 95% CI 1.1-3.7), an observation explained by an association between early seizures and more severe head injuries (as judged by the presence of a non-reactive pupil, a more adverse abbreviated injury scale score for the head, the presence of subdural haematoma or intracerebral haematoma). The authors conclude that although phenytoin was effective in preventing some early seizures, and patients with early seizures had a higher death rate, the reduction of early seizures itself did not have a detectable impact on mortality.

Thus, current evidence form this intervention study and from community-based epidemiology, would indicate that early seizures act simply as a marker for more severe head injuries rather than an important part of a pathogenic process that leads from severe head injury to either late post-traumatic epilepsy or death.

Temkin et al.[9] have conducted a further well controlled study of valproate in the prevention of late post-traumatic epilepsy. They set out to compare the previous beneficial treatment, seven days of phenytoin, with one month's and six months' treatment with valproate. Valproate was chosen because of effectiveness in the prevention of kindling in animal models. The entry criteria were the same as those for the previous study comparing placebo and phenytoin[10]. These included the presence of a depressed skull fracture, penetrating brain injury, or CT evidence of cortical contusion, subdural, extra-dural or intracerebral haemorrhage. The primary analysis compared the incidence of late seizures in the two valproate treated groups with the phenytoin treated group. Slightly under 15% of patients had late seizures when treated with phenytoin compared to approximately 20% of patients receiving valproate treatment (RR 1.4 95% CI 0.8-2.4). Only two patients (1.5%) had early seizures in the phenytoin group compared with 11 (4.5%) in the valproate groups (relative risk 2.9, 95% CI 0.7-13.3). 379 patients entered the study although a further 113 patients, thought to be eligible at the time of randomisation, were subsequently found to be ineligible after randomisation, usually because of a previous history of seizures or previous brain injury that we unknown at the time of randomisation. Nine patients randomised to phenytoin died during follow-up compared with 32 patients randomised to valproate (RR 2.0 to 95% CI 0.9-4.1). The reasons for this increased mortality were not easy to discern either in terms of the severity of head injuries amongst the valproate-treated group or the identification of potentially serious drug adverse effects. The Data and Safety Management Board, for reasons that are not entirely clear, suggested stopping the study even though the planned sample size had almost been achieved. It may be of some interest that the overall mortality rates in each of the randomised groups was considerably lower than the 22% mortality rate observed in the previous study from the same centre comparing phenytoin and placebo[10].

Inevitably, some of the newer generation of antiepileptic drugs may be examined for the prevention of early post-traumatic seizures and late post-traumatic epilepsy, particularly those such as vigabatrin and topiramate that may exhibit antiepileptogenic properties in the kindling model of epilepsy. If clinicians are going to explore the potential benefits of prophylactic AED treatment after head injury, they will need to recognise the need for much larger studies than those currently available. While the Seattle group have contributed relatively large single centre studies, it is clear that multi-centre studies will be required in order to gather sensible information within a reasonable period of time. We might reasonably now ask whether clinical trials in this area have been addressing the right question! It may be more important to know whether prophylactic AED's reduce the prevalence of chronic intractable post-traumatic epilepsy, a question not addressed in any RCT's, than whether they prevent late post-traumatic seizures.

What then can we conclude from this data? There is reasonable evidence that immediate administration of AED's reduces the incidence of early seizures, and that phenytoin has the best evidence to support its use in this role. This effect will, however, carry with it a small risk of acute idiosyncratic reactions, as long as phenytoin is only used during the first post-traumatic week. There is no evidence that prevention of early seizures influences mortality, morbidity or the development of late post-traumatic epilepsy, although existing studies do not have the power to exclude clinically important benefits. Therefore there seems no justification for the routine use of AED's in patients with severe head injuries. The pathway from severe head injury to post-traumatic epilepsy seems to be determined at the time of the injury and cannot thereafter be diverted by the clinician.

- Severe brain injury carries a high risk for the development of epilepsy.
- Early (within the first week), but not immediate convulsions identify patients at risk for late post-traumatic epilepsy.
- Antiepileptic drugs, given in the first week post-trauma, reduce the incidence of early seizures, but there is no evidence that this reduces morbidity, mortality or late post-traumatic epilepsy.
- Neither individual clinical trials, nor a meta-analysis, exclude the possibility of clinically important benefits from preventive treatment with antiepileptic drugs on morbidity, mortality or late post-traumatic epilepsy.

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CHOOSING OUTCOME MEASURES TO PROMOTE CLINICAL EXCELLENCE AND RESEARCH FOR CHILDREN WITH TRAUMATIC BRAIN INJURY

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2. ***Michel Pépin***, Laval University, Québec City, Québec, Canada
3. ***Luc Noreau***, Centre for Interdisciplinary Research in Rehabilitation and Social Integration (CIRRIS), Québec City, Québec, Canada
4. ***Mario Cloutier***, Québec Rehabilitation Institute (IRDPQ), Québec City, Québec, Canada

Objectives:
1. To determine the main outcome measures to be documented in each clinical setting.
2. To derive the most relevant research questions to be addressed with these measures.
3. To implement a network system so that each centre will be able to provide the same measures to create a provincial database.
4. To test the validity of the data collection system.

The purpose of this presentation is to describe an ongoing project that has brought together clinicians, researchers and granting agencies at the “Institut de réadaptation en déficience physique de Québec” (IRDPQ) to promote optimal care and research for children with traumatic brain injury (TBI). The IRDPQ, the largest rehabilitation centre in the eastern part of the Province of Quebec and University Institute, is recognised by the Government of Quebec as an ultra-specialised tertiary care centre for persons with TBI. The clinical program for children cares for an average of 125 children (35 new cases per year) of TBI. Given the preponderance of brain injuries due to automobile accidents, the Sociéte d’Assurance Automobile du Québec (SAAQ), the Provincial Automobile Insurance Agency, has provided financial assistance for the development of a multidisciplinary treatment program. This approach involves a number of professionals including: psychologists, occupational therapists, physical therapists, social workers, physicians, etc. who evaluate the child at admittance into the program before commencing therapy and at specific endpoints and discharge thereafter. This approach dictates the application of a large number of outcome measures by each professional, some mandatory and others by choice. Because this system is relatively new, evaluations based on outcome measure comparisons is not yet systematic and therefore little has been completed on the evaluation of interventions. During the last three years, however, a research team from the CIRRIS Research Centre of the IRDPQ, in collaboration with other colleagues, has implemented a research project funded by the “Conseil québécois de la recherche sociale” in collaboration with the 28 rehabilitation centres in the Province of Quebec involved in the treatment of TBI. The main purposes of this project, known as “TCC-Québec” are 1) to determine the main outcome measures to be documented in each clinical setting;

2) to derive the most relevant research questions to be addressed with these measures; 3) to implement a network system so that each centre will be able to provide the same measures to create a provincial database and 4) to test the validity of the data collection system. This project responds to the need of a better follow up of patients which has been expressed by many authors (Brooks, D. N. & Truelle, J. L., 1994; Hall, K. M., 1997).

Conceptually, the project is based on the model of the Disability Creation Process (Fougeyrollas et al., 1998) which allowed the research team to determine outcome measures for all relevant domains of intervention (organic systems, capabilities, participation, environment). The project is now in the phase of testing data entry in eight pilot centres. The IRDPQ program for children is one of these test centres. Over the next year, the professionals will ensure that the outcome measures, chosen by a team of experts, are properly recorded. After being checked for quality, these data will then be integrated into the database. It is anticipated that this database will provide, in addition to demographic data, outcome measures pertinent to the evaluation of various therapeutic approaches (cognitive, physical, social) as well as social integration and quality of life of the children and families on a long-term basis. The creation of this Provincial Database should facilitate research on prevention, prognosis, rehabilitation approaches, technical aides, cost benefits, social integration and quality of life. Specific details on the treatment approach are presented by Cloutier and Beaupré and of the TCC-Québec project by Pépin et al, in separate poster and oral presentations at this conference.

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METHODOLOGICAL ISSUES IN ASSESSING OUTCOMES OF TRAUMATIC BRAIN INJURY (TBI) IN CHILDREN AND YOUTH

1. ***Jean A. Langlois***, ScD, MPH
2. ***Victor Coronado***, MD
3. ***Karen Gotsch***

Centers for Disease Control and Prevention, Atlanta, GA, USA

Objectives:
1. Review 2 proposed models for studying outcomes of TBI in children and youth.
2. Discuss the pros and cons of existing measures for studying outcomes in young people with TBI.
3. Review other methodological issues and the need for additional research to develop appropriate measures for studying outcomes of TBI in this population.

Advocates, scientists and policymakers agree that there is an urgent need for population-based data on outcomes of TBI in children and youth, from infancy through school-age. This presentation will summarize recommended methods for assessing outcomes of TBI in younger people. In October, 2000, the U.S. Centers for Disease Control and Prevention (CDC) convened a working group of 12 professionals for advice on the state of the art in assessing outcomes and the need for improved information about this population. The participants included advocates, policy-makers, researchers, and clinicians from the U.S. and New Zealand. The CDC presented 1) proposed models for assessing outcomes including factors influencing the receipt of services, 2) a summary of existing measures, and 3) a list of other methodological issues. From the comments and suggested revisions of the expert working group, recommendations for population-based assessment of outcomes in children and youth, as well as for other needed research, were documented. This presentation will provide useful information for researchers and others interested in assessing outcomes of TBI in young people.

INTRODUCING A NEW PEDIATRIC BRAIN INJURY TEST

1. ***Gillian Hotz***, PhD, University of Miami School of Medicine, Miami FL, USA
2. ***Nancy Helm-Estabrooks***, ScD, Boston University School of Medicine, Boston MA, USA
3. ***Nickola Wolf Nelson***, PhD, Western Michigan University, Kalamazoo, MI, USA

Objectives:
1. To introduce a new pediatric brain injury test.
2. To review how the PTBI was developed.
3. To review the specific subtests of the PTBI which were designed to quickly assess neurocognitive functioning across many domains and were chosen for their relevance to the general education curriculum.

The Pediatric Test of Traumatic Brain Injury (PTBI) is a cognitive linguistic screening test that assesses school-aged children in the acute and rehabilitation settings following traumatic brain injury. The test material was selected on the basis of clinical and experimental evidence that these patients demonstrate a wide range of cognitive, language and behavioral deficits first demonstrated in the early stages of recovery. Due to the rising costs of health care and changes in reimbursement patterns there is a great need for a short test that quickly assesses neurocognitive functioning across many behavioral domains. To date no single test has been specifically designed and standardized on children who have sustained a TBI. Out goal, therefore, is to standardize the PTBI so it can be used to establish baseline behaviors and track recovery of functions. Another important feature of the PTBI is that tasks are relevant to the general education curriculum. It will provide profiles of spared and impaired skills that are crucial for selecting and developing treatment programs and planning resumption of educational activities. In this presentation we will describe how the PTBI was developed, discuss the rationales for the specific areas of assessment and provide preliminary data from its use with brain injured children.

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MEASURING OUTCOME FOLLOWING MILD, MODERATE AND SEVERE BRAIN INJURY AMONGST CHILDREN

1. ***Carol A. Hawley***, Senior Research Fellow, University of Warwick, Coventry, UK
2. ***Anthony B. Ward***, Consultant in Rehabilitation Medicine, North Staffordshire Rehabilitation Centre Presenting, Stoke on Trent, UK
3. ***Andrew Magnay***, Consultant in Paediatric Intensive Care, City General Hospital, Stoke on Trent, UK
4. ***Bas Mychalkiw***, Consultant Clinical Neuropsychologist, City General Hospital, Stoke on Trent, UK

Objectives:
1. To identify meaningful measures of outcome following brain injury in children
2. To identify the difficulties associated with long-term follow-up studies of children following brain injury in terms of psychological, emotional, intellectual and cognitive functioning

In a study of all children admitted to hospital with a brain injury over a 6 year period, the usefulness and sensitivity of a number of performance measures was examined at 6 months to 6 years post injury and followed-up one year later. 97 children, aged 5 - 15 years when injured, and 31 control children, together with their parents were assessed. Injury severity was: 47 mild, 17 moderate, 29 severe, and 4 unknown. At follow-up 34% of the injured children were under 18 months post-injury. Assessments included the WISC III-R, Stroop Colour-Word Test, Children's Memory Scale, Wechsler Objective Reading Dimension, Vineland Maladaptive Behaviour Scale, Parenting Stress Index (PSI), Coopersmith Self Esteem Scale, Trail Making Test, and Problem Resolution Scale (PRS) (1,2).

46.4% of parents of brain injured children demonstrated clinically significant levels of stress on the PSI. Levels of stress were similar for all severities of injury and significantly greater (p=0.001) than shown by parents of control children. Similarly on the Difficult Child Domain 50% of mild, moderate and severely injured children scored above the 90th percentile, significantly different from control children (p=0.001).

On the Vineland Maladaptive Behaviour Scale the brain injured group demonstrated significantly more maladaptive behaviours than the controls. Repeat assessment of these measures at follow-up showed no improvement.

On the PRS at follow-up 67% of family-reported problems remained unresolved and 8% had worsened indicating the long standing nature of brain injury. The WISC's usefulness as an outcome measure was questioned as scores were largely unchanged at follow-up.

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PSYCHOLOGICAL AND EDUCATIONAL APPROACH OF THE SCHOOL REINTEGRATION AFTER PEDIATRIC TRAUMATIC BRAIN INJURY (TBI): ASSESSMENT OF AN EXPERIENCE OVER 5 YEARS FOLLOWING

1. ***S. Antonelli***, Academic Head-master, Education Nationale, Lamoralye, France
2. ***A. Boissel***, Psychologist, Association des Paralyses de France, St. Brice sous Forêt, France

After the phase of extensive rehabilitation in hospital, the question of the return in the common school circuit is wandered when T.B.I children are exempted from heavy functional sequelae and when they recover approximately their previous academic standard.

There is then a gap between the previews of the professionals (informed of long-term difficulties for the children with T.B.I) and families and children's hopes in a hurry to leave medical world.

How to follow up these children and these families?

How to work with the professionals of the common schools?

How to negotiate decisive following stages for the orientation and the future of the child?

Hypotheses

The success of the reintegration seems to us required with the implementation until the vocational guidance of children's and teacher's follow-up in common school background. This follow-up must be elaborated with the teaching team, the family and the child before the exit of this last one of the hospital departments of rehabilitation.

Methodology

15 reintegrated children or teenagers in the common school circuit.

Follow-up and analyzes results in 4 or 5 years of the accident

Results

Follow-ups implanted from the phase of rehabilitation allow to set up intervention strategies during the academic time without forget in the course of the years, T.B.I incidences.

In France, there are special means of the Education's Ministry for learning disabilities.

Although not specific for T.B.I, these structures can be sought with profit for children's reintegration

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ASSESSING THE LOSS OF INCOME EARNING CAPACITY IN CASES INVOLVING CHILDREN WITH A TRAUMATIC BRAIN INJRY

Giuseppe Battista, Murphy, Battista,Trial Lawyers Association of British Columbia, Canadian Bar Association, Amerian Trial Lawyers Association, American Bar Assocation, Vancouver, Canada

Objectives:

1. To help the various professionals involved in the care and treatment of a brain injured person understand their role in the process of advancing a loss of future income earning capacity claim.

When a child sustains a traumatic brain injury it is difficult to predict the long - term effects of the injury on his or her ability to earn an income. It is a challenging task for lawyers and the courts to prove and evaluate what that child would have achieved from a vocational perspective had he or she not been injured. The presentation will:

a) Discuss the difficulties in identifying and assessing the nature and extent of the deficits;
b) Discuss the role of the doctors and other professionals in assessing the impact of the injury on the individual's income earning ability, and;
c) Explore the factors that the courts consider in evaluating this type of loss and the approach that judges have taken in these cases.