BNPA abstracts

Proceedings of the Head Injury Conference and Annual Meeting of the British Neuropsychiatry Association, the Institute of Child Health, central London, 12–14 February 2003

Stepping out after brain injury

MANAGEMENT IN THE EMERGENCY DEPARTMENT: PREPARING PEOPLE FOR THE BEST

D. Yates. *University of Manchester*

Emergency departments have changed radically over the past 20 years, both in their mission and in the training and competence of their staff. They are now in a strong position to influence the initial experience of head injured patients and the longer term care of those who do not require neurosurgical intervention. This may not be very evident to those waiting for treatment or, sometimes, to other disciplines in the acute sector that have historically been responsible for inpatient care. A reflection perhaps of the variable integration of services between hospitals and their local communities and the inconsistent application of current knowledge. This is changing.

The management of the patient who has an evident and serious brain injury has been improved by the adoption of a "team approach" to priority assessment and investigation. This has been extended to include regional co-ordination in many centres but problems with critical care bed provision continue to hamper progress. There is good evidence that survival increased after a number of initiatives were introduced in prehospital and emergency department care in the late 1980s but that this progress has now plateaued.

The outcome after less serious injury has been less well documented. Indeed, until 20 years ago most emergency clinicians did not consider the so-called minor head injury to pose a significant medical challenge. Emergency departments do now recognise the problem, but often are unable to do much about it because the necessary support services are either absent or unknown to them. With so many patients presenting with apparently mild head injury and the knowledge that many get better almost despite treatment, it is clearly important to try to identify those who will benefit most from specialist follow up. Tests are required that can be applied quickly and simply in the emergency setting. Althoug I must leave others to discuss the merits of longer term care, it seems clear that the attitude of emergency nurses and doctors and the advice they give to patients who are being discharged must be important. The imminent publication of guidelines on the management of traumatic brain injury by the National Institute of Clinical Excellence (NICE) will accelerate the process of using good quality research data in routine clinical practice, particularly in the critical period of assessment and initial care. They will call for a major increase in the use of computed tomography as a diagnostic tool, largely replacing the triage role of plain radiographs. This should lead to fewer admissions—although this might turn out to be optimistic. There will be a call for closer integration within and between hospitals and closer involvement with community services. We can expect a demand for evidence of adherence to the NICE guidelines and, hopefully, an audit of their effectiveness in improving patient care. However, it is widely accepted that the guidelines, like many before them, rely excessively on consensus because of the paucity of good quality research. It is remarkable that this major public health problem has attracted so little major research funding. Perhaps the methodological success of the on-going CRASH trial (of the effect of a steroid after head injury), which is demonstrating that excellent follow up and outcome analysis can be achieved in head injury research, will act as a stimulus to others and to the research funding bodies to enter the head injury field.

MILD HEAD INJURY: PREDICTING WHO MAY NEED HELP

C. Boake. *University of Texas-Houston Medical School, USA*

Morbidity after mild traumatic brain injury (TBI) deserves attention because mild TBI occurs relatively often, accounting for most TBI hospitalisations and for many disability days. Methodological research problems and clinical referral patterns have fueled a longstanding controversy about the nature of disability after mild TBI. Anxiety and mood symptoms have generally been investigated as part of the post-concussional syndrome. Studies that specifically addressed depression after TBI have not agreed on prevalence and risk factors. The research project "Depression after Mild to Moderate TBI" (Harvey S. Levin, principal investigator), funded by the US Centers for Disease Control and Prevention, aimed to estimate the prevalence, risk factors, and effect on outcome of depression after mild to moderate TBI. 178 patients with non-penetrating TBI and 104 with extra-cranial trauma were prospectively recruited in the emergency centre and inpatient wards of a trauma centre using selection criteria of age > 15 years, lowest postresuscitation Glasgow Coma Scale (GCS) score > 8, blood alcohol level < 200 mg/dL, CT scan within 24 hours of injury (if TBI), and no major antecedent neuropsychiatric disorder. TBI and extra-cranial trauma patients were similar demographically. Outcome measures included a phone interview at one month to administer the Center for Epidemiologic Studies-Depression scale (CES-D) and evaluation at 3 and 6 months with the Structured Interview for DSM-IV (SCID) and neuropsychological and functional measures. The primary outcome of interest was major depressive episode (MDE) at 3 and 6 months post-injury as measured by the SCID.

Prevalence of MDE at 3 months was higher in patients with TBI than extra-cranial trauma. MDE was strongly associated with poorer functional outcome as measured by the Glasgow Outcome Scale and SF-36, and with poorer neuropsychological performance. Prevalence of MDE decreased from 3 to 6 months post-injury. In a logistic regression model predicting 3 month MDE from data available at one month post-injury, the best predictor was the one month CES-D score. Risk of depression is significant and increased after mild–moderate TBI and may be predictable soon after injury. These results are the basis of a new research project entitled "Community Based Case Management of Mild to Moderate TBI" (PI: H. S. Levin) that aims to validate a procedure for early identification of TBI patients at high risk for psychiatric complications and to evaluate in a randomised controlled trial if case management services can improve the psychiatric and functional outcomes of such patients.

Levin HS, Boake C, Song J, *et al*. The validity and sensitivity of the Extended Glasgow Outcome Scale in mild to moderate traumatic brain injury. *J Neurotrauma* 2001;**18**:575–84.

Levin HS, Brown SA, Song JX, *et al*. Depression and posttraumatic stress disorder at three months after mild to moderate traumatic brain injury. *J Clin Exp Neuropsychol* 2001;**23**:754–69.

McCauley SR, Boake C, Levin HS, *et al*. Postconcussional disorder following mild to moderate traumatic brain injury: anxiety, depression, and social support as risk factors and comorbidities. *J Clin Exp Neuropsychol* 2001;**23**:792–808.

EARLY INTERVENTIONS: MINIMISING POST-CONCUSSIONAL SYMPTOMS

R. L. Wood. *University of Wales, Swansea*

Post-concussional symptoms usually include problems of information processing and attention, which have an impact on memory and daily living skills. Many authorities argue that it is unusual for symptoms of minor or moderate concussional injury to persist longer than 3–6 months after injury (Gronwall & Wrightson 1974, 1975; Levin *et al*, 1987b; MacFlynn *et al*, 1984). However, around 15% of cases, what Ruff 1996 and his colleagues refer to as "the miserable minority", display persisting cognitive weaknesses, in addition to somatic complaints such as headache, dizziness, insomnia, and fatigue. These non-specific and subjective complaints may persist for many months, even years (Rimel *et al*, 1981; Rutherford *et al*, 1977,1979) and prove to be a major obstacle to the resumption of pre-accident lifestyle. They can occur in litigants and non-litigants alike. In recent years, the balance of evidence has favoured the view that persisting or late-onset symptoms are unlikely to have an organic cause, but are related to psychological factors such as coping style, attribution

failures, or symptom exaggeration (Karzmark *et al*, 1995; Marsh, 1995; Newcombe *et al*, 1994; Schrader *et al*, 1996; Taylor *et al*, 1996), but some authorities continue to favour an organic interpretation for a minority of cases (eg Ruff *et al*, 1994, 1996). This presentation will review some of the evidence for non-organic factors being responsible for persisting post-concussional symptoms and suggest methods to minimise the risk of a disabling post concussional syndrome developing.

NEUROPSYCHIATRIC SEQUELAE OF TBI: FACT AND FICTION

T. McAllister. *Dartmouth Medical School, USA*

Traumatic brain injury (TBI) is a significant public health problem. Despite this, very little serious attention is devoted to brain injury, and the lay public is quite ignorant of both the enormity of the problem and the nature of the sequelae of TBI. There are many popular misconceptions about the effects of TBI, which result in part from inaccurate portrayals in books, movies, and television. Although many individuals survive TBI, they are frequently left with an array of chronic neuropsychiatric sequelae. The neuropsychiatric sequelae of TBI follow naturally from the typical profile of brain injury sustained in a typical TBI. Changes in personality and emotional control are common, and TBI significantly increases the risk developing a variety of psychiatric disorders. These neurobehavioral sequelae are a source of distress to the injured individuals and their families/caregivers, and often are the most important barrier to returning to pre-injury quality of life. This presentation will give a brief overview of portrayals of TBI in the media, and contrast this with the reality experienced by individuals with TBI. The pathophysiology of TBI will be reviewed and linked to the common neuropsychiatric sequelae of TBI. Important issues in the assessment and treatment of neuropsychiatric disorders in individuals with TBI will be discussed.

SETTING UP AN ACQUIRED BRAIN INJURY SERVICE: WHO TO HELP WHOM?

M. Campbell, J. Garber. *Sheffield Brain Injury Network*

Post-acute services for those living with the effects of acquired brain injury (ABI) have been slow to develop in the UK, despite significant developments in the early medical management of trauma and pathology. Currently, there is limited evidence to support specific interventions or predict detailed outcomes at an individual case level. There is, however, knowledge of the gross characteristics of the population requiring a service, although not of the exact numbers involved. Using knowledge of the primary and secondary effects of ABI on those directly affected and on their support networks, the need for collaborative working across a network of services is advanced. This assertion is supported with reference to the mismatch between the effects of common sequelae and traditional health and social care models of provision; and expert and consensus opinion concerning service models and characteristics.

Particular emphasis is given to desirable staff qualities. Current barriers to achieving the desired level and organisation of service are then reviewed and a brief description of how these issues are being addressed within the Sheffield Brain Injury Network is given, along with contact details for further discussion.

CASE MANAGEMENT: FOR LONG TERM CONDITIONS

C. Johnson. *Rehab without Walls, Milton Keynes*

Ms Johnson will review the background and history of case management in the United Kingdom. She will describe the process of case management and provide examples of long term brain injury case management.

FACTORS DETERMINING RETURN TO WORK AFTER SEVERE TRAUMATIC BRAIN INJURY

Z. Groswasser. *Loewenstein Rehabilitation Hospital, Tel-Aviv, Israel*

Measuring success and outcome following severe traumatic brain injury (TBI) is a must in rehabilitation and is also very much demanded by healthcare providers interested in measuring efficacy of treatment. Most TBI patients are in their second and third decade of life, therefore the yardstick of measuring outcome must be of great significance to their lives. Return to work (RTW) has become, in recent years, an integrative criterion for measuring outcome because it is closely linked to patients' subjective evaluation of quality of life.

Factors influencing outcome can be assigned to various groups. Sex, age, and genetic constitution, like presence of the APOe4 allele, comprise a group of non-treatable non-trauma related factors. TBI related factors like duration of unconsciousness, motor deficits, communication disorders (speech and language), cognitive deficits, behavioral changes, and presence of late post-traumatic seizures have a negative influence on RTW. Logistic regression analysis of these variables has shown that duration of unconsciousness and presence of behavioral disturbances are the significant ones in relation to RTW. Studies have that actual placement of patients at work is lower than the predicted at discharge from rehabilitation programmes.

It was found that somatic elements like headache, dizziness, hypersensitivity to noise, and sleep disturbances, which are part of the "post-concussive disorder" formerly thought to appear in only mild TBI, do appear with time in severe TBI patients and exert a negative influence on RTW. Recent imaging studies have shown that some linear measurements, especially the increase of the width of the third ventricle is negatively associated with RTW and with decrease in overall intelligence.

It is concluded that better understanding of the various variables influencing RTW can be of help in setting realistic goals in the rehabilitation process of TBI patients.

Recovering from head injury

EXPERIMENTAL TRAUMATIC BRAIN INJURY: BIOLOGICAL PROCESSES AND PHARMACOLOGICAL POSSIBILITIES

T.K. McIntosh. *University of Pennsylvania, USA*

The mechanisms underlying secondary or delayed cell death following traumatic brain injury are poorly understood. Recent evidence from experimental models suggests that widespread neuronal loss is progressive and continues in selectively vulnerable brain regions for months to years after the initial insult. The mechanisms underlying delayed cell death are believed to result, in part, from the release or activation of endogenous "autodestructive" pathways induced by the traumatic injury. The development of sophisticated neurochemical, histopathological, and molecular techniques to study animal models of TBI have enabled researchers to begin to explore the cellular and genomic pathways that mediate cell damage and death. This new knowledge has stimulated the development of novel therapeutic agents designed to modify gene expression, synthesis, release, receptor, or functional activity of these pathological factors with subsequent attenuation of cellular damage and improvement in behavioural function. This talk represents a compendium of recent studies suggesting that modification of post-traumatic neurochemical and cellular events with targeted pharmacotherapy and/or cellular transplantation can promote functional recovery following traumatic injury to the central nervous system.

PATHOPHYSIOLOGICAL MECHANISMS OF RECOVERY AFTER TBI: CLUES FROM FUNCTIONAL MRI

T. McAllister. *Dartmouth Medical School, USA*

Traumatic brain injury (TBI) is an enormous public health challenge. The common sequelae of TBI include changes in personality, behaviour, and cognition. Overcoming these challenges often presents the greatest difficulty for individuals in their efforts to return to their pre-injury life. Unfortunately, the degree of disability experienced by the individual with TBI can appear out of proportion to the abnormalities seen on conventional neuroimaging such as computed axial tomographic (CAT) scanning. Although magnetic resonance imaging (MRI) frequently displays more injury-related lesions than does CAT scanning, it is common to find individuals with significant cognitive impairment and normal appearing MRI scans. This has given rise to the suggestion that the persistent sequelae of TBI, particularly mild TBI, result from psychiatric disorders or attempts to gain compensation. We have been using functional MRI (fMRI) to help clarify the pathophysiology of cognitive impairment in TBI one month and one year after injury in individuals with mild and moderate TBI. This talk will present an overview of four fMRI experiments. In Study 1 participants with mild TBI (one month after injury), and healthy controls, performed working memory (WM) tasks while undergoing fMRI. Differences in brain activation were found in the TBI group, despite equivalent task performance. Study 2 used an encoding and retrieval

task in a group one month after injury and a group of healthy controls. Once again differences in task-related brain activation were found in the TBI group. In Study 3, participants were scanned at one month and one year after injury. The TBI group showed persistent, though subtle changes in both cognitive performance and brain activation patterns relative to healthy controls. In Study 4, a dopamine agonist (bromocriptine) was given to participants 2.5 hours before scanning. Both groups showed activation of WM circuitry associated with task performance and localised increased activation of WM circuitry while on bromocriptine. However, the pattern of increased activation differed along an anterior-posterior gradient. Controls showed localised increased activation of anterior DLPFC, whereas the MTBI group showed increased activation in posterior frontal and parietal areas.

Taken together, this work suggests changes in the allocation and modulation of cognitive processing resources in individuals with mild and moderate TBI despite good task performance. These changes evolve but persist one year after injury. Changes in frontal dopaminergic systems may contribute to these changes. The implications of these findings will be discussed.

OUTCOME FOLLOWING CHILDREN'S HEAD INJURY AND ITS IMPACT ON FAMILY LIFE

J. Middleton. *The Radcliffe Infirmary, Oxford*

Head injury in childhood can affect cognition, behaviour, and emotional functioning quite profoundly depending on the severity of the injury. In some ways outcome is similar to that in adults, but it also differs in a number of important ways. Firstly, some effects may not be immediately apparent after injury, and it is only as children develop that the full impact becomes evident. There can be an interaction between the fact that the brain continues to develop, the development of more complex and abstract skills, and the increasing demands made on children as they get older. After initial recovery, problems seem to, and do, get worse in a number of cases. Secondly, new learning is affected, as in adults, but children have far less experience and a reduced knowledge base compared with adults, and their ability to fall back on what they have learnt in the past is therefore less. Consequently they fall further and further behind their peers.

Services immediately following an injury can be supportive and well organised for families. However, as children appear to recover, so available help may diminish considerably. This is particularly the case where physical recovery is good and cognitive and behavioural problems subtle. There is some evidence that there is an inverse relationship between the levels of need as the long term effects of injury become manifest and the amount of support available. The changes that occur in families can be profound and a family that was getting by before an injury may fall apart afterwards.

Family functioning pre-injury is an important factor affecting family functioning post-injury, and children's recovery can be positively enhanced by appropriate family support. This can be at many levels, from basic information about what to expect, to help in getting suitable special education for the child, to psychological therapy for members of the family or the family as a whole.

HEAD INJURY AS A RISK FACTOR FOR ALZHEIMER'S DISEASE

S. Fleminger. *Maudsley Hospital and Edgware Community Hospital*

In a proportion of those who suffer a severe head injury amyloid accumulates in the brain. Head injury therefore might be a risk factor for Alzheimer's disease. Only a very few follow up studies have looked at this, and they have all been follow ups of military cohorts (and therefore almost exclusively men). The findings are not entirely consistent, but one recent study has found an excess of Alzheimer's disease in head injured veterans compared with matched controls who had not suffered a head injury.

A systematic review by Mortimer *et al* in 1991, based on collating data across 8 case-control studies, found that there was greater reporting of previous head injury in people with Alzheimer's disease than in matched controls. This finding was restricted to men. We have recently updated this review on the basis of a further 7 case-control studies published since 1991. Overall these post-Mortimer studies did not find such a strong relationship between head injury and Alzheimer's disease as the Mortimer studies. But they did confirm the sex difference; in men with Alzheimer's disease there was a 2–3 fold greater reporting of previous head injury, whereas in women there was the same reporting of previous head injury in controls and in Alzheimer's. Methodological issues related to case-control studies will be discussed.

Medico-legal aspects of neuropsychiatry

MEDICO-LEGAL ASPECTS OF NEUROPSYCHIATRY

M.A. Jones. *University of Liverpool*

For many years the attitude of the law to claims for compensation involving psychiatric damage has been marked by scepticism. It has always been easier to recover for physical injury than for psychiatric harm. Despite occasional judicial statements suggesting that the law should reflect medical thinking and that the two forms of harm should be treated equally, this has not yet occurred and it is unlikely to occur in the near future. Whereas medicine may approach psychiatric harm as an issue for diagnosis and treatment, the law deals with it in the context of claims for compensation where there are competing policy issues. In particular there is concern that too liberal an approach could lead to a "flood" of actions, both genuine and (possibly) fraudulent. The result is that the law's conceptual foundation is unprincipled and its practical application arbitrary. While there is recognition that psychiatric harm can be just as disabling, if not more so in some cases, than physical injury, and that "deserving cases" should be compensated, there is no agreement on which cases are "deserving", and even where the court accepts that the claimant's claim on our sympathy is strong there may be countervailing policy reasons for rejecting the claim.

Physical harm: The law draws a sharp distinction between psychiatric damage that arises following physical injury to the claimant and psychiatric damage that is not linked to physical injury ("pure" psychiatric harm). Assuming that the defendant can be proved to have been negligent following physical injury, the central issue is simply causation—can the psychiatric harm be causally linked to the defendant's negligence? This is a matter for expert evidence and proof, which will usually depend upon the credibility of the evidence, both the claimant's and the experts'.

"Pure" psychiatric harm: Where the claimant has not suffered physical injury the first hurdle that he/she must leap is to prove that he/she suffers from a "recognised" psychiatric illness, as opposed to merely distress or grief. In the absence of physical injury to the claimant, the courts draw a distinction between psychiatric injury arising from a sudden shocking event and psychiatric injury having a more gradual onset. The latter may be actionable if there is a pre-existing legal relationship (usually contractual) between claimant and defendant. This arises typically in occupational stress cases, but can extend beyond that category of relationships. Again, the legal issue here tends to focus on whether the defendant was negligent (which may depend upon whether psychiatric injury was foreseeable in the circumstances) and proof of the causal link between the negligence and the claimant's psychiatric harm.

Sudden events: Where there is a sudden, traumatic event that either involves the claimant as a "participant" in the event or which the claimant witnesses, the legal focus is on whether the defendant owes the claimant a "duty of care". This is a legal device for excluding certain types of negligence claim from compensation even though the defendant can be shown to have been negligent and that negligence caused the claimant's loss. With sudden events the courts draw a further distinction between those who were in the "zone of danger", and therefore exposed to the risk of foreseeable physical harm, but who fortuitously did not suffer any physical harm ("primary victims"), and those who were merely witnesses of the event ("secondary victims").

"Primary victims": A primary victim is a claimant who was actually exposed to the risk of foreseeable physical injury; or, who reasonably believed he/she was exposed to the risk of physical harm; or, possibly, as a result of the defendant's negligence, reasonably believed that he/she was the involuntary cause of death or injury to another. Formerly, all claimants suffering psychiatric damage in the absence of physical injury had to prove that the psychiatric damage was a foreseeable consequence of the defendant's negligence. Following a ruling of the House of Lords in 1995, those "primary victims" who were exposed to the risk of foreseeable physical injury can recover compensation for psychiatric harm, whether or not the psychiatric harm was foreseeable. Categorisation as a "primary victim" is thus a passport to compensation, subject to proving negligence and causation. Moreover, the claimant does not have to show that it was the risk of physical harm to which he/she was exposed (which is what confers his/her status as a "primary victim") that caused the psychiatric harm.

"Secondary victims": A "secondary victim" is someone who is not within the zone of danger, but suffers psychiatric injury as a result of what he/she witnesses. The "mere witness" will only establish a duty of care if he/she satisfies a number of tests (in addition to proving negligence and causation).

The psychiatric harm must have been foreseeable in an individual of "normal fortitude"; harm attributable to the claimant's peculiar susceptibility to psychiatric damage is excluded, unless a person of reasonable fortitude would have suffered psychiatric illness in the circumstances, in which case even the particularly susceptible individual can recover compensation (and for the full extent of the harm, even if this is exacerbated by his/her predisposition to psychiatric injury).

There must be a relationship of close love and affection between the claimant and a person seen to have been injured or put in peril by the defendant's negligence; there is a rebuttable presumption that the relationship between spouses or parent and child satisfies this requirement; other relationships require specific proof of the closeness of the emotional tie; a bystander is not regarded as falling within the range of foreseeable injury.

The claimant must have been close in time and space to the event, or its immediate aftermath; the "aftermath" can include seeing the victims two hours after the initial accident (provided they have not been attended to by medical staff), but not identifying the victims at a mortuary or in a hospital a few hours later.

The claimant must have witnessed the event with his/her own unaided senses (being told about the event is not sufficient); the perception of events from live television or radio broadcasts will not normally be sufficient.

The psychiatric harm must be shock-induced ("the sudden appreciation by sight or sound of a horrifying event, which violently agitates the mind") rather than the build up over a period of time of a psychiatric response.

Other situations: There are other cases that do not fit neatly into these categories. It is possible that a claimant can claim for psychiatric damage caused as a result of being given accurate but distressing news in a negligent manner, or for negligently being given inaccurate, distressing information. And, negligently exposing the claimant to the risk of developing a fatal disease in the future, which knowledge is the cause of the claimant developing a psychiatric illness, can give rise to a claim for the psychiatric harm.

1 *Walker v Northumberland County Council* [1995] 1 All ER 737: *Hatton v Sutherland* [2002] EWCA Civ 76: [2002] 2 All ER 1.

2 Example of the relationship between solicitor and client: *McLoughlin v Jones* [2001] EWCA Civ 1743: [2002] 2 WLR 1279.

3 *Page v Smith* [1995] 2 All ER 736.

4 *Sion v Hampstead Health Authority* [1994] 5 Med LR 170.

NEUROPSYCHIATRY IN THE CRIMINAL AND APPEAL COURTS

M. Kopelman. *Guy's, King's, St Thomas's School of Medicine, KCL, London*

Neuropsychiatrists have important contributions to make not only in civil courts in compensation and negligence cases, but also in criminal courts and the Court of Appeal. In the present talk, this will be illustrated with regard to four issues: (1) claims of amnesia for an offence, and their related associations; (2) cases of false confession, overturned by the Court of Appeal; (3) the neuropsychiatric aspects of forensic cases; (4) forensic issues in neuropsychiatric cases.

These issues will be discussed and illustrated with respect to a number of high profile cases.

RETROGRADE AMNESIA AND POST-TRAUMATIC AMNESIA: A CRITICAL REVIEW

N. Kapur. *Southampton General Hospital*

This talk will primarily focus on retrograde amnesia and post-traumatic amnesia in patients with closed head injury, but some consideration will also be given to cases of open head injury. Issues of terminology and conceptual issues will be briefly noted. In the case of retrograde amnesia, there are grounds for distinguishing between pre-traumatic amnesia and loss of more remote memories. Severe closed head injury is usually accompanied by some pre-traumatic retrograde amnesia, but severe penetrating head injury may occur without such memory loss. Memory for autobiographical events and memory for public knowledge represent the two major components of retrograde amnesia, and may be dissociably impaired in head injury patients. Although marked retrograde amnesia may be present in some cases of severe closed head injury, its presence following minor head injury is normally associated with the presence of psychogenic factors. Severe, life-long, temporally-ungraded autobiographical amnesia—even in cases of severe closed head injury—is also likely to be due in part to the operation of emotional or motivational factors. In the case of post-traumatic amnesia, this occurs in most cases of severe closed head injury, but may be absent in some cases of penetrating head injury. In severe closed head injury, post-traumatic amnesia generally includes both attentional and memory components, and its duration is closely related to outcome measures. There is relatively good agreement between retrospective and prospective measures of post-traumatic amnesia. There is no strong evidence that the presence of post-traumatic amnesia will necessarily act to protect patients from the occurrence of post-traumatic stress disorder. Specialised assessment procedures may help in the detection of malingering in retrograde and anterograde amnesia. There is only limited evidence for recovery of lost pre-traumatic or post-traumatic memories by pharmacological or hypnotic procedures.

WHIPLASH INJURIES—THE STATE OF THE CONTROVERSY

J.M.S. Pearce. *Hull Royal Infirmary*

Whiplash injuries occur in more than one million people in the United States every year. A small minority complain of symptoms that persist for years, often with no clear demonstrable cause. Most modern work recognises that the late whiplash syndrome is not the result of a chronic injury, and current opinion implicates a biopsychosocial model, which considers it an effect of cultural expectation that generate amplification and misattribution of symptoms.

Logic dictates that explanations for these persisting symptoms can only be: organic pathology caused by injury; psychological illness caused or precipitated by injury; continuation of pre-existing causes of symptoms, eg spondylosis; exaggeration; or in its severest from malingering.

If the injury is strictly defined, and excludes traumatic fractures, subluxations, disk herniations, facet joint disruptions, traumatic radiculopathy, and myelopathy, it is now generally agreed that persisting organic factors do not explain symptoms after the first few days or weeks. Indeed, in several controlled studies, it is clearly shown that acute neck symptoms last for a mean of 3 days, maximum of 3 to 6 weeks.

We have no consistent objective evidence of a persisting physical cause for these symptoms, despite sophisticated investigation. The role of psychological symptoms engendered by the litigation process is acknowledged, but so too is the invariable prospect of financial gain and of symptoms as a source of primary gain and attention. Argument is rife, and doctors are not always free of a biased perspective and possible tertiary gain. The fundamental and final unanswered question is this: should conjecture and speculation about the genuine nature and possible mechanism of symptoms and disabilities stand as scientifically acceptable explanations in the absence of objective findings of physical or sufficient psychogenic illness to account for chronicity?

Members' papers

SEXUAL BEHAVIOUR IN PATIENTS WITH HUNTINGTON'S DISEASE

D. Craufurd, J. Thompson, J.S. Snowden. *St Mary's Hospital, Manchester*

Aims: Previous reports in the medical literature, including that of Huntington himself, have suggested that Huntington's disease (HD) is associated with hypersexuality. However, the only study to examine the issue systematically using modern research methods found no evidence to support this (Fedoroff *et al*, 1994). We therefore investigated libido and sexual behaviour as part of a broader study of behaviour in HD patients.

Methods: 134 patients attending an HD management clinic were interviewed with their principle carer using the Problem Behaviours Assessment for Huntington's disease.

Results: Loss of libido was reported by 61.8% of patients for whom reliable information (corroborated by the partner) was obtained. There was a strong inverse correlation with total functional capacity score (rho –0.459, $p < 0.0001$). Sexually disinhibited behaviour was reported by carers in only 6/104 individuals (3 male, 3 female; 5.8%), while demanding or persistent sexual behaviour was described in just 4/81 cases (3 male, 1 female; 4.9%).

Conclusions: Hypersexual behaviour is rare in HD and affects a small minority of cases only. Loss of libido and hyposexuality are much more common and correlate strongly with other measures of disease progression. The theoretical implications will be discussed.

DE CLERAMBAULT'S SYNDROME AND EXECUTIVE DYSFUNCTION: A CASE REPORT

S. Hudson, L. Chambers, S. Boddington, M. Philpot. *Maudsley Hospital, London*

Case History: A male case of erotomania spanning a 30 year period is described. The patient first presented to psychiatric services at the age of 66 years after breaching a restraining order brought under the Harassment Act 1997. Stalking behaviour included contact with his victim via letters and visits to her home. There was evidence that his contact was increasing in the 2 years before the offence. Additional clinical features were delusions of reference, self-neglect, disinhibition, and micrographia. Symptoms receded on treatment with quetiapine but compliance was poor when he left hospital.

Results: Brain computed tomography was normal for the patient's age and he declined further neuroimaging studies. Neuropsychological assessment demonstrated average IQ with intact verbal memory and language skills. There was impairment of visual memory and executive dysfunction. Six months later there had been a decline in verbal IQ but no change in frontal lobe test performance.

Conclusions: Recent cognitive changes, of as yet unknown origin, appear to have lead to an escalation in stalking behaviour that brought him into contact with psychiatric services late in life. The interaction between the long-standing but stable mental disorder and these changes is discussed.

HYPERSEXUALITY AND PARKINSON'S DISEASE

P. Shaw, A. Blockley, C. Clough, R. Chaudhuri, R. Weeks, A.S. David. *Institute of Psychiatry, London*

Aims: Sexual disorders arising in neuropsychiatric conditions, such as Parkinson's disease, can give insights into the psychobiology of sexual behaviour. We report on six patients with Parkinson's disease who developed a marked increase in both sexual desire and behaviour and consider the possible neuropharmacological and neuropsychological basis of their hypersexuality.

Method: Patients were identified from a large regional neurosciences department and included only if their symptoms caused distress either to the patient or partners. We examined the clinical presentation of the Parkinson's disease, its pharmacological treatment, the phenomenology of the altered sexuality, and associated psychopathology. All subjects had assessments of general intelligence and executive function. Two subjects also completed a task designed to explore the effects of various reward contingencies on behaviour, and their performance compared with subjects with Parkinson's disease who did not experience sexual dysfunction.

Results: In all subjects the hypersexuality involved not only an increase in premorbid sexual activities but an expansion of the repertoire of sexual behaviours (including alterations in sexual orientation and sexual outlets). Paraphilic behaviour occurred in only one subject. The hypersexuality did not occur in isolation: in most subjects it was accompanied by impulse control disorders (eg compulsive gambling and misuse of dopamine agonists). Psychopathology suggestive of mood instability was prominent. Nearly all subjects showed a marked decline in executive function not commensurate with the more modest decline in general intellectual function. There was evidence of an altered perception of reward with pervasive tendency to prefer immediate over delayed gratification—even where this was a disadvantageous strategy. Dopamine precursors and agonists acted as the "fuel" driving the hypersexuality—perhaps both through a direct effect on libido and arousal and indirectly through an interaction with the neural substrate mediating reward.

22Q11 DELETION SYNDROME: A SEARCH FOR CLUES TO THE ORIGINS OF SCHIZOPHRENIA

K.D. Baker, T. Baldeweg, P. Scambler, D.H. Skuse. *Institute of Child Health, London*

Aims: To investigate neurodevelopmental abnormalities in 22q11 deletion syndrome (22q11DS), a group at very high risk for schizophrenia, via multiple methodologies including auditory event-related EEG.

Methods: Adolescents with 22q11DS have been compared to IQ and age-matched controls on auditory ERP measures (N100, mismatch negativity, P300). Cognitive and psychiatric assessments were also conducted.

Results: Adolescents with 22q11DS display aberrant function in neural pathways necessary for detecting change in sound sequences. Individuals with the greatest degree of neural dysfunction display psychological features akin to schizotypal personality. Therefore variation in neural function among individuals with 22q11DS may act as a marker for elevated risk of developing schizophrenia.

Conclusions: It is possible to detect neural dysfunction in a schizophrenia prone group prior to the onset of illness. Heterogeneity within this high risk group may reflect additional genetic or environmental factors that either mediate or protect against the impact of the risk determining gene(s) at the 22q11 locus.

PSYCHOTROPIC MEDICATIONS IN NEUROBEHAVIOURAL TREATMENT OF AGGRESSION AFTER ACQUIRED BRAIN INJURY: EFFICACY AND INCIDENCE OF SIDE EFFECTS

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Aims: Psychotropic medications are frequently used in conjunction with neurobehavioural treatments for aggressive behaviours after neurological injury. Efficacy and side effects for two classes of psychotropic medications are reported.

Methods: All 47 clients treated in one year period at two neurobehavioural rehabilitation units were studied in order to establish the efficacy and side effects of antipsychotic medications and anticonvulsant medications used to treat aggressive behaviours.

Results: Of the 18 admissions to the challenging behaviours unit 72% were admitted taking antipsychotic medications. Eighty-five percent of all persons admitted taking antipsychotic medications without frank psychosis were discontinued from the medication without increases in aggression. Of the participants receiving antipsychotic medications 63% were treated for extrapyramidal side effects, 11% had other physical side effects (tardive dyskinesia or tardive akathesia), and 37% had significant cognitive side effects.

Conclusion: Persons with serious neurobehavioural disorders referred to specialised rehabilitation centres are frequently referred with antipsychotic medications. Their low efficacy for the treatment of aggression and the high levels side effects suggest this does not represent best practice.

DO NEUROPSYCHOLOGICAL MEASURES PREDICT FUNCTIONAL OUTCOME IN HEAD INJURY PATIENTS?

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Aims: To investigate whether standard measures of cognitive function predict level of functional ability in head injury (HI) patients and increase knowledge of factors associated with functional outcome, management, and rehabilitation.

Methods: Retrospective analysis of standard neuropsychological measures including memory and executive function and data from the Assessment of Motor and Process Skills (AMPS) were analysed using correlational statistics for 15 HI patients from the Lishman Unit.

Results: General IQ was correlated with the process component of the AMPS. The cognitive component most associated with the process component was verbal list learning ($p < 0.05$).

Conclusions: This study indicates neuropsychological measures (IQ and verbal learning), may be useful in estimating level of functional ability, and be helpful for determining future management. Verbal list learning has been associated with frontal lobe function. Deficits may be related to problems with declarative memory, ie memory for "knowing what", as opposed to procedural memory, ie memory for "knowing how" to do something. Treatment strategies should focus on preserved ADL procedural skills, modifications to the environment to compensate impaired process skills. For example, reducing the number of options presented during a task or providing salient task items that may cue the correct declarative memory for an ADL.

DEVELOPING A COMPREHENSIVE NEUROPSYCHIATRY SERVICE: THE STOKE MODEL

K. Barrett. *Haywood Hospital, Stoke-on-Trent*

Aims: Provide an account of the North Staffordshire Neuropsychiatry Service.

Methods: Present a description of the components of the service, information on clinical activities over a 12 month period including number, source, and diagnosis of referrals and size and make up of current case load.

HASHIMOTO'S ENCEPHALOPATHY: AN ORGANIC CAUSE OF NEUROPSYCHIATRIC ILLNESS IN ADOLESCENCE

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Aims: Described is possibly the first reported case of Hashimoto's encephalopathy in the child psychiatric literature. We report a 14 year old case with a broad and complex presentation.

Methods: Our patient presented with a 9 month history of tremors, falls, fatigue, transient partial paralysis, blurred vision when reading, headaches, myoclonic jerks, mood swings, episodes of anxiety, panic attacks, and school non-attendance. In the initial stages she was treated for chronic fatigue syndrome. A provisional diagnosis of CFS with neurological features was made and she was treated with anxiolytic medication. Our first contact followed an admission for a generalised tonic-clonic seizure, which appeared to stem out of panic attack with hyperventilation, requiring intubation and ventilation and followed by altered consciousness and delirium. During her admission she had a further seizure and developed psychotic symptoms. Response to steroids was immediate and complete.

Results: A diagnosis of Hashimoto's encephalitis was made on the basis of a neuropsychiatric presentation, high antithyroid antibodies, raised CSF protein, an abnormal EEG, and the response to steroids.

Conclusions: We think this case demonstrates three important issues. Firstly, that Hashimotos encephalitis should be considered an important differential diagnosis when an adolescent presents with psychotic symptoms. Secondly, it adds to the range of clinical presentations within which patients with this very rare disorder can present. Thirdly, it illustrates the necessity to exclude all possible explaining illnesses before making a diagnosis of chronic fatigue syndrome in the presence of hard neuropsychiatric features.

EFFECT OF GALVANIC SKIN RESPONSE (GSR) BIOFEEDBACK TREATMENT IN PATIENTS WITH EPILEPSY

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Aims: Behavioural interventions including biofeedback represent an alternative therapeutic axis in the management of drug refractory epilepsy. Based on increased understanding of the physiological relationship between peripheral autonomic and central cortical arousal, we investigated the effect on seizure frequency of biofeedback training with Galvanic Skin Response (GSR) in patients with treatment resistant epilepsy.

Method: Eighteen patients with drug refractory epilepsy were randomly assigned either to an active GSR biofeedback group (n = 10) or to the sham control biofeedback group (n = 8). Patients received a total of 12 sessions over 4 weeks, representing either real GSR biofeedback training, where subjects were trained to decrease skin resistance using biofeedback, or sham training, where the feedback was unrelated to the subjects' GSR. The primary outcome measure was a proportional reduction in seizure frequency. An intention-to-treat analysis was undertaken.

Results: All patients in the biofeedback group completed the study. However, there were 3 patients in the control group who withdrew in the middle of the treatment. Biofeedback training significantly reduced patients' seizure frequency in the active biofeedback group ($p = 0.004$), but not the control group ($p > 0.10$). This was manifest as a significant between group differences in seizure reduction ($p = 0.007$).

Conclusions: Our findings demonstrate that a behavioural intervention, GSR biofeedback training, may substantially reduce seizure frequency in drug refractory epilepsy, highlighting the potential therapeutic value of this method.

THE TREATMENT OF CAMPTOCORMIA—A CASE EXAMPLE

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Aims: This paper aims to review the different presentations, characteristic features, and available treatment options for the unusual disorder of camptocormia. Also, to describe our experience in using the anti-psychotic medication, risperidone, as a treatment for this disorder.

Methods: The paper is based on literature reviews and illustrated with a case presentation. Particular problems in diagnosis and treatment are examined. The efficacy of the anti-psychotic medication, risperidone, to treat this disorder is explored with the help of a case example.

Results: While the majority of cases have been described in military settings, there is a growing number of civilian cases. The importance of both physical and psychological factors in both assessment and treatment is emphasised. Risperidone, as part of a combined biological, psychological, and social approach to the treatment of this disorder, was used with good results.

Conclusions: Risperidone, when used a part of a biological, psychological, and social approach, may be a useful treatment for camptocormia.

The neuropsychiatry of love (a feast for Valentine's day)

THE ANATOMICAL BASIS OF DESIRE AND ADDICTION

R.N. Cardinal. *University of Cambridge*

Animals work for rewards, such as food and sex, for a variety of reasons. They learn that they like a reinforcer, and that their actions can cause the reinforcer to be delivered to them; integrating these two pieces of information causes them to work for the reinforcer. Animals may also work for reinforcement because it has become habitual to them to do so. Additionally, environmental cues that have in the past been paired with reinforcement (Pavlovian conditioned stimuli) gain motivational significance; animals will work for these cues themselves (termed conditioned reinforcement) and these cues can influence their tendency to work for primary rewards (termed Pavlovian-instrumental transfer). How are these aspects of motivation implemented in the brain, and how might they go wrong in pathological states such as addiction? The neural systems responsible for the motivation to work for rewards have been doubly dissociated from those required for so-called consummatory behaviour (such as eating and copulation). Natural reinforcers and artificial reinforcers such as drugs of abuse activate common motivational circuits within the brain. The nucleus accumbens and its mesolimbic dopamine innervation strongly influences the ability of environmental cues paired with reward to motivate behaviour, and this may be one aspect of motivation that goes awry in addiction. Structures including the amygdala are involved in the mechanism by which environmental cues gain motivational value in the first place. The same circuits are activated when stimuli induce craving in humans, giving insight into human pathological desires and possible therapeutic targets.

EROTOMANIA AND THE PATHOLOGIES OF LOVE

P.E. Mullen. *Victorian Institute of Forensic Mental Health, Australia*

19th century psychiatry recognised three types of erotomania: the love melancholies produced by rejected or unrequited love; nymphomania and satyriasis; and finally delusional beliefs of being loved by someone with whom, in reality, there was little if any connection. Only the latter form of erotomania survived into contemporary classifications of mental disorder and that, until recently, as a rare psychiatric syndrome of curiosity value rather than clinical significance.

The emergence of stalking as a recognised and socially significant form of victimisation radically altered the place of pathologies of love. Though only a minority of stalkers are driven by a pathology of love, that minority is large enough to have shifted this group of disorders out of obscurity into the front line, if not of psychiatry in general, certainly of forensic mental health. The confrontation of clinicians with a wide range of individuals with pathologies of love is overturning the traditional assumptions based on theories illuminated, if at all, by occasional case studies and replacing it with a more complex understanding of these multi-faceted disorders.

CEREBRAL DISORDERS AND DISTURBANCES OF SEXUAL FUNCTION

P. Gautier-Smith. *London*

Lesions of the spinal cord and the peripheral and autonomic nervous systems leading to disorders of sexual function are well recognised, but the same is not true of cerebral conditions, which have to some extent been neglected in this regard, and some possible reasons for this are discussed. Epileptic fits may be provoked by hyperventilation during sexual intercourse, but reflex epilepsy induced by orgasm and feelings of arousal, sexual imagery, and genital sensation may all occur as part of the aura. Automatisms with unstructured sexual

behaviour in temporal lobe attacks are rare. Hyposexuality in temporal lobe epilepsy is more common than the reverse. Non-ictal disorders of sexual behaviour may follow unilateral or bilateral frontal lobe lesions, but the most dramatic are encountered in those in the temporal lobes. The various clinical manifestations are described in personal cases and others in the literature and their physiological significance discussed. Headache occurring during sexual activity is usually vascular in origin and the various forms of it are briefly discussed. The most serious cause is that of a ruptured cerebral aneurysm, which may prove fatal. Headache in this situation may also be due to cervical spondylosis.

BIOLOGICAL BASES OF GENDER IDENTITY DISORDER (TRANSSEXUALISM)

R. Green. *Imperial College, Charing Cross Hospital, London*

Although precise biological origins of transsexualism remain elusive, research is hot on the trail. The Gender Identity Clinic at Charing Cross Hospital, the world's largest, permits research of uniquely sized samples (in the hundreds). Four findings will be discussed.

(1) Hand use preference. This function, reflecting cerebral hemispheric dominance, is organised prenatally and appears to be influenced by sex steroid levels in utero. Male and female transsexuals are more often non-right-handed.

(2) Fingerprint patterns. These are organised prenatally and also may be influenced by prenatal sex steroids. Male-to-female transsexuals, sexually attracted to male partners, differ from other transsexuals or non-transsexuals.

(3) Family tree pattern. Male transsexuals have a dearth of maternal uncles compared to maternal aunts. Genomic imprinting can explain a lethality factor in one generation and atypical psychosexual development in the next.

(4) Sibling order. The odds of a male-to-female transsexual being sexually attracted to male partners increases with each older brother. One explanation is a progressive maternal immune response to each male fetus.

Our research findings complement the finding in The Netherlands of an opposite sex size in a central subdivision of the bed nucleus of the stria terminalis in a small sample (n = 8) of transsexuals' brains, studied postmortem.