Psychiatric illness and subsequent traumatic brain injury: a case control study

J R Fann, A Leonetti, K Jaffe, W J Katon, P Cummings, R S Thompson

.....

J Neurol Neurosurg Psychiatry 2002;72:615–620

Objective: To determine whether psychiatric illness is a risk factor for subsequent traumatic brain injury (TBI).

Methods: Case control study in a large staff model health maintenance organisation in western Washington State. Patients with TBI, determined by *International classification of diseases, 9th revision, clinical modification* (ICD-9-CM) diagnoses, were 1440 health plan members who had TBI diagnosed in 1993 and who had been enrolled in the previous year, during which no TBI was ascertained. Three health plan members were randomly selected as control subjects, matched by age, sex, and reference date. Psychiatric illness in the year before the TBI reference date was determined by using computerised records of ICD-9-CM diagnoses, psychiatric medication prescriptions, and utilisation of a psychiatric service.

See end of article for authors' affiliations

Correspondence to: Dr J R Fann, Department of Psychiatry and Behavioral Sciences, Box 356560, University of Washington, Seattle, Washington, USA; fann@u.washington.edu

Received 26 June 2001 In revised form 29 October 2001 Accepted 7 November 2001 **Results:** For those with a psychiatric diagnosis in the year before the reference date, the adjusted relative risk for TBI was 1.7 (95% confidence interval (CI) 1.4 to 2.0) compared with those without a psychiatric diagnosis. Patients who had filled a psychiatric medication prescription had an adjusted relative risk for TBI of 1.6 (95% CI 1.2 to 2.1) compared with those who had not filled a psychiatric medication prescription. Patients who had utilised psychiatric services had an adjusted relative risk for TBI of 1.3 (95% CI 1.0 to 1.6) compared with those who had not utilised psychiatric services. The adjusted relative risk for TBI for patients with psychiatric illness determined by any of the three psychiatric indicators was 1.6 (95% CI 1.4 to 1.9) compared with those without any psychiatric indicator. **Conclusion:** Psychiatric illness appears to be associated with an increased risk for TBI.

The most common mechanisms of traumatic brain injury (TBI) are motor vehicle collisions, falls, sports and recreational injuries, and assaults.¹⁻⁵ The known risk factors for sustaining a TBI are age 15–24 years (with smaller peaks in the > 60 and < 5 year age groups), male sex, increased blood alcohol concentration, and lower socioeconomic status.^{6 7} The identification of modifiable risk factors and of high risk populations has recently been prioritised by the Department of Health and Human Services' Interagency Head Injury Task Force in the United States⁸ and may lead to prevention programs that help to decrease the incidence of TBI and associated disability and mortality. Although mild cases constitute the vast majority of TBI and may result in prolonged disability,⁹ relatively little is known about the risk factors for mild TBI.

While it is recognised that people are more likely to suffer from psychiatric illness following TBL,¹⁰ it is less clear whether those who sustain a TBI are more likely to have had a psychiatric illness before their injury. Previous studies suggest that psychiatric illness may be a risk factor for traumatic accidents but they did not specifically examine TBI as an outcome.¹¹⁻¹⁷ Furthermore, some of these studies examined small or selected populations and ascertained psychopathology imprecisely. A few studies have examined a narrow range of psychiatric risk factors for TBI in selected populations but their results may not apply to other populations.¹⁸⁻²¹ To date, no large scale studies have looked specifically at the relation between a broad range of pre-existing psychiatric illness and the risk of TBI across all severity levels and age groups.

We undertook a case control study to determine whether markers of pre-existing psychiatric illness were risk factors for subsequent TBI among members of a large staff model health maintenance organisation.

METHODS

The study was conducted using computerised records from the Group Health Cooperative of Puget Sound (GHC), a large staff model health maintenance organisation located in western Washington State. GHC serves approximately 450 000 members, who are generally representative of the area's population in terms of age, sex, race, and marital status.²² Patients received medical care from one of GHC's facilities located in six counties in the Puget Sound area of western Washington State. Data on health plan members were derived from GHC's computerised databases, which included information on all inpatient and outpatient visits and diagnoses, all prescriptions dispensed from GHC pharmacies, age, sex, and insurance type (Medicare, Medicaid, GHC individual or family plan, GHC group plan, or other plan). Each GHC member has a unique number that makes linkage of all utilisations possible. This number is permanent and stays with the person if he or she disenrols or re-enrols. GHC tracks enrolment closely and has a low rate of member disenrolment (13.1% for 1992 through 1993), which made it possible for us to maintain a stable study base. Diagnoses in 1992 were recorded on 95% of all visit records.²² Only about 7% of GHC members have dual insurance coverage, so ascertainment of utilisation is nearly complete. The study was approved by the institutional review boards of GHC and the University of Washington.

Abbreviations: ACG, Johns Hopkins Ambulatory Care Group; CDC, Centers for Disease Control and Prevention; CI, confidence interval; GHC, Group Health Cooperative of Puget Sound; ICD-9-CM, International classification of diseases, 9th revision, clinical modification; TBI, traumatic brain injury

Patients with TBI

Cases were diagnoses of TBI made in the emergency room, hospital, or an outpatient clinic in 1993. The following diagnostic categories and International classification of diseases, 9th revision, clinical modification (ICD-9-CM) codes²³ were used to identify TBI: fracture of the vault or base of the skull (800.0-801.9); other, unqualified, and multiple fractures of the skull (803.0-804.9); and intracranial injury, including concussion, contusion, laceration, and haemorrhage (850.0-854.1). These diagnostic categories for TBI were used by the Centers for Disease Control and Prevention (CDC) in their TBI surveillance studies.²⁴ If a person had more than one visit with a TBI diagnosis in 1993, the first diagnosis was considered the incident TBI. The reference date for cases was the date of this incident TBI diagnosis. Patients were required to have been continuously enrolled at GHC for the year before their TBI diagnosis to ensure that records were available to assess indicators of psychiatric illness. Cases were taken from six counties in the Puget Sound area that provided complete health services utilisation data. To maximise the likelihood of ascertaining incident TBI cases, patients who had an ICD-9-CM diagnosis of TBI in the year before their reference date were excluded from the case group.

Severity of TBI was dichotomised into mild TBI and moderate to severe TBI using the CDC categorisation criteria.²⁵ Cases were considered to be mild if ICD-9-CM codes indicated brief (< 1 hour) or no loss of consciousness and no traumatic intracranial lesions were documented. Cases were considered to be moderate or severe if ICD-9-CM codes indicated prolonged loss of consciousness or a documented traumatic intracranial or brain lesion. Patients whose TBI severity was undetermined were excluded from the study.

Non-TBI control subjects

Three control subjects per patient were selected at random from GHC enrolment files and were frequency matched to patients by sex, age in groups of five years (0–4 years, 5–9 years, 10–14 years, and so on, to \geq 95 years), and enrolment at the time of the case patient's reference date. Similar to the patients, control subjects had to be GHC members on their assigned reference date, had to be GHC members continuously during the year before their reference date, and could not have had a TBI diagnosis during that year.

Psychiatric illness indicators

Psychiatric illness in the year before the reference date was ascertained by three separate methods: presence of a psychiatric diagnosis, filling of a presciption for a psychiatric medication, or utilisation of psychiatric services. Presence of a psychiatric diagnosis was determined by using ICD-9-CM codes recorded in the year before the reference date and was categorised as follows: acute reaction to stress or adjustment reaction (308, 309); alcohol or drug intoxication, withdrawal, or dependence (291.0-292.9, 303.0, 303.9, 304, 305); anxiety (300.0, 300.2, 300.3, 799.2); depression (296.2, 296.3, 296.82, 296.9, 300.4, 311); hyperkinetic syndrome of childhood (314); malaise or fatigue (300.5, 780.7); organic psychotic mental disorders (290.0-290.9, 293.0-294.9); organic non-psychotic mental disorders (310, 780.09); schizophrenia, hallucinations, or paranoia (295, 297.0–299.9, 780.1); somatoform disorders (300.1, 300.6–300.9, 306, 307.8, 307.89); or other psychiatric disorders (307, 316, V40.2–40.9, V62.81, V62.89, V65.9).

Subjects were considered to have filled a psychiatric medication prescription if automated GHC pharmacy data indicated that a prescription for a psychiatric medication in any of the following classes was filled in the year before the reference date: antidepressants, antipsychotics, anxiolytics, lithium, or psychostimulants. Surveys in 1985 and 1986 showed that over 90% of all medications prescribed at GHC were filled in GHC pharmacies and a study found that 97.6% of those treated with antidepressant medications in 1991–92 filled their prescriptions at GHC pharmacies.²² Because antidepressants are commonly used for other indications, they were considered to be for a psychiatric indication only if the prescription was filled within 60 days of a depression diagnosis. Anxiolytics were considered to be for a psychiatric indication if the prescription was filled within 60 days of an anxiety diagnosis. Psychostimulants were excluded if there was a diagnosis of narcolepsy within a year before the reference date.

Utilisation of psychiatric services was ascertained based on computerised records of inpatient psychiatric hospitalisations, outpatient mental health clinic visits, and alcohol or drug treatment inpatient stays or outpatient visits in the year before the reference date. Out of plan mental health services use is rare due to comprehensive mental health services with small service payment responsibilities by patients within GHC.

An overall summary determination of psychiatric illness was made based on the presence of any psychiatric diagnosis, psychiatric medication prescription, or psychiatric utilisation during the year before the reference date.

Medical comorbidity

Medical comorbidity was determined using the Johns Hopkins Ambulatory Care Group (ACG) case mix system (version 4.1),²⁶ which is a measure of relative health status designed to be indicative of expected resource consumption. ACGs use age, sex, and ICD-9-CM diagnoses assigned during patient-health care provider encounters over a period of time (such as a year) to assign a person to one of 106 mutually exclusive categories.²⁷ The ACG system has been found to be useful in predicting both concurrent and subsequent ambulatory care use and costs, as well as subsequent morbidity.²⁸ and has been used extensively to control for medical comorbidity.²⁷⁻²⁹ In this study, ICD-9-CM codes for psychiatric diagnoses were excluded from the ACG category determination to arrive at ACG categories derived solely from medical diagnoses.

Analysis

Odds ratios using unconditional logistic regression³⁰ were used to approximate relative risks of TBI in those with indicators of psychiatric illness relative to those without. The matching variables were included in all models: age was categorised in five year intervals and reference date was categorised as the month of injury. Insurance type, used as a proxy measure of socioeconomic status, and medical comorbidity were examined as potential confounders. Possible effect modification of the association between psychiatric illness and TBI by age and sex was assessed. Possible interaction between ICD-9-CM psychiatric diagnosis and psychiatric medication prescription was analysed.

RESULTS

In 1993, 2250 GHC members had a diagnosis of TBI. Among these, 1641 were enrolled in GHC for at least one year before their index TBI, 1541 were enrolled in the six county study region, and 1445 did not have a TBI in the year before their incident TBI. TBI severity was determined in 1440 patients. These constitute the TBI case group.

Among case patients, 1366 (94.9%) had a diagnosis of intracranial injuries (concussion, contusion, laceration, or haemorrhage); 37 (2.6%) had a diagnosis of fracture of the vault or base of the skull; and 37 (2.6%) had a diagnosis of other, unqualified, and multiple skull fractures. Most cases were classified as mild TBI (n = 1293, 89.8%). Most cases of TBI were diagnosed in an outpatient setting (n = 691, 48.0%) or in the emergency room (n = 582, 40.4%), and 167 (11.6%) were diagnosed in the hospital. The overall annual incidence rate of TBI was 475.2 per 100 000 person years, using a

Psychiatric illness indicator	Cases (n=1440) (n (%))	Controls (n=4320) (n (%))	Adjusted relative risk*	95% CI
Psychiatric ICD-9-CM diagnosis				
Acute reaction to stress or adjustment reaction	48 (3.3)	62 (1.4)	1.9	1.3 to 2.9
Alcohol or drug intoxication, withdrawal, or dependence	34 (2.4)	51 (1.2)	1.6	1.0 to 2.6
Anxiety	26 (1.8)	38 (0.9)	1.5	0.9 to 2.6
Depression	54 (3.8)	87 (2.0)	1.4	1.0 to 2.0
Hyperkinetic disorder	14 (1.0)	48 (1.1)	0.7	0.4 to 1.3
Malaise or fatigue	44 (3.1)	64 (1.5)	1.7	1.1 to 2.6
Organic non-psychotic disorders	10 (0.7)	6 (0.1)	4.4	1.6 to 12.
Organic psychotic disorders	15 (1.0)	11 (0.3)	3.7	1.6 to 8.7
Schizophrenia, hallucination, or paranoia	17 (1.2)	26 (0.6)	1.9	1.0 to 3.7
Somatoform disorders	19 (1.3)	14 (0.3)	2.8	1.3 to 5.8
Other	70 (5.0)	124 (2.9)	1.3	1.0 to 1.9
Psychiatric medication prescription	. ,			
Anxiolytics	27 (1.9)	25 (0.6)	2.4	1.3 to 4.2
Antidepressants	57 (4.0)	89 (2.1)	1.5	1.1 to 2.2
Antipsychotics	18 (1.3)	19 (0.4)	2.7	1.3 to 5.5
Lithium	6 (0.4)	11 (0.3)	1.6	0.6 to 4.4
Psychostimulants	3 (0.2)	13 (0.3)	0.5	0.2 to 1.9
Major psychiatric illness indicators	• •			
Psychiatric ICD-9-CM diagnosis	262 (18.2)	420 (9.7)	1.7	1.4 to 2.0
Psychiatric medication prescription	85 (5.9)	136 (3.2)	1.6	1.2 to 2.1
Psychiatric service utilisation	151 (10.5)		1.3	1.0 to 1.6
Psychiatric diagnosis and medication	66 (4.6)	94 (2.2)	1.7	1.2 to 2.3
Psychiatric diagnosis and utilisation	73 (5.1)		1.3	1.0 to 1.8
Psychiatric medication and utilisation	42 (2.9)	76 (1.8)	1.3	0.8 to 1.9
Diagnosis, medication, and utilisation	32 (2.2)	51 (1.2)	1.3	0.8 to 2.1
Any psychiatric illness‡	349 (24.2)	618 (14.3)	1.6	1.4 to 1.9

*Relative risk adjusted for age, sex, reference date, and Ambulatory Care Group. Reference group is made up of those without each specific indicator. †Lower limit rounded down to 1.0.

⁺Determined by psychiatric ICD-9-CM (International Classification of Diseases, 9th revision, clinical modification), diagnosis, psychiatric medication prescription, or psychiatric service utilisation. CI, confidence interval.

population denominator of 303 001 person years (those enrolled at GHC for at least one year at mid-year 1993).

Almost half of the patients with TBI were 19 years and younger, with 294 (20.4%) less than 9 years and 411 (28.5%) 10–19 years; 115 (8.0%) were 20–29 years, 282 (19.6%) were 30–49 years, 152 (10.6%) were 50–69 years, and 186 (12.9%) were 70 years or older. Among the patients with TBI, 769 (53.4%) were male. Case patients and control subjects were similar with regard to insurance type, with about 80% group plan dependents or subscribers. TBI events were distributed fairly evenly by month, although there was a slightly lower proportion of cases in the winter months. In the year before the reference date, case patients had a mean (SD) of 7.5 (9.7) outpatient visits compared with 5.3 (7.0) visits by control subjects (p < 0.001); these included primary care visits, specialty care visits, hospital outpatient visits, and outpatient mental health services.

As table 1 shows, the relative risks for TBI were generally 1.3- to 4-fold higher for those with preceding psychiatric condition indicators, whether assessed by diagnosis codes, medication prescriptions, or utilisation profiles. Exceptions to this general pattern were seen for hyperkinetic disorder and use of psychostimulants. Among case patients, 24.2% had some indication of a psychiatric illness before TBI compared with 14.3% of control subjects. With an attributable risk of 37.5% for any psychiatric illness indicator, the proportion of TBI cases that might be attributable to psychiatric illness was 9.1%.

A more detailed examination of the subcategories in table 1 shows that all diagnostic categories were associated with an increased adjusted relative risk of TBI except for hyperkinetic disorder of childhood. Those who had a diagnosis of organic non-psychotic disorders had the highest risk of TBI compared with those without such a diagnosis. Those diagnosed with organic psychotic disorders and somatoform disorders also had relative risks of TBI > 2. Groups with a diagnosis of acute reaction to stress or adjustment reaction, alcohol or drug intoxication, withdrawal, or dependence, malaise or fatigue also had significantly greater risks of TBI than those without these diagnoses. Most psychiatric diagnoses were made by non-mental health professionals, with 50% made by family practice physicians.

Three of the five psychiatric medication types were found to be significantly associated with an increased risk of TBI. Compared with those who had not filled a prescription, those who had filled a prescription for anxiolytics and antipsychotics were approximately two and a half times as likely to have a TBI. The relative risk of TBI for those who had filled a prescription for antidepressants was 1.5 (95% confidence interval (CI) 1.1 to 2.2).

ACGs, indicators of medical comorbidity, were found to confound the relation between psychiatric illness and subsequent TBI, decreasing the relative risk estimate for TBI for all indicators of psychiatric illness. Insurance type was neither a risk factor for TBI nor a confounder of the psychiatric illness-TBI relation for all measures of psychiatric illness.

The relation between psychiatric illness and TBI was significantly modified by age only in the category of any psychiatric ICD-9-CM diagnosis (p = 0.03 for test of heterogeneity). For patients with any psychiatric diagnosis versus those who did not have a diagnosis, those in the age group 25–64 years had the highest relative risk for TBI (relative risk 2.2, 95% CI 1.7 to 2.9) followed by those in age group ≥ 65 years (relative risk 1.7, 95% CI 1.2 to 2.4) and those in age group 0–24 years (relative risk 1.3, 95% CI 0.9 to 1.7). The relation between psychiatric illness and TBI was not significantly modified by sex or insurance type for any indicator of psychiatric illness.

There was no significant interaction between the following: alcohol or drug intoxication, withdrawal, or dependence diagnosis and any other psychiatric diagnosis; anxiety diagnosis and depression diagnosis; depression diagnosis and antidepressant prescription; anxiety diagnosis and anxiolytic prescription; and schizophrenia, hallucination, or paranoia diagnosis and antipsychotic prescription.

DISCUSSION

The main finding of our study was that patients with any indicator of psychiatric illness experienced an increased risk of subsequent TBI (relative risk 1.6) compared with those who had no indicator of psychiatric illness. This increase was seen with three different indicators of psychiatric illness: ICD-9-CM diagnoses, psychiatric medication prescription, and psychiatric service utilisation. A significantly greater relative risk for TBI was found for patients who had a previous diagnosis of acute reaction to stress or adjustment reaction; alcohol or drug intoxication, withdrawal, or dependence; malaise or fatigue; organic non-psychotic disorders; organic psychotic disorders; and somatoform disorders compared with those without the specific diagnoses. The presence or absence of alcohol or drug related conditions did not modify the associations between the other psychiatric conditions and subsequent risk for TBI. Previous medication prescription categories of anxiolytics, antidepressants, and antipsychotics were also associated with increased TBI risk.

A few studies have examined the relation between emotional, behavioural, and cognitive problems and TBI in selected populations. While Brown et al¹⁸ found that children with mild head injuries had significantly more premorbid behavioural problems than children with severe head injuries or orthopaedic injuries, Pelco et al19 did not find a higher rate of premorbid emotional and behavioural problems in hospitalised children with mild head injuries compared with children in the general community. The small number of people with hyperkinetic disorder and the presence of comprehensive paediatric care at GHC may explain the lack of a significant association between hyperkinetic disorder and TBI in our study. Dicker²⁰ found that adults who sustained a mild head injury were more likely to have a learning disability before the injury than population controls. Similarly, Teasdale and Engberg²¹ found the relative risk for concussion in the presence of pre-existing cognitive dysfunction among men ages 16 to 24 to be 1.6 (95% CI 1.3 to 1.9) compared with men without cognitive dysfunction.

Previous studies have examined possible associations between psychiatric factors and accidents. Alcohol use has a dose-response relation with an increased risk of injury.11 Children with high accident liability are more likely to have had evidence of personal and social maladjustment, psychosomatic ailments, and referrals to counsellors or psychiatrists before their accidents.¹² Motor vehicle accidents are a major cause of TBI and it has been estimated that 90% of such accidents are caused by driver error.³¹ Studies have suggested that people with psychiatric problems have 1.5 to 2.5 times the risk of a motor vehicle accident compared with other drivers.¹³⁻¹⁷ Transient factors, such as stressful life events, anxiety, depression, functional somatic complaints, alcohol use, and fatigue, may interact with stable traits, such as cognitive impairment, impulsivity, antisocial attitudes, neuroticism, and external locus of control, in causing collisions.³²

The associations found between psychiatric illness and TBI are likely due to complex causal pathways that differ in the various psychiatric indicators. Alcohol, as well as other substances, places a person at a higher risk for TBI through its effects on cognition, coordination, and judgement. Our study found only a minimally significant association between alcohol and drug use disorders and TBI, perhaps in part because of the relatively low numbers of these disorders in the GHC population and the presence of comprehensive substance abuse treatment programs at GHC. People with organic mental disorders were found to be at increased risk for TBI and include those with dementia, delirium, and frontal lobe syndrome. Demented and delirious people have impaired cognitive and physical functioning, making them more prone to accidents and self injury.³³ Driving, for example, is a complex skill in which various cognitive processes such as perception, attention, reaction time, and motor control are involved.³² Cognitive deficits, agitation, impulsivity, and impaired insight and judgement associated with organic mental disorders may lead to risk taking or carelessness, which can lead to injury. Patients with psychotic symptoms, such as hallucinations or delusions, may also have similar impairments, which may lead to situations conducive to TBI.³⁵⁻³⁸

We also found that acute reaction to stress or adjustment reaction, malaise or fatigue, and somatoform disorders may lead to increased risk of TBI. These diagnoses may be proxies for general psychiatric distress, including depression and anxiety, that are modifiable risk factors for TBI. These non-specific conditions are common presentations of psychological distress in the medical setting.³⁹⁻⁴¹ Those with somatoform disorders and chronic fatigue have been found to have a high burden of comorbid psychiatric illness, such as depression and anxiety.^{13 42 43} People with depression suffer from decreased attention and concentration and slowed reaction times,44-46 which may result in motor vehicle collisions, falls, and recreation related injuries that may lead to TBI. Because depressed patients are at increased risk of attempting suicide, it is possible that some of the TBIs in this study were self inflicted. Depression is also associated with impaired overall health related functioning,47 which may result in decreased self care and increased risk of accidents. Anxiety disorders are characterised by distractibility, autonomic hyperactivity, motor tension, shakiness, and fatigue, all of which may contribute to events that cause injury.48

Our results show that use of antidepressants, anxiolytics, and antipsychotics confers significantly increased relative risks for TBI. Studies have reported that use of psychoactive medications such as cyclic antidepressants and benzodiazepines increase the risk of injurious motor vehicle collisions and falls.49-52 These and other psychiatric medications may cause cognitive and motor impairment, sedation, dizziness, and agitation, especially in the elderly.53-55 Psychostimulants may increase alertness in some patients, perhaps explaining our finding of a trend towards decreased risk of TBI. Although it is known from automated data that a prescription was filled, it is unknown how much of the medication was taken. Another consideration is whether an increased relative risk for TBI associated with prescription medication is related to underlying psychiatric illness for which the medication was prescribed or to the pharmacological effects of the medications

Psychiatric diagnosis, prescriptions filled, and utilisations were all recorded before the TBI diagnosis. Therefore, possible bias due to differential recall between case patients and control subjects was eliminated. Because of the completeness and uniformity of the database, the determination of psychiatric illness was not subject to the limitations of incomplete recall or information bias.⁵⁶ Because health care providers at GHC are not influenced by the risk that making a psychiatric diagnosis may not receive full reimbursement, as may be the case in fee for service settings, there was not likely to be reporting bias of psychiatric diagnoses due to reimbursement rates.

Limitations of this study include the paucity of information on TBI history beyond one year before the incident TBI in 1993, as well as possible lack of precision in outcome and exposure measurement. Because those of low socioeconomic status have been shown to have higher rates of TBI,³⁵ there may be residual confounding by socioeconomic status that was not controlled for in the analyses. However, insurance type, which was used as a proxy measure for socioeconomic status, was not shown to be a confounder. TBI is a risk factor for both psychiatric illness and subsequent TBI.⁶⁹ Those with prior TBI would be more likely to have a psychiatric illness, as well as a subsequent TBI, and thus our relative risks may be spuriously increased. The method used to determine psychiatric illness may have been subject to misclassification. Psychiatric diagnoses assigned by non-mental health practitioners may be less sensitive and specific than diagnoses made by mental health practitioners. Subjects could have been incorrectly assigned to diagnostic categories or may not have received a diagnosis when a psychiatric illness was present. However, this potential non-differential misclassification would likely bias effect estimates towards the null.

People with psychiatric distress often present with many unexplained medical complaints and are high utilisers of medical care.⁵⁷ Because those with psychiatric distress visit the doctor more frequently, it is more likely that a mild TBI in these patients would be recognised and recorded, while a person without psychiatric distress may not present to a GHC clinic and a TBI would not be recognised. Several studies have suggested that psychiatric patients or people with previous psychosocial problems such as depression, alcohol abuse, stressful life events, and social difficulties before their TBI may complain of a greater number of or more persistent postconcussive symptoms.^{58–60} These patients may have more frequent medical utilisation and a higher likelihood of having a diagnosis of TBI.

Despite these limitations, our results showed a significant association between pre-existing psychiatric illness and subsequent TBI. Given the impact of TBI on morbidity, mortality, and health care costs, the identification of psychiatric illness as a potentially modifiable risk factor for TBI is important for the prevention of TBI and its sequelae. Increased efforts should be made to educate medical practitioners, patients, and families about the increased risk of TBI in psychiatric populations. Discussions may address ways of decreasing behaviours that can increase the risk of TBI and increasing preventive measures, such as the use of seat belts and bicycle helmets. A better understanding of the relation between specific psychiatric factors and TBI is needed. Also, studies that differentiate the physiological effects of psychiatric medications from the underlying illness that the medications treat should be undertaken.

ACKNOWLEDGEMENT

Funded by a grant from the National Center for Injury Prevention and Control, Centers for Disease Control and Prevention.

- Presented in part at the 45th Annual Meeting of the Academy of Psychosomatic Medicine, Buena Vista, Florida, USA, November 1998. The authors thank David Rubanowice for his computer program-
- ming assistance.

Authors' affiliations

J R Fann, W J Katon, Departments of Psychiatry and Behavioral Sciences, University of Washington, Seattle, Washington, USA *K Jaffe, Departments of Rehabilitation Medicine and Pediatrics, University of Washington, Seattle, Washington, USA A Leonetti, Seattle and King County Department of Public Health, Seattle, Washington, USA

**P Cummings, Harborview Injury Prevention and Research Center, Seattle, Washington, USA

R S Thompson, Center for Health Studies, Group Health Cooperative of Puget Sound, Seattle, Washington, USA

- *Also Harborview Injury Prevention and Research Center
- **Also Department of Epidemiology, University of Washington

REFERENCES

1 Kraus JF, Black MA, Hessol N, et al. The incidence of acute brain injury and serious impairment in a defined population. Am J Epidemiol 1984;**119**:186–201.

- 3 MacKenzie EJ, Edelstein SL, Flynn JP. Hospitalized head-injured patients in Maryland: incidence and severity of injuries. Md Med J 1989:**38**:725-32.
- 4 Thurman DJ, Jeppson L, Burnett CL, et al. Surveillance of traumatic brain injuries in Utah. West J Med 1996;165:192–6.
- 5 Diamond PT. Brain injury in the Commonwealth of Virginia: an analysis of Central Registry data, 1988–1993. Brain Inj 1996;10:413–9.
 6 Kraus JF, McArthur DL. Epidemiologic aspects of brain injury. Neurol Children Strategies (Neurol Children Strategies)
- Clin 1996;14:435-50.
- NIH Consensus Development Panel on Rehabilitation of Persons With Traumatic Brain Injury. Rehabilitation of persons with traumatic brain injury. JAMA 1999;**282**:974–83. 8 Goldstein M. Traumatic brain injury: a silent epidemic [editorial].
- Ann Neurol 1990;27:327.
- Fann JR, Katon WJ, Uomoto JM, et al. Psychiatric disorders and functional disability in outpatients with required to an injuries. Am J Psychiatry 1995;**152**:1493–9.
- 10 Fann JR. Traumatic brain injury and psychiatry. J Psychosom Res 1997;43:335-43.
- 11 Vinson DC, Mabe N, Leonard LL, et al. Alcohol and injury: a case-crossover study. Arch Fam Med 1995;4:505-11
- Manheimer DI, Mellinger GD. Personality characteristics of the child accident repeater. Inj Prev 1997;3:135–45.
 Tsuang MT, Boor M, Fleming JA. Psychiatric aspects of traffic accidents. Am J Psychiatry 1985;142:538–46.
 Waller JA. Chronic medical conditions and traffic safety: review of the Cultific incursion. No. 2014;10:05:072:1412:00
- California experience. N Engl J Med 1965;273:1413–20.
- 15 Crancer A, Quiring DL. The mentally ill as motor vehicle operators. Am J Psychiatry 1969;126:807–13. 16 Eelkema RC, Brosseau J, Koshnick R, et al. A statistical study on the
- relationship between mental illness and traffic accidents. Am J Public Health 1970;**60**:459–69.
- 17 Selzer ML, Payne RE, Westervelt FH, et al. Automobile accidents as an expression of psychopathology in an alcoholic population. *Q J Stud Alcohol* 1967;**28**:505–16.
- 18 Brown G, Chadwick O, Shaffer D, et al. A prospective study of children with head injuries. III. Psychiatric sequelae. Psychol Med 1981;**11**:63–78.
- 19 Pelco L, Sawyer M, Duffield G, et al. Premorbid emotional and behavioral adjustment in children with mild head injuries. Brain Inj 1992;**6**:29–37
- 20 Dicker BG. Profile of those at risk for minor head injury. J Head Trauma Rehabil 1992;7:83–91.
- 21 Teasdale TW, Engberg A. Duration of cognitive dysfunction after concussion, and cognitive dysfunction as a risk factor: a population study of young men. BMJ 1997;315:569-72.
- 22 Saunders KW, Stergachis A, Von Korff M. Group Health Cooperative of Puget Sound. In: Strom BL, ed. *Pharmacoepidemiology*, 2nd edn. New York: John Wiley and Sons, 1994:171–86.
- 23 National Center for Health Statistics. International classification of diseases, 9th revision, clinical modification (ICD-9-CM). DHHS Publication No (PHS) 91–1260. Washington: National Center for Health Statistics, 1991
- 24 Thurman DJ, Sniezek JE, Johnson D, et al. Guidelines for surveillance of central nervous system injury. Atlanta: Centers for Disease Control and Prevention, 1995.
- 25 Thurman DJ, Finkelstein B, Leadbetter SL. A proposed classification of traumatic brain injury severity for surveillance systems. Proceedings of the annual meeting of the American Public Health Association. New York 21 November 1996.
- 26 Anon. The Johns Hopkins University ACG case mix adjustment system, documentation for PC-DOS and Unix, version 4.1. Baltimore: Johns Hopkins University School of Hygiene and Public Health, Health Services Research and Development Center, 1998.
- 27 Weiner JP, Starfield BH, Steinwachs DM, et al. Development and application of a population-oriented measure of ambulatory care case-mix. Med Care 1991;29:452-72
- 28 Starfield B, Weiner J, Mumford L, et al. Ambulatory care groups: a categorization of diagnoses for research and management. *Health Serv* Res 1991;**26**:53–74.
- 29 Henk HJ, Katzelnick DJ, Kobak KA, et al. Medical costs attributed to depression among patients with a history of high medical expenses in a health maintenance organization. Arch Gen Psychiatry 1996;53:899-904
- 30 Breslow NE, Day NE. Statistical methods in cancer research, vol 1. The analysis of case-control studies. Lyon: International Agency for Research on Cancer, 1980:192-246.
- 31 Lewin I. Driver training: a perceptual-motor skill approach. Ergonomics 1982:25:917-24.
- 32 **Elander J**, West R, French D. Behavioral correlates of individual differences in road-traffic crash risk: an examination of methods and findings. Psychol Bull 1993;113:279–94.
- 33 Trzepacz PT. Delirium: advances in diagnosis, pathophysiology, and treatment. Psychiatr Clin North Am 1996;**19**:429–48
- 34 Brody EM, Kleban MH, Moss MS, et al. Predictors of falls among institutionalized women with Alzheimer's disease. J Am Geriatr Soc 1984:32:877-82
- 35 Van den Bosch RJ, Rombouts RO. Causal mechanisms of subjective cognitive function in schizophrenic and depressed patients. J Nerv Ment Dis 1998;186:364-8.

- Schatz J. Cognitive processing efficiency in schizophrenia: generalized vs. domain specific deficits. *Schizophr Res* 1998;30:41–9.
 Cornblatt B, Obuchowski M, Schnur DM, *et al.* Attention and clinical symptoms in schizophrenia. *Psychiatr Q* 1997;68:343–59.
- Weinberger DR, Gallhoffer B. Cognitive function in schizophrenia. Int Clin Psychopharmacol 1997;12 (suppl 4):S29-36.
 Katon W, Williamson P, Ries R. A prospective study of 60 consecutive psychiatric consultations in a family medicine clinic. J Fam Pract 1981;13:47-55.
- 40 Beitman BD, Featherstone H, Kastner L, et al. Steps toward patient acknowledgment of psychosocial factors. J Fam Pract 1982;**15**:1119–26
- Sullivan M, Katon W. Somatization: the path between distress and somatic symptoms. Am Pain Soc J 1993;2:141–149.
 Russo J, Katon W, Sullivan M, et al. Severity of somatization and its relationship to psychiatric disorders and personality. Psychosomatics 1994;**35**:546–56.
- 43 Walker EA, Katon WJ, Jemelka RP. Psychiatric disorders and medical
- 43 Volter LA, kalon VJ, Jenetka K. Tsychiatic disorders and medical care utilization among people in the general population who report fatigue. J Gen Intern Med 1993;8:436–40.
 44 Cornell DG, Suarez R, Berent S. Psychomotor retardation in melancholic and nonmelancholic depression: cognitive and motor components. 1.04 April 1084 02:150-27.
 J Abnorm Psychol 1984;93:150-7
- Kuny S, Stassen HH. Cognitive performance in patients recovering from depression. *Psychopathology* 1995;**28**:190–207.
 Golinkoff M, Sweeney JA. Cognitive impairments in depression. J Affect Disord 1989;**17**:105–12.
- 47 Wells K, Stewart A, Hays RD, et al. The functioning and well-being of depressed patients. JAMA 1989;262:914–9.
- 48 American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th edn. Washington: American Psychiatric Association, 1994.

- 49 Ray WA, Fought RL, Decker MD. Psychoactive drugs and the risk of injurious motor vehicle crashes in elderly drivers. Am J Epidemiol 1992;136:873-83.
- 50 Leveille SG, Buchner DM, Koepsell TD, et al. Psychoactive medications and injurious motor vehicle collisions involving older drivers.
- and injurious motor vehicle collisions involving older drivers. Epidemiology 1994;5:591-8.
 51 Barbone P, MacAdhon AD, Davey PG, et al. Association of road-traffic accidents with benzodiazepine use. Lancet 1998;352:1331-6.
 52 Weiner DK, Hanlon JT, Studenski SA. Effects of central nervous system polypharmacy on falls liability in community-dwelling elderly. Gerontology 1998;44:217-21.
 53 Oxman TE. Antidepressants and cognitive impairment in the elderly. J Clin Psychiatry 1996;57(suppl 5):38-44.
 54 Foy A, O'Connell D, Henry D, et al. Benzodiazepine use as a cause of cognitive impairment in elderly hospital inpatients. J Gerontol 1995;50A:M99-106.
 55 Anon. Physicians' desk reference. Oradell: Medical Economics Co.

- 55 Anon. Physicians' desk reference. Oradell: Medical Economics Co, 2000.
- Stergachis AS. Evaluating the quality of linked automated databases for use in pharmacoepidemiology. In: Hartzema AG, Porta MS, Tilson HH, eds. Pharmacoepidemiology. Cincinnati: Harvey Whitney Books Company, 1991:222–34.
 Katon W, Von Korff M, Lin E, et al. Distressed high utilizers of medical care: DSM-III-R diagnoses and treatment needs. Gen Hosp Psychiatry 1990:12:355 62.
- 1990:12:355-62
- 58 Jacobson RR. The post-concussional syndrome: physiogenesis, psychogenesis and malingering. An integrative model. J Psychosom Res 1995;39:675–93.
- 59 Fox DD, Lees-Haley PR. Base rates of postconcussive symptoms in health maintenance organization patients and controls. Neuropsychology 1995:9:606-11
- 60 Fenton G, McClelland R, Montgomery A, et al. The postconcussional syndrome: social antecedents and psychological sequelae. Br J Psychiatry 1993;162:493–7.