Later investigation of head injury

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Most head injured patients arrive in the accident and emergency (A&E) department within a few hours of the injury. This article concerns the investigation of a group that has received less attention: those whose first attendance is delayed, who reattend, or develop complications after a period of observation.

Delay in the initial presentation or unplanned return to the A&E department after head injury can occur for a variety of reasons. The abuse of alcohol or drugs may lead to signs of an injury being noted only after the patient wakes up or is found still in coma long after the effects of the agent should have cleared. Sometimes seemingly trivial injury leads to attendance some hours, days, or even weeks later because of persisting or worsening symptoms.

Studies in South Africa, England, and Scotland showed that about 2–3% of A&E patients reattend after being sent home with an apparently minor head injury (only 0.2% of the total new A&E attenders). Most of these have responded to advice given at the time of discharge and have developed symptoms such as headache, dizziness, or vomiting prompting reattendance within two weeks of the injury.

In Scotland in 1979 the annual incidence of an intracranial haematoma undergoing surgery was shown to be 4.5 per 100,000 population. Data from a regional neurosurgical unit indicate that one fifth of these are pure extradural haematomas and over two thirds are intradural haematomas, which is the commoner finding at all stages after injury. A report in 1980 showed that over one quarter of surgical intracranial haematomas received surgery after three days (table 1).

Patients who return after discharge, especially those with a head injury, require careful assessment by an experienced doctor. The small number of such patients makes this a realistic aim in most A&E departments. Clinical evaluation of patients presenting late or reattending following head injury should allow identification of three groups:

1. Patients with progressive neurological symptoms or signs (new or worsening), or any evidence of neurological deterioration are likely to have significant intracranial pathology and such patients need immediate discussion with a neurosurgeon and investigation (magnetic resonance imaging (MRI) or computed tomography).

2. Patients with persisting symptoms. These include the elderly confused, suspected child abuse, epilepsy, alcohol and drug related illness, and head injury complications—for example, cranial nerve palsies, cerebrospinal fluid leak, traumatic pneumatocele, momentary positional vertigo, and hearing disturbance. They may require urgent investigation including computed tomography, assessment by appropriate specialists and/or discussion with a neurosurgeon.

3. Patients with resolving symptoms or easily explained minor symptoms—for example, due to scalp wound neuritis, neck strain, viral illness, or psychological upset. These can be allowed home after minimal investigation—for example, cervical spine radiography, with advice and early follow up arranged for physiotherapy, and to attend their general practitioner or appropriate hospital clinic.

Most patients return with symptoms that are listed on head injury warning cards, for example, headache, dizziness, vomiting, drowsiness, disturbance of vision, and fits. These cards are designed to provide advice mainly for the 24 hour period after discharge, with acute complications in mind, but responsible adults accompanying the patient on discharge should be advised of the possibility of later complications. Usually, the longer the time from injury, the less likely there will be a sudden deterioration as a direct result of the head injury. Nevertheless investigation for delayed intracranial haematoma, chronic subdural haematoma, and coincidental intracranial pathology, such as a tumour, must be considered when such symptoms develop.

Headache is the commonest symptom after head injury and may persist for many months. In a review of 271 adults with mainly mild and moderate injury who attended a head injury follow up clinic at an average of three months after injury, headache was present in 66% of patients and 13% described it as "severe". The headache was often caused by benign conditions involving the scalp or neck and there was a significant correlation between such headache and psychological symptoms.
For patients with headache there are a multitude of alternative diagnoses to be considered which may be associated with or coincidental to a recently sustained head injury. In 1989 Fodden et al reported a retrospective analysis of A&E attenders in which 130 (0.36%) had a primary diagnosis of headache of whom 17 were recorded as having a serious intracranial cause (five tumours with raised intracranial pressure, five subarachnoid bleeds, three with intracerebral haemorrhage, two with meningitis, one benign intracranial hypertension, and one subdural haematoma). 10

General assessment
As the head injury itself may not be the only cause of the symptoms it is important to carry out a good general assessment including examination of the neck and cardiorespiratory system as well as a thorough neurological assessment.

Investigations to be considered should include:
- Electrocardiography and measurement of the blood pressure in both arms. Syncope attacks could be cardiac in origin or occasionally be due to subclavian artery stenosis.
- Chest radiography; this may pick up unsuspected infection, injury, or neoplasm.
- Blood tests including full blood count and coagulation screen. Patients on warfarin have a 10 times increased chance of developing a spontaneous intracranial haematoma11 and the case has been made for lowering the threshold for computed tomography to include those on anticoagulant treatment after a minor head injury with persisting headache unrelieved by simple analgesia. 12 In a review of 253 head injured patients requiring serial computed tomograms the likelihood of delayed brain injury from new or progressive lesions was significantly increased if coagulopathy was present. Routine coagulation studies on admission and early follow up computed tomography for those with abnormal studies was advocated. 13
- Blood glucose, urea and electrolytes, liver function tests. Diabetes insipidus and mellitus can both result from head injury and hypoglycaemia must be routinely excluded.

Even mild head injury may lead to metabolic disorder. Inappropriate antiuretic hormone secretion (Schwartz-Barter syndrome) causing hyponatraemia has been reported in 4.6% of head injuries usually associated with moderate or severe head injury but occasionally after mild injury (0.6%). It is characterised by nausea, vomiting, and cognitive impairment. At sodium concentrations <120 mmol/l lateralising signs, seizures, and death may occur. 14 Chronic alcohol abuse, commonly associated with head injury, is also a cause of this syndrome which may be compounded by withdrawal symptoms.

Imaging
The major concern should be to exclude a serious complication of the head injury such as intracranial haematoma or infection. If the clinical features are at all suggestive of intracranial pathology then admission for observation and further investigation is indicated.

SKULL RADIOGRAPHY
Where a scalp wound haematoma or infection is of concern, radiographs (including tangential views) may be useful to reveal an underlying depressed fracture or foreign body.

Voss et al reviewed records of 606 patients who reattended the trauma unit at Groote Schuur Hospital in Cape Town after “minor” head injury. 1 They concluded that all patients who reattend after head injury should undergo computed tomography because at least 14% of scans can be expected to show an abnormality which in 5% of the series led to an operation: in eight for an extradural haematoma, in six for a subdural haematoma, in one for an intracranial haematoma, as well as five intracranial abscesses and seven depressed fractures. A significant predictor for operation was a vault fracture seen on a skull radiograph and this was recommended as an indicator for urgent neurosurgical referral when computed tomography was not possible. In hospitals without 24 hour access to computed tomography it was thought to be justified to observe and to give symptomatic treatment to patients with only subjective features and no fracture—if symptoms did not improve within 12 hours referral for a neurosurgical opinion was recommended. 1

In UK practice, where penetrating trauma is less common, skull radiography is not routinely advised in patients presenting with delayed symptoms after head injury. Thorough clinical assessment by an experienced clinician is essential and computed tomography, admission, or appropriate specialist follow up are likely to be much more valuable.

COMPUTED TOMOGRAPHY
This is widely available and safe in the stable patient therefore has a major role in the investigation of the later presentation of intracranial haematoma.

Chronic subdural haematoma
The initial injury is characteristically minimal and easily overlooked, the typical story is of episodic confusion, drowsiness, and headache and the clinical findings are usually of mild hemiparesis and dysphasia. The condition should be considered in all ages, it is equally common in those under 65 as in those over 65 and needs particular consideration in a patient with chronic alcohol abuse, epilepsy or a bleeding tendency, an infant, or an elderly confused patient.

The outcome is influenced not by age but by the level of consciousness on admission so that the early consideration of the diagnosis and prompt referral for investigation are important. Computed tomography is the main method of diagnosis. Difficulty may arise in the subacute phase in the first two weeks when small isodense collections may not be clearly demonstrated 15 but this situation should be detected from their compressive effects on the adjacent brain.
Delayed extradural haematoma

The increasing early use of serial computed tomography has shown that occasionally a patient with an initial normal scan later develops a “delayed” extradural haematoma. This uncommon condition (6–30% of extradural haematomas) is associated with a fracture in three quarters of cases, is usually diagnosed within two days of injury in 78% and within 13 days in 98%. Although delayed extradural haematomas mainly occur in patients who have sustained severe head injury and multiple trauma, they can occur after an apparently mild head injury with temporoparietal impact. Ashkenazi et al advocates that computed tomography should be routinely performed before discharge for head injured patients who suffer “non-resolving” headache even when there is no neurological deterioration. However, of the six cases presented in that report four were initially in coma (Glasgow coma score 7 or less) and the other two were initially drowsy or confused. Of three patients who regained full consciousness two had a temporal fracture and each developed increasingly severe headache before the successful evacuation of their delayed extradural haematoma four to six days after injury.

It would seem that the onset of severe headache or the development of increasingly severe headache a few days after mild head injury warrants computed tomography in patients without neurological signs, even if their initial scan was normal.

Delayed intracerebral haematoma

Tseng presented 32 patients with delayed traumatic intracerebral haematoma who had undergone computed tomography, in 22 because of failure to recover neurologically, and in 10 because of deterioration. The diagnosis was made at a time interval varying from seven hours to 10 days (mean three days and seven hours); six underwent surgery and 24 (75%) recovered with a good or moderate outcome at one year.

Postoperative computed tomography

The unplanned return of patients who have recently been discharged from a neurosurgical unit after severe head injury should be brought to the attention of the relevant neurosurgeon.

MAGNETIC RESONANCE IMAGING

Although MRI is now generally agreed to be the more sensitive cross sectional imaging technique of the brain parenchyma, it is poor at demonstrating bone and therefore will commonly miss skull fractures. Importantly it has serious practical limitations. Resuscitation and monitoring equipment need to be “MR compatible”. Further risks include inadvertent carriage of ferromagnetic objects into the scan room that may act as projectiles in the magnetic field. MR has been shown to be fatal for patients with cardiac pacemakers and a serious eye injury can occur if an intraocular ferromagnetic foreign body is present, for example in a steel worker.

The rigorous safety precautions are difficult to apply particularly in the confused patient. In selected patients it is of value in assessing non-haemorrhagic areas of brain injury including the posterior fossa, brain stem, and spinal cord but does not disclose additional lesions needing surgery when performed after adequate computed tomography.

Other investigations

CEREBROSPINAL FLUID ANALYSIS

In a patient suspected of a basilar skull fracture it can be difficult to confirm the presence of a cerebrospinal fluid leak. In order to encourage drainage of the fluid from the nose it may be necessary to carry out a provocation test—pressing on the jugular veins with the head down for a few seconds. To distinguish from nasal secretion or tears any fluid obtained should be sent for bacteriology and assayed for $\beta$-transferrin or the sodium chloride concentration estimated (normal cerebrospinal fluid contains sodium 137–153 mmol/l, chloride 119–131 mmol/l). Neurosurgical consultation is warranted in all patients with a cerebrospinal fluid leak. Further investigation such as thin section computed tomography to identify the site of a dural tear will usually be carried out under neuroradiological supervision.

EAR, NOSE, AND THROAT INVESTIGATION

Routine otoscopy and a simple test of hearing should help detect fractures of the temporal bone which may not be evident on initial skull radiography but can be associated with permanent hearing loss. The severity and significance of a hearing defect may not be apparent until some days after injury but requires evaluation and further investigation by an ear, nose, and throat surgeon. Injuries to the ossicular chain may be amenable to surgery. The immediate onset of facial paralysis associated with a temporal bone fracture requires early exploration with a view to repair of the facial nerve in its bony canal. Delayed onset of lower motor neurone facial weakness is likely to be due to oedema of the nerve in its bony canal and should resolve within about two months.

A common type of persisting dizziness after head injury is momentary positional vertigo. This is due to vestibular mechanism disturbance with or without a fracture and, if the patient’s general condition allows, can be reproduced by tilting the patient backwards rapidly with the head turned to the side, placing it at 30 degrees below the horizontal demonstrating nystagmus—the Dix–Hallpike manoeuvre.22 Often self limiting, the symptoms can be alleviated by a violent “liberatory manoeuvre” to free the cupula of its supposed deposits: Epley describes this “canalith repositioning procedure”.23 Sometimes vertigo is amenable to surgery—for example for those with stapedial footplate injury and perilymph leakage. Advice should be given on moving carefully especially in the dark and to be aware that exacerbation may occur with upper respiratory tract and ear infections.
ELECTROENCEPHALOGRAPHY (EEG)
The question of a seizure as a cause or consequence of the head injury may remain after clinical and radiological assessment. The eyewitness account is invaluable in making a diagnosis, as is discussion with relatives and the general practitioner, but an EEG may help confirm this diagnosis in doubtful cases. This can be arranged as an outpatient pending review in a medical or neurology clinic. A normal EEG does not exclude the diagnosis of epilepsy but various activating techniques can be used to increase the diagnostic accuracy to over 90%.

SERUM PROLACTIN
This may be helpful in the differential diagnosis of a seizure. The concentrations rise transiently (peak at 25–20 minutes) after generalised tonic-clonic or partial complex seizures but not after pseudoseizures.

LUMBAR PUNCTURE
There is no place for this investigation after head injury in the A&E department—in doubtful cases of meningitis or cerebral abscess complicating head injury computed tomography should be considered first to exclude raised intracranial pressure. If the diagnosis of post-traumatic meningitis is clear, blood cultures are taken and intravenous antibiotics can be given. In all cases a brief discussion with a neurosurgeon should prevent a patient coming on the end of a spinal needle!

A&E, short stay, or observation ward
Those staff who look after head injured patients on an A&E ward will appreciate the educational value as well as the benefit to these patients in terms of continuity of assessment. The availability of such a ward compared with a general surgery ward is likely to encourage appropriate admission with closer adherence to current guidelines. Such a ward can be efficient in terms of length of stay and economic in the use of investigations. While patients with abnormal computed tomography should be referred to neurosurgery wards the possibility of delayed intracranial haematoma even in those with a normal initial scan requires all ward staff to be vigilant.

Some patients will be considered fit for discharge before their symptoms have fully resolved. Discharge planning should include advice to relatives and further continuity of care can be provided in a review clinic.

Postconcussional syndrome
The threshold for follow up of mild head injury is important because many patients with no abnormalities on brain imaging develop delayed or prolonged neuropsychological symptoms—for example, headache, tiredness, irritability, sleep disturbance, dizziness, poor concentration, or forgetfulness that require careful assessment and management. The “postconcussional syndrome” is multifactorial in origin with organic factors chiefly relevant in the early stages whereas long continued symptoms are perpetuated by secondary neurotic developments often of a complex nature. In the opinion of one of the authors (JJS), early review by an experienced clinician can provide the necessary guidance and reassurance to speed the resolution of symptoms and help the resumption of productive activity for many patients.

Conclusion
The head injured patient who presents late produces a variety of clinical scenarios for the A&E clinician working in the department, ward, or follow up clinic. Those with progressive symptoms require immediate investigation including cross sectional imaging and a neurosurgical opinion. Those with persisting symptoms require urgent investigation and even those whose symptoms are resolving should be considered for follow up when neuropsychological sequelae are anticipated.

As computed tomography becomes more readily available on a 24 hour basis its increasing use can help to identify delayed and chronic intracranial haematoma requiring neurosurgical intervention as well as confirming brain damage that is not life threatening in patients who will benefit from careful follow up.

The selection of patients for timely and appropriate investigation, will continue to rely upon clinical skills and interspecialty cooperation.

Three questions relating to this article are listed in the box 1.

Box 1

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<thead>
<tr>
<th>Question 1</th>
<th>Cognitive impairment several days following head injury may be caused by:</th>
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<tr>
<td>A. Alcohol withdrawal</td>
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<td>B. Hyponatraemia</td>
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<td>C. Delayed extradural haematoma</td>
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<td>D. Chronic subdural haematoma</td>
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<td>E. Meningitis</td>
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<tr>
<th>Question 2</th>
<th>Chronic subdural haematoma is characteristically:</th>
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<td>A. A dark fluid</td>
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<td>B. Associated with a scalp haematoma</td>
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<td>C. Restricted to the elderly</td>
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<td>D. Demonstrated on computed tomography</td>
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<td>E. Dependent upon age for outcome</td>
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<th>Question 3</th>
<th>Useful later investigations for A&amp;E staff to consider in head injury include:</th>
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<tr>
<td>A. MRI scan in a confused patient</td>
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<td>B. Skull radiography for infected scalp wounds</td>
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<td>C. β₂ transferrin assay for cerebrospinal fluid</td>
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<tr>
<td>D. The Dix–Halpike manoeuvre</td>
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<td>E. Serum prolactin</td>
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The key references are Nath et al., Domenicucci et al., and Hadden et al.
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