Post-traumatic amnesia

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ABSTRACT

Post-traumatic amnesia is the transient state of altered brain function that may follow a traumatic brain injury. At a practical level, an individual has emerged from post-traumatic amnesia when he or she is fully orientated and with return of continuous memory. However, the clinical manifestations are often more complex, with numerous cognitive domains commonly affected, as well as behaviour. In the acute setting, post-traumatic amnesia may easily go unrecognised; this is problematic as it has important implications for both immediate management and for longer-term prognosis. We therefore recommend its careful clinical assessment and prospective evaluation using validated tools. Patients in post-traumatic amnesia who have behavioural disturbance can be particularly challenging to manage. Behavioural and environmental measures form the mainstay of its treatment while avoiding pharmacological interventions where possible, as they may worsen agitation. Patients need assessing regularly to determine their need for further rehabilitation and to facilitate safe discharge planning.

INTRODUCTION

Traumatic brain injuries are common; perhaps half the world's population has at least one such injury during their lifetime.[1] Post-traumatic amnesia frequently follows such injuries. Its duration is usually defined as the period between the head injury and the resumption of normal, continuous memory.[2] However, it is a more complex syndrome than this simple definition implies, having variable degrees of disorientation, behavioural disturbance (particularly agitation) and involving other cognitive impairments such as inattention, slowed processing speed and executive dysfunction.

The British neurologists Ritchie Russell and Charles Symonds first described post-traumatic amnesia in detail in the 1930s.[3 4] They emphasised the diffuse nature of post-traumatic neurological dysfunction and also noted considerable individual variation in the time for the amnesia to resolve. These early descriptions recognised the progressive emergence from coma, through post-traumatic amnesia, to normal consciousness.[3 4] Despite the recognition that the full syndrome comprised a myriad of cognitive, behavioural and perceptual deficits, Russell and Symonds proposed that return of memory was the most useful proxy to identify the resolution of the post-traumatic confusional state, and hence coined the term 'post-traumatic amnesia'.[3 4] Since then, various authors have argued that this restrictive implication of the term has led clinicians to focus on the amnestic impairments at the expense of the other cognitive and behavioural deficits. It has therefore been proposed that broadening the terminology to 'post-traumatic confusional state' or 'post-traumatic delirium' would better highlight the full extent of this common syndrome.[5 6]

Neurologists are frequently asked to assess 'confused' inpatients following possible traumatic brain injury or outpatients with neurological problems following previous injury. Identifying the presence and duration of post-traumatic amnesia is important both in the acute setting and retrospectively, as it influences management and prognostication. Its role in prognosis is particularly important, with studies showing that post-traumatic amnesia is the strongest indicator for return to work and outcome compared with other demographic and clinical variables.[7] The prospective evaluation of the duration of post-traumatic amnesia (using one of several available validated tools) can aid accurate assessment. However, these tools can be misleading if used in the incorrect context, for example, in someone with pre-existing cognitive impairment or in someone experiencing other causes of delirium. Therefore, it is

important that clinicians know the contents and limitations of these tools in order to interpret their results correctly. It is also important to identify potentially treatable mimics of post-traumatic amnesia to avoid unnecessary investigations and treatment.

CLINICAL ASSESSMENT

Post-traumatic amnesia can be considered as the period between a traumatic brain injury and the return of full awareness and normal memory function. In practice, someone has emerged from post-traumatic amnesia when they are fully oriented and have regained their ability to form, store and retrieve new memories. However, because permanent cognitive impairments are relatively common following a severe traumatic brain injury, it can sometimes be difficult to identify whether cognitive impairments are ongoing post-traumatic amnesia or the permanent sequelae of traumatic brain injury. In this context, detailed, serial cognitive testing, neuropsychological assessment and imaging can be helpful.

Despite the focus on anterograde memory impairments, post-traumatic amnesia implies more generalised cerebral dysfunction. Cognitively, its characteristic feature is the inability to form new memories (anterograde amnesia). The patient will not be able to recall this period after recovery, although certain islands of memory may return. During post-traumatic amnesia, there is commonly also profound retrograde amnesia (memory loss for events before the injury), but the duration of retrograde amnesia, unlike anterograde amnesia, tends to diminish during recovery. Other cognitive processes besides memory may be impaired in post-traumatic amnesia, including speed of processing, executive functions and attention.[8]

Behavioural disturbance is also common during post-traumatic amnesia, with about half of patients with severe traumatic brain injuries having increased agitation.[9] They often show increased restlessness, confusion and sometimes aggression, as well as other features consistent with a delirium, including hallucinations and sleep–wake cycle disturbance.[5] With such prominent behavioural disturbance, it is easy to misattribute symptoms to a psychiatric cause. We have managed patients initially sectioned for a presumed primary psychosis who were later found to have had a severe traumatic brain injury; their 'psychosis' was actually post-traumatic amnesia.

MEASUREMENT OF POST-TRAUMATIC AMNESIA

There are several clinically validated assessment tools to aid diagnosis, usually including a brief set of questions focused largely on orientation and episodic memory (table 1). In a recent head-to-head comparison of the Westmead Post-Traumatic Amnesia (PTA) Scale, Galveston Orientation and Amnesia Test and Confusion Assessment Protocol, the Westmead PTA Scale, with its greater emphasis on anterograde memory, showed the longest time to emerge from post-traumatic amnesia.[10] In practice, this aspect of the Westmead PTA Scale is useful, as the ability to encode new memories is crucial for engaging in and progressing with rehabilitation, as well as for day-to-day functioning.

However, these tools have limitations. Computerised tests, such as the Paired Associate Learning task, can (in a research setting) more sensitively discriminate which patients are in post-traumatic amnesia and may have a future role in clinical practice.[2 11] In addition, it is important to use and interpret these tests correctly. Many factors can affect the results, including sedative medications, psychiatric comorbidities, extracranial injuries and pre-existing cognitive impairments. In particular, falls and head injuries are more common in people with cognitive impairments,[12] and pre-existing cognitive impairments may not have been recognised or diagnosed before a fall with head injury. Thus, it is important to obtain a detailed collateral history and to review current and previous brain imaging for prior intracranial insults such as strokes, or atrophy suggesting a pre-existing neurodegenerative disorder.

Although the gold standard following head injury is a contemporaneous assessment of amnesia using clinically validated tools, neurologists commonly encounter a patient many weeks, months or even years after the original head injury, often without the detailed information needed to establish the presence and duration of post-traumatic amnesia. Although a Glasgow Coma Scale score of 13 or 14 out of 15 on admission may hint at the presence of post-traumatic amnesia, clinicians must interpret this in the context of other confounders that may cause delirium, for example, concomitant opioid analgesia use. The patient's retrospective estimation of the duration of post-traumatic amnesia (or that of a close witness) may be informative but is often imprecise. This can be confounded by issues such as reduced insight, confabulations or an incomplete collateral history.[13] Thus, although retrospective assessment may help, it cannot be relied on to give an accurate assessment for the duration of post-traumatic amnesia and should be combined with other clinical and imaging markers to help determine the severity of injury.

POST-TRAUMATIC AMNESIA MIMICS

It is often not clear if an acutely confused patient has a recent history of head trauma or not. There are clear similarities between the behavioural and cognitive impairments of post-traumatic amnesia and those of delirium/acute confusional state of any cause; indeed, some authors argue that post-traumatic amnesia should be termed post-traumatic confusional state.[5]

A history of a recent head injury and a progressive recovery of cerebral function, although with the possibility of fluctuation, strongly suggests that post-traumatic amnesia has caused the confusional state. Although there is no precise definition of 'recent', the

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Table 1 Post-traumatic amnesia assessment tools

Post-traumatic amnesia assessment tool	Tool components	Description of tool	Issues
Galveston Orientation and Amnesia Test	Nine orientation questions (eg, What is your name? Where are you now?) and five questions assessing memory (eg, When were you admitted to this hospital? How did you get here?) Generates a score out of 100 with points subtracted for errors	Evaluates both anterograde and retrograde memory and orientation. A score of >75/100 on two consecutive assessments denotes emergence from post-traumatic amnesia.	Incorporates recall of the incident leading to traumatic brain injury, which can be difficult to corroborate. Compared with Westmead PTA Scale, it provides the shortest estimation of post-traumatic amnesia. Does not assess agitation, psychotic symptoms or other areas of cognition beyond memory and orientation.
Westmead PTA Scale	Seven orientation questions (eg, How old are you? What is your date of birth?) and five delayed recall questions (eg, recall of three picture cards presented the previous day) Generates a score out of 12	Assesses anterograde memory and orientation, post-traumatic amnesia present if the score is <12 Emergence from post-traumatic amnesia is considered to have occurred if the patient scores 12 on 3 consecutive days.	Estimates a lengthier duration of post-traumatic amnesia due to its reliance on recall of objects 24 hours after priming Does not assess agitation, psychotic symptoms or other areas of cognition beyond memory and orientation
Confusion Assessment Protocol	Questions to assess seven domains of traumatic brain injury including cognition, orientation, agitation, symptom fluctuation, sleep, daytime arousal, and psychotic symptoms. Generates a score for each domain to define if it is present or not, with an overall score out of 7.	Detailed assessment combining multiple tools. Post-traumatic amnesia is present if four domains are present or three domains if one is orientation.	Infrequently used in research and therefore difficult to compare to other tools Anterograde memory is assessed after 5 min of priming and therefore not as demanding as the Westmead PTA Scale.
Orientation log	Ten orientation questions to time, place and situation Each question scores 0–3, with a maximum score of 30.	Simple measure that is quick to administer. A score of >24/100 on two consecutive assessments denotes emergence from post-traumatic amnesia.	Assesses only orientation, although it does correlate with the Galveston Orientation and Amnesia Test in assessment of length of post-traumatic amnesia.[27 28]

PTA, Post-Traumatic Amnesia.

state of post-traumatic amnesia is time-locked to the head injury itself; that is, it is most apparent directly following the head injury, or there was an immediate loss of consciousness with memory dysfunction directly following emergence from coma.[2] However, there may be no known history of a recent head injury, or the presentation of an acute confusional state may wrongly be ascribed to a head injury, or a patient may have more than one cause. It is therefore important to look for, and treat, other potential causes of delirium.

Table 2 outlines common mimics of post-traumatic amnesia. In a patient with a known recent head injury, common coexisting causes of confusion/delirium include alcohol and/or illicit drug intoxication or withdrawal, Wernicke's encephalopathy, intracranial haemorrhage (figure 1A), fat embolism from long bone fractures (figure 1B), electrolyte imbalance from the syndrome of inappropriate antidiuretic hormone secretion or cerebral salt wasting, medications, seizures or infection including intracranial abscess. In practice, a confused patient with no clear history of a traumatic brain injury needs investigation for any possible cause for delirium. Equally, confused patients with no collateral history are frequently misdiagnosed with conditions such as transient global amnesia or encephalitis; in such cases, imaging evidence of a traumatic brain injury or skull fracture can be helpful (see figure 1C).

Alcohol excess is particularly common in people with traumatic brain injury, with acute intoxication a frequent precipitant to the original accident, and the direct toxic effects of alcohol confounding the assessment. Clinicians should have a low threshold for suspecting alcohol withdrawal and empirically giving high-dose intravenous thiamine to prevent Wernicke's encephalopathy. Rehabilitation may be complicated in people with Korsakoff's syndrome or alcohol-related brain damage, where chronic alcohol misuse has caused generalised and focal atrophy, particularly in the frontal lobes, corpus callosum and cerebellar white matter.[14]

Seizures are also a risk factor and a potential consequence of traumatic brain injury. Clinicians may need to observe closely for seizure activity and consider an electroencephalogram (EEG) to investigate for possible non-convulsive status epilepticus.

In patients with a history of a lucid period followed by reduced conscious level, it is imperative to look for other causes, including delayed presentation of an intracranial haemorrhage, fat embolism syndrome, electrolyte imbalance, concomitant infection and seizure activity.

Table 2 Common differential diagnoses of post-traumatic amnesia

Category	Specific diagnoses	Important aids
Vascular	Ischaemic/haemorrhagic stroke, subarachnoid haemorrhage, subdural/epidural haematoma	Cranial imaging
Infection	Systemic infection and central nervous system infection (eg, meningoencephalitis, cerebral abscess and empyema)	Inflammatory markers, blood cultures, chest imaging, urine microscopy and culture, cranial imaging, lumbar puncture
Traumatic	Brain contusions, diffuse axonal injury, fat embolism syndrome	Cranial imaging
Metabolic	Hypoxia (eg, acute respiratory distress syndrome), electrolyte imbalance, acid–base balance disorders, thiamine deficiency including Wernicke's encephalopathy and Korsakoff's syndrome, constipation, acute hepatic porphyria	Arterial blood gas, chest imaging, urea and electrolytes, full blood count, nutritional screen, treatment trial with B vitamin replacement
Toxins/medications	Alcohol/illicit drug intoxication/withdrawal, opiates, benzodiazepines, anticholinergics	Collateral history, admission blood alcohol level, stigmata of chronic liver disease, liver function test, mean corpuscular volume, clinical signs of alcohol withdrawal (eg, tremor)
Seizures	Ictal and postictal, focal seizures with impaired awareness	Collateral history, seizure chart, EEG
Degenerative	Pre-existing cognitive impairment (eg, underlying Alzheimer's disease, Lewy body dementia and frontotemporal lobar degeneration)	Collateral history, imaging features consistent with neurodegenerative disease (eg, focal hippocampal atrophy)
Psychiatric	Acute psychotic episode, hallucinations, other acute or relapsing chronic psychiatric conditions	Collateral history—may require direct discussion with mental health team who often have separate medical record systems
Miscellaneous	Transient global amnesia	Absence of clouding of consciousness, no evidence of head injury trauma and usually complete resolution within 24 hours

It is also important to consider the mechanism of injury. The index of suspicion for post-traumatic amnesia and traumatic brain injury should be higher in people with more severe mechanisms of injury, while non-trauma-related causes of delirium may be more likely after a more innocuous injury. However, a precise history of injury mechanism is not always available, and even relatively minor appearing injuries (eg, a fall from standing height) can cause significant injuries.

Pre-existing cognitive impairment is important to consider when assessing a patient with persistent 'confusion' following a head injury, especially in those aged over 65 years. Such patients may have a period of 'prolonged post-traumatic amnesia' due to unmasking of a pre-existing neurodegenerative condition, which should prompt appropriate investigation, treatment and future care planning.

It can also be problematic to distinguish the symptoms of transient post-traumatic amnesia from permanent cognitive impairments secondary to the traumatic brain injury. The precise moment that a patient 'emerges' from post-traumatic amnesia is often unclear, particularly following a severe traumatic brain injury, which makes the use of validated scales particularly helpful (see table 1). However, if a patient fails to 'emerge' from post-traumatic amnesia on these simple scales after a couple of weeks, it is important to consider other potential causes and to question whether the traumatic brain injury has caused a more permanent impairment in cognitive function. Formal neuropsychology testing can help to diagnose an individual's persistent cognitive problems but should be performed at least 2 weeks after emergence from post-traumatic amnesia to allow residual cognitive symptoms related to the injury to resolve.

PATHOPHYSIOLOGY

The pathophysiology of post-traumatic amnesia is incompletely understood. It is striking how patients with wide-ranging intracranial scan appearances (including being completely normal) may have similar clinical phenotypes during post-traumatic amnesia. Brain network dysfunction, specifically within the default mode network, is probably central to its mechanism. One study using functional MRI showed evidence of impaired functional connectivity between the parahippocampal gyrus and posterior cingulate cortex in people with post-traumatic amnesia (figure 2).[11] Both these regions are key nodes in the default mode network and have a critical role in encoding new memories. The extent of this functional disconnection predicted performance on neuropsychological tests of memory and processing speed. Diffusion tensor image analysis also identified changes within the subdivision of the cingulum bundle that connects the parahippocampus and posterior cingulate cortex, suggesting that axonal dysfunction following traumatic brain injury may underlie this 'functional' disconnection between brain regions resulting in the clinical syndrome of post-traumatic amnesia.

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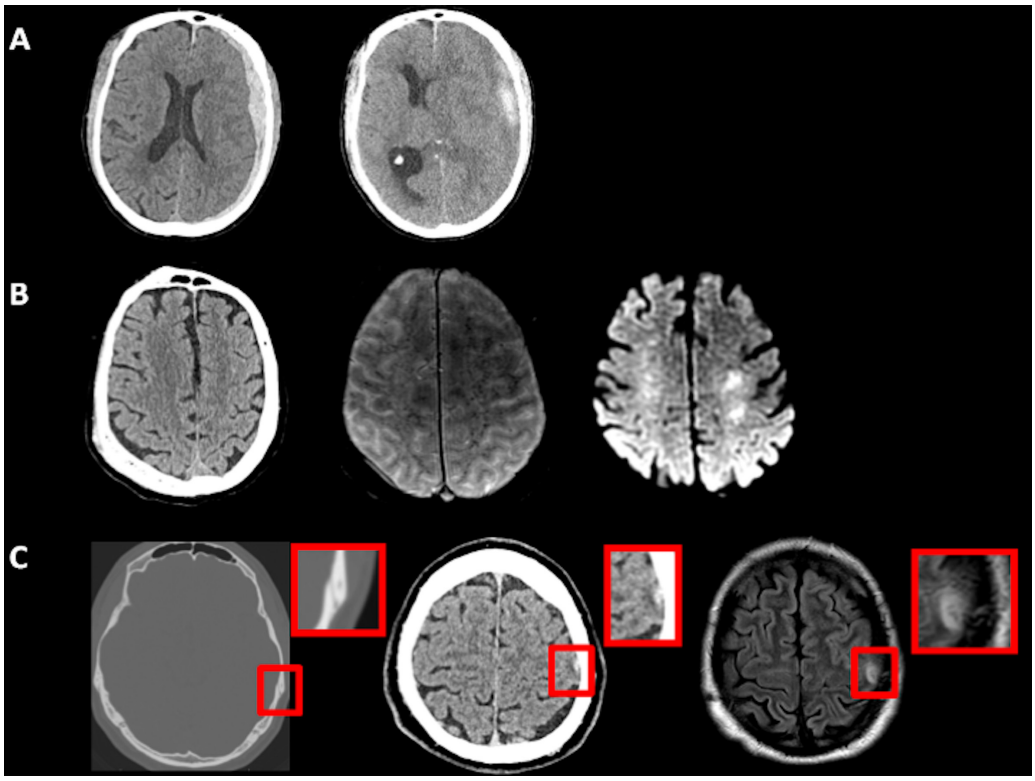

Figure 1 Examples of post-traumatic amnesia mimics and chameleons. (A) 72-year-old man with transient loss of consciousness and head injury. Initial CT scan of head showed left-sided subdural haemorrhage without mass effect. Two weeks later, there was a deterioration in conscious level with repeat imaging showing interval haemorrhage with effacement of the left lateral ventricle and midline shift towards the right. (B) An 87-year-old man who fell from a ladder and was initially lucid followed by a reduction in conscious level in the ambulance on the way to the hospital. Initial CT scan of the head (left) showed no definite intracranial abnormality. Magnetic resonance scan of the brain subsequently showed multiple small foci on susceptibility weighted imaging (middle image) and restricted diffusion on diffusion weighted imaging (right image) throughout the whole brain, caused by fat embolism from a fractured femur. (C) A 36-year-old man was brought to the hospital by a friend who found him confused and repetitively asking questions. He had last been seen well the night before. There was no obvious evidence of head injury on examination, and he was provisionally diagnosed with transient global amnesia. Subsequent cranial imaging found a subtle transverse fracture spanning the sagittal suture, extending through both left and right parietal bones (left) with evidence of underlying contusions in the left postcentral gyrus on MRI (right), which could be seen with retrospect on the initial plain CT scan of the head (centre).

CASE STUDIES

Case 1: a crash in the woods

A healthy 56-year-old man was mountain biking alone but was found confused and wandering by another group of cyclists having apparently crashed his bike. Initial imaging showed multiple rib fractures, a small splenic laceration and left renal haematoma, but a non-enhanced CT scan of the head showed no intracranial bleeding. His conscious level fluctuated with his Glasgow Coma Scale score ranging from 11 to 13 out of 15 (eyes 2–3, voice 4, motor 5–6). Repeat unenhanced CT scan was again normal. He was disoriented in time, place and person but with no focal neurological deficits. A magnetic resonance scan of the brain showed no evidence of traumatic brain injury, and in particular no microhaemorrhages on susceptibility weighted imaging to suggest traumatic vascular injury.

He was admitted to the neurointensive care unit for observation and initially declined further cognitive assessment due to pain and tiredness. On day 5, he scored 29/30 on the Montreal Cognitive Assessment, losing one point for delayed recall, and on day 6 had a normal functional assessment using a Multiple Errands Test. He was subsequently discharged.

At review 3 months after the accident, he could not recall the incident events, nor anything until waking in intensive care at four am the following day. His retrospective estimate of the duration of post-traumatic amnesia was 16 hours.

Figure 3 shows data from his global positioning system (GPS) watch. As he descends a 15 m (49') hill (T_1), he reaches a peak speed of 43.9 km/h (27.3 mph) (T_2), then decelerates to stationary over 13 s (T_3). His heart rate falls over the next minute to a baseline of 72 beats/min. His watch records no speed for 5 min, before his heart rate increases and he begins aimless wandering, seen on the right side of the map (T_4 and panel B).

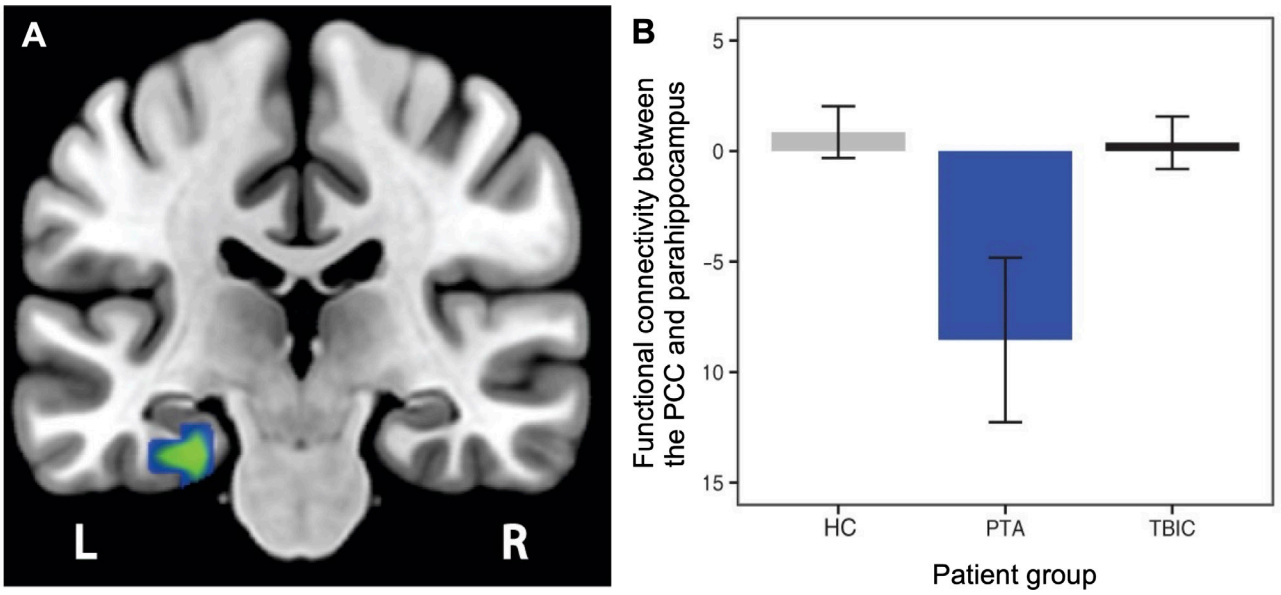

Figure 2 Functional MRI study comparing functional connectivity. (A) Voxel-wise comparison between patients with post-traumatic amnesia and healthy controls showing decreased functional connectivity in the left parahippocampus. (B) Region of interest analysis showing significantly lower parahippocampus–posterior cingulate cortex functional connectivity in patients with traumatic brain injury with evidence of post-traumatic amnesia compared with patients with traumatic brain injury without evidence of post-traumatic amnesia as well as healthy controls (modified from De Simoni *et al*[11]).

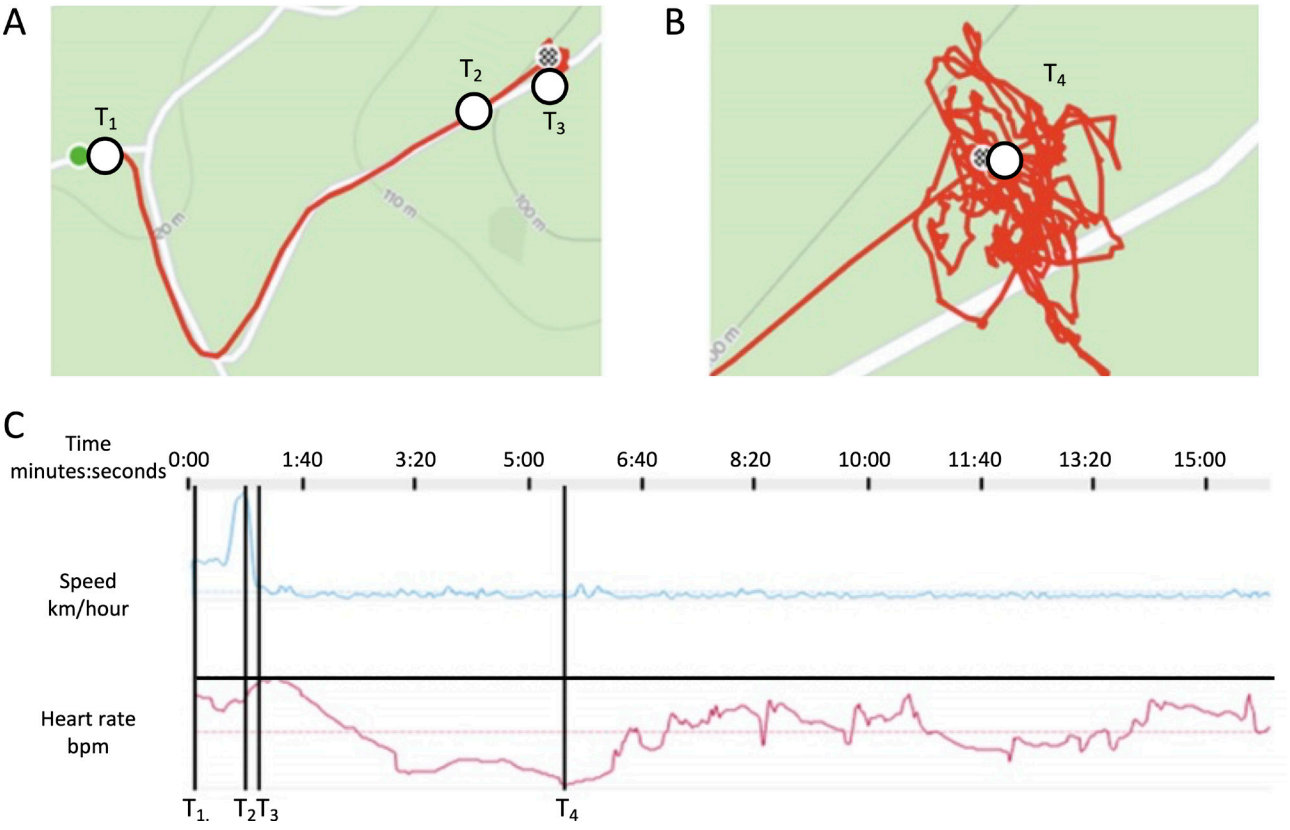

Figure 3 (A) GPS data from the final minutes of a mountain bike ride. (B) Enlarged map showing period of disoriented wandering postcrash. (C) Graph showing time, speed and heart rate during bike ride and postcrash. T_1–T_4 correspond to different time points highlighted on the maps/graph: T_1, start of ride; T_2, maximum speed reached during descent; T_3, point of crash; T_3–T_4, period with no movement; T_4, start of aimless wandering.

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These data suggest that while descending on his bike at speed, he crashed, lay unconscious/motionless for approximately 5 min, before wandering aimlessly in a state of post-traumatic amnesia.

Case 2: 24 hours in A&E

Online supplemental video 1 is taken from the programme 24 Hours in A&E and clearly demonstrates the amnesia and confusion of post-traumatic amnesia. On the first assessment, the woman cannot remember the incident and some months later bases her description on witness accounts (from her 7-year-old son). At the time of assessment, she was clearly disoriented and showed perseverative speech. In online supplemental video 2, she reports a prolonged period of anterograde amnesia and talks of her self-identified methods of dealing with her deficits.

POST-TRAUMATIC AMNESIA DURATION AS A PREDICTOR OF OUTCOME

Several studies have shown that the duration of post-traumatic amnesia can predict the likelihood of return to work. Fleming *et al*'s study of patients with a severe traumatic brain injury found that the duration of post-traumatic amnesia (measured using the Westmead PTA Scale) was the strongest indicator compared with other demographic and clinical variables (including age and premorbid function), whereas the Glasgow Coma Scale score was not predictive.[7] Several other studies have replicated this. Nakase-Richardson *et al*, for example, found that a person's odds of a return to productivity at 1 year after injury fell by 14% with every additional week in post-traumatic amnesia.[15] Furthermore, in a multicentre study with outcome data from over 10 000 patients at 1 year after traumatic brain injury, and over 6000 patients at 5 years after injury, the duration of post-traumatic amnesia was consistently the best predictor of functional outcome.[16]

While longer durations of post-traumatic amnesia do not preclude a successful return to work, some occupations are more difficult to return to due to the nature of the work, especially safety critical (eg, aircrew) or where the role centres on individual professional opinion (eg, judges). However, many people can return to work, especially after vocational rehabilitation and collaboration with the employer's occupational health specialists, potentially including a period of supervision/shadowing (successfully used for medical professionals).

MANAGEMENT

There are published guidelines highlighting recommendations for assessing and managing patients in post-traumatic amnesia (table 3).[17] However, there is only weak evidence to support specific management strategies. We clearly need further research, especially randomised control trials, to inform best treatment.

Since retrospective assessment of post-traumatic amnesia duration is potentially flawed, but its duration is an important predictor of traumatic brain injury severity and outcome, it is important to evaluate post-traumatic amnesia prospectively using a validated tool (see previous discussion).[17] Note, however, that these tools have limited sensitivity and specificity and must be interpreted in the overall clinical context.

Detecting post-traumatic amnesia is also important in determining an individual's decision-making capacity, especially regarding their ability to decide whether to stay in hospital and other healthcare-based decisions in the acute phase. In England and Wales, this is governed by the Mental Capacity Act (2005). A person's inability

Table 3 Post-traumatic amnesia management do's and don'ts based on INCOG Recommendations for Management of Cognition Following Traumatic Brain Injury, Part I: Post-traumatic Amnesia/delirium[17]

Do's	Don'ts
► Allow person to move around as freely as possible ► Maintain a quiet ward environment; ideally provide a side room. ► Promote frequent rest periods. ► Keep staff and physical surroundings as consistent as possible. ► Encourage usual routines and engagement in enjoyed activities where possible. ► Identify most reliable method of communication. ► Perform daily monitoring of post-traumatic amnesia using validated scale (eg, Westmead PTA Scale; *see* table 1). ► Provide regular reassurance that symptoms improve over time. ► Provide orienting and familiarising information, including around the injury and the need for treatment (note: avoid if causes agitation). ► Allow the patient the opportunity to see their neuroimaging. ► Evaluate potential agitation triggers and communicate with relatives how best to avoid these. ► Assess cognitive function in 'real-world' assessments, not just pen and paper tasks.	► Avoid restraint where possible. ► Avoid overstimulation (eg, television and mobile devices/tablet devices). ► Avoid sedative/neuroleptic medication unless felt clinically necessary (especially during daytime). ► Defer detailed neuropsychological testing until 2 weeks of postemergence from post-traumatic amnesia. ► Avoid giving incorrect information in order to manage agitation (ie, informing the patient they will be going home later that day).

to retain information due to anterograde amnesia during post-traumatic amnesia may be sufficient grounds to override the assumption of capacity (see Ashby *et al*[18] for further discussion). Early discharge of patients in post-traumatic amnesia should be avoided where possible as the cognitive and behavioural impairments place them at risk of harm unless there are clear and robust measures in place to prevent this.

Inpatient management of people in post-traumatic amnesia should focus on all facets of the post-traumatic confusional state, including the cognitive impairments, behavioural disturbance, fatigue and altered sleep–wake cycle. The management should focus on behavioural rather than pharmacological interventions wherever possible, with consideration of any physical or psychiatric comorbidities. In addition, other neurological issues related to the acute injury, such as acute symptomatic seizures, headaches and dizziness (eg, benign paroxysmal positional vertigo, which is common and easily treated) need proper assessment and management. Other acute medical issues include nutrition, constipation and assessment and treatment of drug or alcohol withdrawal, including Wernicke's encephalopathy.

Pharmacological treatments are sometimes necessary for people with alcohol (eg, chlordiazepoxide) or opiate withdrawal (eg, methadone). However, it is important to closely monitor and titrate dosing as appropriate, since the underlying brain injury may increase susceptibility to adverse side effects.

The behavioural disturbances in post-traumatic amnesia can be challenging and pose risks to the patient, staff and others. Common behaviours include agitation, impulsivity, perseveration, restlessness, disinhibition and aggression.[19] Although poorly understood, these behaviours appear to relate to the coexisting cognitive impairments, and often the patient is not aware of their injury and impairment. In addition, cognitive dysfunction may impair the normal processing of internal and external stimuli, leading to abnormal behaviours.[20]

Environmental measures to reduce agitation include providing quiet, consistent and calm surroundings, avoiding restraints and minimising staffing changes.[17] In addition, a structured orientation programme may help to calm the patient, including giving time and location information, providing information about the person's injury, diaries, psychoeducation around traumatic brain injury, clear information around the risks of not being treated in hospital, and familiarising cues such as photographs of friends and family.

An altered sleep–wake cycle commonly follows a traumatic brain injury. In addition to physical fatigue, the detrimental effects of poor sleep on cognitive performance are well recognised. It is therefore important to manage sleep actively following a traumatic brain injury, including those in post-traumatic amnesia. Promoting sleep hygiene measures such as a consistent bedtime routine, orientation cues and avoiding overstimulation are all helpful. In a busy hospital ward, a side room or quieter clinical area may help to optimise the environment for sleep. The side effects of most available hypnotics often outweigh their benefits. However, one option is melatonin supplementation. Serum melatonin concentrations fall after traumatic brain injury,[21] and one small randomised controlled trial showed melatonin was safe and improved sleep quality in patients following traumatic brain injury.[22]

No medications have been proven to aid recovery of patients in post-traumatic amnesia,[23] although amantadine can speed recovery of patients with post-traumatic disorders of consciousness.[24] Clinicians often consider using sedating agents in post-traumatic amnesia due to the behavioural disturbance. However, these medications may paradoxically worsen confusion and agitation, impairing functional independence and increasing hospital length of stay.[23] We therefore aim to reduce the pharmacological burden (eg, reducing or stopping opiate analgesia where possible), optimise the non-pharmacological measures discussed earlier and treat other provoking factors/causes of delirium (eg, pain or constipation). If, despite these measures, the behavioural disturbance still put the patient and others at risk, we prescribe the lowest possible dose of a sedating agent. Where possible, we avoid typical antipsychotics such as haloperidol and benzodiazepines, as they can worsen agitation and prolong the duration of post-traumatic amnesia.[25 26] Potential options include risperidone, propranolol and olanzapine, each at the lowest dose possible.

Allied health professionals have an important role in assessing and managing patients in or after post-traumatic amnesia. Clinical neuropsychology evaluation is very helpful where there is diagnostic uncertainty, as well as advising on psychological therapies and education. Nursing staff, physiotherapists and occupational therapists also have essential roles in assessing patients with post-traumatic amnesia, as they often gain insights into daily behaviour changes that may be more informative than a score on a simple cognitive test. We tend to hold a short daily meeting and a more detailed weekly meeting attended by a range of multidisciplinary team members (medical, nursing, clinical psychologists, physiotherapists and occupational therapists) to discuss the progress of individual patients in post-traumatic amnesia, and agree individualised management strategies. Headway (https://www.headway.org.uk/) is a charitable organisation that works to improve life after brain injury and is a useful resource for patients and particularly their next of kin to help understand the consequences of a brain injury.

CONCLUSIONS

Post-traumatic amnesia is often viewed as an impairment of orientation and episodic memory, but it is a far more diverse condition with complex cognitive dysfunction and frequent behavioural disturbance. It is easy to overlook that someone is in post-traumatic

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Key points

- Post-traumatic amnesia commonly follows head injury, and has important management and prognostication implications.
- It is easily missed but clinically validated assessment tools are helpful.
- It has a broad differential diagnosis; other causes of delirium should be considered and can occur concurrently.
- The management of people in post-traumatic amnesia can be challenging but, if possible, should focus on behavioural and environmental aspects rather than using medical interventions.
- The duration of post-traumatic amnesia strongly correlates with the likelihood of return to work and functional independence.

Further reading

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amnesia, despite its immediate management implications and its relationship to long-term outcome. We therefore recommend prospective evaluation of post-traumatic amnesia using validated tools. The pathophysiology underpinning post-traumatic amnesia is poorly understood, although recent brain imaging studies suggest it relates to disruption of neural networks. There is little evidence to guide the management of people in post-traumatic amnesia, but behavioural and environmental measures are recommended, with careful use of medications.

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