ORGANIC DISORDERS

Introduction

This protocol includes three sections with information about the following topics:

- Organic disorders
- Head injuries
- Cognitive impairment due to prescribed medication.

The common theme that links these conditions is the presence of impaired cognitive function.

This is of particular relevance for disability analysts, as much of what we do involves making an assessment of what a person can or cannot do.

Cognitive function enables all the various mental activities that allow us, in a practical sense, to interact with our environment. In short, it determines to a large extent, what we can, or cannot do day to day.

The information in this protocol, although comprehensive, has focused mainly on those conditions which are likely to be encountered frequently in the disability analysis setting, or where it is felt that specific guidance would be helpful.

1. Organic Disorders

As a group, these conditions share the feature that they are due to some demonstrable abnormality of brain structure or function.

Cognition is frequently affected, but they may present clinically as a behavioural or emotional disturbance. The boundaries of these conditions are somewhat blurred, but they can be broadly divided into three categories:

- Delirium
- Dementia
- Specific neuro-psychiatric syndromes.

Many of these conditions are rarities, even in the specialist clinical setting. It is beyond the scope of this protocol to include detail of these. Where a condition is likely to be encountered more frequently, or where there are specific points that merit emphasis, more information is provided.

1.1 Delirium

A short section on delirium is included for completeness. In the past, delirium was often referred to as an acute confusional state. It must be stressed that in the context of disability analysis, these conditions are unlikely to be encountered, apart from after the acute illness, perhaps when the individual is recuperating. These are acute medical conditions, and hospital admission and investigation will usually be necessary to elucidate the cause.

1.1.1 Aetiology

The list of possible causes of delirium is lengthy but can be grouped into the following categories:

- Drugs and alcohol
- Intracranial causes
- Metabolic and endocrine causes
- Systemic infections
- Postoperative states.

Delirium is more common in the elderly, and those who for some reason have reduced cerebral reserve. The latter is commonly due to pre-existing dementia, but other causes include head injury, and cerebrovascular accidents. A significant history of substance abuse also puts individuals at risk.

Amongst elderly people admitted to hospital, the prevalence of delirium is 30%. In general medical or surgical wards, 5 to 15% of patients present with, or develop delirium. In surgical intensive care units, the number rises to 20 to 30%.

1.1.2 Clinical features

The onset of delirium is acute in nature. The clinical course fluctuates with lucid intervals periodically. Relevant clinical features may include the following:

- Drowsiness and disorientation in time and place
- Perceptual abnormalities
- Anxiety
- Poor concentration and impaired memory
- Delusions/paranoid ideas.

For a diagnosis of delirium to be made the following four features should be present:

- Time course as described above
- Altered consciousness
- Change of cognition
- Evidence of a medical cause.

1.1.3 Treatment

Treatment will be of the underlying cause. Good, supportive nursing care is essential in the short term.

Anti-psychotic medication to control extreme anxiety or agitation may be required.

1.1.4 Prognosis

This is again largely dependent on the underlying cause of the delirium. Delirium is associated with a high mortality. Prompt recognition and treatment of the underlying cause increases the chance of survival.

The acute episode is likely to last something in the order of a week, but longer periods of delirium, up to a month in duration, can occur.

1.2 Dementia

Dementia is a chronic syndrome characterised by cognitive impairment **without** altered consciousness. Some specific types of dementia, which are likely to be more commonly encountered, or which have particular features are detailed at **1.2.6** onwards.

The importance of making a specific diagnosis when a patient has dementia has been recognised in recent years, due to the increasing availability of treatment options. Making a diagnosis is still largely dependent on the application of clinical tests, rather than laboratory or other investigations.[1]

For the diagnosis of dementia to be made, two or more of the following cognitive functions must be affected:

- Memory
- Language
- Abstract thinking and judgement
- Praxis (complex movement)
- Visuoperceptual skills
- Personality
- Social conduct.

The impairment will have reached a level that is starting to have an impact socially, or on the ability to function in the workplace.

1.2.1 Aetiology

There are many causes of dementia. Amongst the elderly, by far the commonest are vascular and degenerative disorders. Pre-senile dementia refers to those individuals aged less than 65 years. In younger age groups, a much wider range of possible causes needs to be considered, so that those that are treatable can be excluded. A list of possible causes of dementia is contained in Appendix A.

1.2.2 Prevalence

The prevalence of dementia increases with ageing. As the ageing population in the developed world increases, we can expect the number of people with dementia to show a significant rise. It is estimated that 5% of those aged over 65 have dementia. In those over 80, figures range from 10 to 20%.

Looking at where people live once they are over 65 shows a marked difference. In the community, 5% of those over 65 have dementia, whilst in residential or nursing homes more than 80% of over 65s have dementia.

1.2.3 Clinical features

The usual mode of presentation is poor memory, but altered personality or behaviour can also feature in the early stages. Pre-morbid personality has a large influence on the clinical features. Those with good social skills may retain comparatively good function in spite of intellectual impairment. The elderly, socially isolated and hearing impaired are less able to compensate for intellectual impairment.

The onset is often slow and insidious. Early symptoms may not be recognised by others, or may be difficult to detect. Impaired attention span or reduced concentration are common, non-specific features.

As dementia progresses, difficulty in new learning becomes more conspicuous and recent memory loss more noticeable. Patients become inflexible and less able to adapt to new situations.

When the restricted abilities become stretched beyond their capacity, the person may experience a ‘catastrophic reaction’ comprising an explosive outburst of grief or rage.

Two major behavioural syndromes are recognised, having the following features:

- Apathy, inertia and loss of interest in work and hobbies
- Restlessness, disinhibition, distractibility, and loss of empathy and social skills.

These two pictures may overlap.

Inability to self-care, and disorientation are later features. Behaviour may become stereotypical or feature mannerisms. Irritability, anxiety, aggression and depression are all common features. Insight may be retained to some degree early on. This can cause the person great distress.

Eventually, speech and thought processes become incoherent, to the point where the person can become mute.

Certain sub-types or syndromes have been described. Although there may be overlap clinically and pathologically, these are useful as clinical descriptions.

Subcortical dementia

This is characterised by:

- Slowness of thought
- Difficulty with complex, sequential intellectual tasks
- Impoverishment of affect and personality
- Retention of learning, calculation and language skills.

Cortical dementia

This is characterised by:

- Early and prominent impairment of memory
- Reduced visuospatial abilities
- Difficulty with word finding.

Semantic dementia

This variant can prove particularly disabling. There is selective, progressive loss of meaning for verbal and non-verbal material. Comprehension is impaired, and the person has great difficulty with naming and categorising. Autobiographical memory is not usually affected. Semantic dementia is associated with temporal lobe atrophy.

Differentiating between early dementia (particularly Alzheimer's disease) and age related cognitive change still relies mainly on clinical judgement. In normal ageing the following remain unaffected:

- Accuracy of responses
- New learning
- Verbal abilities.

What may be seen is a gradual slowing of the speed of cognition. Serial neuropsychometric assessments, such as the Mini Mental State Examination (MMSE) or magnetic resonance imaging may be helpful in doubtful cases. The MMSE is described in Appendix B.

In recent years, there has been a move away from looking at human cognitive neurology in terms of topographical location. A network approach is now followed. This is described in Appendix C.

1.2.4 Treatment

Dementia is an incurable condition. In recent years, drug treatment has become available which may improve certain aspects of the condition, such as memory. The drugs currently licensed for use in the UK are acetylcholinesterase inhibitors. Other drugs, such anti-psychotics may be used to control symptoms such as anxiety or agitation. However, evidence to support the continued use of drugs such as **thioridazine** is lacking, and the incidence of adverse side effects high.[2] Concern has also been expressed that anti-psychotics may in fact accelerate cognitive decline.[3] Worthwhile functional gain may result from antidepressant medication, in depressed patients.

Any medication should be prescribed with caution, and in the knowledge that cognitive impairment may be aggravated rather than helped.

1.2.5 Prognosis

Dementia is an irreversibly declining condition. In the USA, it is reported as being the fourth commonest cause of death.

1.2.6 Alzheimer's disease

The commonest cause of dementia in the elderly, it accounts for half of all cases.[4] The condition is slightly more common in women. In the UK, research studies have estimated that there are 400,000 sufferers.[5] This may be an underestimate, as The Alzheimer's Society quotes a significantly higher figure of 700,000.[6] Prevalence is expected to double over the next 50 years.[5] Currently, the point prevalence in over 65's is 2% - 7% (in those moderately or severely affected).

Aetiological factors have yet to be fully clarified. In recent years, research has identified both causative genes and genetic risk factors (the latter can increase the risk of developing the condition by up to eightfold).[7] Other known risk factors include previous moderate or severe head injury as a young adult, and Down's syndrome.[8]

Pathologically, changes in the brain include marked shrinkage of tissue, with widening of the sulci and enlargement of the ventricles. Senile plaques containing amyloid, and neurofibrillary tangles form.[9] The amount of neuropathological change correlates directly with the degree of cognitive impairment.

The onset of Alzheimer's disease is often insidious, and pre-morbid personality may modify the clinical features early on. As the illness progresses, both cognitive and non-cognitive impairments will become more evident. From the time of diagnosis, the mean life expectancy is 7 years.

Once the diagnosis of Alzheimer's disease has been made, management of the patient should focus on a number of issues. Both patient and family will require long term support from a multidisciplinary team. At some point, residential nursing care is likely to be a requirement. The trigger for this is more likely to be a behavioural aspect rather than cognitive decline.[3] For relatives, the commonest 'management' difficulties being severely disrupted sleep pattern or aggressive behaviour.

The management of this condition has changed in recent years with the recognition that the rate of deterioration can be slowed by the use of medication. The drugs used are acetylcholinesterase inhibitors, which delay the breakdown of acetylcholine released into synaptic clefts.

Rivastigmine, **galantamine** and **donepezil** are more efficacious, and have a better side effect profile than earlier drugs used.[10,11,12] Both cognitive function and activities of daily living show measurable improvement on treatment with rivastigmine.[10]

All three drugs have been approved by the National Institute for Clinical Excellence, for treatment of mild and moderate Alzheimer's disease, where the mini mental state examination (MMSE) score is over 12. Other conditions are stipulated, for example certain baseline tests must be carried out and treatment should only be initiated by a specialist physician.[13]

The potential role of statins in lowering the risk of developing Alzheimer's disease has yet to be supported by any valid research trials.[14]

Specific therapy such as Reality Orientation has been shown to benefit both behaviour and cognition, where it forms part of long term management. It can be presented continuously by a carer, or in a 'classroom' setting (using an information display board).[15]

1.2.7 Vascular dementia

In the past, this was referred to as multi-infarct dementia. The pathological changes include enlarged ventricles and multiple areas of infarction in the brain. It is more common in men and there is a strong association with hypertension.

The progression in this type of dementia is usually stepwise, with focal motor signs a common feature. In contrast with Alzheimer's disease, the cognitive impairment is not due to reduced cholinergic function.

Vascular dementia has a shorter life expectancy after diagnosis, compared to Alzheimer's disease, of 4 to 5 years. Many patients with this type of dementia are treated with aspirin. There is no research evidence to indicate that this has any therapeutic impact on the dementia.[16]

1.2.8 Dementia with Lewy bodies

It is thought that this type of dementia accounts for about 20% of cases.[17] The condition has been recognised in patients aged between 50 and 83 years, with a mean age at onset of 75 years. It is slightly more common in men.

The distinctive Lewy bodies were first seen in the substantia nigra of patients with Parkinson's disease. In patients with this type of dementia, they are found in the cerebral cortex.

This type of dementia differs from Alzheimer's disease in that cognitive impairment shows a fluctuating course clinically. Other characteristic features include marked, vivid visual hallucinations and Parkinsonism. Patients may have frequent falls and syncopal episodes.

Anti-psychotic drugs should be avoided as these patients are extremely prone to extra-pyramidal side effects. A few studies have shown cholinesterase inhibitors to be effective in treating dementia with Lewy bodies.[17] As evidence accumulates they may become first line treatments.

1.2.9 Pick's disease

This rare form of dementia usually presents earlier, between the ages of 50 and 60 years. It is slightly more common in women, and some cases are due to an autosomal dominant gene transmission.

Pathologically, the features include atrophy of the frontal and temporal lobes. Microscopic examination of the abnormal neurones reveals the characteristic Pick bodies. These are intracellular inclusion bodies that can be stained with silver.

The clinical picture with this condition is of personality change and social disinhibition. Apathy is often a prominent feature, and as the disease progresses, speech abnormalities are likely to feature.

1.2.10 AIDS dementia complex

Minor cognitive impairment is relatively common in HIV infection which can produce a wide range of neuropsychiatric disorders. It can result in neurological symptoms and dementia, both in the presence and the absence of AIDS.

The virus can directly infect brain tissue, causing AIDS dementia complex. This is usually a later feature of the illness, and occurs in around 30% of patients.

Characteristic symptoms are decreased memory and concentration, or low mood and apathy. It may be difficult to differentiate in the early stages from a depressive illness. The dementia progresses to cause global intellectual impairment with major neurological signs such as ataxia.

As might be expected with a late feature, the prognosis of AIDS dementia complex is very poor.

1.2.11 Prion diseases

There has been particular interest in these conditions in recent years, culminating in the description of Variant Creutzfeldt-Jakob disease (vCJD) in 1996.[18] All the prion diseases remain extremely rare in the UK.

The cases described who were eventually diagnosed as having vCJD were all unusually young in age, and their illnesses tended to be of longer duration compared with other prion diseases, with 50% living longer than a year. The clinical presentation was of mood or behavioural change, with cognitive impairment occurring as a late feature, along with severe cerebellar and extra-pyramidal signs.

Creutzfeldt-Jakob disease affects an older age group of 50 to 70 years, and causes a rapidly progressive dementia usually fatal a few months from onset.

1.2.12 Huntington's disease

This is an autosomal dominant condition affecting men and women in equal numbers. Onset is usually when the person is in his or her thirties, and is likely to comprise involuntary choreiform movements initially. Psychiatric features such as depression or paranoid symptoms are common. Progressive cognitive impairment appears later on in the illness. The rate at which the dementia progresses varies between individuals, but the prognosis for this incurable condition is always poor, with death the expected outcome within 15 years of onset.

1.2.13 Mild Cognitive Impairment (MCI)

In recent years, a separate clinical entity termed mild cognitive impairment (MCI) has been recognised. This comprises measurable memory loss that falls somewhere between normal ageing and mild dementia. Research evidence has shown that patients with MCI have an increased risk of developing Alzheimer's disease. There is no evidence to date to support the use of medication in individuals who may have MCI.[19]

1.3 Specific Neuro-psychiatric Syndromes

This section describes some other organic disorders, which differ in that they do not present with cognitive impairment as a global picture.

1.3.1 Focal cerebral syndromes

Shared features of these conditions are that they:

- Show more selective impairment of cerebral function
- May have specific or localised brain pathology demonstrable.

Frontal lobe

The patient may present with altered personality or inappropriate behaviour due to disinhibition. Judgement and the ability to think abstractly may be affected. They may demonstrate 'perseveration', which is difficulty in switching between tasks. Concentration and attention span will be reduced, but tests of intelligence are usually normal. The patient may have urinary incontinence.

Lack of insight is likely to be a major feature.

Parietal lobe

If the non-dominant lobe is affected there will be visuo-spatial difficulties. Difficulty with daily tasks such as dressing may be apparent. Features of dominant lobe involvement include receptive aphasia, body image disorders and right–left disorientation. Patients may deny that there is anything wrong with them, or claim that affected limbs do not belong to them.

Temporal lobe

A complex clinical picture may present. Along with specific cognitive deficits and neurological signs, there may be personality change and psychotic features. Memory impairment or language difficulties may form part of the picture.

Occipital lobe

Lesions here may present with cortical blindness, homonymous hemianopia, visual disorientation or with complex visual hallucinations.

Corpus callosum

The picture here is of acute and severe intellectual impairment.

Thalamus, basal ganglia and brainstem

Lesions here may present in a variety of ways. Personality, intellect, memory or language can all be affected.

1.3.2 Secondary psychiatric syndromes

Cerebral or systemic disease can result in a number of different secondary psychiatric syndromes. Clinical presentation may be in the form of one of the following:

- Psychosis
- Mood disorder
- Anxiety disorder
- Personality disorder
- Delusional disorder
- Organic hallucinosis – usually visual or auditory
- Obsessive compulsive disorder.

There are many possible underlying causes. More common ones include substance misuse disorders, dementias and epilepsy.

1.3.3 Memory disorders

There are thought to be multiple memory systems in the human brain.

Immediate memory – this can be tested by telling the patient a name and address (previously not known to them). After five minutes, they are asked to recall the name and address.

Recent memory – a typical day history is a good method of testing this.

Long term memory – enquiry about events prior to the presumed onset of the memory disorder will test this.

Recall is not the same as **recognition** and needs to be differentiated. Some patients may retain the ability to recognise information although that they cannot recall information.

Organic disorders usually affect the recall of recent rather than distant events. The loss is usually partial.

Another way of looking at memory, which can help in understanding memory disorders, is by dividing memory function into:

Implicit (procedural) memory

- This involves motor skills, conditional behaviours and repetition priming.

Explicit (declarative) memory

- Short term consciously accessed material. This 'store' is used for example when dialling an unfamiliar telephone number.
- Long term consciously accessed material. This 'store' is split into episodic functions for autobiographical events, and semantic functions for knowledge of the world. Episodic memory is concerned with both past events and new learning.

Causes of amnesia, or memory failure can be divided into:

Transient

- Transient global amnesia
- Transient epileptic amnesia
- Head injury
- Alcoholic blackout
- Post-electroconvulsive therapy
- Post traumatic stress disorder
- Psychogenic fugue
- Amnesia for criminal event.

Persistent

- Amnestic syndrome
- Herpetic encephalitis
- Vascular disorder
- Head injury.

Amnestic syndrome (amnesic/ Korsakov's)

This is a specific impairment of episodic memory. The ability to learn new information and to recall past events are both affected. Intellectual dysfunction is not a feature of amnestic syndrome. Social and occupational functioning are likely to be significantly affected by this condition.

A cardinal feature is that there will be evidence of a general medical condition causing the memory impairment.

A marked degree of this type of memory impairment occurs in the Wernicke-Korsakov syndrome (see protocol on alcohol). Gaps in memory may be filled by confabulation, though this is more commonly seen in delirium or frontal lobe syndrome.

The prognosis is poor if the cause is encephalitis or irreversible brain damage.

Of those with Wernicke-Korsakov syndrome, roughly half will never improve, and a quarter recover fully, with thiamine treatment.

Transient global amnesia

This syndrome needs to be excluded in those patients who present with episodic neurological and psychiatric disturbance. Memory is disturbed for a short period (less than 24 hours). Onset is sudden, and the patient may present as an emergency.

The patient remains alert and neurologically intact. Personal identity is retained (unlike in psychogenic fugue) and they may retain the ability to undertake tasks such as driving competently.

Complete recovery is usual, and further episodes do not usually occur. Transient changes in blood flow in the brain as demonstrated by functional imaging studies, are thought to be a possible underlying cause.

1.4 Main Disabling Effects

In the majority of cases, the disabling effects of cognitive impairment are significant, and there is often a huge impact on the daily life of both patient and family. Once cognitive impairment reaches a certain level, the ability to self care is affected, and increasing levels of supervision may be needed to ensure personal safety of the individual, if behavioural difficulties such as wandering develop.

The concept of mild cognitive impairment (MCI) is also of relevance for disability analysts. A recent longitudinal study comparing patients with MCI with normal subjects, demonstrated that more than 30% had difficulty with tasks of daily living such as toileting and using the telephone. In the past, MCI was thought not to be associated with any change in the ability to cope with activities of daily living.[20]

1.4.1 Assessing the Claimant

The typical day history will provide much of the evidence needed, with regard to the level of cognitive impairment. Information about memory, attention span and concentration can be gleaned from a description of the usual activities of daily living, focusing on what the person can or cannot do.

Clinicians frequently use special tests of cognitive function to assist them in reaching a diagnosis. In the context of disability analysis the use of such tests is not appropriate. A specific diagnosis is not required, and many of the tests could not be applied properly in the available time. A further consideration is that an individual who is cognitively impaired may be unduly distressed by the effort of completing the test, or when confronted by their shortcomings in undertaking the test.

Selected parts of the Mini Mental State Examination (see Appendix B) may in some instances provide useful supporting evidence for the Decision Maker, perhaps in situations where it would seem that the level of disability is less than that claimed. Such situations are likely to occur infrequently, and in most instances ample evidence will be available from the history and observations.

1.4.2 The IB-PCA

Many of those affected by these conditions will be past retirement age, and not in a position to apply for an income replacement state benefit.

Younger individuals may apply for Incapacity Benefit. Social security legislation recognises the severe disabling effects of dementia, in allowing a Decision Maker to determine exemption if documentary evidence of dementia is received. Advice from an approved medical adviser does not have to be sought by the Decision Maker in these circumstances. A significant number of cases will be accepted at the scrutiny stage, where the available evidence indicates that physical functional impairment is likely to be above the threshold.

As such, where a claimant's main medical diagnosis is a type of dementia, it is highly unlikely that they will present for medical examination during the PCA process.

If a claimant does present for examination, there may be scope for advising exemption under one of the following exempt categories:

- *Multiple effects of the impairments of the function of the brain or nervous system causing severe and irreversible motor, sensory and intellectual deficits.*
- *Severe and progressive immune deficiency states characterised by the occurrence of severe constitutional disease or opportunistic infections or tumour formation.*
- *Involving the presence of mental disease, which severely and adversely affects a person's mood or behaviour, and which severely restricts his social functioning, or his awareness of his immediate environment.*

If it is clear that a claimant has a medical condition likely to give rise to severe disability, then early consideration should be given to exemption advice.

If the examining doctor does not feel that the claimant's condition is severe enough to warrant exemption, the mental health assessment should be applied in the usual way.

All 4 areas of mental health functioning are likely to be affected, but the area of 'completion of tasks' particularly so. If the opportunity arises to obtain information from a third party, this can provide invaluable corroborative evidence, as the claimant may have limited insight into their condition.

2. Head Injury

2.1 Introduction

Although head injury is an acute problem dealt with in the Accident and Emergency setting, the after-effects and residual disability can be significant and the resultant functional impairment can at times be devastating.

Head injury may occur in isolation, but is usually seen in combination with other injuries. Head injuries can be fatal, but it must be borne in mind that the consequences of other injuries in a poly-trauma situation can be equally disastrous.

The long-term effects may include epilepsy, neurological deficits, dizzy spells, headaches, poor concentration, impaired cognition, psychiatric syndromes and hydrocephalus.[21]

The extent of medical and nursing care required over the long-term depends upon the severity of the initial head injury, the extent of recovery and the nature and severity of the residual problems.

2.2 Description

2.2.1 Aetiology

The commonest cause of head injury is road traffic accidents. Vehicular failure accounts for only 10% of the accidents, the majority being due to human failure (e.g. influence of alcohol and drugs, false judgement, tiredness and carelessness).

Falls and accidents around the home are a more common cause in children and elderly people.

Head injury is not a commonly reported industrial injury.

2.2.2 Prevalence

About 10% of the cases in an Accident and Emergency Unit will be head injuries.[22] A majority of these head injuries are minor and do not require any major medical intervention. Only about 20% of all head injuries will need admission to the hospital and 2-3% of those admitted will prove fatal.[22]

Of the deaths associated with head injury, 33 to 50% occur before admission to the hospital and about 33% occur within 24hours of admission to the hospital.[22] It must be stressed that death may be a consequence of other related injuries or complications thereof.

Over 50% of those affected are less than 30 years old and there is a male preponderance.[21]

2.2.3 Classification

Head injuries may be classified as **closed** or **open**. There is no communication between the outside environment and the intradural contents in closed injuries whereas there is leak of cerebro-spinal fluid (CSF) in open injuries.

There may or may not be an associated skull fracture. Skull fractures signify high velocity injuries and there is a higher likelihood of primary brain injury in the presence of skull fracture. The skull fracture may be linear or stellate, depressed, open or involving the base of the skull.

2.2.4 Pathology of Internal Injury

Primary Brain Injury

These are further classified into three types:

- **Concussion**: This is associated with transient alteration in level of consciousness and very minor cognitive disturbance, which is also transient. There is no residual neurological deficit.
- **Diffuse Axonal Injury**: Higher cortical functions are affected and they take a very long time to recover, if at all. There is organic and psychological dysfunction including amnesia, memory disturbance, change in personality with either depression or disinhibition.
- **Focal Brain Injury**: Includes injuries to sharply demarcated areas of brain in the region of injured blood vessels or bony prominence. Other causes include depressed skull fractures. There may be contusion, laceration, haemorrhage and haematoma at the injured site. It may lead to secondary brain injury.

Secondary Brain Injury

Further insult to the brain tissue following primary injury constitutes secondary injury and is a direct consequence of the primary injury. It is further classified into the following types:

- **Cerebral Hypoxia**: It results from reduced cerebral perfusion and reduced oxygen in the blood. Cerebral oedema and haematoma cause raised intracranial pressure thereby reducing cerebral perfusion. This causes cellular hypoxia, which potentiates the cellular oedema, and a vicious cycle is set up with rapid worsening of the condition. In addition, in cases where there is co-existent chest injury, blocked airway, respiratory depression, drug or alcohol ingestion and inhalation pneumonia, the level of oxygenation is reduced further. Hypotension due to shock further reduces perfusion.
- **Intracranial Bleeding**: This can be extradural, subdural or intracerebral. A haematoma has the potential to cause detrimental effects by direct pressure effects on the underlying brain tissue. There is also a generalised rise in the intracranial pressure with reduction in cerebral perfusion and danger of herniation or “coning” of the brain stem. About 10% of severe head injuries develop an extradural haematoma. It usually results from rupture of the middle meningeal artery and requires urgent surgical intervention.

Subdural haematoma is more common and occurs in 30% of severe head injuries. In contrast to extradural bleed, there is usually underlying brain injury and mortality is up to 50%. Intracerebral bleed results from primary brain injury and there is associated diffuse axonal injury.

- **Infection**: Meningeal infection following undetected fractures may cause secondary brain injury. Prophylactic antibiotics for open fractures may help prevent this dreaded complication.

2.3 Diagnosis

2.3.1 Symptoms and signs

History is very important in head injuries. There may be amnesia for the event. The duration of unconsciousness and amnesia are proportional to the severity of the injury.

Examination must be comprehensive to rule out other injuries. In case of the poly-trauma patients, assessment is as per the Advanced Trauma Life Support (ATLS) Guidelines.

The level of consciousness is the most important clinical finding in a person with head injury. The Glasgow Coma Scale is the best objective tool for initial and ongoing clinical assessment of people with head injuries and is also a good prognostic tool.[23] The Glasgow Coma scale is reproduced in **Appendix D**.

Accurate assessment of level of consciousness can be performed only after full oxygenation. Neurological assessment is an extremely important part of assessment of head injury. Any evidence of alcohol or drug misuse must be taken in to account.

Change in pupillary diameter is a very sensitive indicator of intracranial bleed. A developing haematoma can lead to pupillary dilatation and loss of light reflex on the same side. However, in the general population there is a finite incidence of pre-existing pupillary asymmetry and it is worthwhile establishing if there were any ocular abnormalities prior to the injury. Bilateral pupillary dilatation with loss of light reflex is an indicator of brain stem injury.

A slowing pulse and rising blood pressure with unilateral pupillary dilatation, against a background of deteriorating consciousness, are signs indicative of rising intracranial pressure.

Most fractures of the skull may be obvious only on X-rays. Clinical signs of a fracture of the skull base include periorbital haematoma, subconjunctival haemorrhage, CSF rhinorrhoea, CSF otorrhoea, and bruising over the mastoid (Battle's sign).

2.3.2 Investigations

Important observations in acute setting include:

- Consciousness (Glasgow Coma Scale)
- Pupil size and response
- Respiratory rate and pattern
- Pulse rate
- Blood pressure.

Observations are recorded on a **special head injury chart**. Further investigations include special skull X-rays and CT scan. There are recommended criteria for request of skull X-rays after recent head injury and these are tabulated in **Appendix E**. Similarly there are criteria for CT scan and/or neurosurgical consultation and these are tabulated in **Appendix E**.

2.3.3 Differential Diagnosis

The effects of alcohol, drugs, epilepsy, diabetes, hypoxia, haemorrhagic shock and other conditions that affect consciousness may all confound the assessing physician faced with a comatose patient with head injury. These co-existing conditions may be wholly, partly or not at all responsible for depression in the level of consciousness. Pre-existing ocular pathology may be responsible for pupillary asymmetry and abnormal pupillary reflex response.

2.4 Treatment

2.4.1 Acute Care and Management

For head injuries the criteria for hospital admission are listed in **Appendix E**. Those who do not merit hospital admission should be advised to spend the first night with a responsible adult and should return to the hospital if there is any deterioration in the clinical condition.

On admission, minor head injuries only need observation. A **special head injury chart** should be used to monitor progress.

Patients with minor head injuries who are fully alert and show no evidence of neurological deficits can be allowed to go home after 24 hours even in the presence of a simple skull fracture. Rest for a week is advised as some cognitive functions like concentration take a few days to recover to pre-injury levels.

All patients with severe multiple injuries are managed as per the ATLS guidelines.

The management of severe head injuries is highly specialised. One or more of the following measures may need to be instituted in cases with severe head injury:

- Intensive monitoring
- Endotracheal intubation and artificial ventilation
- Nasogastric aspiration
- Maintaining fluid and electrolyte balance
- Monitoring intracranial pressure and measures to control it (e.g. intravenous mannitol or controlled hyperventilation)

Intracranial haematoma may need to be drained surgically, especially if there is focal neurological deficit due to pressure effect from the haematoma or there is a risk of impending herniation of the brain stem.

2.4.2 Rehabilitation

Recovery from traumatic brain injury is complex and variable and can take a very long time. Initial recovery is very rapid but various functions may recover over different time scales making it a very complex problem to assess and manage.[21] Recovery is not only due to anatomical reorganisation but also due to behavioural compensation and functional adaptation.

Rehabilitation is necessarily a team approach and because of the complex recovery process it needs to be tailored to individual needs. It is imperative to involve the individual and the family as active members of the team right from the start.[21,24]

The team members (physiotherapist, occupational therapist, psychologist etc.) have to gain an in-depth knowledge of the individual in context of their pre-morbid level of functioning and abilities and this includes their personality and behaviour as well.

Although there usually is a substantial degree of spontaneous recovery, there is a need to learn to adapt to the residual problems and to get back to independent existence. New ways of performing tasks and interacting with others have to be learned.

Psychological support needs to be provided. Behavioural difficulties, cognitive and emotional problems all have to be addressed in a sensitive manner.

Goals should be set for the short and long term and these goals need to be realistic and meaningful for the individual. The therapist's goals may be worthless to the individual and compliance in such cases may be minimal at the best.

Post-concussional syndrome may present after head injury (usually severe enough to result in loss of consciousness) and includes symptoms like fatigue, memory loss, short attention span, irritability, executive dysfunction, learning problems, lack of initiative and poor insight. Loss of self-esteem and fear of permanent brain damage can lead to anxiety or depression. The aetiology of this syndrome is thought to be twofold – organic genesis with psychologically driven persistence. Some patients are hypochondriacal, adopting a permanent sick role and searching for a diagnosis and cure. Compensation motives are not always a feature. The symptoms may vary in severity and can prevent the individual from effectively functioning in their previous roles. Despite recovery, some level of residual functional loss is likely and a sensitive readjustment of career goals while addressing these problems is called for.

The ultimate aim of rehabilitation is successful integration of the individual in the community.

Severe residual problems may need life-long care in a small number of cases.

2.5 Main Disabling Effects

The disabling effects of head injury and consequent traumatic brain injury can be myriad and variable. The nature and severity of the problems and their various permutations not only depend on the extent of the injury but also on the pre-morbid characteristics of the individual.

Focal neurological deficits may never recover and can cause physical impairment of varying severity. However, this protocol deals only with the psychological aspects of brain injury.

2.5.1 Mild disability

This is usually a consequence of minor brain injury where loss of consciousness has been less than 20 minutes and posttraumatic amnesia less than 1 hour.[25]

The varying symptoms reported include headache, dizziness, fatigue, reduced speed of thought, poor concentration, poor memory, irritability, depression and anxiety. Most individuals with minor brain injury return home within a few days and the symptoms subside over days to weeks. Cognitive deficits resolve within 3 months in the majority of individuals.[25]

A very small minority has some cognitive deficit for several years after the injury. Many of the non-specific physical symptoms like headache and fatigue are assumed to result from the chronic effort to overcome or cope with the persisting cognitive deficits.

Though apparently normal, it may be difficult for these individuals to hold down jobs or maintain relationships. Ignorance about the cognitive deficits and the problems arising therefrom leads to feelings of frustration, guilt and anxiety.

It must, however, be reiterated that these long-term residual cognitive deficits are minor and present only in a small minority of individuals after minor brain injury. The majority of individuals return back to pre-morbid level of function within a period of 3 months.

2.5.2 Moderate to severe disability

This is usually a consequence of moderate to severe traumatic brain injury where unconsciousness has lasted for more than 20 minutes and post traumatic amnesia for over 1 hour.[26]

In these cases there are persistent residual cognitive and behavioural problems leading to significant functional impairment. Commonly reported long-term deficits include:

- Attention deficit / reduced concentration
- Fatigue
- Learning and memory problems
- Impaired planning and problem solving
- Concrete thinking (inability to deal with abstract concepts)
- Lack of initiative
- Inflexibility
- Dissociation between thought and action
- Impulsivity
- Irritability and temper outbursts
- Physical aggression
- Communication problems
- Socially inappropriate behaviour and disinhibition
- Self-centred behaviour and egocentricity
- Changes in affect (flat affect, inappropriate emotions and mood)
- Lack of insight

A wide range of symptoms may be reported with the resultant functional impairment varying in severity. Innumerable permutations of the above deficits and their manifestations are likely in these individuals with moderate to severe disability.

2.6 Assessing the Claimant

2.6.1 General Considerations

Poor attention and memory may affect the ability to cope with tasks and with pressure. A previously active individual may become inactive and sit for hours doing nothing. There may be a tendency to forget the risks and hazards of daily life. Poor concentration may lead to an inability to pursue previously enjoyed leisure activities.

Short attention span may be evident, as the claimant would need lots of prompting. Short attention span may also affect the ability to read.

Ability to learn and retain new skills is likely to be affected. There may be impairment in problem solving and planning. Complex tasks involving strategies, long-term planning and error checking may be affected.

Adapting to new and unfamiliar situations is difficult. Abstract thinking may be impossible. It may be difficult to generalise from a single example. The ability to understand humour and indirect language may be lost.

There may be a tendency to rely on rigid routines. Anxiety and irritability on change of routines may be a feature.

There may be a failure to control, regulate and monitor thoughts and behaviour.

Lack of initiative may lead to self-neglect and avoidance of routine tasks.

Fatigue may cause the claimant to feel overwhelmed by multiple tasks and the individual tends to leave tasks incomplete.

Behavioural problems and temper outbursts may lead to interpersonal problems and an inability to interact with people. They may also have a tendency to physical aggression. Frustration may cause irritability and a resultant preference to be left alone. There might also be problems in communication.

2.6.2 History

A detailed history of the head injury and the functional problems should be obtained. The information and evidence on the claimant's file should be noted. A careful account of the typical day should be noted. An attempt must be made to establish the level of pre-morbid function, abilities and behaviour.

2.6.3 Informal Observation

Physical and psychological deficits may become evident on observation during the assessment. Informal observation of the claimant's mood, emotions, concentration, initiative, ease of communication and insight must be made. Much valuable information may be gained by this method.

2.6.4 Physical Examination

This may be appropriate if there are any reported or observed physical functional disabilities.

2.6.5 IB-PCA Considerations

The Mental Health Assessment must always be done in individuals with functional impairment following head injury.

It must be borne in mind that such individuals may have low tolerance and a very small minority may be prone to temper outbursts and very rarely physical aggression. See the CME module **"Dealing with Potentially Violent Situations"** for detailed guidelines.

Those with severe and irreversible motor, sensory and intellectual deficits arising from traumatic injury may be exempt from the PCA if there is enough evidence to support such a decision at file scrutiny stage. However, such individuals may sometimes be called for assessment if evidence in the IB55 is lacking. If the criteria for exemption are satisfied then the disability analyst should terminate the assessment following the guidelines outlined in the CME module **"Exemption advice at the examination stage"**.

Answers to the questions in the Mental Health Assessment can be drawn from the history, account of the typical day and the informal observations made.

3. Cognitive Impairment due to Prescribed Medication

3.1 Introduction

The range of drugs available for medical practitioners to prescribe is vast, with new preparations being added to the list at a steady rate. The number of serious side effects caused by prescribed drugs is very small when consideration is given to the number of prescriptions issued to patients.

The majority of patients being treated for a disabling condition will be prescribed some form of medication by their general practitioner, and many will be on more than one preparation. Many patients report side effects which they attribute to their medication. Where the medication is needed short term, and the side effects rated as of 'nuisance value' only, the patient may be encouraged to persevere with the treatment by their doctor. If the treatment is intended to be longer term then consideration of an alternative preparation is likely to be made.

Side effects are frequently dose related, or can be expected to diminish once the patient has been taking the medication for a period of time. A good example of this is the well-documented anti-cholinergic effect exerted by tricyclic anti-depressants, which usually disappears after about 3 weeks on treatment.

Many drugs may cause cognitive side effects but the main purpose of this section of the protocol is to highlight the approach that should be taken in the context of disability analysis. Therefore, a detailed description of all individual drugs that can cause such side effects will not be included. Drugs causing specific psychiatric symptoms such as depression have been mentioned in the relevant protocol.

3.2 Specific Side Effects

Side effects, which have the potential to affect cognitive function, include the following:

Drowsiness/Fatigue: This is the commonest side effect likely to be reported by the claimant. Prescribed drugs that can cause this include benzodiazepines, opioid analgesics, anti-histamines, major tranquillisers and anti-depressants. It would be expected that some of these drugs would only be prescribed in the short term (benzodiazepines), or intermittently (anti-histamines).

Confusion: This is less likely to be reported but is noted as a possible side effect of many prescribed drugs. It can arise as a side effect of all the anti-epileptic drugs in common use, benzodiazepines, opioid analgesics, anti-histamines, major tranquillisers, corticosteroids, thyroxine and anti-depressants.

Impaired memory and/or concentration: Few drugs have been documented as causing these side effects. Lithium is one example where impaired memory is reported with long term use. Other drugs include buserelin, zolpidem and some anti-epileptics.

Irritability: Though primarily not a direct cognitive effect, it can lead to mild impairment of concentration and is not uncommonly reported by claimants. Drugs most likely to cause this are anti-epileptics, anti-depressants, naltrexone, acetazolamide, and aminophylline.

3.3 Assessing the Claimant

Enquiry about specific daily activities may be helpful. For example, it is useful to know whether the claimant has been advised by their GP not to drive or operate machinery. It would be highly unusual for medication causing significant cognitive impairment to continue to be prescribed without clear therapeutic benefits or alternatives being tried. A detailed recent drug history should be taken.

In some instances it may be extremely difficult to ascertain whether a claimed side effect is due to prescribed medication or the underlying condition. This is particularly so with symptoms which are more difficult to define or quantify, such as fatigue or drowsiness. In a number of instances, both the condition and the treatment can make the patient feel tired and lacking in energy. This should not influence unduly the functional assessment made, but may require a brief explanation for the decision-maker if the cause of cognitive impairment is unclear.

3.4 IB-PCA Considerations

In the context of the IB-PCA, significant cognitive impairment due to prescribed medication is likely to be encountered very infrequently.

Enquiry about the side effects of medication is a requirement in all IB-PCA assessments. Where a claimant reports a side effect that may give rise to cognitive impairment, the disability analyst should complete a Mental Health Assessment as a matter of course.

The usual approach must be taken when applying the Mental Health Assessment. Evidence from all available documents, clinical history, account of a typical day, informal observations and examination of the mental state will all contribute to descriptor choice. Careful attention should be paid to variability, when assessing the cognitive side effects of symptoms.

Cognitive impairment can affect all 4 areas of mental functioning, but where significant it would be expected that there will be particular impact on the ‘completion of tasks’ section.

An examining doctor may feel that a claimant's history of cognitive impairment due to medication is implausible. This may be because there is a strong consensus of medical opinion about the possible side effects arising from the particular drug. If it is decided not to proceed with the Mental Health Assessment, the justification should include pertinent information about the drug, and relevant features of the mental state to support that decision.

In doubtful cases it is always best to apply the Mental Health Assessment so that clear advice can be formulated for the Decision Maker.

Appendix A - Causes of Dementia

There are many possible causes of dementia. These can be usefully grouped into the following categories:

- Primary degenerative
- Genetically determined
- Vascular
- Traumatic
- Inflammatory, autoimmune or infectious
- Metabolic, endocrine or toxic
- Vitamin and other nutritional deficiency
- Neoplastic, post-radiation or post-anoxic.

Appendix B - Mini Mental State Examination

This is one of the most widely used tests of cognitive function. It can be particularly helpful in monitoring or following cognitive performance over a period of time when applied at intervals to an individual. It has also been used extensively in the research environment as a method of evaluating the effects of specific treatments for dementia. A number of standard questions are asked, with points accrued for correct answers up to a maximum of 30. The NICE guidelines stipulate a MMSE score of at least 12 before certain drugs can be prescribed.

Orientation

The patient should be asked if they can recall the following facts:

1. Year
2. Season
3. Date
4. Day
5. Month
6. State (in the UK the country or region might be used)
7. County
8. Town or city
9. Hospital (or name of the building in which the interview takes place)
10. Floor.

Registration

Tell the patient the name of 3 objects (at one second intervals).

Ask the patient to repeat them (score one point for each correct answer). Repeating the objects until they are learnt.

Attention and calculation

Serial ‘7’s scoring one point for each correct answer, stopping after 5 answers.

The patient can, as an alternative be asked to spell WORLD backwards.

Recall

Ask the patient to recall the '3' objects, scoring one for each recalled.

Language

Ask the patient to name a watch and a pencil as you point to them. (2 points)

Ask the patient to repeat the phrase 'No ifs, ands, or buts'.

Ask the patient to follow a three-stage command. For example – 'Take a paper in your right hand. Fold the paper in half. Put the paper on the floor.(3 points in total)

Ask the patient to read and carry out the following instruction. CLOSE YOUR EYES.

Ask the patient to write a simple sentence of their choice, containing a subject and an object. The sentence should make sense, but spelling errors can be discounted.

The last part of the test involves asking the patient to copy a diagram of two 5 sided figures overlapping by one corner each. The figures should have sides of 1.5 cm in length, and the intersecting sides should form a quadrangle. If all sides are preserved, and the intersecting sides form a quadrangle, one point is added to the score.

Appendix C - Neurocognitive networks

Traditionally, cognitive function was mapped topographically. These days the concept of neurocognitive networks within the brain has widespread acceptance. The five main networks are as follows:

- Right hemisphere, posterior parietal cortex and frontal eye fields for spatial awareness.
- Left hemisphere including Broca's and Wernicke's areas for language.
- Hippocampus, amygdala and cingulate cortex for memory and emotion.
- Prefrontal and posterior parietal cortex for working memory and executive function.
- Temporo-parietal and temporo-occipital cortex for face and object recognition.

Appendix D - Glasgow Coma Scale

	SCORE
EYE OPENING	
Spontaneous	4
To verbal command	3
To pain	2
None	1
MOTOR RESPONSE	
Obeys Commands	6
Localises pain	5
Flexion withdrawal to pain	4
Abnormal flexion (decorticate)	3
Extension to pain (decerebrate)	2
None	1
VERBAL RESPONSE	
Orientated	5
Confused conversation	4
Inappropriate words	3
Incomprehensible words	2
None	1
SEVERITY OF INJURY INDICATED BY SCORE AT ASSESSMENT AFTER RESUSCITATION	
Severe head injury	3 - 8
Moderate head injury	9 - 12
Mild head injury	13 - 15

Appendix E - Criteria for Skull X-ray, CT scan and hospital admission after head injury

Criteria for Skull X-ray after head injury (Based on guidelines of the working party on head injuries, Royal College of Surgeons of England).

1. Loss of consciousness or amnesia at any stage.
2. Any neurological symptoms or signs.
3. Cerebrospinal fluid or blood emanating from the nose or ear.
4. Suspected penetrating injury or marked scalp bruising or swelling (simple scalp laceration alone is not a criterion for skull X-ray)
5. Alcohol intoxication.
6. Difficulty in assessing the patient (e.g. young patients or epileptics).

Criteria for CT scan and / or Neurosurgical referral after head injury (Based on guidelines of the working party on head injuries, Royal College of Surgeons of England).

1. Fractured skull in combination with any of the following:
   - Confusion or depression of conscious level
   - Focal neurological signs
   - Fits or seizures.
2. Confusion or other neurological disturbance persisting for more than 12 hours, even if there is no skull fracture.
3. Coma continuing after resuscitation.
4. Suspected open fracture of the vault or base of the skull.
5. Depressed skull fracture.
6. Deterioration of conscious level or other neurological signs.

Criteria for hospital admission after head injury (Based on guidelines of the working party on head injuries, Royal College of Surgeons of England).

1. Any depression of conscious level or confusion at time of examination (a short period of unconsciousness or amnesia by itself is not a criteria for admission).
2. Severe headache or vomiting.
3. Any neurological abnormality
4. Skull fracture
5. Difficulty in assessing the patient (e.g. alcohol intake, children, epileptics)
6. Homeless or those living alone (i.e. inability to discharge for overnight care by responsible adult).

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