

# Organic Psychiatric Disorders

## ***Introduction***

The central clinical feature of most organic psychiatric disorders is impaired cognitive functioning. Cerebral dysfunction can however also cause organic mood states, personality change as well as organic psychotic and even neurotic states. The cerebral dysfunction may be caused by a disruption of brain structures or by alterations in neurophysiology. A vast array of medical and surgical conditions are capable of producing this disruption, they may be systemic or originate within the brain.

The disorders most frequently seen by psychiatrists are the neurodegenerative conditions associated with brain ageing which cause the chronic organic brain syndrome of the dementias (dealt with in the Psychiatry of Old Age section). Delirium, an acute organic brain syndrome is frequently encountered amongst medical and surgical inpatients.

## ***Overview***

### **General Principles**

Cognitive impairment can be broken down into deficits in attention, orientation, memory, intelligence, and higher executive functions. The pattern of deficits is affected by whether the pathology is acute or chronic and whether it is diffuse or focal .

In assessing a patient where an organic psychiatric disorder is suspected a standard assessment of mental state should be carried out and in addition greater attention paid cognitive function testing and other specific details.

### **History**

An informant may be needed to allow a full and clear history to be elicited.

### **Mental State Examination**

**Appearance and Behaviour:** Look for evidence of deterioration in self-care or dressing dyspraxia, repetitive or perseverative behaviour.

**Speech:** Look out for perseveration, impaired fluency, evidence of receptive or expressive dysphasia.

**Thought:** Content may be impoverished with loss of abstract reasoning. Delusions If present are likely to be simpler, less systematised and less well maintained than in a functional disorder such as schizophrenia.

**Perception:** Look for evidence of misinterpretation, illusions and hallucinations in any modality. Visual hallucinations may be vivid, colourful and detailed, size distortions may occur and frequently they are more troublesome in poorly lit environments.

**Cognitive testing:** This is central to the examination.

**Concentration.** The capacity for sustained attention and affects performance of all other tests. Look for evidence of distractibility, fluctuating alertness, difficulty grasping elements of conversation. Formal tests include, counting backwards from 20 or the months of the year backwards.

**Orientation** is assessed in time, place and person. Time: Test for orientation for time of day, day of week, month, year, an estimate of the length of time of the interview up to that point and the sequence of recent events. Place: Enquire about the name and location of the place where you are, but more importantly type of place e.g. home, hospital, day centre. Person: Can the patient identify surrounding persons and indicate their status e.g. doctor, spouse, friend.

## Memory

- **Immediate Memory:** This includes registration of new information and can be tested by digit span and registration of a word list or name and address.
- **New Learning:** The learning of new information (retention and recall). To test formally 5 minute recall of name and address or a word list. Impairment of new learning is often the first deficit noted in a dementing process.
- **Remote Memory:** This should be assessed for personal and impersonal information. It covers anything that has been learned separate from the time of testing. Ask about recent history/events, personal history, general knowledge. In dementia memories are lost in the order of most recent to most distant (Ribots law), consequently demented individuals often seem to be living in the past. Where a discrete neurological event has occurred the concepts of anterograde and retrograde memory are important.

Formal tests for parietal and frontal lobe function should also be performed but are not described here.

## Acute v.'s Chronic

	<b>Acute</b>	<b>Chronic</b>
<b>Example</b>	Acute confusional state secondary to drug intoxication, cerebral anoxia, liver failure etc.	The dementias e.g. Senile Dementia of the Alzheimer's Type.
<b>Onset</b>	Rapid	Insidious
<b>Course</b>	Usually short lived, fluctuating picture	Chronic, progressive course
<b>Consciousness</b>	Impaired	Clear
<b>Perception</b>	Usually altered causing misperception or hallucinations	Hallucinations may occur in the later stages
<b>Sleep</b>	Sleep-wake cycle often disturbed	Sleep-wake cycle usually intact
<b>Memory &amp; Orientation</b>	Impaired	Impaired

A brain suffering an acute insult is likely to show signs of impaired consciousness. The degree of impairment varies from slight problems with concentration and attention to coma.

## Focal v.'s Diffuse

Organic mental states are caused by both focal pathology (e.g. tumour, stroke) and diffuse pathological conditions (e.g. Alzheimer dementia, generalised cerebral anoxia etc.). However focal lesions may cause general cerebral dysfunction and hence cause global cognitive impairment, as may systemic disease.

There is considerable localisation of brain function. Hence a discrete lesion confined to the posterior part of the inferior frontal gyrus on the dominant lobe (Broca's area) will cause an expressive dysphasia, and a lesion confined to the posterior part of the superior temporal gyrus (Wernicke's area) causes a receptive dysphasia. When widespread cortical neurodegeneration occurs, as is the case in dementia, these brain areas are frequently affected as well and language functions are often impaired. Language deficits occurring in dementia are quite variable and various combinations of expressive and receptive deficits may occur.

## Definitions

- **Aphasia** (c.f. dysphasia): failure of language output (may be motor, sensory, syntactical, nominal, receptive, expressive).
- **Agnosia**: failure to identify or recognise objects despite intact sensory function.
  - Anosagnosia**: failure to recognise ones own physical disability
  - Autotopagnosia**: misidentifies parts of own body
  - Topographical agnosia**: unable to orientate to familiar geography
  - Hemisomatagnosia**: ignores half of body
  - Tactile agnosia**: unable to recognise letters or shapes traced on hand
  - Astereognosis**: does not recognise familiar objects by touch alone
- **Apraxia**: failure to carry out motor function despite intact sense organs and peripheral motor system.
  - Constructional apraxia**: unable to copy design using pen/matchsticks, blocks etc. (may be a visuospatial agnosia).
  - Dressing apraxia**: unable to dress self.

## Localisation of Cortical Function

For convenience this has been organised per lobe:

### Frontal lobe.

The frontal lobe connects with motor and sensory areas and the limbic system.

	Functions	Dysfunctions
<b>Motor area</b>	Controls contralateral movement.	Contralateral spastic paresis.
<b>Broca's area</b>	Speech	Motor aphasia
<b>Prefrontal area</b>	Critical for personality, abstract thought, judgement.	"Frontal lobe syndrome": disinhibited, facetious humour, apathy, distractible perseveration, urinary incontinence.

### Temporal lobe.

	Functions	Dysfunctions
<b>Hippocampus</b>	Episodic memory	Bilateral lesions cause an amnesic syndrome.
<b>Wernicke's Area</b>	Comprehension of language	Sensory aphasia

### Parietal lobe.

Functions	Dysfunctions
Receives and identifies tactile information.	Cortical sensory loss e.g. (impaired two point discrimination).
Processes visual and auditory sensations.	Agnosias and apraxias. Dominant: right-left orientation, literacy and numeracy impaired.
Planning and sequencing of motor acts.	Non-dominant loss visuospatial and body awareness, L-spatial neglect, anosagnosia and autotopagnosia.

### Occipital lobe

Functions	Dysfunctions
Interprets visual images.	Disturbed spatial orientation. Visual illusions and hallucinations. Cortical blindness.

## **Delirium**

Delirium (a.k.a. Acute Confusional State) is an organic mental syndrome characterised by acute onset and fluctuating course. It occurs in the context of physical illness. Its duration is brief and the prognosis (either death or recovery) depends on the underlying physical condition.

## **Epidemiology**

10-20% of hospital inpatients, in general wards, manifest some degree of delirium.

Elderly people seem particularly likely to develop confusion in response to a wide range of stimuli - either physical insults or sudden social change. This presumably reflects the reduced ability of the aged brain to cope with such events, particularly if it is additionally damaged by a dementing process. An acute confusional episode may sometimes be the first evidence of an underlying dementia

## **Clinical Features**

The cardinal feature is impaired consciousness. This may manifest as impaired or fluctuating attention.

- Disorientation (time, place and person)
- New Learning impaired.
- Disorganised thinking. Conversation may be rambling or incoherent.
- Perceptual disturbances are frequent misinterpretations, illusions and hallucinations. (Benign stimuli may be misinterpreted as threatening).
- Diurnal fluctuation.
- Sleep wake cycle disturbed.
- EEG shows diffuse slow wave activity.
- In elderly people apathy, under-activity and clouding of consciousness are more common presentations of delirium, than the florid, overactive restless, hallucinating states usually described in relation to younger patients.

Acute confusion should be regarded as indicative of underlying disease and investigated medically. Untreated it has a 40% mortality rate.

## **Aetiology**

High risk groups include the very young and the elderly, individuals with pre-existing organic brain disease (e.g. dementia), alcohol and drug abusers.

Cause is usually multifactorial e.g. in postoperative delirium such factors as patients age, the stress of surgery, pain, insomnia, medication, electrolyte imbalance, fever, infection and the dim ward lighting, may all be contributory.

Intracranial causes include:

- Epilepsy and post ictal states
- Brain tumour
- Infection
- Haemorrhage.
- Extracranial causes include:
- Drugs (medical and recreational).
- Endocrine dysfunction.
- Disease of non-endocrine organs e.g. heart, liver, kidney or respiratory failure.
- Deficiency states e.g. thiamine deficiency.
- Electrolyte imbalance.
- Withdrawal states - e.g. delirium tremens.
- Operations, e.g. (black patch delirium following eye operations).
- Catastrophic social situations, e.g. move into residential care

## **Assessment**

A full physical assessment mandatory.

Sometimes it is difficult to distinguish delirium from dementia but if in doubt assume it is delirium so that an immediate and vigorous therapeutic response is ensured.

## **Treatment**

Identify and treat cause.

Nursing care. Provide reassurance and explanation. If sensory deprivation is a factor improve lighting conditions.

A neuroleptic such as haloperidol may calm an agitated, restless patient and benzodiazepines are useful in the treatment of some withdrawal syndromes.

## ***Amnesic Syndrome***

A syndrome of memory loss with relative sparing of intellect and personality. The memory deficit is mainly of new learning.

## **Clinical features**

New learning is lost but immediate memory is intact enabling the patient to carry a conversation. Insight is often lacking and the patient may compensate for the failure of memory by confabulating. The individual cannot recall events that occurred subsequent to the time of onset of the illness (anterograde amnesia).

The onset is usually sudden and once established treatment brings improvement only to a minority. It is usually a persistent disorder.

## **Aetiology**

Thiamine deficiency due to chronic alcoholism, malnutrition, hyper-emesis gravida, Ca stomach etc. (Where thiamine deficiency is the cause the syndrome may be preceded by Wernicke's encephalopathy which is characterised by confusion, ataxia, ocular palsies, nystagmus and peripheral neuropathy. This condition passes after a number of weeks often leaving an amnesic syndrome. The term Korsakoff's syndrome is often used in this context.)

- Herpes encephalitis.
- Tumours.
- Trauma.
- Subarachnoid haemorrhage.
- Carbon monoxide poisoning.

## **Pathology**

Punctate haemorrhage in the mamillary bodies and diencephalon are found in the Korsakoff syndrome.

Bilateral hippocampal damage, as occurs with post-encephalitic states.

## **Treatment**

Treat the underlying pathology.

Prevent Wernicke-Korsakoff syndrome by treating alcoholics with large doses of thiamine and other B vitamins. (Remember that there are other causes of Wernicke-Korsakoff syndrome and consider treating individuals with these disorders too.)

Supportive measures once the syndrome has become fixed.

## ***Psychiatric Consequences of Epilepsy***

Epilepsy is a common neurological disorder having a prevalence of around 1%. It is estimated that between 30-50% of epileptics have significant psychiatric difficulties. The incidence of psychosis and affective disorders is high in this population and personality disturbance is also more common than in the general population. Epileptics are at greater risk of developing schizophrenia particularly those with temporal lobe epilepsy. Personality disturbance is also more common in the group with focal temporal lobe seizures. Aggressiveness of an explosive nature is characteristic and libido is often reduced. Impulsiveness, moodiness and suspiciousness have been described but these findings are open to criticism.

## **Classification of Epilepsy**

### **Definition**

Epilepsy is a brain disorder characterised by recurring excessive neuronal discharge, manifested by transient episodes of motor, sensory or psychological dysfunction, with or without loss of consciousness or convulsive movements. The seizure is associated with marked electroencephalographic (EEG) changes.

### **Classification**

Current classification follows the following structure:

#### **Generalised seizures**

These involve all brain structures and may be primary (generalised from the outset) or secondary (arising secondary to a partial seizure).

- **Tonic-clonic** (grand mal)- either phase can occur alone. In the tonic phase respiratory arrest, tongue biting and bladder emptying can occur.
- **Absence** (petit mal) - transient loss of consciousness with retention of postural tone, lasting a few seconds, More common in childhood. Automatic movements in 80% of patients.
- **Myoclonic**
- **Atonic**

#### **Partial seizures**

The seizure originates in a focal area of cortex or sub-cortex. They may become secondary generalised seizures.

- **Simple partial seizures** - marked by simple motor or sensory symptoms **without** impairment of consciousness.
- **Complex partial seizures** - there is disturbance of consciousness. Most commonly temporal lobe in origin (TLE) but may be frontal. Two types of onset; simple partial onset or impaired consciousness at onset. Automatisms may be present.

## **Psychiatric complications of epilepsy:**

### **Peri-ictal.**

The period surrounding the seizure.

**Precipitation of seizures** - precipitants can be external (reflex epilepsy; photic epilepsy) or internal (stress; anxiety, hyperventilation; fatigue; sleep loss; withdrawal from drugs such as benzodiazepines, antiepileptics, alcohol; drugs which lower the seizure threshold such as antidepressants, neuroleptics).

**Prodrome** - changes in mood (particularly dysphoria and irritability) may appear minutes to days before a seizure and are not directly related to seizure activity.

**Auras** - these represent the initial focal onset of the seizure, last only seconds to a few minutes and typically have a stereotyped form. In temporal lobe epilepsy (TLE) these may include hallucinations in any modality, epigastric sensations, déjà or jamais vu. These can comprise the sole feature of a complex partial seizure.

**Ictal** - automatisms can occur when the seizure starts in the periamygdaloid region and spreads bilaterally. 80% last less than 5 minutes and ictal automatisms never last more than 1 hour. More protracted automatisms can occur due to complex partial status.

**Post-ictal** - some degree of post-ictal confusion is common after any generalised seizure. Examination would reveal confusion, reduced attention, disorientation and impaired co-ordination.

### **Inter-ictal.**

The period between seizures.

**Schizophreniform psychosis** - this has been described as a psychosis with characteristic features of schizophrenia, on average starting some 14 years after the onset of epilepsy. Visual hallucinations appear more common than in schizophrenia. Incidence may be around 2% of epileptics, the majority having left temporal lesions. It appears to be a non-specific effect of underlying brain damage rather than directly due to seizure activity.

**Affective psychosis** - in epilepsy overall there is a 5-fold increase in the risk of suicide, but in TLE this figure rises to 25-fold.

**Non Epileptic Attack Disorder** - previously known as pseudoseizures, these are attacks which may be mistaken for epilepsy but are not of epileptic origin. Most patients have or have had epilepsy. Clinical pictures include atypical pattern of the attacks, rarity of injuries or incontinence (note these do happen in this disorder). Aetiologies include previous sexual abuse, hyperventilation syndrome etc.

**Cognitive impairment** - this can be temporary, due to continuing seizures plus/minus the effects of antiepileptic medication, or progressive, probably due to brain injury from continuing seizure activity.

**Personality** - whilst an "epileptic personality" has been described in older literature this construct appears to have little actual validity. Irritability and impulsive behaviour (including aggression and violence) may be part of an episodic dyscontrol syndrome secondary to complex partial seizures.

## **Psychiatric Consequences of HIV Infection**

The mental disorders seen in people with HIV infection are similar to those that occur in individuals suffering from other potentially fatal conditions that have an unpredictable course. They include a range of normal psychological reactions (such as shock, denial and distress), as well as abnormal responses (such as suicidal behaviour and major depression). There are, however, differences in comparison with other disorders, due to features specific to HIV infection:

Brain-related complications of HIV can give rise to organic psychiatric disorders, including dementia.

Individuals with HIV infection are at increased risk of developing mental health problems (there is often a history of psychological and social difficulties prior to acquiring HIV).

The social stigma associated with HIV and AIDS often adds to the problems faced by those trying to adjust to the physical consequences of the condition.

Identification of factors associated with the development of significant psychiatric morbidity should help in recognising those at risk and in providing effective interventions. Such factors include:

- HIV-related factors - notification of HIV infection, decline in health, AIDS diagnosis, disfiguring or disabling symptoms.
- History of psychiatric problems.
- Lack of social supports.
- Avoidance and denial as a habitual way of coping.
- Exposure to grief due to AIDS, and other adverse life events.
- Personal characteristics: older age, reduced 'brain reserve', ethnicity and gender, historical/current injecting drug use.

People with HIV infection can develop a wide range of mental disorders:

- Abnormal psychological reactions - adjustment disorders, manifestations of personality disorder.
- Mood and other disorders - major depression and other depressive syndromes, suicidal behaviour, manic episodes, sexual dysfunction, anxiety disorders, obsessive compulsive disorder, eating disorders, association with child sexual abuse.
- Organic brain syndromes - acute and sub-acute brain syndromes, HIV-associated dementia, HIV-associated minor cognitive impairment.

### **Abnormal psychological reactions**

Symptoms of anxiety, insomnia and depression, as well as social impairment, are common in response to the discovery of HIV infection or the development of complications. Whilst usually mild and self-limiting they can be severe and disabling.

Adjustment disorder is one of the most common diagnoses in people referred to mental health services (~30%) whilst personality disorder is often an associated diagnosis (usually avoidant, dependent, narcissistic or histrionic features, sometimes made worse by substance misuse).

### **Mood and other disorders**

Suicidal ideas are common in people with HIV, and there is a risk of both deliberate self-harm and suicide.

Severe depression has been reported in about 15% of referrals to mental health specialists.

The risk of manic episodes seems to be increased in HIV, mania being the most frequent reason for psychiatric hospitalisation among people with HIV. In some cases, illicit drug use or iatrogenic causes are implicated, as can be the chance association of HIV infection and bipolar affective disorder, but in the majority of cases no obvious aetiological factors are identified. Most cases of new-onset mania occur in advanced HIV disease and they are often

associated with the presence of substantial cognitive impairment. New-onset mania in severe symptomatic disease is predictive of reduced survival.

Sexual dysfunction is common in HIV, organic and iatrogenic factors being important in advanced disease stages. Anxiety disorders, including phobias and panic attacks, and obsessive-compulsive disorders are occasionally seen.

Eating disorders can complicate the management of HIV.

## **Organic brain syndromes**

Neuropsychiatric syndromes are common in HIV infection. Immune suppression can lead to a variety of secondary complications affecting the brain, including opportunistic infections such as cerebral toxoplasmosis and progressive multi focal leucoencephalopathy, and tumours such as cerebral lymphoma.

Acute and sub-acute syndromes (delirium) often occur as a result of systemic disorder and secondary infections. However, even in the absence of secondary complications, HIV infection can be associated with adverse effects on brain function.

Primary HIV-related brain disorders include HIV-associated dementia and minor cognitive disorder.

### **HIV-associated dementia**

This is characterised by substantial memory and intellectual decline, with often marked psychomotor slowing, and the possible presence of motor abnormalities, in the absence of delirium or secondary HIV-related disorders.

Recent reports suggest a prevalence of up to 15% in advanced disease.

Dementia tends to develop over a relatively short period of time, and once present it is associated with poor prognosis (as a rule, dementia is a syndrome of the final year of life, although on occasion it can develop earlier).

### **HIV-associated minor cognitive disorder**

This develops mostly in symptomatic patients whether or not they have a diagnosis of AIDS.

Clinical features include poor memory and attention, slowed information processing and difficulty with abstract thinking.

While 5% of newly diagnosed AIDS cases have been found to be impaired, the proportion increases to 60% in late AIDS.