NEUROLOGICAL EMERGENCY

Acute behaviour disturbances

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Psychiatric disorders are commonly encountered in neurological practice and most neurologists accept the need to assess and manage behavioural problems. The more complicated cases require expert psychiatric intervention and, for optimum care, it is essential that there is close collaboration between neurologist and psychiatrist.

Many neurological conditions, particularly those with cerebral involvement, increase the risk of developing a psychiatric disorder. Furthermore psychiatric disorders can be associated with symptoms such as headache, dizziness and weakness which suggest neurological disease but for which no organic explanation can be found. This phenomenon, known as somatisation, is being increasingly recognised and accounts for a substantial proportion of patients who are referred to neurological departments.

Schiffer[1] evaluated a consecutive series of 241 patients attending an American neurology service and found that 101 (41·9%) had symptoms sufficient to warrant a psychiatric diagnosis according to DSM-III criteria. Of 57 inpatients, 10 were considered to have a primary psychiatric disorder and five of these had no neurological illness. Among 184 outpatients, 32 had a primary psychiatric disorder and 30 of these had no neurological illness. Kirk and Saunders[2] had previously described a retrospective survey from a neurological clinic in northeast England and reported that, during a four year period, 358 (13·2%) of 2716 patients had a psychiatric disorder with no evidence of neurological illness.

If the psychiatric disorder underlying the neurological symptoms is not recognised, patients can be subjected to unnecessary and expensive investigations[3]. They may receive symptomatic treatment which fails to alleviate their condition and they will become dissatisfied with the result of their treatment and try to consult other specialists in the hope of finding a more effective remedy.

Some of the psychological symptoms and behavioural disturbances which the neurologist encounters are of sudden onset and require urgent attention. This review describes the clinical features and management of those disorders which are most likely to give rise to acute behavioural problems in neurological practice.

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Affective disorders

The fundamental disturbance is a change of mood (or affect), either depression or elation. However, mood disturbance is not necessarily the most prominent symptom and it may be masked by a wide range of other abnormalities which at first sight suggest the presence of organic disease. Affective disorders have a tendency to recur. When the recurrences always take the same form, the condition is referred to as unipolar affective disorder; when the mood change varies between depression or elation it is described as bipolar affective disorder.

DEPRESSIVE DISORDER

Depression not associated with organic disease usually presents to the neurologist with symptoms such as headache, dizziness, disturbance of higher mental function and facial or bodily pain.[4] Psychological symptoms, emotional conflicts, and life stresses may not be volunteered and are only elicited by tactful, direct questioning on the part of the examining doctor. Fitzpatrick and Hopkins[5] assessed a series of patients referred to a neurologist with headaches not due to structural disease and found that 37% had an affective disorder of at least mild severity. The preoccupation with pain may dominate the clinical picture to such an extent that the patient does not appear depressed and does not readily admit to feeling depressed. This has been described in relation to facial pain[6]; the cognitive features of depression such as self reproach, suicidal thinking and psychomotor retardation were uncommon but, in addition to pain, the patients complained of insomnia, fatigue, irritability, and agitation.

Depression also complicates existing neurological disease. In some cases—for example, spinal cord injuries,[7 8] depression appears to result largely from the personal and social implications of the neurological disability. In diseases in which there is cerebral involvement, the aetiology of depression is more complex. The mood disturbance may be triggered by the emotional impact of the disease but may also result from structural pathology, possibly through interference with neurotransmitter pathways within the brain. Depressive disorders with cerebral involvement are classified as the organic mood disorders by the International Classification

of Disease (ICD-10).[9] One of the most significant features of these disorders is that the mood disturbance, either depression or mania, may be the first manifestation of underlying neurological pathology.[10] Suspicion of unrecognised physical illness should be particularly high if there is no clear psychosocial precipitant, if the mood disorder first presents in middle or late life and if there is no family or personal disposition to psychiatric illness.[11]

Depression has been described as a presenting feature of cerebral tumours,[12] multiple sclerosis,[13 14] Parkinson's disease[15 16] and Huntington's chorea[17 18] but most attention has been given to its association with cerebrovascular disease. Eastwood *et al*[19] reported that 10% of patients in a stroke rehabilitation inpatient unit had a major depressive disorder, while 40% had symptoms of minor depression. Robinson *et al*[20] studied patients admitted to hospital after an acute stroke and found that 27% demonstrated symptoms of major depression, while 20% had symptoms of minor depression. The association between mood disturbance and severity of physical impairment is not a strong one and it has been proposed that the site of the vascular lesion is important in determining post-stroke depression. Left-sided lesions in the frontal lobe or basal ganglia have been particularly implicated.[21] Other reports have not confirmed this hypothesis and have found a lower prevalence of depressive illness, especially when the survey has included patients not admitted to hospital.[22]

Some patients with cerebrovascular disease experience mood disturbances that are too brief to justify the diagnosis of a depressive illness.[23] This phenomenon, known as pathological emotionalism, is manifest by rapid changes of mood, sudden episodes of crying, and inappropriate and uncontrollable laughter. This type of mood change has been observed more commonly in patients with lesions in the left frontal and temporal areas.

Assessment of suicide risk

The risk of suicide is one of the reasons why depression can become an acute medical problem and it must be considered when assessing any patient who is thought to be depressed. The risk is increased in several neurological disorders,[24 25] particularly multiple sclerosis, epilepsy, head injury, and spinal cord lesions.

The most important consideration is whether the patient is expressing active suicidal intent, either spontaneously or in response to direct questioning. Table 1 shows the profile of demographic factors that increase the risk of suicide, which has emerged from various studies of people who have attempted suicide; a knowledge of these is important when assessing a depressed patient.[26–28]

If the risk of a suicide attempt is considered to be significant, or if there is doubt about this, the neurologist should consult urgently with a psychiatrist colleague who should evaluate the patient as soon as possible. A number of management options, including outpatient or day hospital treatment, are available according to the perceived risk and availability of social support. When the risk is high and the patient has little in the way of support, admission to a psychiatric ward should be advised; this may have to involve invoking the Mental Health Act if the patient cannot be persuaded to accept voluntary admission.

Table 1 Risk factors for suicide in depressed patients

● Previous suicide attempts
● Male sex
● Increasing age
● Living alone
● Social isolation
● Recent bereavement
● Unemployment
● Drug and alcohol misuse
● Personality disorder

Stupor

Stupor is another serious complication of depression. It is a term that leads to disagreement between neurologists and psychiatrists, who use it in different ways. It is sometimes used to describe an intermediate stage on the spectrum of impaired consciousness that eventually leads to coma. Lishman[29] argues that this is an incorrect use of the concept which he believes is more appropriately defined as a syndrome in which the patient is conscious but makes no spontaneous movement and shows little response to external stimuli; in the most advanced form of stupor the patient is completely mute and immobile. Consciousness is inferred by the fact that there may be purposeful eye movements, following the actions of other people in the vicinity, and also by the patient's recall of events once recovery has occurred. In psychiatric practice, depression and schizophrenia are the commonest causes of stupor.[30 31] The diagnosis depends on eliciting a history of the relevant symptoms during the weeks before the onset of stupor. In the case of depression there is a history of progressive psychomotor retardation in addition to the psychological symptoms of low mood, guilt and self reproach. A full neurological evaluation, including CT of the brain, is essential before a diagnosis can be made with confidence. Cerebral disorders that can give rise to the clinical picture of stupor include dementia, encephalitis, and cerebral tumour or cyst in the upper midbrain or posterior diencephalon causing increased intracranial pressure.

Management of depressive disorder

Depression usually responds to a combined approach of antidepressant medication and cognitive therapy. Tricyclic antidepressants (amitriptyline, imipramine) are still the mainstay of treatment but are poorly tolerated in association with physical illness and prescribing practice is slowly changing in favour of the selective serotonin re-uptake inhibitors (SSRIs) (fluoxetine, paroxetine) because of their lower risk of side effects. Monoamine oxidase inhibitors (phenelzine, tranylcypromine) are also effective in depressive illness. They are used by some psychiatrists as the preferred drug when depression is accompanied by anxiety, phobic symptoms, weight gain, hypersomnia, and fatigue. For many years they have been underused because of fears of their interaction with tyramine-containing foods, potentially resulting in a catastrophic rise in blood pressure. Various

foods, alcoholic drinks, and drugs have had to be avoided by patients taking monoamine oxidase inhibitors. This problem has been overcome by the introduction of reversible and selective inhibitors of monoamine oxidase subtype A, such as moclobemide.

All antidepressants take up to two to three weeks to produce a clinical effect. In some clinical situations this delay may be dangerous and a speedier response to treatment is required. Electroconvulsive therapy (ECT) is the preferred treatment in these situations, particularly when the risk of suicide is high, or when the patient is stuporose and is not maintaining adequate nutrition or fluid intake. ECT may have to be given under the terms of the Mental Health Act if the patient is incapable of giving informed consent to treatment; in England and Wales this involves obtaining a second opinion from a psychiatrist appointed by the Mental Health Act Commission. Table 2 shows the main indications for ECT in depressive disorder.

Table 2 Indications for electroconvulsive therapy in depressive disorder

- Severe depression with high risk of suicide
- Depressive stupor
- Depressive disorder with psychotic symptoms
- Failure to respond to an adequate course of an antidepressant
- Inability to tolerate side effects of antidepressants
- If physical illness makes antidepressants less safe than ECT

There are no absolute contraindications to ECT. A decision on treatment should be taken after careful consideration of the risks of the various treatment options weighed against the risks of continuation of the depressive disorder.

MANIA

The symptoms of mania are in many ways the opposite of those of depression. There is elevation of mood accompanied by an enhanced sense of well being, physical and mental overactivity, pressure of speech, flight of ideas, increased self confidence, and a loss of social inhibitions. Some manic patients are predominantly irritable rather than elated, particularly when other people do not share their views of their own abilities. In severe forms of mania, the inflated self esteem may develop into delusions of a grandiose or religious nature and there may be auditory or visual hallucinations in keeping with the prevailing mood. Lesser degrees of mania are referred to as hypomanic episodes when the severity of symptoms does not lead to severe disruption of work or social rejection. Mania is a serious condition if the symptoms are not treated adequately. Impaired judgement can lead to accidents, catastrophic overspending, and promiscuity with its attendant risks of infection. Sustained overactivity eventually leads to exhaustion or cardiovascular collapse.

Mania is a much rarer problem than depression in neurological practice and only small series of cases have been described. It has been reported to complicate cerebrovascular disease, brain tumour, and head injury. Most reported cases have had lesions involving the limbic or related areas of the right hemisphere; there is also an increased prevalence of a family history of affective disorder suggesting that both genetic and anatomical factors are important in the development of mania secondary to neurological disease.[32] A study of mania following closed head injury reported that six of 66 patients (9%) had features of mania at some stage during a 12-month follow-up period, this figure being higher than has been reported in other brain-injured people. In this study mania was associated with temporal basal polar lesions; no links were established with the severity of brain injury or previous personal or family history of psychiatric disorder.[33]

Management of acute mania

Treatment is best carried out in hospital; if the patient is already in a neurology ward, transfer to a psychiatric ward is desirable. Because of the lack of insight which is characteristic of mania compulsory admission may have to be arranged. The three groups of drugs which are used regularly in acute mania are phenothiazines (such as chlorpromazine), butyrophenones (for example, haloperidol) and lithium. Chlorpromazine can be given orally starting at a dose of 25–50 mg eight hourly, increasing to a maximum daily dose of 1500 mg according to the clinical response. When oral treatment is not accepted an intramuscular injection of up to 150 mg can be given but this should not be repeated because of its irritant effect on muscle tissue. Haloperidol can be started at an oral dose of 5 mg eight hourly increasing to a maximum daily dose of 100 mg. When speedier control of symptoms is required, an intramuscular injection of 10–20 mg can be given. Lithium carbonate, in a daily dose of 800–1200 mg, is well tolerated but has a slower onset of effect than the other drugs. It is most effective when given in combination with chlorpromazine in acute mania but its main use is as a long term prophylactic drug in recurrent affective disorder.

Phenothiazines and butyrophenones are likely to produce a variety of extrapyramidal side effects, especially in patients with existing brain damage. Akathisia, Parkinsonism, acute dystonia and tardive dyskinesia are well established complications. Akathisia, a subjective sense of motor restlessness, leads to hyperactivity and an inability to relax. It can be wrongly attributed to a worsening of the manic condition, so an inexperienced clinician may increase the dose of neuroleptic rather than reduce it and consequently the akathisia symptoms worsen. A β-adrenergic blocking drug such as propranolol is an effective remedy once the condition is diagnosed. Acute dystonias can also give rise to diagnostic confusion. They can present with tongue protrusion, torticollis, oculogyric crisis or opisthotonos and these are often wrongly

diagnosed as dissociative (hysterical) reactions. Rapid relief of symptoms can be achieved with an intravenous dose of 5–10 mg of procyclidine.

If manic symptoms do not respond to medication, ECT can prove to be a quick and effective treatment.

Anxiety and stress-related disorders

Patients with anxiety and allied conditions may present to neurologists if their symptoms are intermittent and of sudden onset.

PHOBIC ANXIETY DISORDERS

A phobia is an abnormal, disproportionate fear of an object or situation which leads to avoidance of the object or situation that precipitates it. Agoraphobia is the commonest phobia encountered clinically and symptoms attributed to neurological disease are particularly prominent; these include headache, impaired concentration, dizziness, and a fear of falling. The clue to the diagnosis comes from eliciting a link between the symptoms and specific situations which invariably precipitate them. In the case of agoraphobia the commonest triggers are open spaces, crowded streets, supermarkets, and public transport.

PANIC DISORDER

The characteristic features are recurrent episodes of overwhelming anxiety which are unpredictable and not confined to a particular situation. The somatic symptoms of anxiety, notably palpitations, chest pain, hyperventilation, and dizziness, usually dominate the picture but there is also intense psychological anxiety, often involving a fear of going mad or dying from a heart attack or brain tumour. This may be accompanied by depersonalisation which the patient describes as an altered sensation in his body which feels lifeless or unreal as if he has lost his feelings and is observing himself from the outside. Panic attacks may be mistaken for the aura of temporal lobe epilepsy, vestibular disease or the early manifestation of multiple sclerosis. Non-neurological conditions that need to be considered in the differential diagnoses are hyperthyroidism, phaeochromocytoma, hypoglycaemia, and supraventricular tachycardias.

A typical attack lasts only a few minutes but tends to recur. Patients become apprehensive of recurrent episodes and some learn to avoid certain places where an attack would be particularly embarrassing, such as a public place far from home from which there is no easy escape route. Thus panic attacks come to be associated with agoraphobic behaviour in some patients.

POST-TRAUMATIC STRESS DISORDER

This syndrome is a delayed response following exposure to a stressful experience of an exceptionally threatening nature which is quite outside the range of everyday experience and which is likely to cause distress to almost everyone. There is a perceived danger of death or severe physical trauma but the injuries actually sustained may be quite trivial. The experiences which evoke the response include natural disaster, road traffic accidents, military or terrorist activity and being the victim of torture or rape. The disorder has been estimated to occur in 10% of patients who sustain multiple injuries after road traffic accidents.[34]

There is a delay between the traumatic event and the onset of symptoms which ranges from a few weeks up to six months. The typical symptoms include recurrent intrusive memories of the event ('flashbacks'), disturbed sleep, nightmares of the event, emotional blunting, avoidance of cues that evoke memories of the original trauma, depression, irritability, and autonomic arousal.

Management of anxiety and stress-related disorders

Behaviour therapy, involving relaxation and gradual exposure to the precipitating situation, is of proven value in phobic disorders and in panic disorder when there is avoidance behaviour. A clinical psychologist should assess the patient and organise treatment if behaviour or cognitive therapy are considered appropriate. Phenelzine is a useful adjunct to psychological methods of treatment. Drug treatment is more important in spontaneous panic attacks; both phenelzine and imipramine have been shown to be effective.

There has been considerable interest in the psychological treatment of people who have been involved in accidents or disasters and who are at risk of developing post-traumatic stress and other psychiatric disorders.[35] Early intervention is believed to be important.[36] Various techniques have been used including psychotherapy and cognitive therapy. The essence of treatment is to allow the patient to relive the experience and ventilate the emotional response in a cathartic manner and to prevent the development of avoidance behaviour. If symptoms have become established, either tricyclic antidepressants or monoamine oxidase inhibitors can provide symptomatic relief.[37]

Dissociative (conversion) disorder

This group of disorders is also known as conversion hysteria. The term hysteria is a controversial one and is used in several different ways; as a result it has been omitted from both DSM-III and ICD-10 but it is still widely used as a diagnostic category in clinical practice.

Dissociative disorders are characterised by symptoms that suggest lesions in the motor or sensory pathways of the voluntary nervous system. There is loss or distortion of neurological function which cannot be adequately accounted for by organic disease.[38] Psychiatrists would also want to establish positive evidence that the symptom is linked to psychological factors,[39] either previous severe stress, emotional conflict, or an associated psychiatric disorder. It is assumed that the

symptoms are not intentionally produced, as in malingering, but are a result of unconscious motives. This, however, is a notoriously difficult distinction to make and it often appears that the degree of insight into the nature of the disability varies from time to time.

Dissociative disorders are thought to be declining in incidence. A survey at the National Hospital, London, indicated that the diagnosis was made in approximately 1% of inpatients.[40] The commonest symptoms, which are usually of acute onset, are motor weakness, altered sensation, gait disturbance, and pseudoseizures.[41] The neurological examination reveals characteristic abnormalities that enable the experienced neurologist to make a confident diagnosis in most cases.[42] Weakness usually involves whole movements rather than muscle groups and it affects the extremities much more often than ocular, facial, or cervical movements. Various clinical techniques can be employed to show that weakness of a limb is associated with simultaneous contraction of opposing muscle groups. There is discontinuous resistance during testing of power ("give way weakness"), muscle wasting is absent, and reflexes are normal. Sensory loss or distortion is often inconsistent when tested on more than one occasion and incompatible with peripheral nerve or root distribution. There may be discrete patches of anaesthesia or hemisensory loss that stops abruptly in the midline. Visual symptoms include monocular diplopia, triplopia, field defects, tunnel vision, and bilateral blindness associated with normal pupillary reflexes. Dissociative gait disturbance, astasia-abasia, is recognised by its bizarre character and intermittent pattern; the patient walks normally if he thinks he is not being observed. In some cases, when being observed, the patient actively attempts to fall and this contrasts with the patient with organic disease who tries to support himself.

Pseudoseizures are more difficult to evaluate because they are episodic and often coexist with true epilepsy. An account from a reliable observer is invaluable but it is essential for the clinician to witness an attack before making a firm diagnosis. The clinical features, which simulate epilepsy to a varying degree, have been described in detail.[43] During an attack there is marked involvement of the truncal muscles with opisthotonos and lateral rolling or jerking of the head or body. All four limbs may exhibit random thrashing movements which increase in amplitude if restraint is applied. Cyanosis is rare unless there is deliberate breath holding. Corneal and pupillary reflexes are retained although they may be difficult to elicit because the eyelids are kept firmly closed. Tongue biting and urinary incontinence are uncommon unless the patient has some degree of medical knowledge and has learned from experience that they are characteristic features of epilepsy.

In contrast to true epilepsy, pseudoseizures usually occur in the presence of other people and not when the patient is alone or asleep. Some episodes simulate partial motor seizures or simple faints. Others occur in rapid succession without recovery of consciousness and they may be accompanied by deliberate tongue biting or incontinence so the clinical picture mimics status epilepticus. In addition to the clinical features of the attacks, certain demographic characteristics help to distinguish pseudoseizures from true epilepsy. Patients with pseudoseizures are more likely to have a family history of psychiatric illness, a personal history of psychiatric illness, previous suicide attempts, sexual maladjustment, and current affective disorder.[44]

Dissociative, or psychogenic, amnesia can also create diagnostic problems.[45] There is a sudden loss of memory, usually in relation to a markedly stressful event. The amnesia is selective and predominantly involves the inability to recall emotionally charged memories. The ability to learn new information is relatively preserved as are cognitive skills such as reading and writing. A characteristic feature is a loss of personal identity; the patient is unable to recall his name, age, address, occupation, and family details and may fail to recognise his relatives when they visit. Recovery is usually rapid and complete. In some cases, however, dissociative amnesia lasts for several days or weeks and is accompanied by an apparently purposeful wandering away from home or place of work. During this condition, known as a dissociative fugue, a new name and identity may be assumed. Self care is maintained and the patient's behaviour may appear completely normal to people who do not know him. Recovery occurs abruptly and there is amnesia for the period of the fugue. Organic conditions that need to be considered in the differential diagnosis of dissociative amnesia included head injury, delirium, epileptic fit, Wernicke's encephalopathy, alcoholic blackout, and transient global amnesia.

Any patient suspected of having dissociative symptoms should be examined carefully by a neurologist and psychiatrist. Special investigations, such as an MRI and EEG telemetry, are required in some cases before a confident diagnosis can be made and, in a few, a decision has to be deferred until the symptoms can be reviewed after a suitable time interval. The presence of organic disease does not rule out a diagnosis of dissociative disorder. Indeed it is now recognised that neurological lesions and dissociative symptoms occur together more frequently than can be explained by chance. Although the neurological lesion cannot explain the presenting symptom, coexisting disease of the nervous system may facilitate the emergence of dissociative mechanisms and provide a model for the symptom.[39] This may explain the frequent occurrence of pseudoseizures in patients who also have genuine epilepsy.

Management of dissociative disorder

Once the diagnosis of dissociative disorder has been established, the results of the clinical

examination and special investigations should be discussed with the patient together with reasons for considering the symptoms to have a psychological basis. This should not be conducted in a confrontational manner. It is best to explain that the symptoms are due to stress, that they are familiar to the clinician, that serious disease has been excluded and that full recovery can be expected. Requests for further investigations should be resisted. A reassuring and encouraging approach is beneficial. Any associated psychiatric disorder such as depression should be treated appropriately and interpersonal conflicts discussed with the patient and key relatives. These patients are often highly suggestible so they respond well to predictions of recovery. Given that they are preoccupied with physical complaints, their recovery can be assisted by providing a physical framework for improvement. Physiotherapy is particularly effective in restoring the power to a paralysed or weakened limb. Using such an approach with patients with hysterical gait disorders, about half recovered completely during a three to four day hospital stay.[46]

In resistant cases an abreaction interview under intravenous sedation is useful. Several drugs can be used to provide the required level of sedation but most experience has been gained with sodium amytal.[47] The procedure involves slow intravenous injection at a rate of no more than 50 mg per minute until the sedation threshold is achieved as judged by drowsiness, slurred speech, and rapid horizontal nystagmus. This usually occurs with a dose of 150—350 mg after which the level of sedation should be maintained by further slower injection to a maximum total dose of 500 mg. During the interview the patient is encouraged to explore and ventilate any emotional problems that have hitherto not been discussed. This can have a cathartic effect and is often followed by dramatic improvement which is facilitated if the interviewer predicts recovery before and during the interview. An interview using amytal can also be useful to clarify the diagnosis and gain access to undisclosed psychopathology. It appears particularly useful for dissociative amnesia and should be used if the memory impairment does not begin to improve within a few days of its onset. Hypnosis can be employed as an alternative procedure if the clinician has sufficient experience of the technique.

Somatoform disorders and neurasthenia

These disorders tend to be chronic and are associated with repeated consultations for physical symptoms and requests for medical investigations despite previous negative findings. They are unlikely to present as emergencies but as part of a lengthy history, at times exceeding 30 years, of chronic ill health and contact with medical services.

Acute psychotic disorders

Psychotic symptoms are common complications of neurological and other medical illnesses. In most cases they are due to delirium and represent the acute effects on mental functioning of intracranial lesions, or metabolic or toxic disturbance elsewhere in the body. The characteristic features are alterations of consciousness, muddled thinking, mood change, psychomotor abnormalities—either retardation or excitability—and perceptual disturbances in the visual or tactile senses. In severe cases there are florid visual and tactile hallucinations. The aetiology and management of delirium have already been reviewed in this series.[48]

Delirium arising during treatment of Parkinson's disease can often lead to diagnostic problems because psychological symptoms may be due to the disease itself or to prescribed drugs. Anticholinergic drugs such as benzhexol, procyclidine, and orphenadrine can precipitate acute delirium which resolves when the drug is withdrawn. Quicker resolution can be achieved by parenteral administration of the anticholinesterase, physostigmine. Delirium also occurs during treatment with levodopa but other acute psychological reactions can develop with this drug including depression, hypomania, hypersexuality, delusions and hallucinations in clear consciousness. Clozapine has been recommended as an effective treatment for psychotic disorders associated with levodopa therapy.[49] This drug is currently available for the treatment of resistant schizophrenia; its use is restricted because of the appreciable risk of agranulocytosis which occurs in at least 1% of cases. The white cell count has to be monitored weekly for the first 18 weeks of treatment and fortnightly thereafter. Other side effects of clozapine include convulsions and hypersalivation.

Occasionally transient psychotic disorders develop in clear consciousness in the absence of other features of delirium. The most striking clinical symptom is a well developed delusional system, nearly always of a paranoid nature. These psychotic disorders are seen most commonly in patients who have been admitted acutely to hospital and who have been treated in intensive care or coronary care units which they perceive as bewildering and threatening. The delusions typically involve the nursing or medical staff treating the patient who may accuse them of trying to poison—or even kill him. In response to these delusions there may be sudden outbursts of aggression towards members of staff or attempts to leave hospital against medical advice. Psychoses of this nature are probably precipitated by the emotional impact of the illness and by the disruption it causes to the patient's familiar environment.

Acute psychoses without features of delirium or dementia can occur in association with a wide range of cerebral disorders, notably epileptic foci, cerebral tumour, cerebral trauma, Wilson's disease, Parkinson's disease, and Huntington's chorea. The psychosis can take the form of a schizophrenic or affective disorder. The site of the lesion has been considered to influence the pattern of

symptoms. A schizophrenic presentation has been particularly associated with temporal lobe lesions in the dominant hemisphere, whereas affective disorders have been linked with lesions in the non-dominant hemisphere. Not all studies have demonstrated such an association with laterality.[50]

Management of acute psychosis

When the psychosis is believed to be triggered by the perceived stress of the treatment environment it is advisable to transfer the patient as soon as possible to a less intimidating environment—for example, from an intensive care unit to a general ward or from a general ward to the patient's home, if care is available there. Medication may be needed if there is aggressive or disruptive behaviour. Chlorpromazine or haloperidol may be given in doses similar to those used in acute mania. If very rapid control of symptoms is required, droperidol can be given intravenously in a dose of 5–15 mg and repeated every four to six hours.

These neuroleptic drugs are well recognised as having unpleasant extrapyramidal side effects. The most dangerous of these, but fortunately the least frequent, is the neuroleptic malignant syndrome in which extrapyramidal rigidity and akinesia develop abruptly or within a few days.[51] Pyrexia is always present, together with autonomic disturbances including profuse sweating, increased salivation, hyperventilation, tachycardia, and labile blood pressure. Laboratory investigations show a leucocytosis and a raised creatinine kinase. The incidence of the condition is not known but is probably well under 1% of all cases treated with neuroleptics if strict diagnostic criteria are applied.[52] Neuroleptics should be stopped immediately once the condition is diagnosed. Rapid improvement is seen if a dopamine agonist, such as bromocriptine, is administered and the mortality rate has been considerably reduced by this treatment.

Personality change due to cerebral disease

A change in personality is likely to follow diffuse, severe damage to the brain of the sort that is commonly seen after major head injury but which can also occur in association with cerebral tumours, cerebrovascular disease, dementias, and encephalitis. The changes in behaviour are persistent and often develop gradually but they may lead to sudden outbursts of impulsivity which create acute problems. A survey of patients who had survived a severe head injury reported that the prevalence of personality change increased with time; at five years after the injury, 74% of patients were described by their relatives as having undergone a personality change. Threats or gestures of violence had occurred in 54% while there had been an actual assault on a relative in 20% of cases. Other problems which had occurred included trouble with the law (31%), childishness (38%) and being upset by minor changes in routine (38%).[53]

Brain injury can produce an exaggeration of premorbid personality traits so that a person with an obsessional personality becomes even more meticulous and preoccupied with detail, whereas someone with a psychopathic personality becomes more impulsive and irresponsible. Among a group of patients with Wilson's disease, psychopathic personality traits were significantly related to the severity of neurological symptoms, particularly dysarthria, bradykinesia, and rigidity.[54] If the damage is localised to particular parts of the brain, the personality changes tend to be more specific.[55 56] Frontal lobe damage is associated with apathy, lack of initiative, tactlessness, irritability, euphoria, and disinhibition. Although the patient's demeanour is predominantly listless there may be unpredictable outbursts of aggression or sexually disinhibited behaviour. Social skills tend to be lost with a failure to consider the feelings of other people and the impact of tactless remarks. The ability to plan ahead is impaired with the result that irresponsible decisions may be taken with little concern about their outcome.

Irritability and aggressive outbursts are especially associated with temporal lobe pathology. Herpes simplex encephalitis has an affinity for the temporal lobes, so behavioural manifestations are common during the acute stages of the illness and after recovery if there is residual brain damage. Patients who survive temporal lobe damage may manifest the features of the Kluver-Bucy syndrome which include hypersexuality, aggressive outbursts, excessive oral behaviour and visual agnosia.

Management of aggressive behaviour

Table 3 summarises the conditions in which unpredictable outbursts of aggression may occur.

Patients who are potentially violent should not be interviewed in an isolated room; when the risk is particularly high the doctor should not be alone with such patients. Adequate staff should be available nearby. The immediate aims are to control the risk of violence, to diagnose the underlying disorder and to administer specific treatment. A detailed history and full physical and mental state assessment are rarely possible; immediate treatment has to be arranged before complete information is available.

Table 3 Neurological and psychiatric disorders associated with aggression

- Acute delirium
- Brain damage—especially to frontal and temporal lobes
- Epilepsy
- Functional psychoses—schizophrenia, bipolar affective disorder
- Alcohol abuse—intoxication, withdrawal
- Drug abuse—intoxication, withdrawal, threatening behaviour to obtain drugs
- Personality disorder

Every effort should be made to calm patients by sympathetic understanding and reassurance. Violence is often a response to paranoid experiences and patients can be pacified if they believe that the doctor appreciates the reasons for their behaviour. If this can be achieved medication may be accepted voluntarily; otherwise compulsory treatment becomes unavoidable if patients are endangering themselves or others. Physical restraint should be applied with the assistance of security staff; the safety of all involved is best ensured by having more than a sufficient number of staff available. At least one person should restrain each limb while another administers medication.[57 58] Haloperidol 10–20 mg intramuscularly or droperiodol 5–15 mg intravenously are the preferred drugs, except for cases of alcohol or drug misuse or patients with serious physical illness. Benzodiazepines should then be given instead—for example, diazepam 10 mg by slow intravenous injection or lorazepam 2 mg intramuscularly. Once the risk of aggression has been controlled it is nearly always necessary to arrange admission to an appropriate inpatient unit to begin a diagnostic evaluation and specific treatment for the underlying condition when the diagnosis has been clarified.

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