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frightened, and unable to comprehend sensory inputs. Aetiology Table 26.5.1.1 lists common causes of delirium. The profile of a high- risk patient is well-established: older people with existing dementia, sensory impairment, and a history of previous stroke, depression, alcohol abuse, or delirium. Precipitating factors may be directly and sufficiently causal; for example, a new anticholinergic drug, status epilepticus, and se- vere alcohol withdrawal. In many cases the precipitating factor may seem insufficient to provoke a delirium, leading to suspicion that a vulnerable brain (for example due to dementia or previous stroke) combined with an abnormal neuro-inflammatory or al- tered neurotransmitter response to external insult has triggered the syndrome. 26.5 Specific psychiatric disorders

SECTION 26 Psychiatric and drug-related disorders 6476 Animal and human experimental work increasingly support this hypothesis; thus insults such as an anticholinergic drug, or using high dose lipopolysaccharides to mimic the effects of sepsis, lead to clinical syndromes resembling delirium in vulnerable animals. In humans, stress responses to insults, including elevated circu- lating glucocorticoids, or exaggerated blood and cerebrospinal fluid (CSF) cytokine responses, are hypothesized to mediate the physio- logical pathway leading to the clinical signs of delirium, which may themselves be mediated by altered cholinergic and monoaminergic neurotransmission. There may be multiple causes. Epidemiology Delirium is probably the psychiatric condition most frequently rec- ognized in general hospitals, though cases occur in other settings. It has been most studied in general hospitals, where prevalence figures of 15–25% are typically found on orthopaedic, geratology, and gen- eral medical wards. The highest prevalence (up to 50%) is found in postsurgical intensive care unit (ITU), and in palliative care wards. The prevalence in nursing home settings is around 10–15%. In the community the prevalence is probably only 1–2%. Clinical features Box 26.5.1.1 outlines the diagnostic features of delirium. The car- dinal criteria are inattention and reduced awareness of the environ- ment. Recent criteria emphasize disorientation to the environment as evidence of reduced awareness, acute change compared to base- line function, and the broadening of aetiologies to include sub- stance misuse and toxins. Fluctuation, often worse at night, and visual hallucinations, often of a frightening nature, are common. The patient may exhibit a hyperactive form with agitation and dis- turbed behaviour, or sometimes a hypoactive form with reduced activity Differential diagnosis The main differential diagnosis is from dementia. Other differentials to consider are brain lesions such as concussion and cerebral malig- nancy (primary and secondary). Delusions and hallucinations occur in acute functional psychoses (though less often visual in nature). The overactivity of mania may suggest hyperactive delirium and the inactivity of depression hypoactive delirium, a key difference being the reduced consciousness level in delirium. Drug and alcohol in- toxication can cause a similar picture to delirium, but their presence is often obvious. Clinical investigation Delirium is often not recognized and as many as two-thirds of cases are missed. The diagnosis is more likely to be made if the patient has the overactive rather than underactive form. Clinical experience is often essential to recognize the signs in more subtle presentations. Key to the diagnosis is the recognition of the core clinical features, especially inattention and fluctuating conscious- ness. These are emphasized in the published diagnostic criteria such as ICD-10 and DSM-5. There are instruments available to support the diagnosis such as the Confusion Assessment Method (CAM) which focuses on acute onset and fluctuating course, and inattention, supported by disorganized thinking and altered con- sciousness. At the very least, a test of attention (e.g. counting back from 20) and of orientation (e.g. year/season/month/date/day) should be attempted. Characteristically, scores on such tests will vary over time. Medical assessment should seek the causes, beginning with the commonest

causes of delirium such as infection or dehydration. An electroencephalogram (EEG) is sometimes used: typically there will be diffuse slowing and increased θ and δ-activity. This can help dif- ferentiate delirium from dementia but is rarely of significant prac- tical value. There may also be a need to assess for hearing or visual impairment. Table 26.5.1.1 Common causes of delirium Examples Predisposing factors (established risk factors from prospective studies) Older age Dementia Alcohol misuse Stroke Depression Hearing impairment Visual impairment Multimorbidity Functional impairment Urinary catheter Precipitating factors (selected) Polypharmacy Specific medications Infections Renal impairment Hypothermia Dehydration Trauma Infection Hypoxia Hypoglycaemia Alcohol and drug withdrawal Anticholinergics Sedatives Antiparkinsonism drugs Urinary tract infection Respiratory tract infection Delirium tremens Box 26.5.1.1 Diagnostic features of delirium • Reduced attention (i.e. reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment) • Acute onset of a change from the previous cognitive functioning • Fluctuation during the course of a day, and often worse at night • Other psychological disturbances (e.g. memory deficit, disorientation, language, visuospatial ability, or perception), hallucinations (often visual) • Evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal, or exposure to a toxin

 Management Prevention The high prevalence and incidence of delirium in general hospital patients has led to considerable interest in its prevention. The onset of delirium is particularly common in orthopaedic postoperative patients and among unwell patients in intensive care units. Pharmacological methods with potential to prevent delirium in- clude antipsychotic agents, cholinesterase inhibitors, melatonin, or anti-inflammatory agents. To date, however, there is inadequate evi- dence to recommend this strategy. Prevention should instead focus on identifying higher risk patients and using complex interventions to reduce their risk of delirium (see Table 26.5.1.2). These measures are supported by evidence but require considerable multidisciplinary effort to implement. Some could be considered simply good clinical care (regular checks for dehydration, constipation, early mobilization, adequate pain relief). Others require a change in staff behaviour, for example use of orientation devices, regular checks on sensory aids like spectacles and hearing aids, pharmacist review for medications, with anticholinergic effects and allowing family to provide care outside visiting hours. Implementation of these meas- ures can only happen if there is a ward- or hospital-wide awareness of delirium and a willingness to actively identify, prevent, and treat the condition. Reducing the duration of delirium The treatment of delirium is an emergency. It is now clear that pro- longed delirium is associated with worse outcomes. Patients may come to harm from wandering or falls, and long periods of delirium may exacerbate dementia. The principles of management are there- fore to keep the patient from harm while seeking and treating the underlying cause. In a significant minority of cases no specific cause is found, and clinicians may be tempted to give antibiotics, especially if infective causes are suspected (e.g. when inflammatory markers are raised). There is at best equivocal evidence for the effect of antipsychotics and lorazepam in reducing the duration of delirium, and modest evidence from at least one controlled trial that rivastigmine (a cho- linesterase inhibitor) may lengthen duration of episodes. Routine use of psychotropic medication to reduce delirium is therefore not supported. Other proposed strategies, for example routine use of anti-inflammatory medication, are also unsupported by evidence. Treating the symptoms of delirium Given the lack of evidence that pharmacological treatment can re- duce the length of an episode, its role is limited to treating distressing symptoms. Chlordiazepoxide (or equivalent doses of diazepam, or other long-acting benzodiazepine) is recommended for the treatment of alcohol withdrawal and its major

complication of delirium tremens. For other causes, there is modest evidence to support the use of olanzapine, risperidone, and haloperidol in treating aggression, dis- tress, and psychotic symptoms in delirium. Local protocols usually suggest dosing regimens; as most patients are frail, lower doses are used than with younger patients with schizophrenia or mood dis- orders (the usual indications). These treatments should be withdrawn when symptoms re- solve, as there is doubt about the risk/benefit ratio after this time. Benzodiazepines (usually short acting agents like lorazepam) are sometimes used for behavioural control in distressed or aggressive patients, but there is little evidence to support this. Preventive and treatment interventions for delirium overlap con- siderably. They include direct support of brain function including reversing hypoxia and stopping deliriogenic drugs (see Box 26.5.1 and Table 26.5.2). Indirect support focuses on the environment of care: using orientation clocks, involving family in care, promoting sleep at night (e.g. using earplugs, side rooms), using single staff members during shifts. The environment of care includes staff and family carers, and responsible staff need to ensure that all carers clearly understand the diagnosis and prognosis. Outcome Delirium is a strong independent predictor of prolonged hospital stay, increased mortality, and of institutionalization. Delirium often indicates an as-yet undiagnosed dementia, while among patients with delirium who do not currently have dementia, the risk of de- mentia developing later is substantially increased. For those patients with established dementia, incident delirium is shown to accelerate the deteriorating course of dementia. For all of these outcomes, it ap- pears that length of delirium is predictive of poor outcomes, making shortening delirious episodes a legitimate management goal. It is now also clear that delirium is by no means always transient. Meta- analyses show that nearly half of hospital cases are not yet resolved at the point of discharge from hospital, while up to 20% remain deli- rious six months after the index episode. FURTHER READING American Psychiatric Association: (2013). Diagnostic and statistical manual of mental disorders, 5th edition. Arlington, VA. Cole MG (2010). Persistent delirium in older hospital inpatients. Curr Opin Psychiatry, 23, 250–4. Inouye SK, Westendorp RG, Saczynski JS (2014). Delirium in elderly people. Lancet, 383, 911–22. Maclullich AM, et al. (2013). New horizons in the pathogenesis, assess- ment and management of delirium. Age Ageing, 42, 667–74. Table 26.5.1.2 Preventive measures for delirium Routine Specific Avoid use of urinary catheters Use hearing aids and spectacles Detect and treat constipation Detect and treat pain Early mobilization Review nutritional and hydration status Stratification for delirium risk Secondary prevention—screening (e.g. using CAM) Orientation clocks Pharmacy review for drugs that cause delirium Sleep promotion Proactive management of alcohol and drug withdrawal

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