Transient Cognitive Disorders (Delirium, Acute Confusional States) in the Elderly

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Transient cognitive disorders (delirium and pseudodelirium) are highly prevalent among the elderly, especially those with brain damage. Delirium is a common feature of physical illness or drug intoxication in elderly patients and requires prompt medical attention. While potentially reversible, delirium may herald death. Pseudodelirium may be induced by psychosocial stress or accompany a functional mental disorder. Transient cognitive disorders must not be confused with dementia, a chronic syndrome. The author discusses clinical features, etiology, pathogenesis, diagnosis, and treatment of delirium and related transient disorders of cognition in the elderly.

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Delirium and intoxication may be considered transient effects, from temporary causes, of that condition of sensorium which, more deeply fixed and longer continued, obtains the name and produces all the aspects of mental derangement.

—Henry Holland (*Medical Notes and Reflections*, 1839)

Cognitive disorders are on the rise. Their growing importance as a medical and psychiatric problem reflects the continued increase in the number of the elderly worldwide (1, 2). In the United States, there are 25.5 million persons aged 65 years and older. Dementia and delirium, the main cognitive disorders, are most common among the elderly. Recent medical editorials speak of dementia as a "quiet epidemic" and "one of the greatest problems facing modern society" (3, 4). The elderly, and especially the demented, are uniquely prone to transient cognitive disorders, usually referred to in the literature as delirium or acute confusional states (5). As the prevalence of dementia is expected to rise in the coming years because of the aging of the population, so the incidence of delirium is likely to follow suit. While dementia has attracted growing attention and been reviewed in this journal (6, 7), delirium in the elderly continues to be neglected (8, 9). A recent report of the Royal College of Physicians emphasizes that insufficient attention has been paid to this common and important mental disorder, one whose onset in an elderly patient usually heralds physical illness and hence calls for immediate medical evaluation (8). Furthermore, delirium is still often mistaken for an irreversible dementia. The present overview may help prevent such grave diagnostic errors in the future and stimulate sorely needed research on transient cognitive disorders.

EARLY STUDIES

In 1870, Hood (10) reported on several cases of "senile delirium" and concluded that it could develop in elderly persons free of prior "mental debility," required prompt treatment of the underlying cause, and was potentially reversible but could result in death from exhaustion. In 1904, Pickett (11) stressed the importance of distinguishing "confusion" and "delirium," two similar and curable mental disorders of the elderly, from senile dementia. Delirium, he believed, always had an organic cause, while confusion could result from bereavement, for example. This proposed distinction between delirium and confusion has remained an unexplored issue until today. By 1939 the concept of transient cognitive disorders in the elderly had been clearly formulated and the importance of differentiating them from senile dementia fully recognized (12). Unfortunately, progress in this area since then has been hampered by terminological chaos and lack of explicit diagnostic criteria. As a result, case identification, elucidation of causative factors and pathogenetic mechanisms, and clinical management have suffered. To facilitate communication, research, and teaching, the semantic muddle must be overcome.

DEFINITIONS: A WAY OUT OF THE SEMANTIC MUDDLE

Overlapping, inconsistently used, and poorly defined terms have bedeviled this area of psychiatry for

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decades. "Senile delirium," "acute confusional states," "acute brain syndrome," "acute brain failure," "pseudosenility," and "clouded states" are all terms that have been used to designate delirium in the elderly. Currently, "acute confusional states" and "delirium" are the most often used designations. The former term has been recommended by the World Health Organization and defined as a syndrome characterized by "features of delirium" (13). Derived from "confusion" (14), an ambiguous quasi-medical term that is widely used and all too loosely applied to the elderly (15), "acute confusional states" is a designation favored by geriatricians and neurologists. It refers to some combination of spatiotemporal disorientation, difficulty in thinking coherently, memory impairment, and bewilderment (15–17). In psychiatry, the terms "confusion" and "acute confusional states" have no established meaning, may be used in reference to a person of any age, and lack a clear etiological connotation of either organicity or psychogenicity (16, 18). In *DSM-III*, "delirium" has replaced "acute confusional states" and is listed among organic brain syndromes. Ambiguity persists, however, as some writers include among acute confusional states both organic and presumably psychogenic transient cognitive disorders (15, 16), while others use the word "delirium" in a narrow sense to denote a syndrome featuring hyperalertness, hyperactivity, frightening hallucinations, and high autonomic arousal (19).

To clear up this semantic muddle I propose that 1) the term "delirium" be used exclusively for those transient global cognitive disorders that may occur at any age and that are judged to be of *organic* etiology, 2) the use of the ambiguous term "acute confusional states" be discouraged, and 3) the delirium-like cognitive disorders judged to be functional be referred to as "pseudodelirium," by analogy with pseudodementia, until their nosological status has become clarified. (Such disorders cannot be called delirium, since *DSM-III* requires evidence of a "specific organic factor" for the latter.)

Delirium is defined as an organic brain syndrome characterized by global cognitive impairment of abrupt onset and relatively brief duration (usually less than 1 month) and by concurrent disturbances of attention, sleep-wake cycle, and psychomotor behavior (17, 20, 21, *DSM-III*). (For the sake of clarity, I will use the term "delirium" here even where the original source referred to used one of its synonyms.)

INCIDENCE AND IMPORTANCE OF DELIRIUM

Nearly every physical illness may give rise to delirium in an elderly person. As one geriatrician put it, "Acute confusion is a far more common herald of the onset of physical illness in an old person than are, for example, fever, pain or tachycardia" (22, p. 24). "Confusion" is one of the most frequent reasons for referral of a patient to a geriatrician (23). Failure to diagnose delirium and to identify and treat its underlying causes may have lethal consequences for the patient, since it may constitute the most prominent presenting feature of myocardial infarction, pneumonia, or some other life-threatening physical illness.

Few epidemiological studies have been done on delirium in the elderly. Its incidence is claimed to be four times higher in persons more than 40 years old (24) and is highest among those older than 70 years (25). A geriatric multicenter British study found that 35% of patients aged 65 years and over had delirium on admission or developed it during the index hospitalization (26). An even higher incidence, 80%, was found among 5,000 patients 65 years and older admitted to the Oxford Geriatric Unit (27). Of 534 patients aged 60 or older admitted to psychiatric wards of San Francisco General Hospital in 1959, 55% were diagnosed as having "acute organic brain syndrome" (i.e., delirium), which in about 80% of cases was associated with chronic brain disease (28). Two studies of elderly patients admitted to general medical wards reported an identical incidence of delirium, 16% (29, 30). Furthermore, other studies have shown that about 25% of those elderly patients who are judged to be cognitively intact on admission may be expected to develop delirium during the first month of hospitalization (8, 26). Thus between one-third and one-half of the hospitalized elderly are likely to be delirious at some point during the index admission. If one considers that in 1980 the elderly occupied 38% of all beds in nonfederal short-stay hospitals (31), it seems likely that the incidence and prevalence of delirium in these patients are higher than is often realized. Several investigators have reported that 10%–15% of general surgical patients aged 65 or older become delirious after their operation (32, 33). Clearly, the syndrome is sufficiently frequent among elderly medical and surgical patients to pose a formidable diagnostic and therapeutic challenge to all physicians, including general hospital psychiatrists.

Delirium is by definition transient, but this should not obscure the fact that in the elderly it is often a prelude to death or, probably less often, to dementia. Bedford (27) reported that fully 33% of 4,000 patients exhibiting delirium on admission to a hospital died within a month. Of the survivors, 80% recovered in less than a month and only 5% remained confused for more than 6 months. (By *DSM-III* criteria these patients would be likely to have a diagnosis of dementia.) In both the multicenter British study and the San Francisco study about one in four delirious elderly patients died within a month of admission (26, 28). This mortality rate was about twice as high as that for comparable nondelirious patients (26). In the two general hospital studies, 37% and 18%, respectively, of the delirious patients died (29, 30). Patients with the lowest mental test scores on admission, indicating most severe cognitive impairment, are likely to have a poor outcome (30). The development of delirium in an elderly person must be regarded as a grave prognostic sign.

CLINICAL SYNDROME: PSYCHOPATHOLOGY AND COURSE

Studies of the phenomenology of delirium in old age are almost nonexistent, and the relevant literature is mostly anecdotal. The core features of the syndrome may be assumed to be the same in all age groups, however, and include disorders of cognition, attention, sleep-wake cycle, and psychomotor behavior (5, 12, 17, 20, 32, 34–38).

Disorder of Cognition

Global disturbance of cognition is one of the core features of delirium (17). Perception, thinking, and memory—the three main aspects or phases of cognition—are all abnormal to some extent in delirium, as the adjective "global" implies. Cognitive processes whereby the individual acquires knowledge and guides his or her behavior are disorganized, rendering him or her helpless to some degree. Acquisition, processing, retention, retrieval, and utilization of information are all impaired, resulting in what both medical and lay observers often refer to as "confusion." This impairment may range from slight to profound; in the latter, organized cognitive activity practically ceases. As a result, the patient is less capable than normally of making sense of his or her environment and situation, of reasoning and solving problems, and of sustaining goal-directed behavior for any length of time (17, 20, 34, 35).

Disorganization and fluctuating efficiency of cognitive processes in delirium appear to facilitate the emergence of certain abnormal cognitive phenomena such as misidentifications, illusions, hallucinations, dream-like (oneiric) thinking, delusions, and confabulations. The patient is always at least mildly disoriented in time and, in more severe delirium, also for place and person, but he or she practically never loses the sense of personal identity (34). Typically, the disoriented delirious individual tends to misidentify unfamiliar places and persons as familiar ones (39). The cognitive disorder characteristically fluctuates in severity over the course of a day, with so-called lucid intervals interspersed irregularly throughout, and is usually most pronounced during the night.

The elderly delirious patient often fails to show the more elaborate and flamboyant features such as hallucinations, oneiric thinking, and confabulations (8, 32). Many of these patients suffer from concurrent dementia, which appears to limit their capacity to elaborate these symptoms.

Perception in delirium is marked by reduced ability to discriminate and integrate percepts and to distinguish them clearly from imagery, dreams, and hallucinations (17). The patient may perceive objects as distorted, i.e., too big (macropsia), too small (micropsia), reduplicated (polyopsia), or misshapen (dysmorphopsia) (40). Stationary objects may be perceived as moving or flowing together. Illusions and hallucinations in any modality, but especially visual or mixed visual and auditory ones, are common but are neither invariably present in nor diagnostic of delirium. While some observers claim that only about 40% of the delirious elderly hallucinate (28), others assert that most of these patients can be found to do so if one questions them systematically (41). In about half of the patients hallucinations occur only at night (42). They are typically experienced as real, three-dimensional, moving, bright, and colored images of people or nonhuman objects of natural size (42). Most patients react to them with fear or anger and attempts at flight or fight, but some may actually enjoy them, especially the visions of cherished relatives or friends.

Thinking is disorganized and fragmented. Delirious patients of any age tend to experience uncontrolled and often disturbing dream-like imagery or disjointed thoughts. The elderly delirious patient, however, is more likely to have only impoverished and incoherent thought processes. His or her ability to reason, use abstract concepts, judge, solve problems, and plan action is reduced. To think sequentially and logically, to grasp the meaning of words and define them, to see similarities and differences, and to direct thought at will are all abilities that have become difficult or impossible for the patient. A frankly paranoid attitude is common, and persecutory delusions, which tend to be poorly worked out, fleeting, changeable, and bound to the immediate stimuli, occur in 40% to 55% of the delirious elderly (28).

Memory is impaired in all its aspects: registration, retention, and retrieval. Immediate recall is impaired, probably because of reduced attention span. Recent memory is more impaired than remote memory, and the ability to learn is reduced. Both retrograde and anterograde amnesia of some degree are present (17). Unless the patient is also demented, he or she is likely to have relatively intact remote memory for both personal and public events.

Disorder of Attention and Wakefulness

For about a century delirium has been linked with the concept of disordered or "clouded" consciousness, its alleged cardinal feature (17). *DSM-III* includes clouding of consciousness among the diagnostic criteria for delirium and equates it with "reduced clarity of awareness of the environment," accompanied by "reduced capacity to shift, focus, and sustain attention to environmental stimuli" (*DSM-III*, p. 107). For reasons discussed elsewhere (17, 35), I shall use the more clearly defined and precise terms "attention" and "wakefulness," rather than "clouding of consciousness," a metaphorical concept that, in my view, is obsolete, vague, and redundant.

In delirium, attention is disordered in all of its main aspects: alertness (vigilance) or readiness to respond to stimuli, selectiveness, and directiveness. The patient shows diminished ability to respond to stimuli selectively, to mobilize, sustain, and shift attention at will,

and to direct mental processes (17). Alertness or vigilance may be abnormally increased or decreased, but capacity to deploy attention selectively and directively is invariably reduced (17, 34, 35). Some writers regard abnormalities of attention as basic to and responsible for cognitive disorganization (43). Basic or not, the characteristically fluctuating attentional disturbances in delirium enhance disruption of cognitive processes, impede communication with the patient, and render his or her behavior and performance erratic.

Disordered wakefulness and sleep-wake cycle are among the essential features of delirium (17, *DSM-III*). Wakefulness is usually reduced during the day and abnormally increased at night. Night sleep is usually fragmented and reduced. For centuries many medical writers have viewed delirium as the "dreams of waking persons" (44) and as a manifestation of disordered sleep (17). Kennedy (36) pointed out the similarities between delirium and dreaming, both of which are associated with EEG slowing and lessened attention. A typical elderly delirious patient is awake, restless, agitated, bewildered, and often hallucinating during the night and may oscillate between sleep and waking and between dreaming and hallucinating (12, 17, 34, 45). In one study, about half of the patients were found to be "disturbing at night" (28). The close association between fragmentation of the normal sleep-wake cycle and delirium is one of its most characteristic distinguishing features (17).

Disorder of Psychomotor Behavior

A delirious patient may be predominantly hyperactive or hypoactive or may repeatedly switch from one of these extremes to the other in the course of a day, as Soranus (46) had already observed in the second century A.D. His description of the patient's psychomotor behavior has not been surpassed:

> Quiet or loud laughter, singing or a state of sadness, silence, murmuring, crying, or a barely audible muttering to one's self; or such a state of anger that the patient jumps up in a rage and can scarcely be held back, is wrathful at everyone, shouts, beats himself . . . or seeks to hide in fear, or weeps, or fails to answer those who speak with him, while he speaks not only with those who are not present but with the dead. (46, p. 23)

Both speech and nonverbal behavior are disordered and may range from catatonia and lethargy to incessant aimless activity and vocalizations. About 10% of patients may display violence toward others (28). Hypoactivity may predominate during the day, its opposite at night. Speech may be slurred, hesitating, disjointed, repetitious, circumlocutory, and paraphasic, and the patient's writing may show neographisms and spelling errors (47, 48). Tremor, such as asterixis, and choreiform movements may occur, especially in metabolic encephalopathies and drug withdrawal delirium.

Associated Features

A variety of emotions, but most often fear, apathy, rage, or depression, may accompany delirium (36, 38, 49). Autonomic, especially sympathetic, nervous system hyperarousal, manifested by flushed face, dilated pupils, tachycardia, and sweating, may occur in association with fear and hyperactivity (17). Incontinence of urine and feces is not uncommon in the delirious elderly.

Course and Outcome

Delirium comes on acutely over a few hours or days, often manifests itself first at night, and in the majority of cases clears up completely in 1 to 4 weeks. In the elderly, on the whole, it tends to last longer than in the younger patients (12). About half of the survivors leave the hospital within a month (26). For many patients delirium is a terminal event, as was mentioned earlier. The survivors have a good prognosis, as transition from delirium to dementia appears to be relatively uncommon. The favorable outcome is influenced by early detection and effective treatment of the syndrome. A demented delirious patient who is adequately treated is likely to return to his or her previous level of cognitive functioning, unless additional brain damage has occurred.

MULTIFACTORIAL ETIOLOGY

By definition, delirium is an organic brain syndrome, i.e., a mental disorder whose occurrence requires cerebral dysfunction due to one or more organic etiologic factors (8, 9, 17, *DSM-III*). The latter include systemic or cerebral disease, exogenous physical or chemical agents, and withdrawal from certain substances of abuse. The presence of one or more of these factors is a necessary, but not always sufficient, condition for delirium to occur. Some writers have emphasized the etiologic importance of psychosocial variables in delirium of the elderly (15, 36, 37, 50). Kennedy (36), for example, asserted that impaired brain function due to physical causes renders an elderly individual exquisitely vulnerable to a wide range of psychosocial stressors, such as bereavement, relocation to an unfamiliar environment, or destruction of the home, all of which may help precipitate delirium. Kral (37, 50) postulated that brain damage and lowered resistance to stress predispose many elderly persons to cognitive disorganization in response to both psychological stressors, such as loss of spouse or rejection, and physical ones, such as a hip fracture.

Etiologic organic factors could be identified in 80% to 95% of reported cases of clinically diagnosed delirium in the elderly (26, 28, 51–53). The remaining 5% to 20% represent an unsolved puzzle. They could include patients with pseudodelirium as well as those in whom organic etiology escaped detection. Some

investigators have reported relatively frequent concurrence of delirium and depression (26, 29, 52, 54–56). Elderly depressed patients are often malnourished and physically ill and are likely to be taking a variety of drugs, including tricyclic antidepressants. All of these factors are potentially deliriogenic (26, 52, 54). A transient cognitive disorder has been reported to occur in 12% of elderly patients suffering from an acute affective psychosis (56), and it may accompany other functional mental disorders as well (57). It is not clear at this time what proportion of these cases represents pseudodelirium rather than delirium.

Etiology of delirium in the elderly is typically multifactorial, in the sense that several causative organic factors are often implicated and their deliriogenic effect is frequently enhanced by such psychosocial stressors as bereavement, transfer to an unfamiliar environment, or excessive or deficient sensory inputs (17). Sleep loss and fatigue may also play a contributory role. The elderly are highly vulnerable to the cognitively disorganizing effects of all of these factors because of certain ubiquitous predisposing conditions (58).

Predisposing Factors

Aging processes, brain damage or disease, and impairment of vision and hearing predispose the elderly to delirium (8, 17, 26, 32, 34). Aging of the brain and of the special senses appears to facilitate cognitive disorganization in response to both physical and psychosocial stressors. Brain damage and disease, especially vascular and degenerative, increase predisposition to delirium even further. Capacity for homeostatic regulation and for resistance to stress is reduced in the elderly, possibly as a result of age-related changes in hypothalamic nuclei (50, 59). Circadian rhythms undergo changes. Certain parts of the brain on whose integrity normal cognitive processes depend are susceptible to aging and show selective cell loss. There is loss of cells and reduction in the dendritic tree in the cerebral cortex (59). Frontal cortex, hippocampus, and locus ceruleus are among the structures selectively involved (60). Destruction of locus ceruleus or raphe nuclei or both has been blamed for the occurrence of nocturnal delirium in demented elderly patients (61). The central cholinergic system is affected by aging, and even more so by degenerative brain disease, with resulting reduction in acetylcholine synthesis (62). Since adequate functioning of this system is needed for normal memory, learning, attention, wakefulness, and sleep-wake cycle, its deficiency is likely to predispose to delirium. Some authors claim that reduced acetylcholine synthesis due to various systemic disorders is a common denominator in metabolic encephalopathies (63). The normal elderly show reduction of cerebral blood flow and glucose metabolism, changes that are much greater in the presence of even mild and asymptomatic arteriosclerosis and are most pronounced in senile dementia (64–66). The aging brain is highly vulnerable to hypoxia of any origin.

These changes related to aging, cerebral disease, or both are compounded by additional factors such as increased general susceptibility to, and a high frequency of, episodes of disease and high prevalence of chronic diseases. Furthermore, impaired mechanisms of drug metabolism render the elderly highly susceptible to drug-induced delirium (17, 34).

Associated Physical Conditions

The most common physical illnesses associated with delirium in the elderly include congestive heart failure, pneumonia, urinary tract infection, cancer, uremia, malnutrition, hypokalemia, dehydration and/or sodium depletion, and cerebrovascular accidents (26, 28, 30, 51, 52, 56). Systemic diseases are much more often implicated than the primary cerebral ones. Intoxication with medical drugs is probably the most frequent single cause (8, 9, 17, 34).

The elderly are singularly prone to adverse drug reactions as a result of age-related changes in the metabolism and distribution of drugs in the body (34, 67). High drug consumption and frequent polypharmacy are notorious consequences of the current prescribing practices for elderly patients and no doubt increase the incidence of drug-related delirium (34, 67). Drugs with anticholinergic properties are among those most frequently prescribed for the elderly (68) and probably constitute the single most common cause of delirium in this age group. Delirium is especially likely to be induced when several anticholinergic drugs are administered concurrently (68). Table 1 lists the more important organic causes of delirium in the elderly (5, 17, 28, 34, 51, 53, 63, 68, 69).

PATHOGENETIC MECHANISMS

Almost 50 years ago, Hart wrote: "Of the precise processes by which delirium is mediated we know nothing. In discussing at the present time the possible pathogenesis of delirium we have therefore to leave the sphere of knowledge and enter that of hypothesis and speculation" (70, p. 747). This statement still holds. Several mechanisms are proposed to account for the occurrence of delirium in the elderly, and they may be classified as neurochemical, stress, information input, and sleep-wake cycle disturbance hypotheses.

Engel and Romano (71) postulated that a general reduction of cerebral metabolism underlies all cases of delirium and is reflected in concurrent cognitive impairment and a slowing of EEG background activity. Any factor reducing the supply, uptake, or utilization of substrates for oxidative metabolism could thus result in delirium. Blass and Plum (63) hypothesized that impairment of brain oxidative metabolism results in reduced synthesis of neurotransmitters, especially of acetylcholine, whose relative deficiency in the brain appears to provide a major, if not the only, pathogenetic mechanism in delirium. This hypothesis is sup-

TABLE 1. Common Organic Causes of Delirium

Cause	Type
Drugs	Diuretics, sedative-hypnotics, analgesics, antihistaminics, antiparkinsonian agents, antidepressants, neuroleptics, cimetidine, digitalis glycosides
Alcohol intoxication or withdrawal	
Cardiovascular disorders	Congestive heart failure, myocardial infarction, cardiac arrhythmias, aortic stenosis, hypertensive encephalopathy, orthostatic hypotension, subacute bacterial endocarditis
Infections	Pneumonia, urinary tract infection, bacteremia, septicemia, cholecystitis, meningitis
Metabolic encephalopathies	Electrolyte and fluid imbalance; hepatic, renal, and pulmonary failure; diabetes and other endocrine diseases; nutritional deficiency (especially of vitamin B complex); hypothermia and heat stroke
Cerebrovascular disorders	Transient ischemic attacks, stroke, chronic subdural hematoma, vasculitis
Cerebral or extracranial neoplasm	
Trauma	Head injury, surgery, burns, hip fracture

ported by the ease with which delirium may be induced clinically or experimentally with anticholinergic drugs (17, 68, 72) and be reversed by physostigmine, a cholinesterase inhibitor (17). Itil and Fink (72) have proposed that imbalance of central cholinergic and adrenergic mechanisms, affecting both the medial ascending reticular activating system and the medial thalamic diffuse projection system, underlies delirium. Increased central noradrenergic activity has been found in delirium tremens and probably accounts for its characteristic features, such as hyperalertness and hyperactivity (73), as well as for the absence of EEG slowing (74). Thus, imbalance of cerebral neurotransmitters, notably of acetylcholine and noradrenaline, appears to be implicated in many, possibly in most, cases of delirium.

Kral (37, 50) hypothesized that delirium in the elderly represents a reaction to acute stress, one mediated by abnormally high levels of circulating corticosteroids, or by increased vulnerability of the hypothalamus to their effects, or by concurrence of both of these conditions. In the elderly, secretion and plasma levels of cortisol are preserved while the rate of its degradation is reduced (75). In response to stress, patients suffering from senile dementia show higher and more sustained increase in plasma cortisol levels than do the normal elderly (50). Kral has speculated that excessive cortisol brings about delirium by virtue of its deleterious effect on the brainstem centrencephalic system. Cortisol does affect both cerebral and mental function and appears to interfere with selective attention and hence with processing of information (76), but its role in delirium is still speculative. Adrenaline and noradrenaline levels tend to remain elevated for a longer time in stressed elderly people than in young individuals (77). Since increased secretion of catecholamines leads to increased cerebral blood flow and oxygen consumption in experimental animals (78), one may postulate that a stress-related increase of such secretion in the elderly, especially the demented, could accentuate cerebral metabolic demands and further reduce oxygen tension, thereby facilitating onset of delirium.

Levels of sensory stimulation needed for optimal cognitive activity decline with age, rendering the elderly susceptible to sensory overload and its disorganizing effects on cognition (15, 79). Some authors believe that sensory deprivation contributes to delirium in the elderly (9, 15, 36). Recent work using positron computed tomography indicates that cerebral glucose metabolism in human beings decreases with reduced sensory inputs (80). Cameron (81) observed that senile patients prone to nocturnal delirium could be made delirious by being put in a dark room in the daytime. He postulated that memory impairment rendered these patients dependent on uninterrupted input of visual cues, and abolition of such cues in the dark elicited spatial disorientation, anxiety, and delirium. Sensory deprivation has also been invoked to explain delirium in elderly patients undergoing cataract surgery (17).

Nocturnal delirium, common in demented patients, has been ascribed in part to sleep pathology. Feinberg and associates (82) observed that demented elderly patients tended to awaken abruptly from dream periods (REM sleep) and to display agitated delirium for up to 10 minutes. These researchers postulated that nocturnal delirium could in some cases be due to abrupt transition from dreaming sleep to wakefulness, with consequent intrusion of dreams into the waking state. More recently, Japanese researchers proposed that nocturnal delirium in dementia could be due to the occurrence of stage 1-REM (REM sleep without muscle atonia) caused by destruction of serotonergic neurons in raphe nuclei, or of noradrenergic neurons in locus ceruleus, or both (61). There is evidence of selective loss of noradrenergic neurons in locus ceruleus in senescence and especially in senile dementia (83). Diurnal sleep-wake patterns in elderly subjects tend to be fragmented, with a high prevalence of sleep apnea, excessive daytime drowsiness, and microsleeps (84). Disruptions of the sleep-wake cycle appear to be common in the elderly, especially in the presence of cerebral disease, and it is likely that they play an important part in the genesis of transient cognitive disorders. Psychosocial stress and sensory understimulation or overload, singly or in combination, may play a contributory role in some cases.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis of delirium involves two essential steps: 1) recognition of the syndrome and 2) identification of its cause (or causes) (17). The syndrome is recognized on

the basis of its clinical features, described earlier, and by applying *DSM-III* diagnostic criteria. The patient's cognitive function and attention are tested as part of the psychiatric examination (69). One of the clinical scales may be used for this purpose, such as the Mental Status Questionnaire (85) or the Mini-Mental State (86). Once delirium is diagnosed or suspected, the search for causative factors must start at once. All medical drugs that the patient is taking should be carefully scrutinized for their deliriogenic potential. Evidence of physical illness, either acute or chronic with acute exacerbation, must be looked for, as the patient's life may be at stake. *It must never be assumed a priori that a patient's transient cognitive disorder is due to a reported life change or psychosocial stress alone.* Such an assumption could result in failure to search for, diagnose, and treat an underlying physical illness, with possible fatal consequences for the patient. Physical, including neurological, examination and selected laboratory tests are called for in all cases. The choice of tests must depend on the clinician's judgment and may include some or all of the following: blood chemistries, hemogram, urinalysis, serology, ECG, chest X-ray, blood culture, toxic drug screen, CSF examination, EEG, and CAT scan.

The EEG has been claimed to be the most sensitive and reliable indicator of cerebral metabolism generally and of its derangement in delirium in particular (71). Bilateral diffuse slowing of EEG background activity has been shown to correlate positively with the degree of the delirious patient's cognitive impairment (71). The EEG may help distinguish delirium from pseudodelirium and intracranial from systemic causes of the former (87). In the elderly, however, the diagnostic value of the EEG is limited by the fact that generalized slowing is seen not only in delirium but also in primary degenerative dementia (88). Repeated tracings may be needed to distinguish between these two conditions, since in delirium the EEG tends to normalize as the patient's cerebral disorder improves.

Differential Diagnosis

Delirium needs to be distinguished from dementia and pseudodelirium. Such differential diagnosis may at times be very difficult, because delirium in the elderly is often superimposed on dementia; i.e., there is an organic brain syndrome that also features global cognitive impairment. Furthermore, a demented patient may suffer from both depression and delirium or pseudodelirium at the same time. An adequate history may be unavailable for a patient who lives alone or with a demented spouse, and the patient is usually a poor historian. Because information about the mode of onset of the disorder (i.e., whether abrupt or insidious) and about the nature and duration of the symptoms is crucial for the clinical diagnosis, history must be sought from all available sources. As a general rule, a patient who by all accounts has functioned well intellectually and then suddenly develops a cognitive-attentional disorder that fluctuates in severity over the course of a day and becomes most marked at night is suffering from delirium unless proven otherwise.

TABLE 2. Differential Diagnosis of Delirium and Dementia

Feature	Delirium	Dementia
Onset	Rapid, often at night	Usually insidious
Duration	Hours to weeks	Months to years
Course	Fluctuates over 24 hours; worse at night; lucid intervals	Relatively stable
Awareness	Always impaired	Usually normal
Alertness	Reduced or increased; tends to fluctuate	Usually normal
Orientation	Always impaired, at least for time; tendency to mistake unfamiliar for familiar place or person	May be intact; little tendency to confabulate
Memory	Recent and immediate impaired; fund of knowledge intact if dementia is absent	Recent and remote impaired; some loss of common knowledge
Thinking	Slow or accelerated; may be dream-like	Poor in abstraction, impoverished
Perception	Often misperceptions, especially visual	Misperceptions often absent
Sleep-wake cycle	Always disrupted; often drowsiness during the day, insomnia at night	Fragmented sleep
Physical illness or drug toxicity	Usually present	Often absent, especially in primary degenerative dementia

Delirium Versus Dementia

It is important to distinguish delirium from dementia, as the two syndromes carry different prognostic implications (89). It is a grave error to attach the label of senile dementia to a patient suffering only from delirium, yet such misdiagnosis is not uncommon and may cause disastrous consequences for the patient and his or her family (90, 91). To avoid misdiagnosis one needs to consider the evolution of the patient's symptoms over time as well as his or her behavior and cognitive performance at the time of examination (69, 89). Table 2 may assist the reader in the diagnostic process.

Differential diagnosis is often complicated by the concurrence of delirium and dementia, since about one-third of hospitalized demented patients are likely to suffer from superimposed delirium (8). Moreover, the two syndromes, as they are currently defined in *DSM-III,* do overlap and are not sharply delimited. In an already demented patient the features of delirium tend to be modified. Complex hallucinations, dream-like mentation, and confabulations are frequently lacking, and the patient exhibits mostly apathy interrupted by noisy restlessness, as well as more or less severe deficits of memory, general knowledge, and thinking ability. As a general rule, acute and fluctuating worsening of cognitive functioning in a patient known to

suffer from dementia should suggest the onset of delirium and lead to appropriate medical management. It is not clear at this time how often pseudodelirium occurs in the course of dementia in response to stress.

Delirium Versus Pseudodelirium

A transient cognitive disorder is not always delirium as this syndrome is defined in *DSM-III*. As noted earlier, in 5% to 20% of elderly patients with apparent delirium, no organic causative factor could be detected. The term "pseudodelirium" has been proposed to designate a delirium-like transient cognitive disorder occurring in the absence of demonstrable organic causes (92). Such disorders have been described in the literature for over a century and have been called "acute confusional insanity or state," "acute delirious mania," and "psychogenic delirium" (17, 70). The elderly, particularly those with some degree of chronic cognitive impairment, are especially prone to exhibit pseudodelirium as a feature of an affective, schizophrenic, brief reactive, paranoid, or atypical psychosis (56, 57). Disorientation, bewilderment, incoherence, and perceptual and psychomotor disturbances of rapid onset appear to characterize pseudodelirium. In contrast to delirium, however, laboratory evidence of diffuse cerebral dysfunction is absent. The incidence of pseudodelirium is unknown. Its clinical features have not been systematically studied and contrasted with those of delirium. This is an unexplored area of geropsychiatry, one clamoring for research.

To differentiate pseudodelirium from delirium on clinical grounds alone may be impossible at times, because the two syndromes resemble each other too much. Signs of physical illness, such as fever or asterixis, should suggest delirium. In the latter, the patient's level of awareness tends to fluctuate irregularly in the daytime and is usually lowest during sleepless nights. Pseudodelirium, like pseudodementia, may be suggested by inconsistencies in cognitive performance, as when a patient claims to be unaware of his or her whereabouts yet is oriented for time and has no trouble finding his or her way around (93). The probability of pseudodelirium being present is increased if the patient has a past history of psychiatric illness, displays marked depressive or manic features, is grossly and consistently delusional, or appears to be unmotivated to perform on bedside cognitive tests rather than truly impaired. Persistent questioning may reveal that the patient is cognitively more or less intact but is too preoccupied with his or her own thoughts, too agitated, or too withdrawn to perform on request.

If doubt persists about whether delirium is present, additional tests, such as the amobarbital interview, the EEG, and the dexamethasone suppression test (DST) may help (93, 94). In pseudodelirium cognitive performance tends to normalize under amobarbital, the EEG is normal (unless dementia is also present), and DST results are likely to be abnormal if a major depressive disorder underlies the transient cognitive disorder in the absence of advanced primary dementia or inanition (94).

TREATMENT

Therapy of delirium is two-pronged. First and foremost, one must look for the putative organic factors and treat or remove them. Drugs that the patient has taken may have to be withheld or their dosage reduced. Concurrently, symptomatic and supportive treatment must proceed (9, 15, 17, 30, 34, 58, 69, 95). Fluid and electrolyte balance and nutrition must be maintained. A proper sensory environment for the patient needs to be secured, one that guards against both extremes of sensory input. A quiet, well-lighted room, a clock and a calendar, and a few familiar objects or photographs may help reduce anxiety and cognitive disorganization. Good nursing—consistent, supportive, and orienting—is essential (15). Sleep may be aided by a hypnotic such as temazepam, 15–30 mg at bedtime. It must be kept in mind that practically all hypnotics may at times increase confusion. Finally, sedation may be necessary for an agitated patient who could sustain a fracture or some other serious injury, or even be killed, as a result of an attempt to run away or fight, especially at night when confusion tends to peak. Haloperidol is the drug of choice in this situation (17). It is effective and relatively safe and has only mild anticholinergic activity. Its main side effects in the elderly are extrapyramidal. It may be given orally or intramuscularly in doses of .5–5 mg twice daily, depending on the degree of the patient's agitation.

COMMENT

Fifteen years ago I called delirium the Cinderella of American psychiatry: taken for granted, ignored, and seldom studied (20). The situation has changed little since then, and the syndrome, despite its high frequency among the elderly, remains almost totally neglected by researchers. Much still needs to be learned about its incidence, phenomenology, pathogenesis, and prevention. The current upsurge of interest in geropsychiatry, occasioned by the continued aging of the population as a whole, may at last include delirium and the conditions that mimic it and stimulate badly needed clinical research. The relation of delirium to psychological stress, disordered biorhythms, sleep pathology, dreaming, dementia, and acute functional psychoses has never been properly explored, yet its nature touches on basic issues of the mind-body problem.

Application to this syndrome of the newest techniques for studying the brain-behavior relationship, such as positron emission tomography, may help clarify its pathogenesis in the future. Such methods may finally allow us to establish the theoretically and practically important relation of delirium to pseudodelirium. Can psychological stress alone give rise to acute

cerebral dysfunction manifested behaviorally as delirium? Is the *DSM-III* definition of this syndrome too restrictive by requiring presence of a "specific organic factor" as a necessary diagnostic criterion? Hart (70) raised the former question 50 years ago and thought that such a possibility was logically tenable, but this issue has never been satisfactorily resolved. Meanwhile, however, epidemiological and clinical studies should be undertaken. A recent study by a group of nurses, though flawed, points in the right direction (96). Liaison psychiatrists are strategically placed to carry out such research in collaboration with geropsychiatrists and neuroscientists (97).

In clinical practice, the often difficult task of differentiating delirium from pseudodelirium and dementia will occur with growing frequency, as the number of very old persons will predictably increase in the coming years. The importance of this task is underscored by the authors of a recent study: "Psychiatrists and other mental health professionals must be able to differentiate organic brain syndrome from functional psychiatric illness in order to avoid increased mortality and morbidity in elderly patients" (98, p. 432). Such differentiation presupposes knowledge of the distinguishing clinical features of the syndromes involved and highlights the need for appropriate research and teaching. To differentiate delirium and conditions that mimic it from dementia is a matter vitally important for the elderly patient's welfare and even survival, and should be a required skill for mental health workers and all other health workers. Since liaison psychiatrists and geropsychiatrists are, on the whole, the physicians most familiar with transient cognitive disorder, they should play a key and active role in the requisite teaching (97).

Finally, there is need to attempt prevention of delirium whenever possible. Liaison psychiatrists have demonstrated that this can be achieved, with resulting reduction of the length of hospitalization and hence its cost (99). Considering that the elderly, on the average, stay in the hospital for treatment of acute disorders 30% longer than younger patients do (31) and that the development of delirium tends to prolong their stay, preventing its onset could result in substantial savings and in avoidance of protracted disability due to cognitive impairment (97, 99, 100). The patient would also avoid the risk of being branded as demented, with all the undesirable consequences of such a label. Efforts to prevent delirium must involve a revision and reform of current uninhibited prescribing practices for elderly patients, as the syndrome is so readily induced by medical and psychotropic drugs (69).

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