

Medical Aspects of Catatonia

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This column reviews medical aspects of catatonia, including medical disorders that can cause catatonia as well as medical complications of acute and chronic catatonia. Before the introduction of antipsychotics, most cases of primary psychiatric catatonia were due to (catatonic) schizophrenia. Presently, affective disorders (bipolar disorder, severe major depressive disorder) are more common than schizophrenia as a cause of catatonia.

DIAGNOSIS

Although it is a relatively rare condition, catatonia may be acute or chronic. Catatonia is now recognized as a syndrome that may be encountered in a wide range of conditions including primary psychiatric disorders, metabolic disorders, neurologic disorders and brain injury, and drug-induced disorders.¹ Idiopathic catatonic states such as periodic catatonia have also been described. The core features of catatonia are stupor, motoric immobility, mutism, negativism, excitement, catalepsy, and posturing. The core features are the same regardless of whether the condition occurs in the context of a mood, psychotic, or medical state. The characteristic signs of catatonia and its diagnosis are reviewed in detail elsewhere.² The Bush-Francis Catatonia Rating Scale is a validated rating scale that is very useful clinically and in research.^{3,4} While the diagnosis of catatonia can easily be missed when its most classic signs are absent (eg, waxy flexibility with posturing), catatonia is also misdiagnosed. In psychiatric settings, catatonia may be mistakenly diagnosed in patients with a severe negative symptom or deficit state of schizophrenia, in those suffering the effects of prolonged institutionalization, or in withdrawn patients with dementia who have diminished social interaction. Medical, neurologic, and substance-induced conditions that may be mistaken for catatonia are discussed below.

PROGNOSIS

The prognosis in catatonia, both in response to treatment and after multiple episodes, is determined by the nature and severity of the disease state of which catatonia is a manifestation. Classical catatonic signs, such as mutism, stupor, negativism, and excitement, do not by themselves indicate the etiology or determine the prognosis. The prognosis is better for catatonia occurring in a mood disorder than in schizophrenia. Among medical causes of catatonia, the prognosis is better in metabolic or substance-induced disorders than in those involving injury to the brain. The prognosis is poorer in very chronic cases, those with incomplete recovery after electroconvulsive therapy (ECT), those with early relapse, and when catatonia is accompanied by dementia.

DIFFERENTIAL DIAGNOSIS OF CATATONIA IN MEDICAL SETTINGS

The evaluation and differential diagnosis in catatonia are challenging because a history and those aspects of the physical examination requiring cooperation are usually not obtainable from a catatonic patient; thus, collateral sources of information must be obtained. Other disease states can mimic catatonia and should be considered in the differential diagnosis.¹ These conditions include stiff-person syndrome,⁵ akinetic Parkinson's disease, malignant hyperthermia,⁶

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locked-in syndrome,⁷ selective mutism, conversion disorder, and other hyperkinetic and hypokinetic states.⁸

Stiff-person syndrome is an uncommon autoimmune disorder with progressive muscle stiffness, rigidity, and spasm, slowly progressive over the course of years.⁵ Akinetic Parkinson's disease also can produce a state similar in some respects to catatonia (ie, mute, immobilized), but it occurs well after the diagnosis of Parkinson's disease has been established, later in the course of the disease. Malignant hyperthermia is a familial disorder and may include some of the characteristics of catatonia, but it only occurs following general anesthesia. Selective mutism shares only that one feature with catatonia and is mainly found in young children.⁹ In adults, selective mutism occurs as a manifestation of personality disorder, factitious disorder, or malingering.¹⁰

Catatonia has been reported to result from a variety of medical conditions, including metabolic, neurologic, and substance-induced disorders. A review¹¹ of 261 published cases of catatonia found that in 75% of cases there was no relevant psychiatric disorder associated with the catatonic state. The most common cause of "organic catatonia" (ie, catatonia resulting from a general medical condition) is injury to the central nervous system (CNS), whether from stroke, trauma, vasculitis, tumor, or anoxia.¹² Strokes involving the anterior cerebral circulation may cause akinetic and apathetic states.^{13,14} Bilateral infarction of the medial frontal lobes¹⁵ or parietal lobes¹⁶ has been reported to result in catatonia. Catatonia has occurred with a variety of CNS tumors in various brain locations¹⁷⁻²⁰ and as a manifestation of a paraneoplastic syndrome.²¹ A catatonic-like state may also be caused by epilepsy,²² rarely as a post-ictal phenomenon,²³ and even as an adverse effect of anticonvulsants.²⁴

Metabolic disorders causing catatonia have included hypo- and hyperthyroidism, hypo- and hyperadrenalism, and vitamin B₁₂ deficiency.¹ Numerous drugs and toxins have been reported to cause catatonia, including neuroleptics, dopamine-blocking antiemetics, corticosteroids, cyclobenzaprine, disulfiram, and tetraethyl lead poisoning.^{1,25}

Hallucinogens can cause an excited catatonic state, including 3,4-methylenedioxymethamphetamine (ecstasy) and phencyclidine (angel dust). Carbon monoxide poisoning can cause catatonia due to damage to the basal ganglia. Neuroleptics can cause a parkinsonian catatonic state or catatonia as part of neuroleptic malignant syndrome as well as aggravate catatonia due to other causes.² If a patient with catatonia of any etiology is treated with a neuroleptic, it can be difficult, if not impossible, to discriminate the original catatonia from neuroleptic-induced catatonia, with a subsequent high risk of evolution into neuroleptic malignant syndrome. Neuroleptic malignant syndrome

can be particularly difficult to distinguish from "acute lethal catatonia," ie, severe agitated primary catatonia.²⁶ As if this was not diagnostically and therapeutically challenging enough, catatonia has also been reported as a result of withdrawal from clozapine,²⁷ anticonvulsants,²⁸ and benzodiazepenes.²⁹

MEDICAL COMPLICATIONS OF CATATONIA

Not all patients with chronic catatonia fit the classic picture of the rigid stuporous patient in a fixed posture, but it is such patients who are most at risk for medical complications. Chronic catatonic patients are particularly vulnerable as their physicians may fail to diagnose or treat medical complications or coincident medical illnesses, for several reasons. First, recognition and treatment of medical problems are difficult due to patients' mutism or other communication impairments, inability to cooperate, and prolonged immobility. A second reason is related to physician attitudes. Swartz and Galang³⁰ described three cases with long delays before serious medical problems were recognized because the patients were mislabeled as hopelessly demented, leading to therapeutic nihilism. Even when a patient's chronic catatonia is clearly recognized, physicians sometimes become passive in the pursuit of treatment and prevention of complications, out of pessimism and a sense of helplessness. Third, chronic catatonic patients typically reside in long-term care facilities (eg, nursing homes, state psychiatric hospitals) where general medical care resources may not be adequate. For all of these reasons, care providers for catatonic patients must be vigilant for medical morbidity. The following reviews the major medical complications frequently encountered in chronic catatonic patients.

Pulmonary Complications

The most common pulmonary complication of catatonia is aspiration. While the frequency of aspiration and its contribution to mortality in catatonia have not been quantified, aspiration is the most common cause of death in patients with dysphagia caused by neurologic disorders and the most common cause of death in patients on tube feedings.³¹ Aspiration can result in pneumonitis (inflammation caused by aspirated acidic gastric contents) and/or pneumonia (bacteria infection). Prophylactic antibiotics are not beneficial in patients who are considered at high risk for aspiration. In patients who do have aspiration pneumonia, broad-spectrum antibiotics that include coverage against gram negative organisms are recommended.³¹ While corticosteroids are often prescribed for aspiration pneumonitis, the practice is not supported by the available data.³¹ Based on

a single case of fatal aspiration pneumonitis in a patient with catatonia, Bort³² recommended prophylactic administration of antacids in catatonia. However, this is not a good idea. While antacids would neutralize gastric acidity and, therefore, would be expected to reduce pneumonitis, gastric acidity normally keeps stomach contents sterile. Hence, neutralization with antacids may promote colonization by pathogenic organisms, making aspiration pneumonia more likely and more severe.³¹ The same rationale would argue against routine administration of histamine-2 blockers or proton pump inhibitors.

Other forms of pneumonia are also common in patients with chronic catatonia. Multiple risk factors include malnutrition, weak cough, poor respiratory effort, atelectasis, and crowded institutional settings which also makes infection more likely with an antibiotic-resistant organism. When respiratory effort is extremely poor, respiratory failure may result.³³

Pulmonary embolus is another common cause of death in patients with catatonia. Prolonged inactivity promotes venous stasis which, in turn, may lead to thrombosis. Dehydration is another factor promoting venous thrombosis. In a retrospective study, Carroll³⁴ found 6% of catatonic patients developed venous thrombosis. Case reports^{1,2,35,36} of thrombosis often leading to pulmonary embolism have been frequent in catatonia. A review³⁷ of 22 cases of pulmonary embolism in psychogenic catatonia found that death from pulmonary embolus occurred only after the second week of catatonic symptoms, often without warning. Potential preventive measures include hydration, physical therapy, support hose, and prophylactic anticoagulation. Low-dose subcutaneous heparin and low molecular weight heparin are well supported by studies in post-surgical patients at high risk for deep vein thrombosis and pulmonary embolus. However, the risk appears to be lower in chronically immobilized patients,³⁸ for whom prophylactic anticoagulation is not standard care. Whether anticoagulation would reduce morbidity and mortality in catatonia is unknown but worthy of study. Pulmonary emboli are not a contraindication for ECT, and ECT has been safely administered to catatonic patients after they had pulmonary emboli.^{39,40}

Malnutrition and Gastrointestinal Complications

Malnutrition and gastrointestinal complications are also common in chronic catatonia. Reduced oral intake leads to dehydration and malnutrition, which, in turn, promote other complications, especially infection and skin breakdown.⁴¹ Dehydration also leads to constipation or ileus, which can be profound in catatonia.⁴² For all of these reasons, it may become necessary to provide enteral feeding, either via nasogastric (NG)

or percutaneous gastrostomy (PEG) tubes. Unfortunately, both carry risk of significant morbidity. As noted above, aspiration is the most common cause of death in patients with feeding tubes. NG tubes' complications include esophagitis, pneumothorax, empyema, and bronchopleural fistula. Feeding tubes are associated with diarrhea, dependent edema, and bacterial colonization of gastric contents.⁴¹ While duodenal placement of a PEG or a jejunostomy may be more effective in providing nutrition, they too can cause aspiration. Potential adverse metabolic consequences of feeding tubes include hypoglycemia, hypercapnia, and electrolyte abnormalities. The difficult decision regarding whether the benefits of enteral feeding outweigh its burdens may be avoided altogether if catatonia is treated early, aggressively, and effectively with ECT.

Oral and Cutaneous Complications

Chronic catatonia also may result in numerous adverse oral effects. Dental caries and gum disease are frequent. Poor oral hygiene promotes colonization with bacteria more pathogenic than normal flora, in turn making aspiration more likely to result in serious pneumonia. Frequent administration of antibiotics to treat infection leads to antibiotic-resistant bacteria and secondary oral fungal infections. Skin breakdown is extremely common. Stasis, immobility, and pressure all contribute to the development of decubitus ulcers.

Genito-urinary Tract Complications

Genito-urinary tract complications are frequent as well, including urinary retention due to bladder distention and urinary incontinence, requiring catheterization or diapers. Malnutrition, poor hygiene, and an indwelling catheter create high risk for urinary tract infection. Carroll³⁴ found urinary tract infections in 8% of catatonic patients, but this is likely an underestimate. Institutionalized patients are more likely to develop urinary tract infections with resistant organisms. When feasible, intermittent catheterization is preferable to an indwelling catheter. Despite precautions, the majority of patients with indwelling catheters for >2 weeks will eventually develop bacteriuria. Treatment of symptomatic bacteriuria is always indicated. For the asymptomatic patient, removal of the catheter and a short course of antibiotics are usually successful. If the catheter cannot be removed, antibiotic therapy for asymptomatic bacteriuria is unlikely to be successful and may just result in infection with a resistant strain.

In females with chronic catatonia, menstrual hygiene may be neglected, leading to vaginal infection. Treatment with antibiotics for other infections may result in candida vaginitis.

Neuromuscular Complications

Last, neuromuscular complications are also common in immobilized patients with chronic catatonia, including flexion contractures and postural nerve palsies. Both can be prevented through physical therapy and mobilization. Prolonged immobilization is a risk factor for rhabdomyolysis. While rhabdomyolysis has been recognized as a complication of acute lethal catatonia, neuroleptic malignant syndrome, and severe acute neuroleptic-induced dystonia, the incidence of rhabdomyolysis in chronic catatonia is unknown. **PP**

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