Review article

Catatonia

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Summary

Catatonia was first described by Karlbaum in 1874. It was subsequently catogorised as a subtype of schizophrenia. With the recognition of NMS as a form of catatonia and awareness that catatonia could occur in a number of psychiatric and medical illnesses, catatonia is now increasingly identified in clinical practice. Irrespective of the cause, the treatment of catatonia is the same as that of NMS. Benzodiazepine such as lorazepam in high dose are effective in the treatment of catatonia and ECT should be used in patients who do not respond adequately to benzodiazepines. In addition the underlying condition should be treated and in NMS antipsychotics should be withdrawn.

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Introduction

If one were to ask a psychiatrist, what catatonia is, the answer might be, "it is a type of schizophrenia". Karl Karlbaum in 1874 described catatonia in 26 patients who were inmates of the asylum where he worked. The patients described by Karlbaum were a heterogeneous group with multiple aetiologies. Among his patients were some with neurological disorders, epileptic seizures and medical conditions such as tuberculosis. Two decades later, Emil Kraepelin, an influential figure in psychiatry, grouped together catatonia, hebephrenia and paranoid psychosis as dementia praecox (1). He described catatonia as a "secondary accompanying phenomena" rather than a fundamental clinical sign of dementia praecox. Due to his influence and extensive writing, the view that catatonia is synonymous with schizophrenia has persisted to this day. In the 1980s it was thought that catatonia had become rare. This apparent decline was probably due to change of treatment setting from asylum to modern day-care units. Not conducting detailed physical examinations and lack of awareness about catatonia in mood disorders also contributed to the decline in the number diagnosed with catatonia (2).

Because catatonia is considered a subtype of schizophrenia, clinicians automatically assume that patients with catatonia have schizophrenia. This could result in antipsychotic treatment which can harm the patient if the catatonia is due to neuroleptic malignant syndrome. Associating catatonia with schizophrenia can also prevent the clinician from looking for other causes of catatonia. It is therefore important that psychiatrists are knowledgable about catatonia.

Pathophysiology

The early writers, Karlbaum, Kraepelin and others thought that catatonia was a problem of volition or will. This was based on the tripartite model of mind function. The mind or brain was thought to have three main functions; emotion, will or volition and thinking. Kraepelin described deficits in all three areas in dementia praecox (3). In manic depressive psychosis he described that the primary deficit was of emotions. He thought in catatonia, the primary deficit was in will or volition. A patient with catatonia remained fixed in a posture because he could not will himself from that posture. He was also not able to resist the manipulations of the examiner because he lacked will. This was described as the cause of the phenomenon known as waxy flexibility. This model of catatonia is now considered outmoded, but the exact mechanism which causes catatonia has remained elusive. Though the symptom complex is distinctive, the aetiology is varied and it is likely that the syndrome has a final common pathway. However it is puzzling as to why only a percentage of patients with conditions know to predispose to catatonia develop the disorder. For example, although mood disorder is implicated in the aetiology in about 50% of patients with catatonia, only 10-20% with mania develop catatonia.

The pathophysiology of catatonia is most likely linked to a dysfunction of the motor system (3). Early studies focused on the basal ganglia because subcortical structures are involved in initiating movement. Early postmortem studies of patients with catatonic schizophrenia showed lesions in the basal ganglia. It is uncertain whether these changes were due to catatonia or schizophrenia. Functional imaging which allows the study of cortical functioning during motor activity, shows involvement of the parietal cortex with additional changes in the orbitofrontal cortex (4).

It is important to explore the relationship between catatonia and neuroleptic malignant syndrome (NMS) as these syndromes may have the same pathophysiological basis. In both catatonia and NMS, rigidity and akinesia is present but tremor is prominent in NMS (5). Waxy flexibility, motor anosognosia and affective and behavioural changes are more likely in catatonia.

The role of dopamine has been of primary interest in catatonia. In early studies, increased levels of metabolites of catecholamines have been found in the urine of patients with catatonia. This suggests that the