

Clinical overview

Does persisting fear sustain catatonia?

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Objective: To examine the psychological substrate of catatonia.

Method: Reviewing the historical descriptions and explanations of catatonic behaviours by clinicians from its delineation in the 19th century to the present.

Results: Patients with catatonia are often haunted by fears and terrors; this has not been widely appreciated, and certainly was lost from view in the days when catatonia was considered a subtype of schizophrenia. The report contributes to resolving a major question in catatonia: is the mind in stupor *inactive*, as the blank state that we picture in anesthetized patients, or is the mind *active*, so preoccupied as to exclude all other influences.

The main finding: Persistent fear occupies the mind of catatonic patients.

Conclusion: The signs of catatonia are adaptations to persistent fear, akin to tonic immobilization. The relief afforded by sedation supports this interpretation.

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Clinical Recommendations

- Catatonia is an identifiable, verifiable and treatable behaviour syndrome that is no longer considered a type of schizophrenia.
- In acute hospital settings 7% or more of admissions meet criteria for two or more signs of catatonia for 24 hours or longer.
- The psychology of catatonic stupor, mutism, posturing, and other signs is dominated by persisting fear and terror, explaining the role of anxiolytic sedatives.
- Clinical skills allows the identification of homogeneous population samples for much needed physiology, neuroendocrine and psychology studies of catatonia.

Additional Comments

- Fear emerges as central in catatonia. Does this give us new insight into the physiological and psychiatric sequelae of trauma, stress, and sexual violence?
- Diverse forms of catatonia are included in the syndrome, verified by their common treatment response. Do biologically distinct subforms of catatonia exist – seen in endocrine and physiologic measures – or are these clinical variations?
- The clinical signs and treatment response of catatonia and melancholia overlap. Is there a common neurophysiological basis for this overlap? As well, neuroendocrine abnormalities are well defined for melancholia. Similar explorations for catatonia are needed.
- Catatonia is considered a psychiatric disorder. Yet it is commonly found among general medical, emergency room, and neurology hospital services. Should it be reclassified as a general medical illness with some psychiatric manifestations? Like neurosyphilis, should its place in medicine be changed?

The acute onset behaviour syndrome of catatonia is commonly seen as stupor and lack of responsiveness, and less often in an excited delirious form. It is

recognized by the presence of two or more signs identified in its listings of behaviours for 24 h or longer. The most common behaviours are mutism,

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negativism, posturing, rigidity, staring, and repetitive acts. The diagnosis is verified by the rapid symptomatic relief following the acute administration of a barbiturate or a benzodiazepine sedative. The diagnosis is validated when catatonia is relieved by the known effective treatments of sedation or electroconvulsive treatment.

Catatonia's features bear little relationship to other syndromes in the dictionary of medical diseases. It is not an infection, nor a metabolic disease, nor a cancer, nor a genetic error, and is not a disease of a single body organ or of the brain. After recovery, which is commonly full in patients treated in a timely fashion, no damage to the body remains even in the most lethal conditions (1, 2).

Patients in catatonic stupor are unresponsive, wakeful, with open eyes following events around them. They do not respond to questions, to commands, or to pinprick or other painful stimuli. A puzzling question asks, is the mind in stupor inactive, as the blank state that we picture in anesthetized patients, or is the mind active yet so preoccupied as to exclude all other influences? An extensive case literature supports the belief that the patients are preoccupied by heightened distress and by active fears that fully occupy their consciousness. This history helps us understand the mechanism behind our effective treatments

The first description of patients under the label 'catatonia' in 1874 by the German neuropsychiatrist Karl Kahlbaum stressed the motor elements of posturing, rigidity, staring, and repetitive speech and movements occurring in stupor and occasionally in excited states. When Emil Kraepelin recognized catatonia in his patients, he pigeonholed them within his picture of dementia praecox. The Swiss psychiatrist Eugen Bleuler relabeled these conditions as 'schizophrenia', establishing 'schizophrenia, catatonic type' as its single formulation in the psychiatric nomenclature, where it has lain for most of the 20th century. When catatonia responded to barbiturate sedatives and then to the induced seizures of ECT in the 1930s, these treatments were found unhelpful in other forms of schizophrenia, casting doubt on the association of the two syndromes. Increasingly, Scholars found catatonia signs more often in mood-disordered and systemically ill subjects, adding additional doubts as to the tie of catatonia and schizophrenia. Increasingly, catatonia became seen as a behaviour syndrome in association with systemic medical diseases, as a toxic response to neuroleptic and hallucinogenic drugs, and to seizures and trauma (3). By 1994, the commissioners of the DSM-IV psychiatric classification added a class of *catatonia secondary to a medical condition*, and in 2013, the

DSM-5 commissioners discarded the class of catatonia secondary to schizophrenia and retained catatonia as secondary to a medical condition, supporting its present image as an independent systemic syndrome.

Catatonia's characteristics make it unique among the disorders in the medical dictionary. A survey of its history brings attention to a theme, described over and over again, of persistent concerns with death and dying, of fear and fright of overwhelming experiences that cannot be escaped or defeated, suggesting that catatonia is best understood as an adaptive syndrome outside the common accepted causes of the body's disorders. It is often the case that subjects experience a severe threat that they cannot escape, from which they cannot hide, that they cannot defeat. The dread persists and the subjects seek to survive by denial, by dissimulation in extraordinary postures and repetitive behaviours, by withdrawing from the environment in stupor, or running, hiding, or threatening behaviours in activated terror. Even when the episode is not precipitated by fear and dread, fearful images haunt many of the patients during stupor.

In his descriptions of 26 cases of catatonia, Kahlbaum reports the patients as '*astonished*' or '*thunderstruck*'. Catatonia appeared

after very severe physical or mental stress... such as a terrifying experience'; 'the patient remains motionless, without speaking, and with a rigid masklike facies, the eyes focused at a distance... devoid of any will to move or to react to any stimulus'. 'The general impression conveyed... is one of profound mental anguish, or an immobility induced by severe mental shock'. To the extent that Kahlbaum concedes a psychology to catatonia, it is to mental pain and fright (4).

In a report of 1833 when psychiatry trainees asked patients what they recalled from their stupors, *Mme C*, much upset by the July Revolution in Paris of 1830 reported:

Soon her reason left her. She hears the cannon, sees the wounded, the blood, the dead. She gradually becomes overwhelmed. She stays motionless, her eyes fixed, mute, barely murmuring a few incoherent words (5)

In 1862, the French psychiatrist Henri Dagonet quoted Monsieur X, emerging from a stupor saying:

I imagined that both my parents were dead, that my native city had been flooded; it seemed to me that a new deluge had submerged all the land. While that was going on, I was always hungry, and I believed that people were going to let me die of inanition. In the garden where I was taken for walks, I was afraid of the patients around

me; to my eyes they looked like brigands and assassins, and I lived in continual anxiety. In walks outside the asylum I would walk slowly, with loathing; I was convinced that people wanted to drown me, or else have me crushed on the railway tracks that passed nearby(6)

August Hoch, a New York psychiatrist working on Ward's Island, described 36 patients with 'benign stupor' that meet the criteria for catatonia. For 35 of the cases, he reported '*thoughts of death or closely related conceptions*' or similar fears in each case (7). A decade later, a study of 100 patients with 'catatonic dementia praecox' admitted to the Hudson River State Hospital between 1927 and 1931, reported that 48 had ideas 'expressing fears of being killed (18), vague fears (15), of castration (10), of homosexuality (6) and of being buried alive (5)' (8).

The picture sharpens with the introduction of the sodium Amytal interview: with a few grains, the patients come awake, begin to speak, often requesting food (after they had been tube fed for weeks), and generally behaving amicably (after weeks of negativism). In this narrow window, which opened only for an hour or two, they had a chance to relate what they had been immediately experiencing during the stupor. In 1932, Erich Lindemann, at the Psychopathic Hospital of the State University of Iowa, reported that fear was the overriding theme. A woman, age 38, who had been mute, refusing food and muttering to herself for four weeks, '*lost the suspicious and apprehensive expression in her face*', two minutes after the Amytal injection. She appeared relieved, looked around in an interested way, and spontaneously asked the examiner, '*Where am I? Who brought me here? Where are my children? Can I go home?*' She reported that something terrible was pressing down on her, that she had a tremendous fear of impending danger, that she heard God's voice talk to her, that she was in a coffin which was prepared to take her to hell, and that she had a profound feeling of guilt with reference to autoerotic habits of her early adolescence. She remained alert for about two hours, then relapsed into her previous condition (9).

In experimental studies, Georg Northoff compared the thoughts and motor signs of akinetic catatonia patients with those with Parkinsonism. Of 22 catatonic patients, 13 were relieved by lorazepam within 24 h. On day 21 after treatment, the patients were asked for recollections of their akinetic states. Compared to the Parkinson patients, the catatonics were more concerned about the loss of control of their movements, overwhelmed by anxiety and the blockade of their movements by

emotions and feelings of isolation from their environment, and were not fully aware of the deficits in their motor functions (10).

After the prolonged immobilization of cardiac surgery, a patient was '*immobilized and almost like a statue*'; another '*was frozen and expressionless. She spoke barely audibly in a monotone with long pauses and made no spontaneous comments*' (11).

A 'resignation syndrome' marked by stupors unto death is now reported among refugee children coming to Sweden from Syrian wars (12). In the Uganda conflicts, a stuporous, repetitive 'nodding syndrome' progressing to death is reported. Some patients in this Uganda study were relieved by lorazepam, the effective treatment for catatonia (13).

Sexually assaulted women recall their overwhelming fear, feeling physically restrained and immobile, unable to move or resist. Such behaviour is labeled 'tonic immobility'—an involuntary, reflexive state of muscular rigidity, inability to move, with suppressed vocal behaviour (14). It is reported when humans are confronted by an inescapable, fear-inducing situation.

Tonic immobility is a behaviour described in animals. A rigid motionless posture is elicited by slowly and quietly stroking an animal, gradually releasing, with the animal now remaining immobile with limbs in the unusual postures in which they are placed. The phenomenon is demonstrated in prey animals, such as chickens and other fowl, rabbits, frogs, snakes, and guinea pigs (11). A tradition of pretending to be dead is described as the behaviour of the Virginia opossum—'playing possum', in childhood play.

Recognition that clinical catatonia is present in 10% of acutely ill psychiatric in-patients, that it is relieved by anxiolytic drugs, and that patients give the appearance of intense anxiety led the psychologist Andrew Moskowitz to propose catatonia as 'a relic of ancient defensive strategies, developed during an extended period of evolution in which humans had to face predators in much the same way many animals do today and designed to maximize an individual's chances of surviving a potentially lethal attack' (15).

The history of catatonia directs attention to fear as the precipitating and sustaining component. Examining our treatment efforts, we can understand the benefits of the sedative drugs, the barbiturates, benzodiazepines, and the resolutions of catatonia after anesthesia for surgery as interrupting the fear state and encouraging a more normal appreciation of the behaviour state. The resolution of the fear state by induced seizures can also be understood as an interruption by seizures or the sedatives.

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We know little of the neuroscience of catatonia. For decades, these cases have been lost within schizophrenia samples or rejected from studies by their unique features and negativism. Yet, among behaviour syndromes, catatonia is an identifiable, verifiable, and eminently treatable syndrome, making it suitable for study within homogeneous study samples. Attention to the role of fear and the syndrome's psychology offers additional opportunities to better understand the syndrome.

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