

Catatonia: A Recognizable and Treatable Systemic Syndrome

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Abstract:

Catatonia, a motor dysfunction of acute onset, expressed as mutism, negativism, posturing, staring, repetitive speech and others, was demarcated in 1874. Useful barbiturate treatments were described in 1930 and the benefits of inducing grand mal seizures (ECT today) was described in 1934. Many forms of the illness are now recognized: stupor, mutism, malignant catatonia, delirious mania, self injurious behavior in autism.

The diagnosis is made when subjects exhibit two or more signs for 24 hours or longer. The diagnosis is verified by the acute relief afforded by a benzodiazepine test. Treatment is by increasing dosages of benzodiazepines, and when this fails, ECT is effective. Optimized treatment algorithms are described.

Once thought to be a form of schizophrenia, this is no longer accepted in the latest DSM-5 where it is classified as “Catatonia secondary to a general medical condition.” Catatonia is a recognizable, verifiable, and treatable behavior syndrome that is found in up to 10% of hospitalized patients.

Keywords: catatonia, mutism, malignant catatonia, neuroleptic malignant syndrome, delirious mania, self-injurious behavior, toxic serotonin syndrome;

Barbiturate, benzodiazepine, lorazepam, zolpidem;

Electroconvulsive therapy, flumazenil;

Catatonia rating scale; benzodiazepine relief test

Catatonia: A Recognizable and Treatable Systemic Syndrome

An unkempt unmoving man, standing silently and stiffly, staring at the ceiling, arms tightly at his sides, paying no heed nor responding to the persons about him.

A child is mute, rocking to and fro, hitting his head against the bed's headboard, requiring restraints to protect him from self-harm.

A distraught and disheveled woman runs through the ward, repeatedly shouting "Don't kill me, don't kill me!" requiring restraint and sedation.

An acutely ill unresponsive young man is brought to the hospital Emergency Room in stupor, febrile, sweating, stiff, and dehydrated.

Each subject is suffering from catatonia, an acute systemic syndrome that can be effectively treated once it is recognized. For more than a century catatonia has been cloistered as a marker of the Kraepelin/Bleuler concept of schizophrenia. In the past 40 years, however, catatonia has been divorced from this marriage and is increasingly accepted as an independent, identifiable, and treatable syndrome distinct from schizophrenia. (Fink and Taylor 2003; Taylor and Fink 2003; Fink 2013; Shorter and Fink, 2018).

Before Kraepelin's descriptions of dementia praecox and manic depressive insanity as principal diseases among the chronically ill patients in his university academic hospital, an acute illness of abnormal motor behaviors had been delineated by Karl Kahlbaum in his sanitarium in Görlitz, Germany (Kahlbaum 1874; Kahlbaum 1973). In a small book of 110 pages, "*Die Katatonie oder das Spannungsirresein: eine klinische form psychischer Krankheit*," Kahlbaum presented 26 vignettes of immobile and stuporous patients; some rigid and posturing; others moving and pacing continually; some repeatedly hitting a wall or themselves; or standing and staring, unblinking, repeating words or phrases over and over, sometimes in whispers or repetitive shouts. Many were excited, grandiose, delirious, delusional or in stupor, but the behaviors that marked them together were their motor behaviors. Some also suffered with syphilis, tuberculosis, or epilepsy.

Kahlbaum delineated his patients so well that many authors quickly recognized the unique syndrome. Kraepelin found Kahlbaum's signs among his long-stay chronically ill. Shuffling index cards in which he had written his patients' histories, he found catatonia most often among those with *dementia praecox*, the life-long illness that progressed slowly to dementia and rarely remitted. In the 6th edition of his textbook in 1899, catatonia became a subtype of dementia praecox (Shorter 1997).

When the Swiss psychiatrist Eugen Bleuler renamed the illness to *schizophrenia* in

1908 he, too, described catatonia as an identifying marker. When psychiatric illnesses were formally classified in 1952 by the American Psychiatric Association in the *Diagnostic Statistical Manual* (APA 1952) the Kraepelinian tradition of citing catatonia only as *schizophrenia, catatonic type* continued. In the subsequent DSM revisions catatonia continued to be referenced as a singular type of schizophrenia (APA 1968, 1980, 1994).

For a century clinicians could only assign the diagnosis of *schizophrenia, catatonic type*, for any patient with catatonia. Following treatment guidelines for schizophrenia, the designation recommended the prescription of potent neuroleptic drugs. Some catatonic subjects developed an acute neurotoxic syndrome with escalating fever, hypertension, tachycardia, sweating and delirium. Some died. By 1980 the illness was labeled the “neuroleptic malignant syndrome” (NMS) (Caroff 1980). Believing NMS to be the result of the dopaminergic blockade of neuroleptic drug action, dopamine agonists were prescribed. And, erroneously seeing the possibility that muscle weakness was related to malignant hyperthermia, treatment with dantrolene was prescribed. But these treatments did not affect NMS. Improvement came when the administration of neuroleptics was discontinued.

By the 1980s, benzodiazepines and ECT were accepted as the effective treatments.

The connection of catatonia with schizophrenia in the psychiatric classification was partially broken in 1994 in DSM-IV when *catatonia secondary to a medical condition* was listed as an identifiable diagnosis. The connection of catatonia with schizophrenia was fully broken in the DSM-5 in 2013 with the deletion of the class *catatonia type of schizophrenia* and retention of *catatonia secondary to a medical condition*. Catatonia is now identified as a systemic medical, not as a primary psychiatric, syndrome (Fink et al., 2016). This designation strengthens the consideration of catatonia in patients referred to modern consultation-liaison services.

When to consider catatonia

Catatonia is to be considered in the differential diagnosis of any patient reporting an acute change in motor behaviors – mutism, excitement, delirium, stupor, abnormal speech, posturing and repetitive rhythmic acts. The alternation of agitation and stupor is

characteristic of catatonia. The diagnosis is aided when patients are examined using a Catatonia Rating Scale (Fink and Taylor 2003). My personal experience is with the examination developed at Stony Brook University Hospital, cited as the Bush-Francis CRS, published in 1996 (Bush, Fink, Petrides et al. 1996a,b) (See Appendix 1-3).

The CRS examination, which usually requires less than five minutes, lists 23 items, scored on either a 4-point or 2-point scale.

The first 14 items are recommended as a “screening instrument” to identify subjects with total scores of 2 or more who warrant further testing. In hospital-wide surveys using the CRS, most patients score 2 or less. Positive catatonia scores range from 4 to 14 on the screening instrument.

Body temperature is the second guide to the diagnosis. Fever and autonomic abnormality mark malignant catatonia, a life-threatening condition. Febrile patients warrant a detailed systemic medical review.

Verifying the Diagnosis

Catatonia is quickly relieved, albeit temporarily, by the administration of a benzodiazepine (lorazepam, diazepam), a barbiturate (amobarbital), or a GABA agonist (zolpidem). An intravenous injection of 1 mg or 2mg lorazepam or the oral administration of 10mg zolpidem brings a reduction of catatonia signs within a few minutes. A reduction of the CRS score of 50% or greater verifies the diagnosis of catatonia.

Treating Catatonia

Catatonia is relieved by high doses of benzodiazepines and induced grand mal seizures (ECT). About 80% of catatonia cases respond to sedative drugs; the remainder recover by inducing seizures.

Benzodiazepines

In 1930 the American physician William Bleckwenn described the rapid relief of catatonia by injections of high doses (0.5 to 2.0 Gm) of amobarbital (Amytal sodium). Catatonia quickly improved, as he reported and demonstrated in an historic silent black and white film (Bleckwenn 1930 a,b). Full relief followed the administration of large daily doses.

The favorable response of catatonia to a barbiturate contrasted sharply with the poor response of schizophrenic patients without catatonia to such treatment, raising doubts as to the connection of the two disorders. These doubts led to the separation of catatonia from schizophrenia and its recognition as a systemic disorder (APA 2013)

Since Dr. Bleckwenn's experience, amobarbital has been replaced by a benzodiazepine, with lorazepam and diazepam most frequently used. These compounds are considered safer than the barbiturates and less likely to be used in suicide.

For patients with the sedated stuporous form of catatonia marked by mutism, withdrawal and inhibition of movement, the dosage of lorazepam begins at 1 to 3mg orally, progressing rapidly until symptom relief is seen. For the severely ill, dosages of 15mg to 30mg/day have been necessary. (Diazepam dosages are calculated at ratios of 5mg to 1mg of lorazepam; Zolpidem dosages are quoted to 40mg/day.)

Induced seizures (ECT)

In 1934 the Hungarian neuropathologist Ladislas Meduna observed a decrease in glia in the autopsied brains of schizophrenic subjects and a surfeit in those with epilepsy. As some clinical studies had reported an absence of schizophrenia among hospitalized epileptic patients, he conceived of increasing glia in schizophrenic patients by inducing seizures using the chemical pentylenetetrazol (Metrazol, Cardiozol) (Meduna 1937). Many of his first patients were catatonic and these were the ones that showed the most relief (Gazdag et al 2009).

Chemically induced seizures were inefficient, however, as many injections failed to elicit a seizure. By 1938 the chemical induction was replaced by electricity, a more efficient and more secure method, that is the modern technique of electroconvulsive therapy (Shorter and Healy 2007). With the benefits of these treatments and the increasing

use of psychoactive drugs, the sanatoria emptied and catatonia seemed to disappear from psychiatric practices (Mahendra 1981).

The choice of treatment, whether benzodiazepines or ECT, is determined by the severity of the illness and the degree of fever and autonomic instability. For the more severely ill, i.e., those who are febrile and exhibiting the systemic signs of a malignant illness, ECT is preferred (Fink and Taylor, 2003).

1. What is an “effective seizure”? While we do not understand why or how grand mal seizures have the favorable consequences that permit us, essentially, to “fight one disease with another,” we know that a “full” grand mal seizure is a necessary marker of effective treatment. Heart rates increase; hypothalamic and pituitary hormones are released into the blood and cerebrospinal fluid (most easily measured by serum prolactin at 20 minutes post-seizure [Swartz 1985]); and EEG recordings show patterned rhythms. The seizure, not the currents or amounts of electricity, are the basis for a favorable outcome (Fink 1979, 2009).

Not all seizures are equal in efficacy. Whether the treatment was effective is best determined by the pattern and duration of the seizure EEG. (Modern ECT devices include sophisticated EEG recording instruments.) The electrical stimulus momentarily blocks the recorded brain rhythm, followed by a gradual build-up of ever higher amplitudes and slowing of frequencies (15-25 sec), the interspersed spikes and slow waves (10-15 sec), a period of high amplitude slow waves (10-20 sec), ending in a sharp end-point and flattening of energies. For such events to reflect an effective treatment the minimum overall durations are greater than 30 seconds, often averaging 40 to 80 seconds. Shorter seizure durations are associated with poorer clinical outcomes (Fink 2000, 2009). (See Figure 1).

An effective EEG seizure

The duration and the EEG patterns of the seizure are affected by the placement of electrodes, energy dosing, types of currents, age of patient and pre-treatment medications. The protocols for the parameters to induce the seizure vary with the illness being treated. In adult and elderly depressed patients the common belief that the immediate cognitive effects are worsened by the electrical energy leads to protocols designed to decrease the

energy dosing, unilateral electrode placement, ultra-brief currents and 2/wk schedules. Such schedules are inefficient in treating catatonia, an illness of greater severity than most mood disorders. To treat catatonia successfully and rapidly, the following guidelines are recommended:

a. Bitemporal electrode placement: Treatments are optimized by bitemporal (or bifrontal) electrode placement, and dosing by half-age calculated energies in U.S. marketed devices (Petrides and Fink 1996). Since the 1960s, repeated comparisons of the efficacy and safety of unilateral and bilateral electrode placements compel the conclusion that treatment efficacy is impaired in unilateral electrode placement (Fink and Taylor 2007). Considering the mortal risks of dehydration, blood stasis, thrombosis and death in incompletely treated catatonia patients, there is little justification for the use of RUL placements in patients with catatonia.

b. Daily seizures: In patients with malignant catatonia (NMS, delirious mania) daily treatments are useful. Indeed, for patients with high fevers, daily treatments of two seizures a day are life-saving (Arnold and Stepan, 1952).

c. Flumazenil: Benzodiazepines inhibit seizures making higher energies necessary for an effective treatment. Since catatonic patients are commonly prescribed benzodiazepines before referral for ECT, greater stimulus energies are needed to induce full grand mal seizures. The intravenous administration of the benzodiazepine antagonist flumazenil (0.5 mg IV) quickly blocks seizure inhibition by the benzodiazepine, encouraging a full seizure to develop. Flumazenil is injected in conjunction with the adjuvants for amnesia, muscle relaxation and anticholinergic vagal blockade.

d. Ketamine: The commonly used amnesic agents methohexital, thiopental, etomidate and propofol inhibit both seizure durations and quality. Intramuscular ketamine offers better premedication, especially in excited patients. Ketamine lowers seizure thresholds enhancing seizure quality and is a preferred medication.

e. *Continuation (maintenance) ECT*: For a variety of historical reasons, many patients are prescribed a fixed number of seizures for their treatment course. The number is incorporated into the consent signed by the patient (or by the justice when ECT is endorsed by a court). But such numbers are guesstimates that are founded only on historical averages. The number of seizures, like the dosage of psychoactive medications, varies widely. Since the 1970s continuation ECT has been encouraged and more and more patients are being treated for many months and occasionally for years (Fink et al.1996). C-ECT is recognized as necessary in sustaining the benefits in treating major depression (Fink 2014b) and in catatonia (Wachtel and Shorter 2013).

Accompanying medications and catatonia

Neuroleptics: The prescription of neuroleptics is not recommended in catatonia, indeed their use is best interdicted. High potency neuroleptics like haloperidol precipitate a malignant illness in catatonic patients that is often fatal (Shalev and Munitz 1989). The widespread use of intramuscular haloperidol in severely agitated and excited patients is too risky to be encouraged; high dose benzodiazepines are to be preferred to sedate the severely agitated and excited patient. There is little evidence that catatonia is relieved by either typical or atypical neuroleptics. Unfortunately, such use is still common, because of the historical association of catatonia with schizophrenia.

Anticonvulsants: Because many catatonia patients exhibit rhythmic repetitive movements, these have been seen as evidence of seizure disorders and anticonvulsants prescribed. Case reports describe carbamazepine, valproate and lithium as favorable augmenting agents during ECT treatment and during prolonged continuation treatments.

The many faces of Catatonia

Once catatonia was defined by reliable diagnostic criteria, numerous syndromes were brought into the catatonia tent, first by recognition of catatonia signs and then by the efficacy of the catatonia treatments. In patients with two or more catatonia signs for 24

hours or longer, the quick relief from an acute administration of a benzodiazepine verifies the presence of catatonia, and both induced seizures and high dose benzodiazepines were found clinically effective (Fink and Taylor 2001).

The common image of catatonia is the mute, stuporous, posturing, rigid, staring and negativistic patient. Persistent mutism is described in many different forms including *selective mutism* and *persistent refusal syndrome*, the latter commonly described in the UK (Fink 2013).

A febrile neurotoxic lethal form that follows the administration of high potency neuroleptic agents, known as the *neuroleptic malignant syndrome* (NMS), was increasingly recognized in the 1980s. The pathophysiology was thought to result from dopaminergic blockade and the agonist bromocriptine was prescribed. Muscular weakness was related to malignant hyperthermia and the muscle relaxant dantrolene was also prescribed (Caroff, Mann, Francis and Fricchione 2004). But these treatments were ineffective. When the connection to catatonia was seen and benzodiazepines and ECT were prescribed, outcomes were much better. NMS is successfully treated as a form of catatonia (Fink and Taylor 2003).

The *toxic serotonin syndrome* (TSS) is an acute syndrome with motor and vegetative signs similar to NMS that follows the use of serotonergic agents. It is responsive to catatonia treatments (Fink 1996).

Malignant (lethal, toxic) *catatonia* was described before the advent of neuroleptic agents, occurring as an acute syndrome with fever, dehydration and excitement. A life-threatening form labeled *delirious mania* (DM) is identified that is remarkably responsive to daily induced seizures (Fink 1999).

Self-injurious behaviors (SIB) are increasingly recognized among adolescents with mental handicap, autism and autism spectrum disorders (Wachtel et al. 2013). The same repetitive acts are features in Gilles de la Tourette syndrome and obsessive compulsive disorders. When such patients are seen as ill with catatonia they have been successfully treated by ECT (Trivedi et al. 2003; Fink et al. 2013).

Since 2007 an auto-immune encephalitis identified by an abnormality of the NMDA receptor in serum or CSF tests is characterized by catatonia. When identified, its treatment as catatonia is successful (Dhossche et al. 2011).

Each of these behavior syndromes is identified by catatonia signs and successful treatment validates the diagnosis of catatonia. The range of behaviors recognized as catatonia, the increasing recognition of catatonia as a systemic medical illness and the divorce from the century-long association with schizophrenia has moved catatonia from its consideration as a psychiatric disorder to the increasing recognition as a systemic medical disorder. When surveys of the numbers of patients with catatonia are done in academic hospitals and emergency rooms, using Catatonia Rating Scales, from 9% to 20% of the populations show two or more catatonia signs (Fink and Taylor 2003).

Such association, with its effective treatments, has done much to relieve catatonia in emergency rooms, medical and neurologic clinics and consultation and liaison services. Identifying catatonia as an independent syndrome and its effective relief by available treatments is an important milestone in the history of medicine.

The Biology of Catatonia

How are we to envision catatonia among the systemic illnesses that make up the body of medicine? Catatonia is a disorder of posture, movement and speech, with many patients reporting intense anxiety and fear (Fink 2013; Fink and Shorter, 2017; Shorter and Fink, 2018). It does not result from a structural defect in a single body organ nor is it associated with a physiologic dysfunction. It is not the consequence of a brain lesion. It occurs in the context of general medical illnesses. After catatonia is relieved we see no residuals; it is as if the blackboard has been erased with a few smudges left at the corners. It is a behavior of the whole organism, arising suddenly and vanishing without a trace. It is better likened to an inherited behavior like sleeping or crying or coughing or sneezing.

Catatonia is distinct from other behaviors defined as psychiatric illnesses. It is not a disorder in thought or emotion, although such accompaniments are common. Some authors consider catatonia as an “end-state” whole-body response to imminent doom, a behavior inherited from ancestral encounters with carnivores, an adaptation that remains an inherent feature of living (Moskowitz 2004). This would make catatonia an atavism.

Kahlbaum described his 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.” He continues: *“The general impression conveyed . . . is one of profound mental anguish, or an immobility induced by severe mental shock”* (Kahlbaum 1973). Citing fear as the central theme of the syndrome, Kahlbaum titled his book *Catatonia, The Tension Insanity*.

An Australian psychiatrist described four patients with systemic physical illness who exhibited catatonia in association with intense fear (Perkins 1982). Although the patients improved with treatments for catatonia, recovery depended, he believed, on the treatment of the psychological stressors. He regarded catatonia as regression to a primitive state of mind elicited by overwhelming fear.

Georg Northoff and his colleagues in Frankfurt, Germany assessed the experiences three weeks after recovery in 24 patients (15 excited, nine inhibited) who met the criteria of four or more catatonia signs (Northoff et al. 1996). The patients' greatest fears were their inability to control intense anxiety. They felt threatened and feared dying. They were less concerned about their lack of control of body movement or of self-care.

Reactions characteristic of catatonia are encountered after immobilization for cardiac surgery. One 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.”* The postoperative reaction has been described as experiencing a catastrophe, the patients resembling *“the photographed faces of survivors of civil disasters, the countenances present staring and vacant expressions of seeming frozen terror. Immobile, apathetic, and completely indifferent to their fate, they respond to inquiries in monosyllables devoid of affect”* (Gallup 1977).

A “resignation syndrome,” marked by severe stupors and death, is reported among refugee children coming to Sweden from the Syrian wars (Sallin et al. 2016). In the Uganda conflicts, a stuporous, repetitive “nodding syndrome” progressing to death is reported (Kakooza-Mwesige et al. 2016). Some patients in this Uganda study responded to lorazepam.

Are concepts of “tonic immobility” and “negative conditioning” in animals relevant? Tonic immobility is the rigid posture 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 chickens and other fowl, frogs, snakes, guinea pigs and rabbits (Marx et al. 2008). A tradition of pretending to be dead is described as the behavior of the Virginia opossum – “playing possum,” as it is described in childhood play (Galliano et al. 1993).

Intense fear is the evolutionary basis for both tonic immobilization and for catatonia. Recognition that catatonia is present in 10% of acutely ill psychiatric inpatients, that it is relieved by anxiolytic drugs and that patients give the appearance of intense anxiety, led the New Zealand psychologist Andrew Moskowitz (2004) to propose that catatonia is “*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.*”

The principal defenses of prey animals are flight, fight and dissimulation. Flight occurs when a predator is at a distance; fight is an option when escape is not possible; and hiding, dissimulation and absence of movement occur when the predator is at a distance that would permit not seeing an immobile prey. The core catatonic symptoms of stupor, mutism and immobility can be linked to tonic immobilization with prominent examples in rape assault and cyber-bullying experiences (Fink 2013).

Stupor, rigidity, posturing and mutism of catatonia are analogous to tonic immobilization. Repetitive words and acts, posturing and grimacing are dissimulations – attempts to appear other than oneself. Fright and discomfort stimulate crying that brings nursing, cuddling and relief. Older children develop a repertoire of calls, screams, cries, postures, hiding, throwing and breaking of objects to bring similar relief. Being mute and not responding brings desired attention. The behaviors may be active or passive; for each the caretakers and other children respond, and soon postures, grimaces, repetitive acts, repetitive speech and withholding of speech become learned behaviors.

Catatonia is a behavior that is identified by observation. Changes in movement, posture and speech are sufficient to identify the syndrome. No damage to the body remains after recovery, indicating that the abnormal behavior is an exaggeration of a normal state. Such observations encourage us to view catatonia as an inherited adaptive syndrome outside the conventional causes of the body’s systemic disorders.

L'envoi

After more than a century of being seen as a marker of schizophrenia, catatonia's distinction as an unique motor syndrome was finalized in 2013 in the official DSM-5 published by the American Psychiatric Association. Our present understanding is an example of applying the medical model of diagnosis to define a systemic syndrome and effective treatments (Taylor et al. 2010; Taylor 2013). Populations of catatonia patients are biologically homogeneous, characterized by measurable motor behaviors that are relieved by known interventions. By contrast, populations labeled as major depression, bipolar disorder and schizophrenia are necessarily heterogeneous, lacking both verification tests and assured outcomes with the recommended treatments.

The decision to lump all depressive mood disorders under a single large umbrella of "major depression" in DSM-III lost the useful distinction between psychotic (melancholic) and psychoneurotic (anxious) depression. Melancholia is analogous to catatonia, in that both are identifiable, both have verification tests and both are treatable. Their common response to induced seizures and the frequent appearance of catatonia in patients with melancholia makes the connection between the two syndromes a challenge. It is timely to characterize melancholia as a distinct mood disorder with reliable identification of symptoms and course, verification by tests such as the dexamethasone suppression test and application of the known successful treatments of induced seizures and tricyclic antidepressant drugs. The lesson of our catatonia experience is that it is time to develop melancholia as a primary systemic disorder and discard its consideration mainly as a specifier in the DSM (Taylor and Fink 2006; Shorter and Fink 2010).

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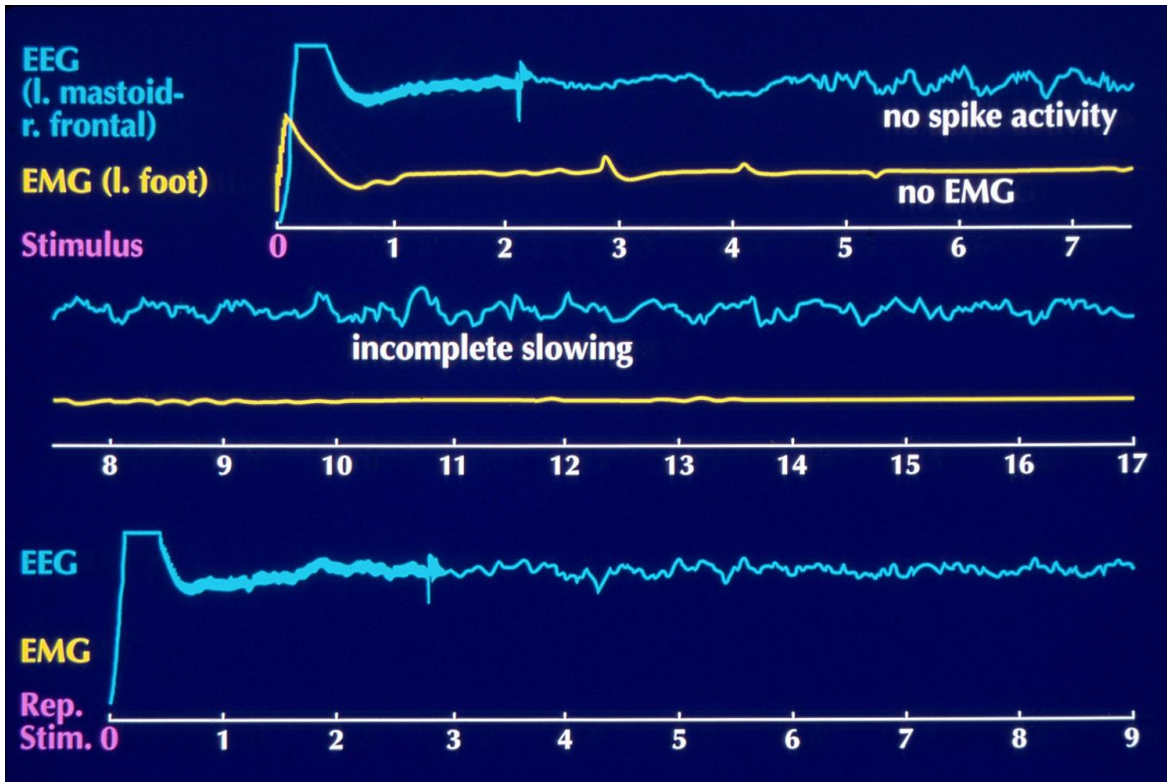
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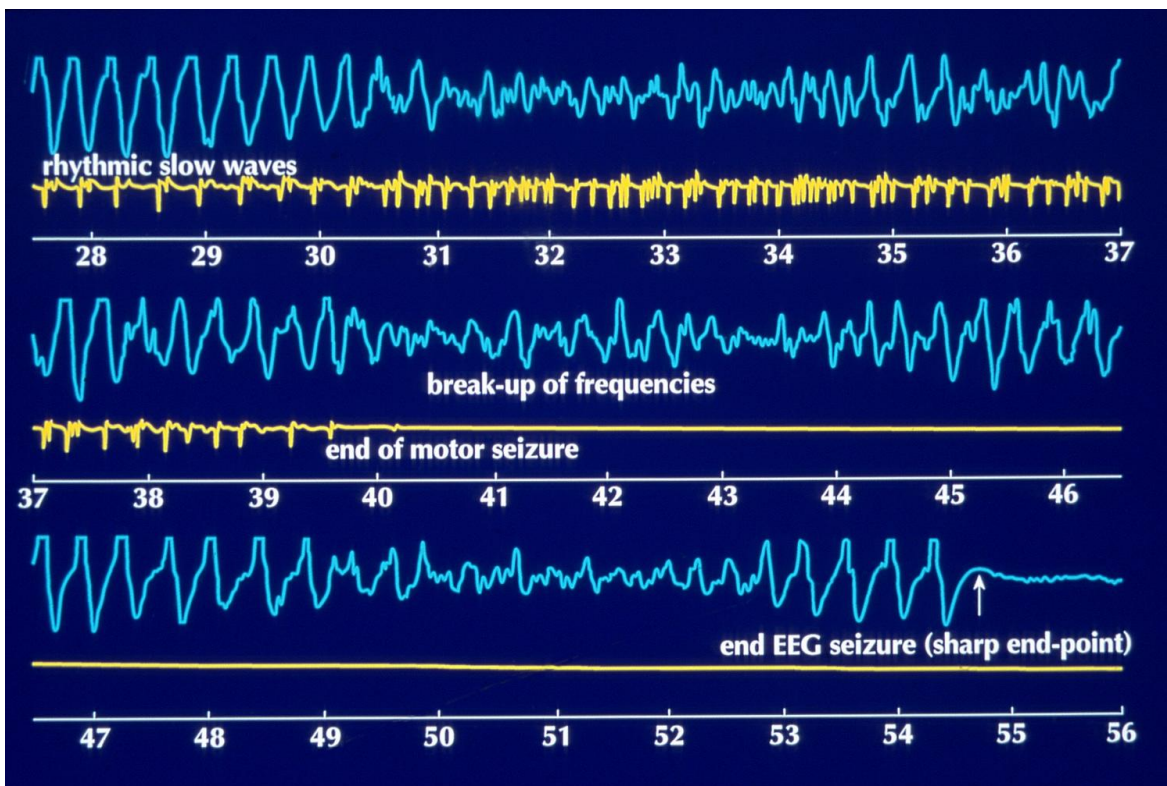
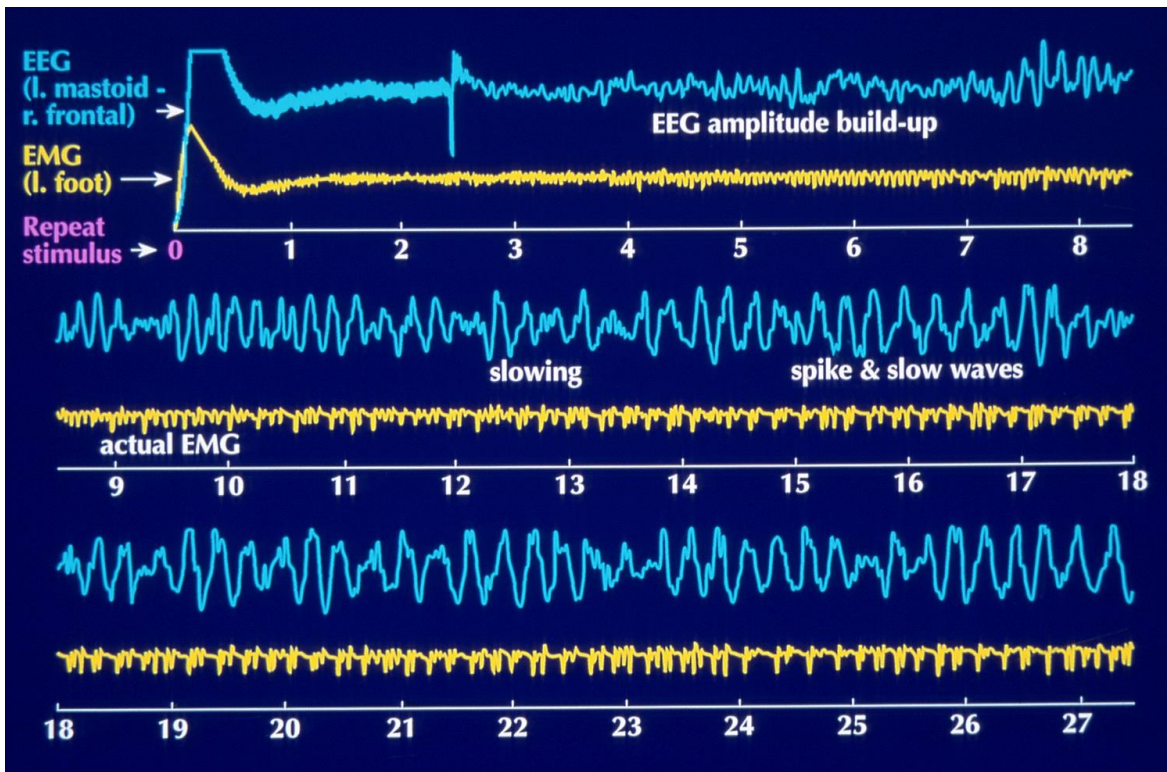
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Figure 1: Image of an Effective Seizure





APPENDIX 1: Examination for Catatonia

- The method described here is used to complete Catatonia Rating Scales.
- Ratings are made based on the observed behaviors during the examination, with the exception of completing the items for ‘withdrawal’ and ‘autonomic abnormality’, which may be based upon either observed behavior and/or chart documentation.
- Rate items only if well defined. If uncertain, rate the item as ‘0’.

| | |
|---|---|
| <p>1. Observe patient while trying to engage in a conversation.</p> | <p>Activity level, abnormal movements, abnormal speech</p> |
| <p>2. Examiner scratches head in exaggerated manner.</p> | <p>Echopraxia</p> |
| <p>3. Examine arm for cogwheeling. Attempt to reposition, instructing patient to "keep your arm loose". Move arm with alternating lighter and heavier force.</p> | <p>Rigidity, Negativism, Waxy Flexibility</p> |
| <p>4. Ask patient to extend arm. Place one finger beneath hand and try to raise slowly after stating, "DO NOT let me raise your arm".</p> | <p>Passive obedience</p> |
| <p>5. Extend hand stating, "DO NOT shake my hand".</p> | <p>Ambitendence</p> |
| <p>6. Reach into your pocket and state, "Stick out your tongue, I want to stick a pin in it."</p> | <p>Automatic Obedience</p> |
| <p>7. Examine for the grasp reflex.</p> | <p>Grasp Reflex</p> |
| <p>8. Examine the patient's chart for oral intake, vital signs, and unusual incidents.</p> | |
| <p>9. Observe the patient indirectly for a brief period each day.</p> | |

APPENDIX 2: Catatonia Rating Scale

CATATONIA RATING SCALE

- Use the presence or absence of items 1-14 for screening purposes.
- Use the 0-3 scale for items 1-23 to rate severity.

1. Excitement.

Extreme hyperactivity, constant motor unrest which is apparently non-purposeful. Not to be attributed to akathisia or goal-directed agitation.

0= Absent

1= Excessive motion, intermittent.

2= Constant motion, hyperkinetic without rest periods.

3= Severe excitement, frenzied motor activity.

2. Immobility/ Stupor.

Extreme hypoactivity, immobility. Minimally responsive to stimuli.

0= Absent

1= Sits abnormally still, may interact briefly.

2= Virtually no interaction with external world.

3= Stuporous, not responsive to painful stimuli.

3. Mutism.

Verbally unresponsive or minimally responsive.

0= Absent

1= Verbally unresponsive; incomprehensible whisper.

2= Speaks less than 20 words/5 minutes.

3= No speech.

4. Staring.

Fixed gaze, little or no visual scanning of environment, decreased blinking.

0= Absent

1= Poor eye contact. Gazes less than 20 seconds before shifting of attention; decreased blinking

2= Gaze held longer than 20 seconds; occasionally shifts attention

3= Fixed gaze, non-reactive.

5. Posturing/ Catalepsy.

Maintains posture(s), including mundane (e.g., sitting or standing for long periods without reacting).

0= Absent

1= Less than one minute.

2= Greater than one minute, less than 15 minutes.

3= Bizarre posture, or mundane maintained more than 15 min.

6. Grimacing.

Maintenance of odd facial expressions.

0= Absent

1= Less than 10 sec.

2= Less than 1 min.

3= Bizarre expression(s) or maintained more than 1 min.

7. Echopraxia/ Echolalia.

Mimicking of examiner's movements/ speech.

0= Absent

1= Occasional.

2= Frequent.

3= Continuous.

8. Stereotypy.

Repetitive, non-goal-directed motor activity (e.g. finger-play; repeatedly touching, patting or rubbing self). (Abnormality is not inherent in the act but in its frequency.)

0= Absent

1= Occasional.

2= Frequent.

3= Continuous.

9. Mannerisms.

Odd, purposeful movements (hopping or walking tiptoe, saluting passers-by, exaggerated caricatures of mundane movements). (Abnormality is inherent in the act itself.)

- 0= Absent
- 1= Occasional.
- 2= Frequent.
- 3= Continuous.

10. Verbigeration.

Repetition of phrases or sentences.

- 0= Absent
- 1= Occasional.
- 2= Frequent, difficult to interrupt.
- 3= Continuous.

11. Rigidity.

Maintenance of a rigid position despite efforts to be moved (Exclude if cog-wheeling or tremor are present.)

- 0= Absent
- 1= Mild resistance.
- 2= Moderate.
- 3= Severe, cannot be repostured.

12. Negativism.

Apparently motiveless resistance to instructions or to attempts to move/examine patient. Contrary behavior, does the opposite of the instruction.

- 0= Absent
- 1= Mild resistance and/or occasionally contrary.
- 2= Moderate resistance and/or frequently contrary.
- 3= Severe resistance and/or continually contrary.

13. Waxy Flexibility.

During reposturing of patient, patient offers initial resistance before allowing himself to be repositioned (similar to that of a bending a warm candle).

- 0= Absent.
- 3= Present.

14. Withdrawal.

Refusal to eat, drink and/or make eye contact.

0= Absent.

1= Minimal oral intake for less than one day.

2= Minimal oral intake for more than one day.

3= No oral intake for one day or more.

15. Impulsivity.

Patient suddenly engages in inappropriate behavior (e.g. runs down hallway, starts screaming, or takes off clothes) without provocation. Afterwards can give no or an incomplete explanation.

0= Absent

1= Occasional

2= Frequent

3= Constant or not redirectable

16. Automatic Obedience.

Exaggerated cooperation with examiner's request, or repeated movements that are requested once.

0= Absent

1= Occasional

2= Frequent

3= Continuous

17. Passive obedience (mitgehen)

Raising arm in response to light pressure of finger, despite instructions to the contrary.

0= Absent

3= Present

18. Negativism (Gegenhalten).

Resistance to passive movement that is proportional to strength of the stimulus; response seems automatic rather than willful.

0= Absent

3= Present

19. Ambitendency.

Patient appears "stuck" in indecisive, hesitant motor movements.

0= Absent

3= Present

20. Grasp Reflex.

Strike open palm of patient with two extended fingers of examiner's hand. Automatic closure of patient's hand.

0= Absent

3= Present

21. Perseveration.

Repeatedly returns to same topic or persists with same movements.

0= Absent

3= Present

22. Combativeness.

Usually in an undirected manner, without explanation.

0= Absent

1= Occasionally strikes out, low potential for injury

2= Strikes out frequently, moderate potential for injury

3= Danger to others

23. Autonomic Abnormality.

Circle: Temperature

Blood Pressure

Pulse rate

Respiratory rate

Inappropriate sweating.

Table 2

0= Absent

1= Abnormality of one parameter [exclude pre-existing hypertension]

2= Abnormality of 2 parameters

3= Abnormality of 3 or greater parameters

Appendix 3: The Catatonic Syndrome

| <i>The Catatonia Syndromes</i> | |
|--|---|
| | <i>Some Eponyms</i> |
| Retarded Catatonia Benign Stupor | Kahlbaum Syndrome (KS) |
| Excited catatonia Delirious mania | Manic excitement Manic delirium Bell's mania |
| Oneiroid state | <i>Onirisme, Oneirophrenia</i> |
| Malignant catatonia (MC) | Lethal catatonia Pernicious catatonia |
| Neuroleptic malignant syndrome | NMS; MC/NMS <i>Syndrom malin</i> Neuroleptic induced catatonia |
| Toxic serotonin syndrome | Serotonin syndrome; TSS |
| Repetitive Syndromes | Tourette's Syndrome Post-Encephalitic Parkinsonism Self-injurious Behavior Anti-NMDAR encephalitis |
| Periodic catatonia | |
| Mixed affective state | Rapid cycling mania |
| Primary Akinetic mutism | Apallic syndrome Stiff man syndrome Locked-in syndrome |

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