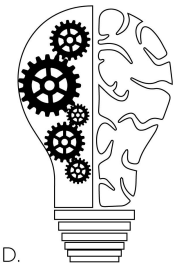
Episode 073: Catatonia: Diagnosis and Treatment

Alex Ramos B.A., Michael Cummings M.D., Jerry Trotter M.D., David Puder, M.D.



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There are no conflicts of interest for this episode.

What is Catatonia?

Catatonia is a severe motor syndrome. **It is a secondary response to an underlying illness that requires quick diagnosis and treatment**.

It has different causes:

- Structural brain disease (epilepsy, toxic, metabolic or infectious diseases)
- Systemic disorders involving the brain
- Psychiatric disorders (schizophrenia, affective psychoses, severe anxious states/emotional shock)
- N-methyl-D-aspartate receptor (NMDAR) encephalitis
    - The most common cause of autoimmune catatonia, and is a disorder of the immune system.
    - It also entails a very high risk of neuroleptic malignant syndrome (a disorder with prominent activation of the innate immune system). (Rogers, 2019)

After discovering that you have someone who is catatonic, it is essential to get a full history, collateral, and **look at reasons** this person developed catatonia.

History of the catatonia diagnosis

In 1847 Karl Kahlbaum gave the first clinically accurate description of catatonia which he described in 4 phases

1. A short stage of immobility with waxy flexibility and posturing

2. Stupor or melancholy

3. Mania with pressured speech and hyperactivity

4. Dementia

In 1893 after being influenced by Kahlbaum, Kraeplin included catatonia into the group of deteriorating psychotic disorders. He also noted differences between catatonia associated with mood disorders, versus the one associated with chronic psychosis. He found that their symptoms, course and prognosis were significantly different.

For the most part, catatonia was strongly associated with schizophrenia until 1990's when new criteria for mood disorders with catatonic features and the catatonic type of schizophrenia were proposed by the DSM-IV.

Evaluation and Diagnosis

A number of rating scales can be used to screen for catatonia including:

- Rogers Catatonia Scale
- Bush-Francis Catatonia Rating Scale (BFCRS)
- Northoff Catatonia Rating Scale
- Braunig Catatonia Rating Scale

Of these, the most widely used is the BFCRS. This rating scale consists of 23 items: the first 14 items are used for screening 0= absent and 3 = present. The first 14 items include symptoms like: immobility/stupor, mutism, staring, posturing/catalepsy, stereotypy, meaningless repetition of words or phrases etc. **Two or more of the 14 screening symptoms must be present for 24 hours or longer in order to diagnose catatonia.** The total score of the 23 items indicates severity of catatonia.

Clinical Features and Assessment

Clinical presentation varies depending on the phase of illness and its etiology. However, catatonia is present in about **10% of acutely ill psychiatry patients**, only a small minority have schizophrenia (30%) while a majority have bipolar disorder (43%).

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While no test can confirm the diagnosis, accurate personal and familial history taking and some procedures (EEG, brain imaging etc.) can point to the etiology. **The most important thing to do is a full workup in order to identify the cause, as catatonia is always secondary to another illness and that primary source should be treated.**

For example, a patient could present with a history depression that has been worsening over the last 3-4 months, they then stop talking as much, stop eating as much, begin to have speech delay. By the time they are brought into the ER by family the catatonia has already been evolving for some time.

Mutism and stupor are the principle signs, but if someone has one sign, they often have 4 or more. According to Fink and Taylor, the symptoms need to be present for an hour or longer, and reproducible on two or more occasions. As the disease progresses, patients become malnourished, dehydrated, lose weight, develop muscle wasting, contractures and develop bed sores. Sometimes because they stay so still, they can die from DVT or PEs.

- This is most often seen in patients with major depressive disorder, melancholic depression, severe schizophrenia, or severe bipolar depression.
- This state is considered a medical emergency—without treatment patients can eventually die from the catatonia.

Dr. Puder remembers a patient with bipolar type 2 who became catatonic; he stopped drinking and eating. At an appointment with his primary care provider, this patient fell over due to hypotension. It was then found that he had not been talking at home as much, and had long speech delays. His hypotension stemmed from him losing the urge to drink fluids. This points to the importance of spotting the more subtle signs of catatonia before it evolves to its severe form.

For a diagnosis, there needs to be 2 or more of the classic signs (Fink & Taylor, 2003)

1. Mutism - decreased speech, silent and unresponsive in speech, not always accompanied by stupor or immobility
2. Stupor - persistently unresponsive, unaware of surroundings, can be difficult to distinguish from mutism, they may become energized for a few hours in something, looking like a manic excitement
3. Posturing
4. Waxy flexibility
5. Stereotypy

6. Automatic obedience
7. Ambitendency
8. Echophenomena
9. Mannerisms

How does catatonia progress?

Classic signs of catatonia include mutism and stupor. In its extreme form, the patient is in a frozen state and mute—this is difficult to miss. However, in its subtle form, it is often missed and recognizing the signs is important for early diagnosis.

Early signs of catatonia:

- Increased speech latency (patient responds, but there is an increased length of time for response to begin).
- Gradually evolves into partial mutism (patient responds only sometimes).
- Echolalia may start subtly; it may begin with the patient repeating the last words of the examiner then they may give an answer, the more severe if becomes it will evolve to them only repeating the examiners words.
- Motor activity also follows this course; the patients movement will initially appear slowed and will start evolving into holding unusual postures for long periods of time. Eventually they will become “frozen”, where they can be put in a position by someone else and they will hold that position (waxy flexibility).
- Echopraxia may also start subtly; it can begin with the patient *occasionally* mimicking the examiners movements, but eventually evolve to them *completely* mirroring the examiners movements; this gives way to a loss of movement all together.

What are some of the differential diagnostic things to consider?

What is Neuroleptic Malignant Syndrome (NMS) and how is it different than catatonia? (Northoff, 2002)

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Catatonia may be confused with the early stages of NMS. While they have clinical similarities, there are important differences between them:

- **Catatonia:** characterized by **akinesia** (loss or impairment of the power of voluntary movement). These patients show posturing, keeping their limbs and/or head in a position against gravity. Catatonic patients do not realize their movements are altered. These patients show strong, intense, uncontrollable emotions. They tend to have **overwhelming anxiety** that can lead to their catatonia. Catatonia is also characterized by bizarre behavioral abnormalities (perseveration, stereotypes, automatic obedience, negativism, etc.).
- **NMS:** a life-threatening neurological emergency associated with the use of antipsychotic medications. It is characterized by a clinical syndrome of mental status change, rigidity, fever and dysautonomia. **Patients with NMS experience cog-wheel rigidity and also show akinesia. In contrast to catatonia, these patients are aware that their movements are altered, and do not show posturing or the behavioral abnormalities seen in catatonic patients.**
    - Unlike catatonic patients, the anxiety seen in patients with NMS is not as intense and uncontrollable and the anxiety is more likely related to their awareness of their motor immobility.

What is Neuroleptic Malignant Syndrome (NMS)?

- A form of “malignant catatonia”, it develops quickly and can be fatal within a few days if left untreated.
- Etiology is thought to be due to a severe decrease in central dopaminergic activity usually due to exposure to dopamine antagonist medication (i.e antipsychotics).
- Clinical Picture
    - Autonomic Dysfunctions: Tachycardia, hyperthermia (temperature of 104-108 F is not uncommon in these patients), high blood pressure
    - Rigidity, mutism, stupor and increased CPK leading to risk of acute renal failure
    - Things that increase the risk of developing NMS
        - Treatment with neuroleptic drugs
        - History of catatonia
        - History of schizophrenia
        - Mood disorders (especially postpartum depression)
        - Alcoholism or substance abuse disorder
        - Agitation, dehydration, and exhaustion

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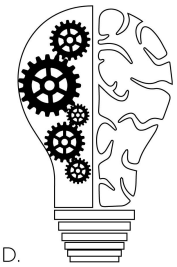

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    - Basal ganglia disorders (Parkinson's, Wilson's disease, Huntington's disease, and tardive dystonia)
    - Low serum iron in the course of catatonia increases risk when neuroleptics are prescribed
    - Male gender
- **Treatment**
    - Withdraw antipsychotics immediately!
    - Start dopamine agonists: bromocriptine, dantrolene, and life supportive therapy before evaluating for the use of lorazepam or ECT.

What is Serotonin Syndrome and how is it different than catatonia?

- **Serotonin Syndrome (SS)**:
    - Associated with a high dose of serotonergic medications such as Tricyclic (TCA's), SSRI's. Most cases are due to mixing monoaminoxidase inhibitors with other serotonergic medications, leading to potentially fatal consequences.
- **Signs of SS**:
    - Confusion, anxiety, irritability, euphoria and dysphoria, GI symptoms (nausea, vomiting, diarrhea, incontinence), restlessness and agitation, neurologic findings (ataxia/incoordination), tremor, myoclonus, hyperreflexia (ankle clonus and muscle rigidity), and autonomic abnormalities (hyper or hypotension, tachycardia, excess sweating etc.)
- **Treatment:**
    - Include: Withdraw medication that caused SS immediately! Life supportive interventions for respiratory and cardiovascular systems, and proper hydration in order to prevent renal failure.
    - Benzodiazepines can be used to manage agitation.
    - 5-HT receptor antagonists such as cyproheptadine and mirtazapine have been proposed but are not considered a first line choice.

What is delirium and how is it different than catatonia?

Delirium is a waxing and waning of attention. It is a brain disturbance characterized by the DSM as requiring a disturbance of consciousness or attention, a change in cognition that develops acutely and tends to fluctuate. These patients have lucid periods followed by periods where they become very disoriented. Delirium is a common medical issue with a high rate in the ICU in

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hospitals. They have trouble with things like the serial 3's (counting down from 21 by 3's), drawing a clock, and spelling world backwards.

- **There are 4 subtypes of delirium: Hyperactive, hypoactive, mixed, and without motor symptoms.**
    - **Hyperactive**: characterized by motor agitation, restlessness, and aggressiveness. These are the patients that are pulling out their IV lines, yelling, throwing things, and seeing visual hallucinations.
    - **Hypoactive**: characterized by motor retardation, apathy, slowing of speech, patients can appear sedated.
    - **Mixed**: combination of hypo and hyperactive
    - **Without motor symptoms**: patients only experience the cognitive symptoms of delirium

Delirium represents a critical breakdown in brain function due to severe metabolic disturbance of some kind. Exposure to anesthesia is a common cause of delirium in hospitals, it is estimated that postoperatively 40% of people experience delirium, however most cases are transient and short lived.

- **Catatonia and Delirium** (Grover, 2014)
    - Catatonia and Delirium share many clinical features. Due to this, catatonia may be misdiagnosed as delirium, managed as delirium, or can co-occur with delirium. They both have motor variants that can overlap with each other. **It is important to differentiate between catatonia or delirium because a benzodiazepine will help with catatonia but will make delirium worse**. For the most part benzodiazepine are contraindicated in delirium, except in the case of alcohol withdrawal delirium.
    - Patients with hypoactive delirium have features (apathy, slowing of speech, etc.) that are similar to negativism and mutism seen in catatonia. For those with hyperactive delirium, the overlap is in symptoms like excitement/agitation.
    - Grover, states that this overlap suggests that there is merit in evaluating for catatonic symptoms in patients with delirium to expand its clinical picture. The study conducted found that patients with delirium frequently have catatonic features like excitement, immobility/stupor, mutism, negativism, combativeness, withdrawal, staring and impulsivity. The frequency of these symptoms varied from 8.3% to 72.7%.

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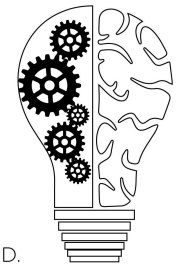

- If you suspect a patient may have delirium, it is important to have them do things like listing the serial 3's, drawing a clock, and spelling world backwards to differentiate between delirium and catatonia. A catatonic patient may take a long time to do these things, but will give you accurate answers unlike a patient with delirium. Another clinical pearl is the difference seen when administering ativan (benzodiazepine) to a catatonic versus a delirious patient. If ativan is given to a catatonic patient they will "wake up" in a sense, their speech will change, and their movements will improve. However, giving ativan to a delirious patient will put them to sleep and worsen their delirium.
- Benzodiazepines are known to treat catatonia, but are also useful in its diagnosis.

Lorazepam Challenge Test

- After the patient is examined for signs of catatonia, 1 or 2 mg of lorazepam are administered via IV. **Patient is reexamined in 5 minutes**, if there is no change another dose is administered and the patient is reexamined. A reduction in catatonic signs indicated a positive response and confirms the diagnosis. Generally it is a pretty dramatic response—they can go from being in a frozen stuporous state before administration of lorazepam to completely alert and interactive after administration.
- Things to look for after administration:
  - Speech latency and if there is improvement
  - If echolalia and echopraxia have improved
  - Is their ease of movement improved
- If giving an IV dose is not possible in a particular setting, giving lorazepam IM is the next best option, if the patient improves it confirms the catatonia diagnosis. Improvement can be seen **15 minutes after injection**. If they have a positive response, treatment with a benzodiazepine can be started until the primary illness is under control, then the benzodiazepine can be gradually withdrawn.
  - Lorazepam should not be given orally in order to diagnose. Uptake is too slow and may not give a confirmatory response.

Pathophysiology

- Catatonia is thought to be due to a basal ganglia disruption at the thalamocortical traits, including:

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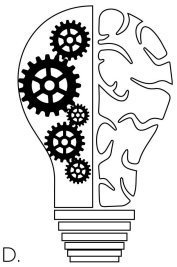

    - Motor circuit → rigidity
    - anterior cingulate/medial orbitofrontal circuit → akinetic mutism
    - lateral hypothalamic connections → hyperthermia and dysautonomia
    - lateral orbitofrontal circuit → imitative and repetitive behaviors
- These disruptions basically lead to decreased **dopamine** transmission in these circuits.
- Other sites of possible pathophysiology include decreased activity at GABA-A receptors and increased activity at NMDA receptors. This suggests the use of benzodiazepines (GABA agonists) and Amantadine (NMDA antagonists) as possible therapy.
- "Neuroimaging studies of patients with a history of catatonia indicated poor functional activation of SMA, primary and secondary motor cortices, inferior parietal cortex, and basal ganglia during self-initiated movements, lower cerebral blood flow in right prefrontal and parietal cortex, and reduced γ-aminobutyric-acid (GABA)-A-receptor density in the left sensorimotor cortex." (Walther, 2019)
- "MRI studies reported that orbitofrontal cortex dysfunction, which is partly reversible by lorazepam administration."
- "Thalamocortical hyperconnectivity (to M1) was linked to ratings on the BFCRS (ie, increased connectivity in patients with more severe catatonia symptoms)."
- 'Hyperactivity within the SMA and preSMA (which are critically involved in motor control, movement selection, initiation, timing, and inhibition) has been associated. The SMA is thought to initiate the inhibitory processes in downstream brain areas such as the basal ganglia, and could result from a feedforward stimulation of the subthalamic nucleus, or other areas exerting inhibitory control.'
- Genetics (Walther, 2019)
    - "Lifetime morbidity risk of catatonia for first-degree relatives of patients with periodic catatonia is 27%, with an autosomal dominant linkage to chromosome 15q15, and heterogeneous linkage to chromosome 22q13."
    - "A study of multiplex families reported substantial heritability estimates for catatonia symptoms, symptom endorsement frequency, and syndrome severity. The most frequent signs, such as psychomotor retardation and excitement, showed a moderate degree of familiality, whereas classical signs, such as mutism or rigidity, showed a high degree of familiality."

Prognosis

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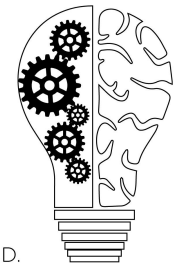

Catatonia is a potentially life-threatening condition, often requiring hospitalization on either a medical or psychiatric ward. 'If left untreated, it can lead to deep vein thrombosis or pulmonary embolism; decubitus ulcers, muscle contracture, and rhabdomyolysis (which can lead to renal failure).' Prognosis is usually good if diagnosed early and with aggressive treatment.

- Data suggests that mood disorders associated catatonia have a better prognosis than the catatonia that is associated with psychotic disorders.
    - In a randomized double blind, placebo-controlled trial in 18 patients with chronic schizophrenia with catatonic features, 6 mg of lorazepam a day given for 12 weeks had no effect on their catatonic symptoms.
    - Another study showed that 73% of 24 patients with catatonia had remission within 6 days after starting benzodiazepines, those that only had partial response (6/24) all had schizophrenia.
    - Authors suggest that "chronic catatonia" in the context of schizophrenia is very different and much less responsive to ECT and Benzodiazepine therapy, so it carries a less favorable prognosis than the acute forms of catatonia.
- Immune System & Catatonia (Rogers, 2019)
    - Activation of the innate immune system is associated with mutism, withdrawal, and psychomotor retardation, which constitutes the neurovegetative features of catatonia.
    - Evidence is sparse and conflicting for acute-phase activation in catatonia, and whether this feature is secondary to immobility is unclear.
    - Infections agents (viral, bacterial, and parasitic) have been associated with catatonia, but are primarily linked to CNS infections.
    - Autoimmunity appears to cause catatonia less by systemic inflammation than by the downstream effects of specific actions on extracellular antigens. The specific association with NMDA Receptor encephalitis supports a hypothesis of glutamatergic hypofunction in catatonia.

Treatment

- Catatonia is associated with significant morbidity and mortality rates, therefore quick diagnosis and treatment is necessary.
- Supportive care

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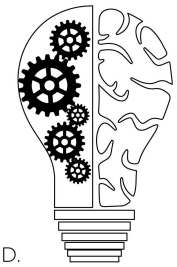

    - Due to the many complications that come with catatonia (i.e. aspiration pneumonia, dehydration, nutritional deficiencies, electrolyte disturbances, and venous thromboembolisms) patients require a high level of nursing care, IV fluids and or nasogastric tube feeds.
    - 'General preventive recommendations for patients with catatonia include pharmacological prophylaxis of venous thromboembolism, skin assessments / frequent repositioning to prevent pressure ulcers, daily stretching to prevent muscle contractions, and tube feeding and intravenous fluids when necessary to avoid dehydration and malnutrition.'
- Benzodiazepines
    - Benzodiazepines (lorazepam especially: 79% remission rate), are proven to be effective in the acute management of catatonia and are **considered first line management** regardless of the etiology with remission rates as high as 70-80%
    - Considered the most effective and safe drug for the management of most acute phases of catatonia.
        - Most authors suggest starting at 1-2 mg of lorazepam every 4-12 hours while adjusting the dose to relieve the catatonia. Care should be taken to not give too high a dose, which can lead to sedation and respiratory compromise. A mistake that is commonly seen is clinicians going up on benzodiazepine dose too slowly. Some clinicians will start catatonic patients on 1 mg 3x per day and see if there is a response. Dr. Cummings states you have to be more aggressive with benzodiazepine treatment. He states the optimal way is to give them 2 mg every 4 hours, re-examine them and if they show response (no longer frozen, no longer stuporous, they have fluid movements) then at the end of a 24 hour period you should have a good idea of how much of the benzodiazepine it took to get them to a good response state and you can continue them on that treatment.
        - If you see the patient responding to lorazepam, the dose should be increased until they have a full response. It is important to note that patients can begin to have **lateral nystagmus** once they reach their threshold of how much medication they can tolerate.
        - Those with longer half lives are preferred over those with short half lives. Switching from IV administration of lorazepam to oral or nasogastric administration maintains the therapeutic effect.

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    - A minority of patients are more prone to catatonia; these patients will end up requiring a high potency benzodiazepine on an ongoing basis. For the majority, once their primary illness is under control, their catatonic symptoms will disappear and they will no longer require a benzodiazepine.
    - While very effective for most cases of catatonia, **research has shown repeatedly that chronic catatonia associated with schizophrenia is less responsive to benzodiazepines**. Those that have the best response to benzodiazepines are those with a mood disorder associated catatonia.
    - Once a patient is at their therapeutic dose and their primary illness is being treated, patients can begin being tapered down their benzodiazepine. **Tapering should be gradual (.5 mg per month)**. If tapering is not gradual, the patient will just re-enter into catatonia. As you taper watch for rebound symptoms.
- Electroconvulsive Therapy
    - Electroconvulsive Therapy (ECT) is an even more effective non-pharmacologic intervention especially if it has been refractory to pharmacologic treatment.
    - There is still some stigma surrounding the use of ECT but it can be very transformative for patients that have been unresponsive to benzodiazepines. ECT is a minor medical procedure done in an outpatient setting. It involves putting the patient to sleep and paralyzed, they then have a stimulus applied that leads them to have a seizure that lasts less than a minute. They are usually up and walking around in about a half hour.
    - ECT becomes the first treatment of choice if the underlying condition (e.g. psychotic depression) warrants ECT, or in life threatening situations (malignant catatonia or NMS).
    - If lorazepam is not effective, ECT should be considered
        - 2-3 ECT are usually effective to remove the catatonic state
        - 4-6 recommended to prevent recurrence
        - 10-20 may be needed in resistant cases
    - Study looking at the efficacy of ECT: 50 patients presenting with catatonic symptoms (23 of whom were diagnosed with schizophrenia) received either ECT or a benzodiazepine as first line treatment. Only 1 in 41 patients responded fully to benzodiazepines, and 19 responded partially. All 17 patients who received ECT achieved remission.

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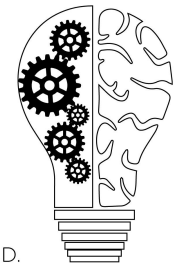

    - Bilateral standard pulse ECT with a stimulus dose substantially above the seizure threshold is recommended most and considered the most efficacious compared to unilateral ECT.
- Antipsychotics
    - First Generation
        - The use of first generation antipsychotics for catatonia is ambiguous, but for the most part it is encouraged to discontinue them due to the risk of developing Neuroleptic Malignant Syndrome.
        - A prospective follow up of 82 patients found that in patients who received antipsychotics while catatonic, 3.6% developed NMS, a much higher incidence when compared to all antipsychotics treated patients .07-1.8%.
    - Second Generation
        - Caution should be taken even when prescribing second generation antipsychotics (which are generally known for reducing the risk of extrapyramidal syndromes) since cases of NMS have been linked to these as well.
- Glutamate Antagonists
    - **Amantadine or memantine** are NMDA antagonist properties and have been found to be effective in catatonia.
    - One case report has described amantadine being used in an adolescent girl whose catatonia was resistant to ECT and improved after the addition of amantadine. However, amantadine needs to be studied further in order to determine its efficacy.
- Mood Stabilizers
    - Some catatonic cases have also been shown to benefit from lithium preventative therapy (although it could precipitate catatonia at toxic levels). Carbamazepine and valproic acid have been shown to be effective in some catatonic cases as well.

Summary:

There are many different things that lead to catatonia, so finding out the underlying cause is very important. Treatment may differ if patients' etiology is a mood disorder versus an anxiety disorder, versus psychosis like schizophrenia.

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For the most part, it has a great prognosis if identified and treated early, but prognosis does vary depending on etiology. Chronic catatonia associated with schizophrenia is known to be more resistant to treatment and have a worse prognosis than acute catatonia.

There are different treatment options available, however, benzodiazepines are considered first line treatment and are very effective with a 79% remission rate. If benzodiazepines are not effective, ECT is very effective in treatment resistant cases. ECT becomes the first treatment of choice if the underlying condition (e.g. psychotic depression) warrants ECT. It is important to maintain a high index of suspicion of catatonia, paying attention to patients movements, seeing if they have speech latency, these are the early signs that could be seen and identifying these could be life saving for patients with catatonia.

Further Reading:

Bush-Francis Catatonia Rating Scale:http://turkpsikiyatri.org/arsiv/bush-francis_catatonia_rating_scale.pdf

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