

# SHOCKING RESULTS: A Case of Malignant Catatonia

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## INTRODUCTION

Catatonia is a disorder that affects both behavior and motor functions. It is divided into multiple categories and can be hard to distinguish from other disorders (Figure 1). The incidence of catatonia in psychiatric inpatient setting is 5-20%, with the most common cause being bipolar depression.

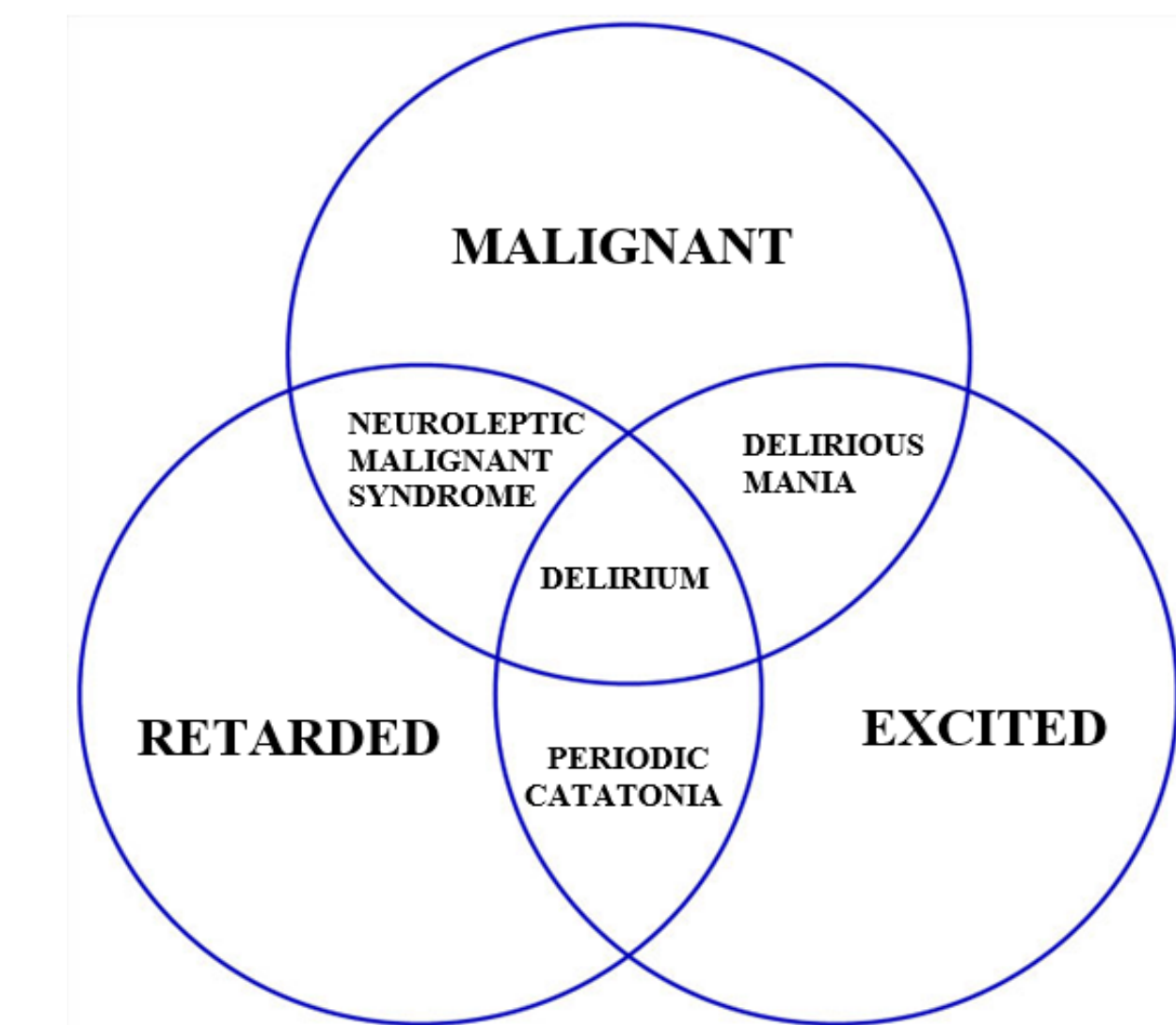


Figure 1. Three main subtypes of catatonia (malignant, retarded, excited) with differential diagnosis (NMS, delirious mania, periodic catatonia).

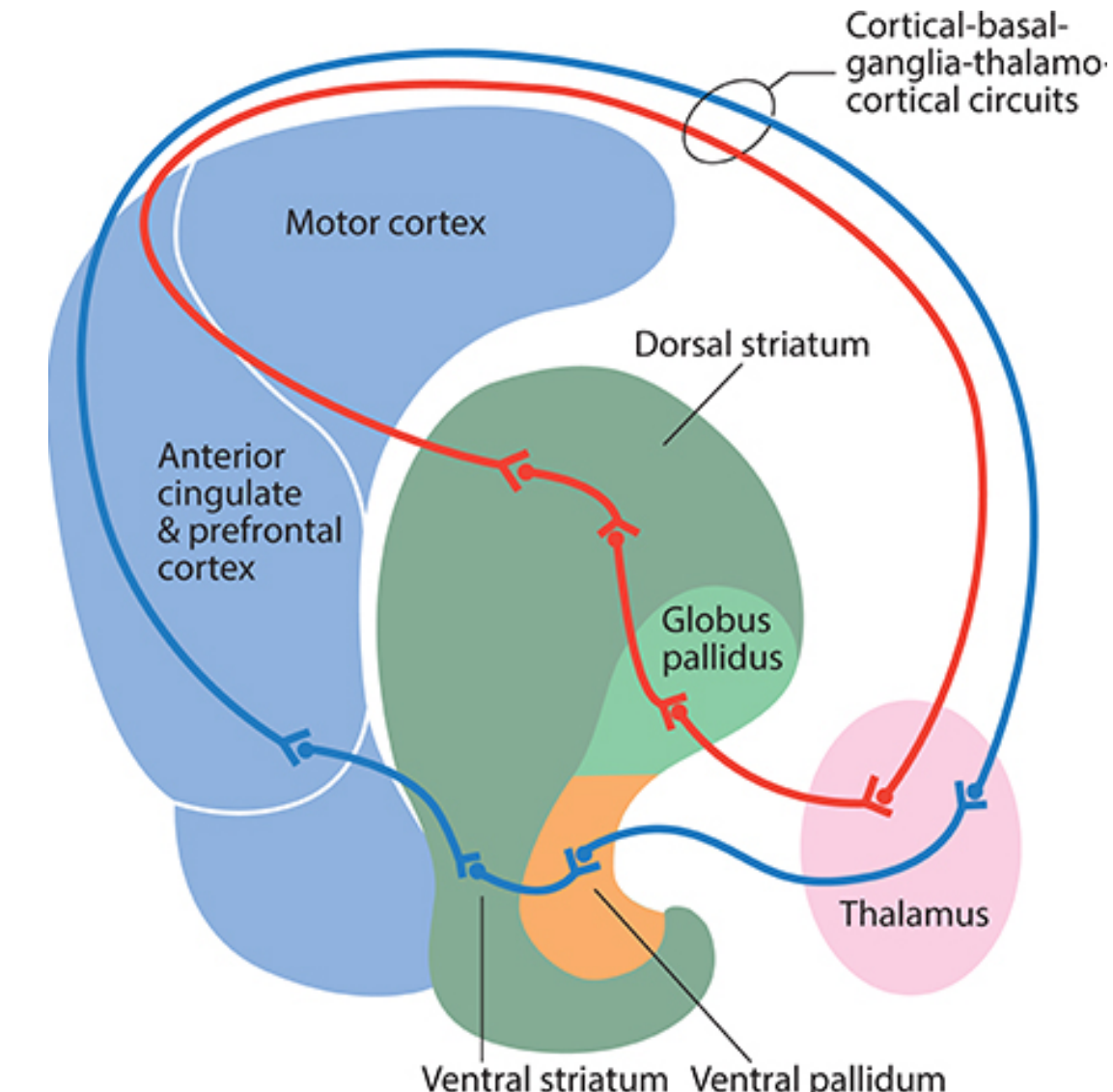


Figure 2. Cortical-basal ganglia-thalamo-cortical circuit path. <https://www.frontiersin.org/articles/10.3389/fnbeh.2016.00059/full>

Catatonia may be associated with changes in the cortical-basal ganglia-thalamo-cortical circuit (Figure 2) associated with:

- Low levels of gamma-aminobutyric acid (GABA)
- Low dopamine D2 receptor binding
- High levels of N-methyl-D-aspartate receptors

Catatonia includes three or more of the following symptoms:

Symptom	Description
Stupor	No psychomotor activity; not actively relating to environment
Catalepsy	Passive induction of a posture held against gravity
Waxy Flexibility	Slight, even resistance to positioning by examiner; often will remain in the new position
Mutism	No, or very little, verbal response (exclude if known aphasia)
Negativism	Opposition or no response to instructions or external stimuli
Posturing	Spontaneous and active maintenance of a posture against gravity
Mannerism	Odd, circumstantial caricature of normal actions
Stereotypy	Repetitive, abnormally frequent, non-goal-directed movements
Agitation	Does not appear to be influenced by external stimuli
Grimacing	Contortion of facial features
Echolalia	Mimicking another's speech
Echopraxia	Mimicking another's movements

Malignant catatonia can include:

- Autonomic instability (fever, hypertension, tachycardia)
- Severe muscle rigidity
- Nonspecific lab findings of leukocytosis and elevated creatinine kinase

## CASE PRESENTATION AND TREATMENT

An 18 year old African American male with no past psychiatric history, a family history of schizophrenia, good social support, and no pertinent past medical or surgical history presented to the Laureate Inpatient Psychiatric Hospital with new onset of mania and psychotic symptoms including bizarre, disorganized behavior and emotional outbursts.

During admission, he was started on antipsychotic medication for control of his symptoms. Eventually, he started to exhibit slow responses and rigidity in left arm along with waxy flexibility in bilateral upper extremities. With the onset of these symptoms, catatonia was added to the differential. There were no autonomic changes. A lorazepam challenge was positive. His antipsychotic medication was discontinued and he was put on scheduled Lorazepam at 1mg every six hours for five days with resolution of catatonia symptoms.

Pt had ongoing symptom of racing thoughts, increase energy, poor sleep, psychosis, irritability, and response to internal stimuli. Therefore was re-trialed on antipsychotic medication. Afterwards, the pt had continued mood and psychotic symptoms and a return of waxy flexibility, sleeping for nearly 20 hours each day, restricted affect with minimal speech, and a refusal to eat or drink.

Due to concern about medical instability, the patient was transferred to Saint Francis Hospital to provide aid in treatment of dehydration, malnutrition, and access for further medical interventions if necessary. With the start of autonomic instability (blood pressure of 184/106) and elevated creatine kinase at 5,506 (normal range less than 225). Pt was diagnosed with malignant catatonia.

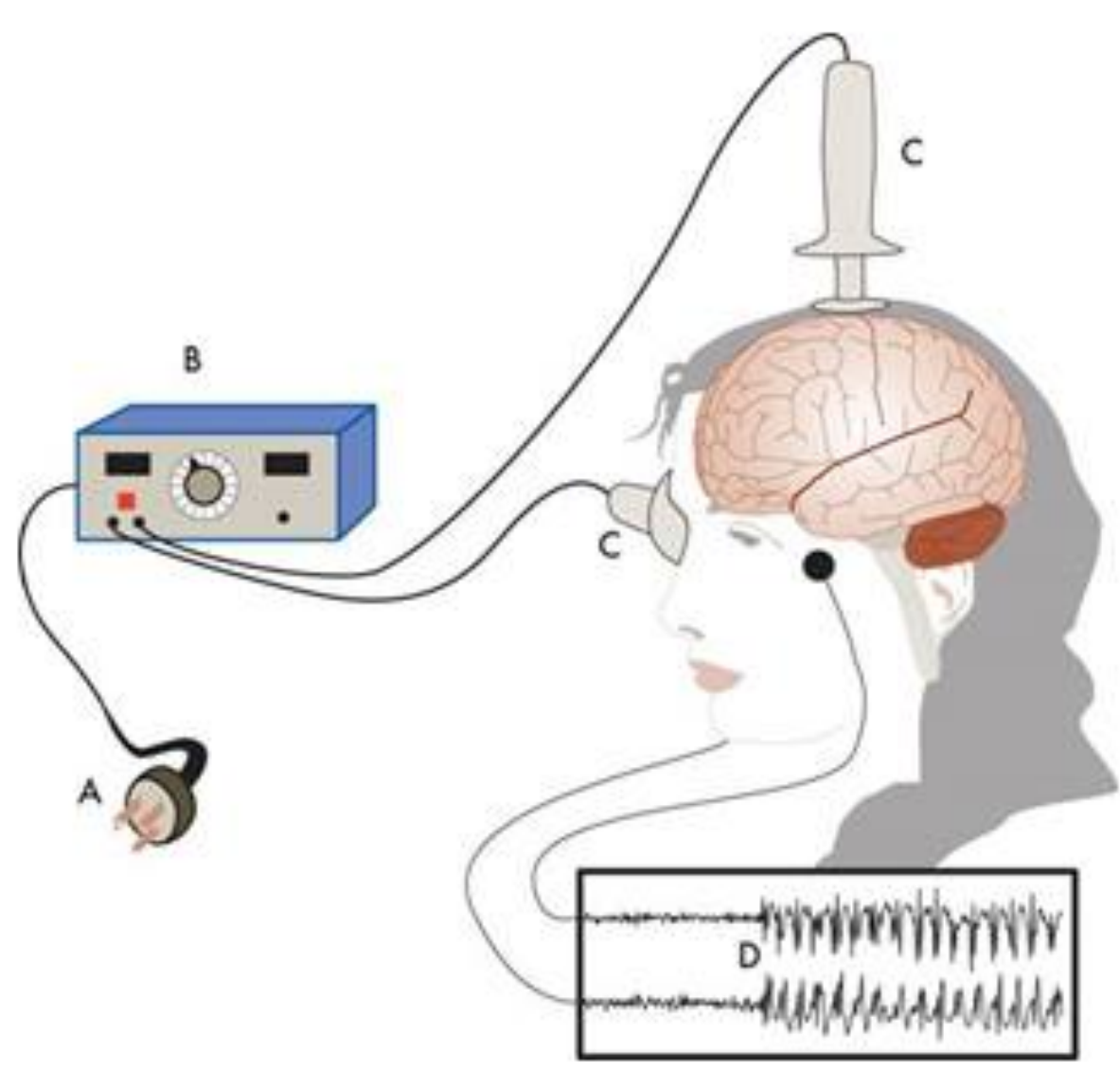
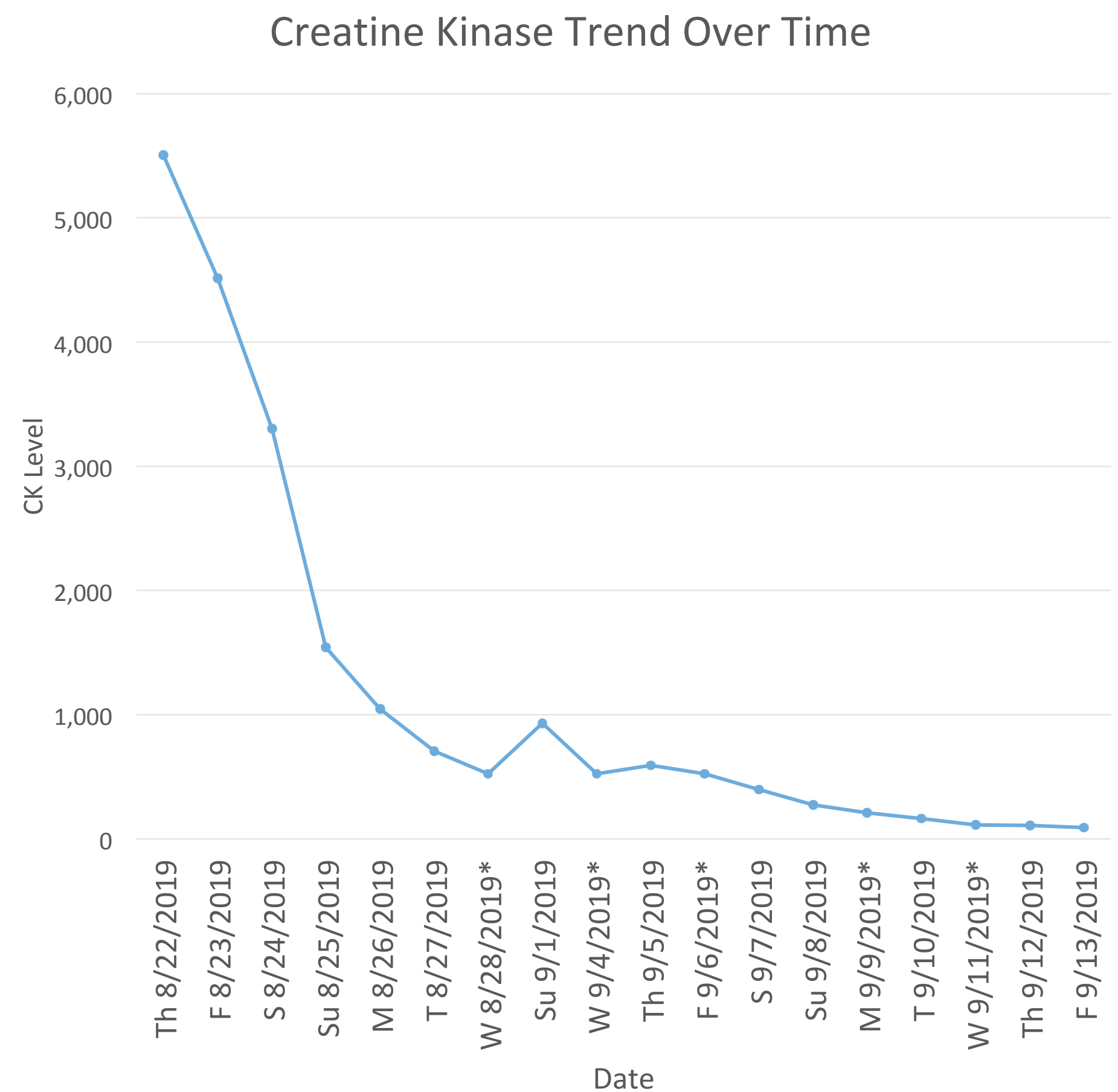


Figure 4. Unilateal placement of electroconvulsive therapy probes. <https://medicine.musc.edu/departments/psychiatry/divisions-and-programs/divisions/bsi/ect>

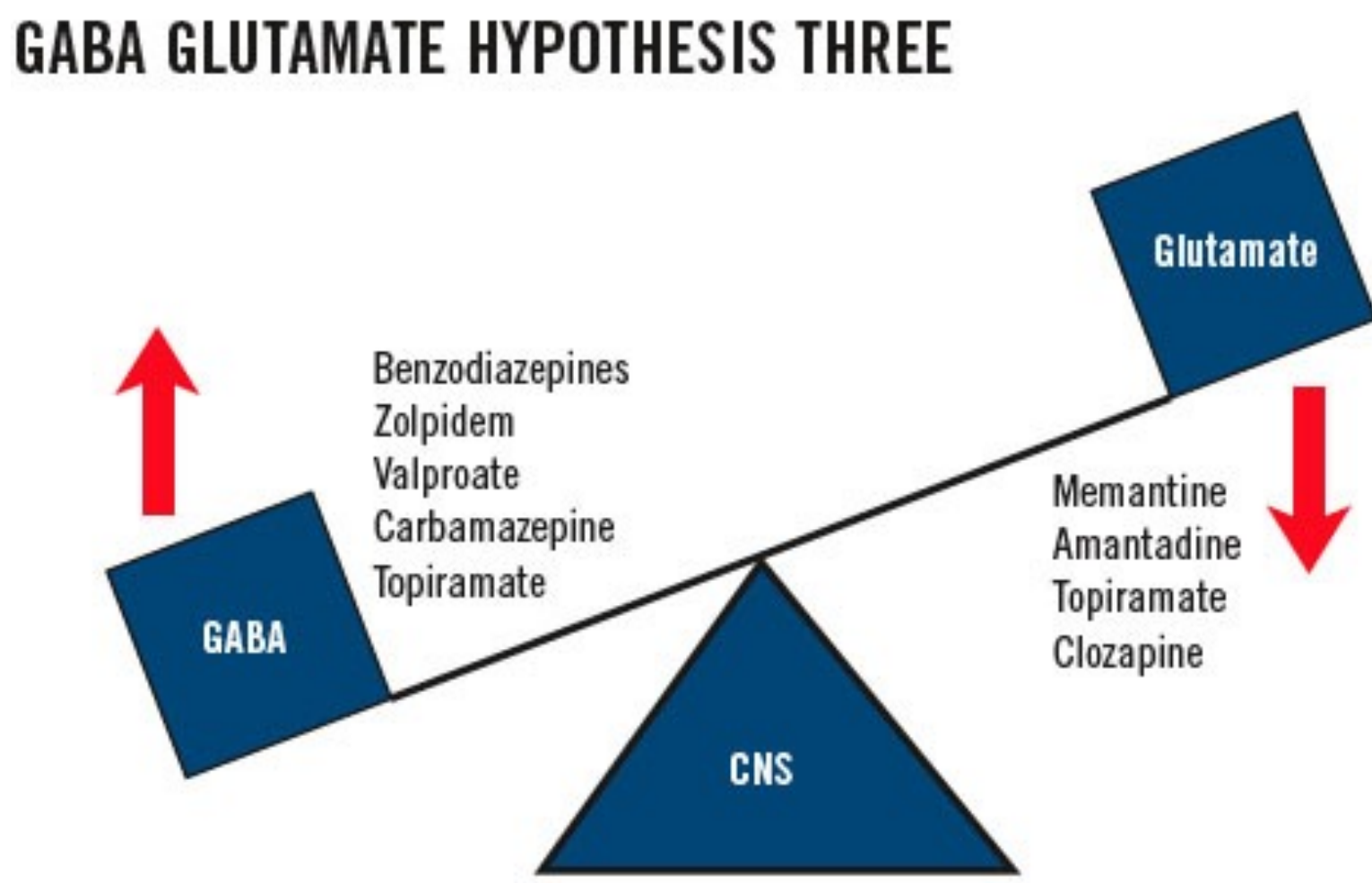


A consult for electroconvulsive therapy (ECT) was placed and initiated treatment of malignant catatonia (figure 4). He responded well to six sessions that occurred Mon., Wed., Fri., for two weeks with total resolution of symptoms and normalization of autonomic instability and creatine kinase levels, as seen in figure 3.

On termination of ECT, he was on valproic acid 1000mg two times a day, olanzapine 10mg at night, temazepam 15mg at night, and lorazepam 1 mg two times a day to help maintain control of his symptoms. After discharge, he was admitted to our community assertiveness program and he continued to do well on his medications. Over several months, his outpatient psychiatrist slowly tapered and discontinued the lorazepam and he continues to have good control of his mood and psychotic symptoms with no return of catatonia symptoms.

## DISCUSSION

First line treatment is scheduled lorazepam doses, at 6-22 mg per day. Other medications can be used to restore GABA-glutamate imbalance (Figure 5).



According to this hypothesis, pharmacologic treatment tend to restore GABA-glutamate imbalance, decreasing glutamate transmission or increasing GABA transmission. Decrease of dopaminergic transmission may act indirectly, increasing GABA-glutamate imbalance.

Figure 5. GABA-Glutamate Imbalance <http://primarypsychiatry.com/the-pharmacotherapy-of-catatonia/>

In severe cases, ECT should be initiated within the first five days to lower mortality rates. Mortality rates of malignant catatonia is 20%, and response rate is higher with ECT (89%) than lorazepam alone (40%). Recommendation is daily treatments of ECT for at least six treatments to reduce risk of sudden relapse. ECT is terminated after the acute catatonic episode, but maintenance ECT sessions have been successful in treating periodic catatonia. For benzodiazepine treatment, recommendation is to maintain the dose for 6 months then taper slowly to prevent relapse.

Psychiatrist and Internist should work closely to prevent complications:

- Monitor autonomic instability
- Daily CK measurements
- Hydration, nutrition status: For arrhythmia and evaluation for feeding tube
- Ins/Outs: For urinary retention
- Frequent repositioning: To prevent ulcers, deep vein thrombosis, pulmonary embolism, contractures and aspiration

## CONCLUSION

Catatonia should be on a differential diagnosis for patients presenting with mental status changes to ensure timely treatment to decrease morbidity and mortality. Dopamine blocking drugs are contraindicated in malignant catatonia as they can precipitate and worsen catatonia and put a patient at increase risk for developing neuroleptic malignant syndrome.

There is a need for more ECT trained physicians to help decrease mortality of catatonia and to increase access to a invaluable treatment. With the underdiagnoses of catatonia, further research is warranted to aid in diagnosis and treatment of this disorder.

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