

Psychiatric Emergencies: **Catatonia**, **Neuroleptic Malignant** **Syndrome**, and **Serotonin Syndrome**

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Medical Psychiatry: A Comprehensive Update
9/17/2026

Disclosures: None

Objectives



UNDERSTAND
EXAMINATION FOR
CATATONIA



DESCRIBE TYPICAL
DIFFERENTIAL
DIAGNOSIS OF
CATATONIA, NMS,
AND SS

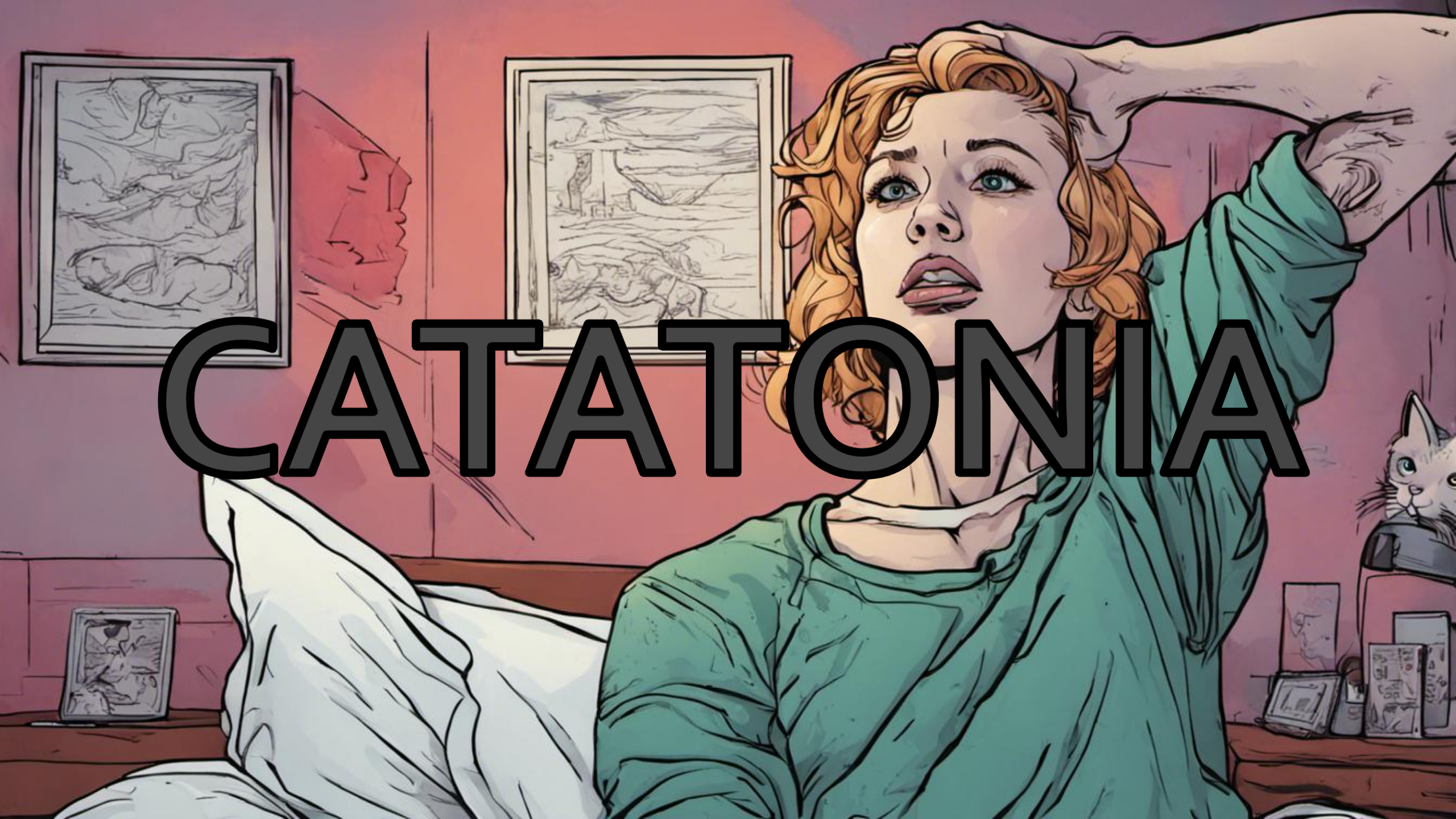


APPRECIATE KEY
DISTINCTIONS
BETWEEN NMS AND
SS



DESCRIBE
TREATMENT
APPROACHES,
INCLUDING WHEN
FIRST-LINE
TREATMENTS FAIL

CATATONIA



History (Shorter and Fink 2018)

- Described by Kahlbaum in 1847
- Kraepelin and Bleuler thought 2/2 *dementia praecox* → schizophrenia
- Fink and Gelenberg later reclassified as a **syndrome with many causes**

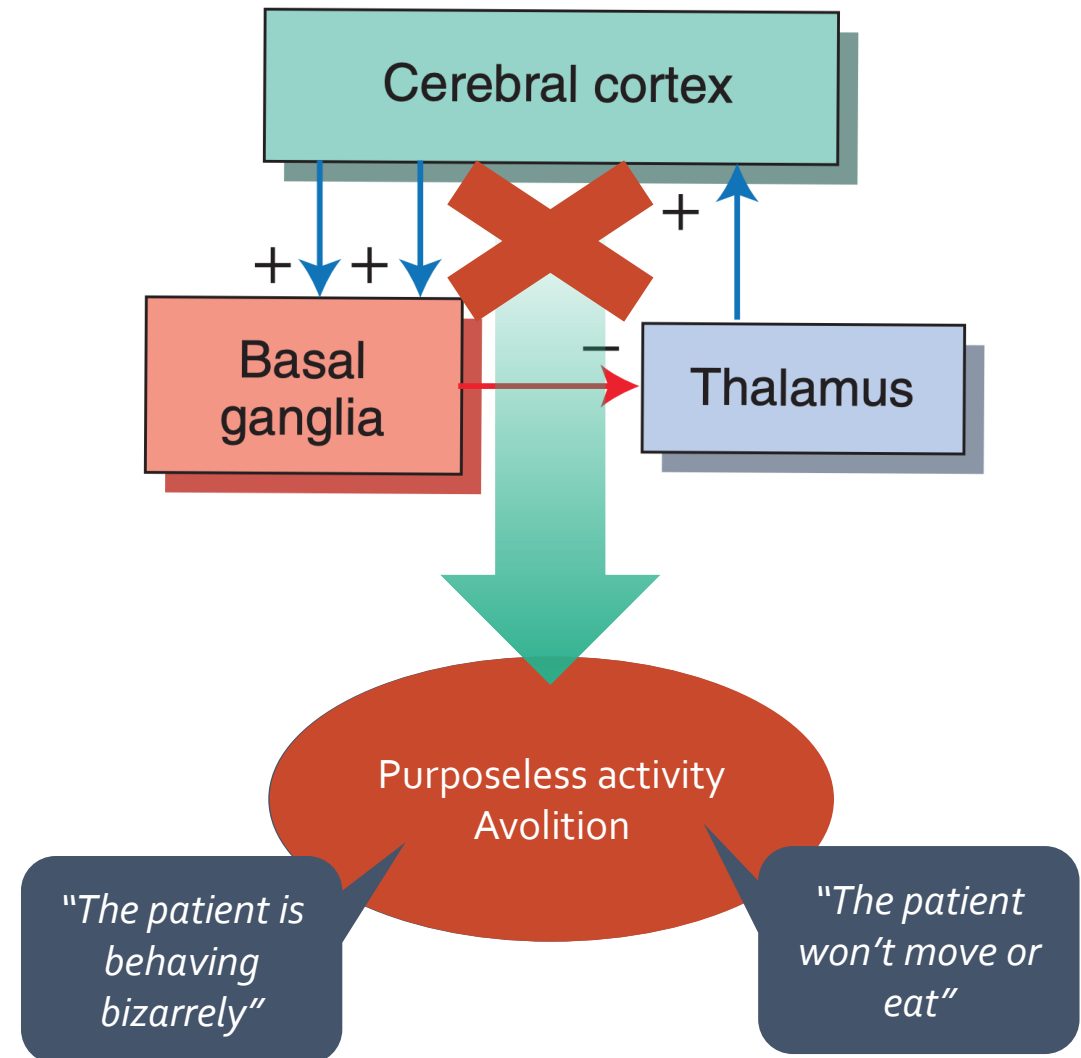


Definitions

Catatonia: a syndrome of disturbed **movement** and **behavior** associated with affective and cognitive changes

Aberrancy in the **function** of brain networks involved in motor **initiation**, **termination**, **motivation**, and **selection** at any level may result in catatonia

From *Essential Neuroscience*, 2nd Ed. Ch 20: Motor Systems.



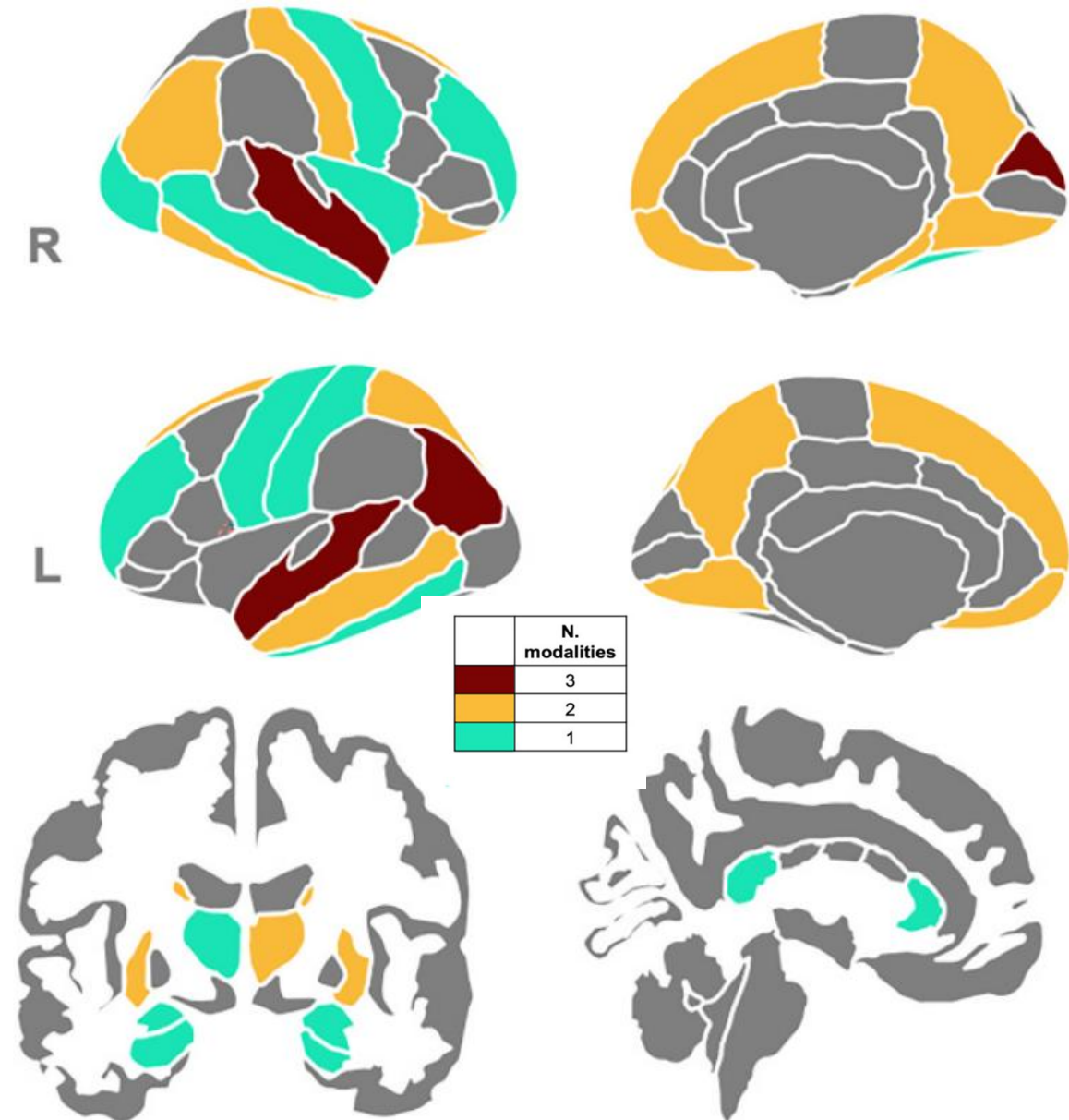
Psychomotor activity	Assessment	BFCSI (2 of 14)	BFCRS (8, 28) (severity measure)	ICD-11 (44) (3 of 15)	DSM-5-TR (39) (3 of 12)		
Increased	Observe	Excitement		Increased Psychomotor Activity	Agitation		
			Impulsivity				
			Combativeness				
Abnormal	Observe	Grimacing					
		Mannerism					
		Stereotypy					
		Verbigeration					
	Elicit	Posturing/Catalepsy	Posturing				
			Catalepsy				
		Echolalia/Echopraxia	Echolalia	Echolalia/Echopraxia	Echolalia		
			Echopraxia		Echopraxia		
			Automatic obedience				
							Mitgehen
							Gegenhalten
							Perseveration
							Grasp reflex
	Observe/Elicit						
	Exam						
Decreased	Observe/Elicit	Waxy flexibility					
		Stupor					
		Mutism					
		Withdrawal					
		Ambitendency					
	Elicit	Negativism					
	Observe	Staring					
Exam	Rigidity						
Other	Exam/Collateral		Autonomic abnormality				

From Wilson et al. 2025

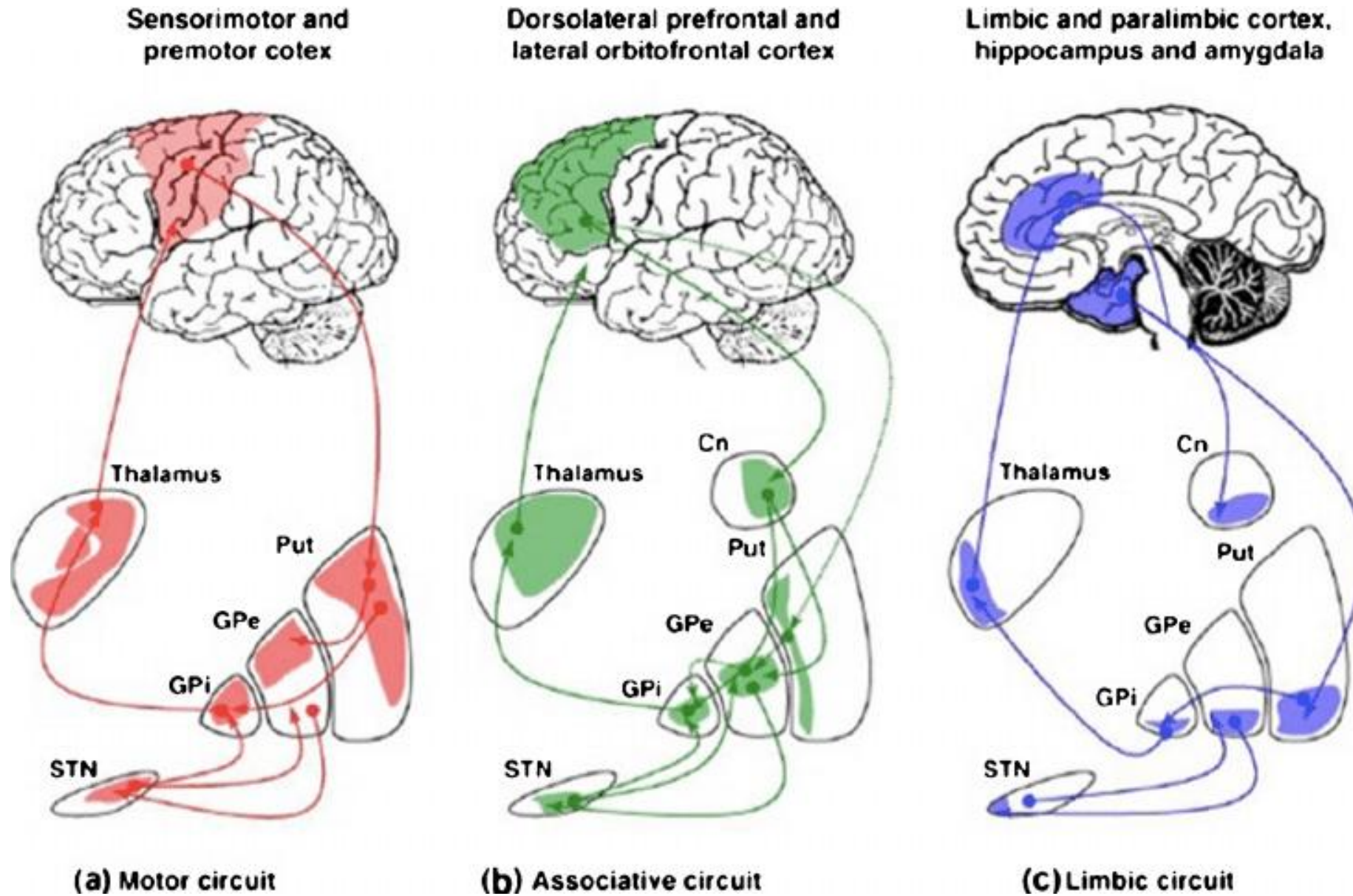
Pathophysiology

(Cattarinussi et al. 2022)

- Pathophysiology hypothesized but not proven to date
- Neurotransmitters: GABA, glutamate, dopamine, norepinephrine
- Neuroanatomy: widely distributed, involving **fronto-parietal networks**, **CSTC loops**, **salience network**, and **DMN dysfunction**
- Other studies have also implicated the **cerebellum** (particularly posterior hemispheres and vermis)

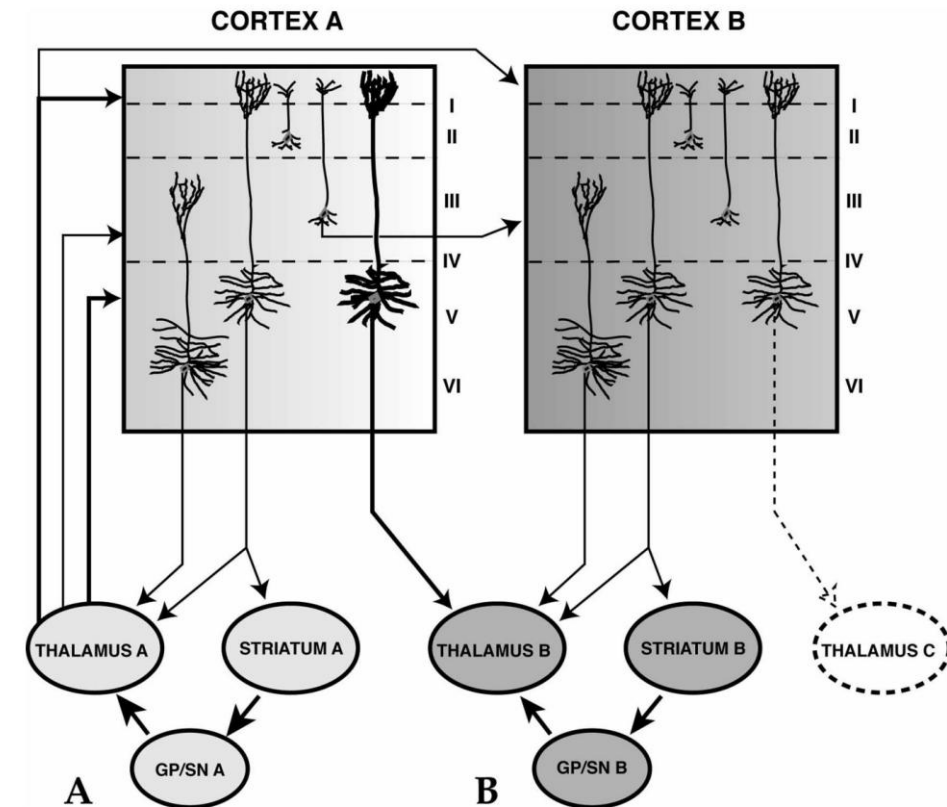


From Lapidus et al. (2014), based on Alexander's 5 parallel CSTC loops



Pathophysiology (Northoff et al. 2002, Moyal et al. 2025)

- **“Top down”**: altered modulation across the “motivation to action” pathway through cortico-cortical connections leads to aberrant motor programming vs CSTC
- **“Bottom up”**: limbic overdrive or hypodopaminergic states
 - Mammalian fear response?
 - Inflammatory hypothesis
 - Neuroleptic malignant syndrome



Epidemiology

(Oldham 2018, Wilson et al. 2017, Luccarelli et al. 2025, Cuevas-Esteban et al. 2025, Mormando et al. 2026)

Rare syndrome: 4-5 episodes per 100,000 population
Nearly always “secondary”

9-20% of
psychiatric
inpatients

13-31%
of mood disorder
patients
(esp BPAD)

17%
of ASD patients

7-30%
of schizophrenia
patients

22.4% of FEP,
a/w worse
psychopathology

25-30%
of hospitalized
delirious patients
(catatonic
symptoms may
respond to
lorazepam)

20% of catatonia
presentations
are
nonpsychiatric—
**majority CNS
illnesses**

Epidemiology (Oldham 2018, Conti et al. 2025)

TABLE 3. Features Suggestive of Primary or Medical Catatonia

Features favoring medical catatonia

- Comorbid delirium (81% vs 0%)¹⁸
- Autonomic change (38% vs 5%)¹⁸
- Catatonic excitement (50% vs 11%)¹⁸
- Grasp reflex (26% vs 0%)¹⁸
- Pneumonia (57% vs 0%)¹⁸
- Known history of a neurological condition (68% vs 37%)¹³
- History of seizures (42% vs 14%¹³; 48% vs 0%¹⁸)

Features favoring primary catatonia

- History of psychiatric illness (90% vs 47%)¹³
- Female gender (65% vs 40%)¹³
- Presenting neuroleptic use (52% vs 16%)¹³

TABLE 4. Medical Causes by Category

Miscellany	59 (19.5%)
Inflammation of the CNS	87 (28.8%)
Neural injury	58 (19.2%)
Developmental disorder	6 (2%)
Structural CNS pathology	29 (9.6%)
Epilepsy	28 (9.3%)
Toxin or medication	35 (11.6%)
Total	302

Endocrine disorders

Metabolic disturbances (including hypovitaminoses)

Electrocution

Genetic syndromes (Phelan-McDermid, 22q11 deletion, Trisomy 21)

Epidemiology (Oldham 2018)

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Infectious meningoencephalitis: HSV, HIV, SSPE, COVID-19, malaria, prion, etc.

Limbic encephalitis: autoimmune encephalitis targeting limbic regions, associated with regulation of emotion and motion (**NMDAR**, LGI1, GAD65, CASPR2, GABA)

Lupus encephalitis and MS

Epidemiology (Oldham 2018)

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Traumatic brain injury

*Hypoxemic-ischemic and post-hypoxic
leukoencephalopathy*

Neurodegenerative disease (including late iPD)

PRES

Epidemiology (Oldham 2018, Rogers et al. 2024)

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Consider non-convulsive status epilepticus originating from the medial temporal lobe. There are no reported cases of catatonic NCSE discovered with depth electrodes, although this is a theoretical possibility.

May also present in a post-ictal or interictal pattern, usually associated with psychosis.

Epidemiology (Oldham 2018)

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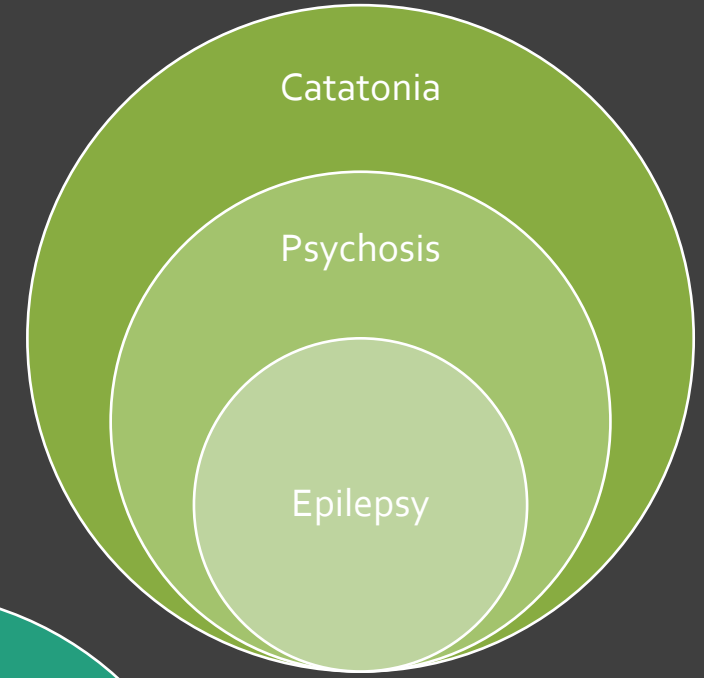
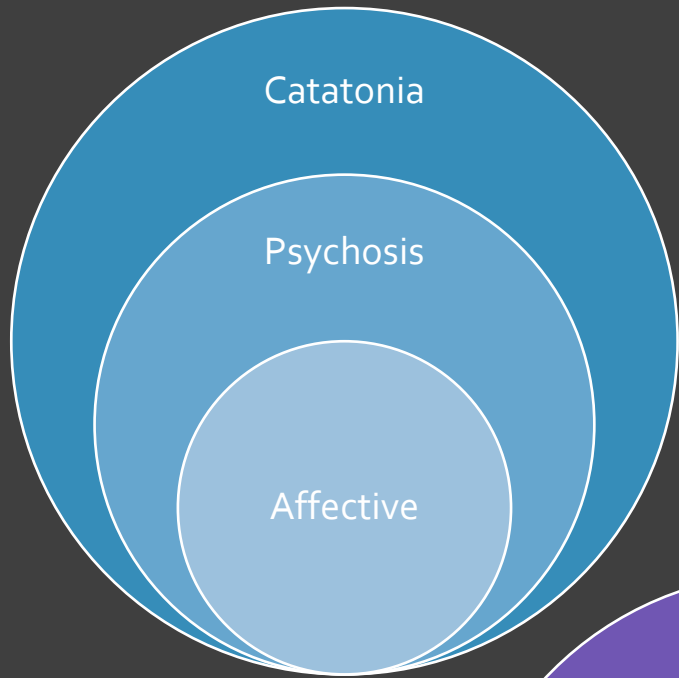
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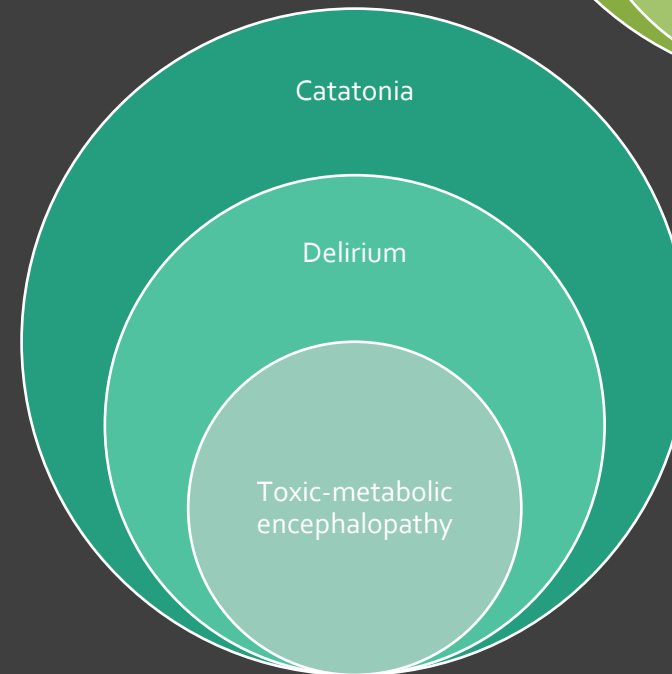
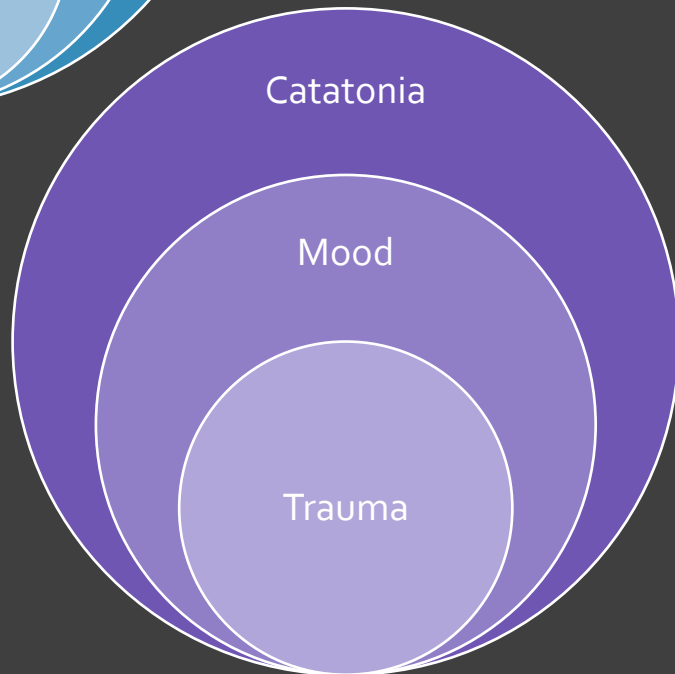
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DA⊗	Sedatives	Others	Abuse
APs	GABA w/d	Disulfiram	Stimulants
Antiemetics	Z-drug w/d	Antibiotics	K ₂ /MJ
Clozapine withdrawal	Baclofen	Immunomodulatory (tacrolimus, steroids)	PCP and inhalants
HXY, BSP	Gabapentin	Lithium	Opioids and CO



"Onion" Model



Prototypical Presentations (Rogers et al. 2023)

Type	Hypokinetic	Hyperkinetic	Malignant
Psycho-motor activity	Stuporous or obtunded—although not comatose	Prolonged, purposeless psychomotor activity	Typically quite rigid, may be hyper- or hypoactive
Symptoms	Immobility Mutism Withdrawal Staring Rigid	Mannerisms Stereotypies Verbigeration Echophenomena Impulsivity Combateness	Hyperthermia VS changes (↑T, ↑HR, ↑↓BP) Diaphoresis Rigidity
DDx and subtypes	Hypoactive delirium Akinetic mutism NCSE (TLE or FLE) Disorders of consciousness Functional coma Parkinsonian crisis Stiff person syndrome	Hyperactive Delirium Acute and delirious mania NCSE (TLE or FLE) Akathisia	Neuroleptic malignant syndrome Serotonin syndrome Meningoencephalitis
Notes	May not respond to pain Incontinence or retention		CK very commonly elevated, sometimes without rigidity (atypical NMS)

Related Syndromes and Subtypes

(Rogers et al. 2023, Karmacharya et al. 2008, Fusunyan et al. 2022)

Periodic catatonia

- Onset typically in adolescence, but described in older adults
- Considered an “idiopathic” catatonia, strong genetic basis
- Acute, weeks-long episodes followed by prolonged remission
- Rapid switches between hypo- and hyperactive
- Lithium may be treatment of choice for maintenance

Delirious mania

- Specific type of hyperactive catatonia
- Acute mania with delirious features with purposeless motor activity
- Other canonical features: *denuditeness*, *inappropriate toileting*, and *water obsession*
- Tempo is often more rapid than typical mania
- Treatment: High-dose lorazepam and ECT, then low-potency neuroleptics (clozapine, quetiapine)

Related Syndromes and Subtypes

(Rogers et al. 2023, Karmacharya et al. 2008, Fusunyan et al. 2022, Smith et al. 2024, Munir 2025)

Akinetic Mutism

- Immobility, mutism, paratonia (indicative of frontal release) due to profound motivational deficit
- Lacks affective and hyperkinetic features of catatonia
- Typically follows neurological injury (particularly to ACC) but also inflammatory states
- Does not respond to lorazepam; may improve with zolpidem, amantadine, or stimulants

Neurodevelopmental Disorders (eg ASD)

- Common decompensated state among patients with ASD and genetic syndromes
- MUST reflect a marked departure from baseline with **2/5 proposed core criteria**:
stupor/immobility, new mutism, posturing/catalepsy, stimulus independent excitation/agitation
- Stereotypies, verbigeration, echophenomena often present at baseline
- Treatment is similar, although benzodiazepines may be challenging to wean

Complications

(Clinebell et al. 2014, Park et al. 2017, Rodriguez-Cabo et al. 2026, Pathak et al. 2025, Hsu et al. 2026)



Vascular
thromboses

50% mortality → 10%
with ECT/BZDs for
MC/NMS



Pressure ulcers



Myolysis

Marker of
mortality and
other adverse
outcomes, ~50%
relapse in 2 years



Muscle
contractures



Nutritional
deficiencies



Hyperthermia and
death

Evaluation: History



PRODROMAL
FEATURES



TEMPO OF
ONSET



AFFECTIVE
SYMPTOMS



NEUROLOGIC
SYMPTOMS

Evaluation: Data

(Hosseini et al. 2023, Blackman et al. 2023, Ross et al. 2021)

Labs

- Definitely:
 - CMP and CBC
 - TSH
 - CPK
 - Tox
 - B12/folate
 - Serum iron
- Consider:
 - AM cortisol
 - Heavy metals
 - Vitamin C, zinc

EEG

- Useful to distinguish psychiatric from GMC/N catatonia and to rule out NCSE
- *Any abnormality: SN 0.76, SP 0.67 for GMC/N*

MRI

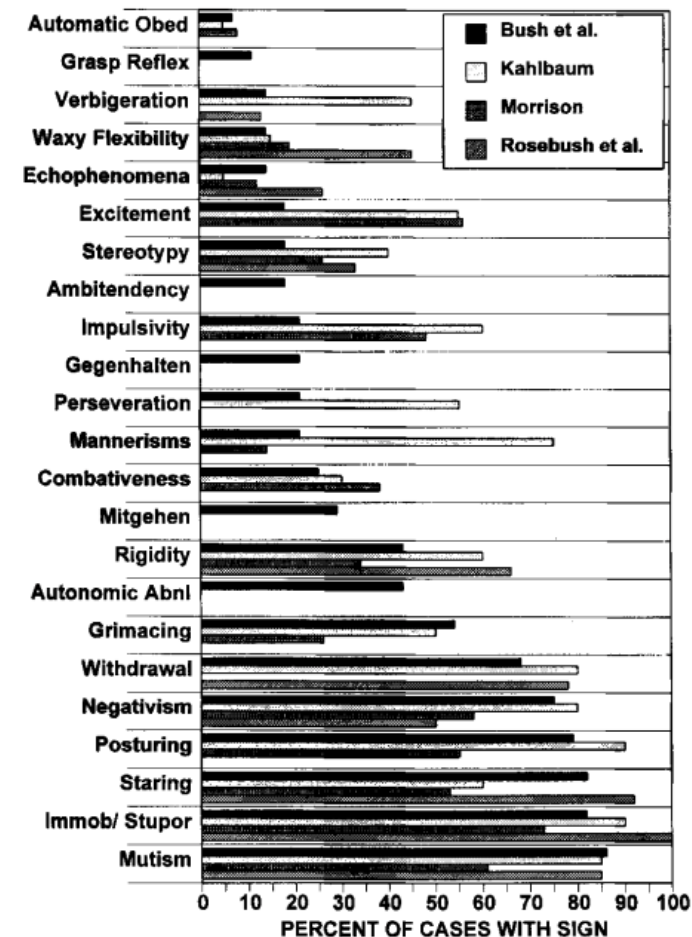
- If first lifetime episode OR concerning history
- *5.9% findings relevant in FEP, and motor findings such as catatonia enhance cost-effectiveness*

LP

- If concerns for meningitis or encephalitis
- *May be cost effective to screen for autoimmune encephalitis in any FEP, particularly with motor features*

Evaluation: Exam (Bush et al. 1996)

Screen Symptoms	Scoring Symptoms
Excitement	Impulsivity
Immobility	Automatic obedience
Mutism	<i>Mitgehen</i>
Staring	<i>Gegenhalten</i>
Posturing/catalepsy	Ambitendency
Grimacing	Grasp reflex
Echophenomena	Perseveration
Stereotypy	Combativeness
Mannerisms	Autonomic disturbance
Verbigeration	
Rigidity	
Negativism	
Waxy flexibility	
Withdrawal	



Evaluation: Exam

1. Observe the patient from the door. Maintain safety at all times.
2. Tone exam, rotating arm (fulcrum wrist, fulcrum elbow)—fast and slow
3. Ask to hold arms up but maintain them in the same position, and then push upward
4. Position arms in an odd position, and ask them to let them fall
5. Give conflicting instructions (put hand out but tell them “do not shake my hand”)
6. Rub hand along palm to fingers distally
7. Provide a movement to copy (e.g. scratching head)
8. Odd command: “I have a pen in my pocket. Stick your tongue out so I can write my name on it.”
9. As much a cognitive and elemental neurological exam as patient will tolerate

The Bush-Francis Catatonia Rating Scale Assessment Test Patient



Evaluation: Diagnostic Challenge

Complete BFCRS or other standardized diagnostic exam (e.g. Northoff)

Administer 1-2 mg IV lorazepam (consider SE risk: elderly, resp compromise)

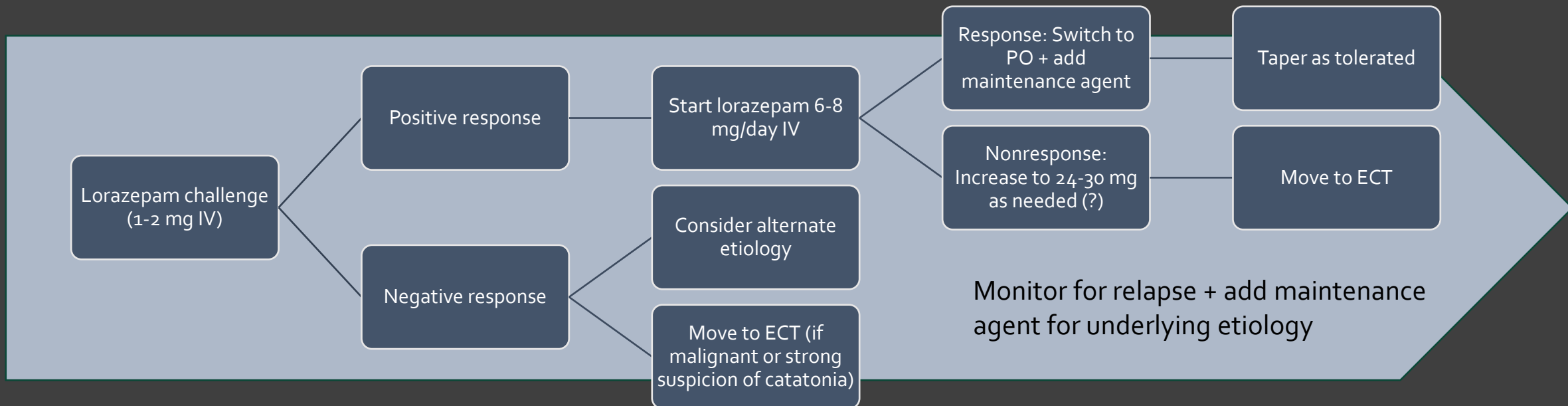
Repeat BFCRS in 30-120 minutes (response may range widely)

Continue lorazepam 1-2 mg q6-8h if the patient improves, or if there continues to be a very high suspicion of catatonia

Treatment: Lorazepam

(Rogers et al. 2023, Ungvari et al. 1999, Luccarelli et al. 2026)

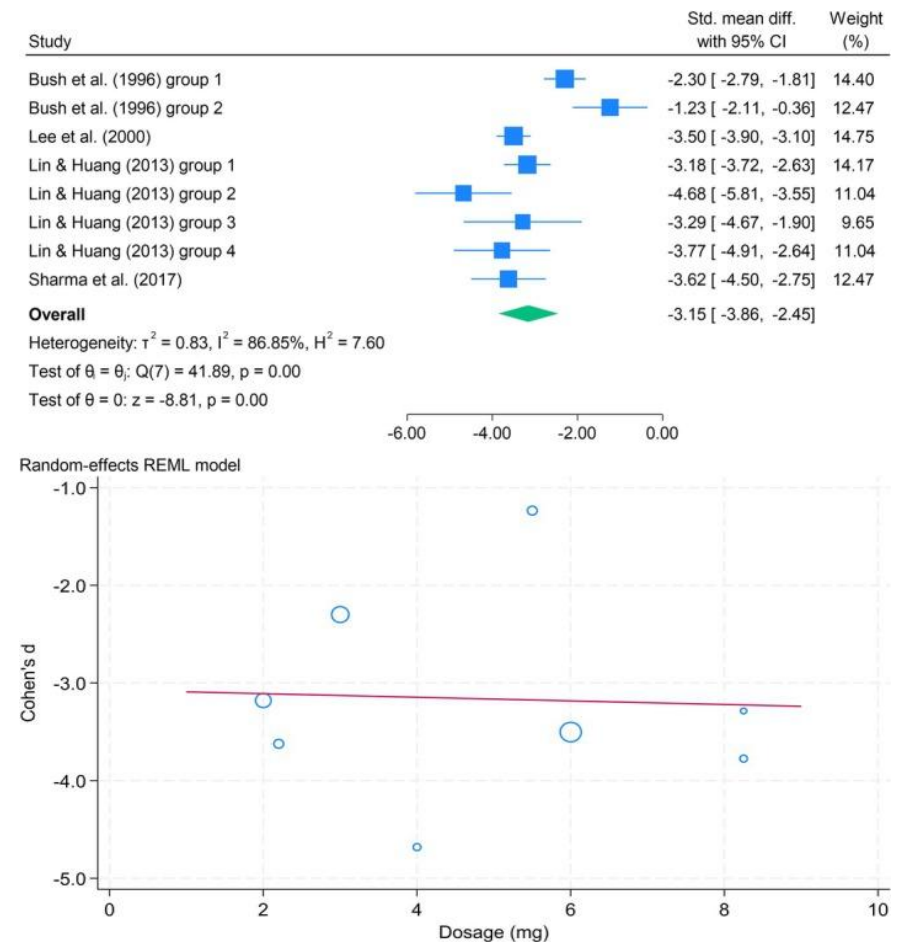
- Response rate: Historically estimated **66-100%**, typically within 10 days
- *Ungvari et al. found no benefit in the only DBRCT of lorazepam in catatonia (population: chronic catatonic schizophrenia)*
- Other benzodiazepines like midazolam and diazepam may suffice



Treatment: Lorazepam (Bot et al. 2025, Duque et al. 2026)

Bot et al. (2025): meta-analysis of 20 studies and 138 patients

- Effect: -11 ± 4 on BFCRS, Cohen's $d=2.8-3.0$ (very large)
 - Response = 77%
 - Remission = 53%
- No correlation with response/remission and dose up to 9 mg
- General medical or neurologic cause was less treatment-responsive than primary psychiatric, congruent with other studies
- Weaknesses
 - Doses of lorazepam analyzed did not exceed 9 mg due to lack of data
 - No control group



Treatment: ECT (Rogers et al. 2023, Mann et al. 1990, Beach et al. 2017)

- Response rate: **59-100%**
 - Potentially higher in malignant catatonia
 - May response within 2-3 treatments, although 20+ occasionally required
- Bilateral, three times weekly is most effective
- Drawbacks
 - Limited availability
 - Complicated consent process (substituted decision-making in emergencies)
- Indications
 - Malignant catatonia
 - Limited response to lorazepam after 2-5 days
 - Very poor self-care
 - Underlying MDD or BPAD

Treatment: **NMDA-receptor antagonists**

- Small case series level of evidence, reviewed by Beach et al. (2017); a supportive retrospective study from Duque et al is forthcoming
- Thought to inhibit NMDA receptors (inhibitory for DA signalling), which may be involved in catatonia
- Amantadine 100 mg daily to BID, increased to 600 mg TDD over 3-4 days
- Memantine 5-10 mg BID, increased to 10 mg BID over 3-4 days
- A small systematic review of case reports documenting success without significant adverse events with ketamine treatment (Caliman-Fontes et al. 2024)
- No reports on dextromethorphan

Treatment: Others

(Beach et al. 2017, Almeida 2021, Sienaert et al. 2014, Gunther et al. 2025)

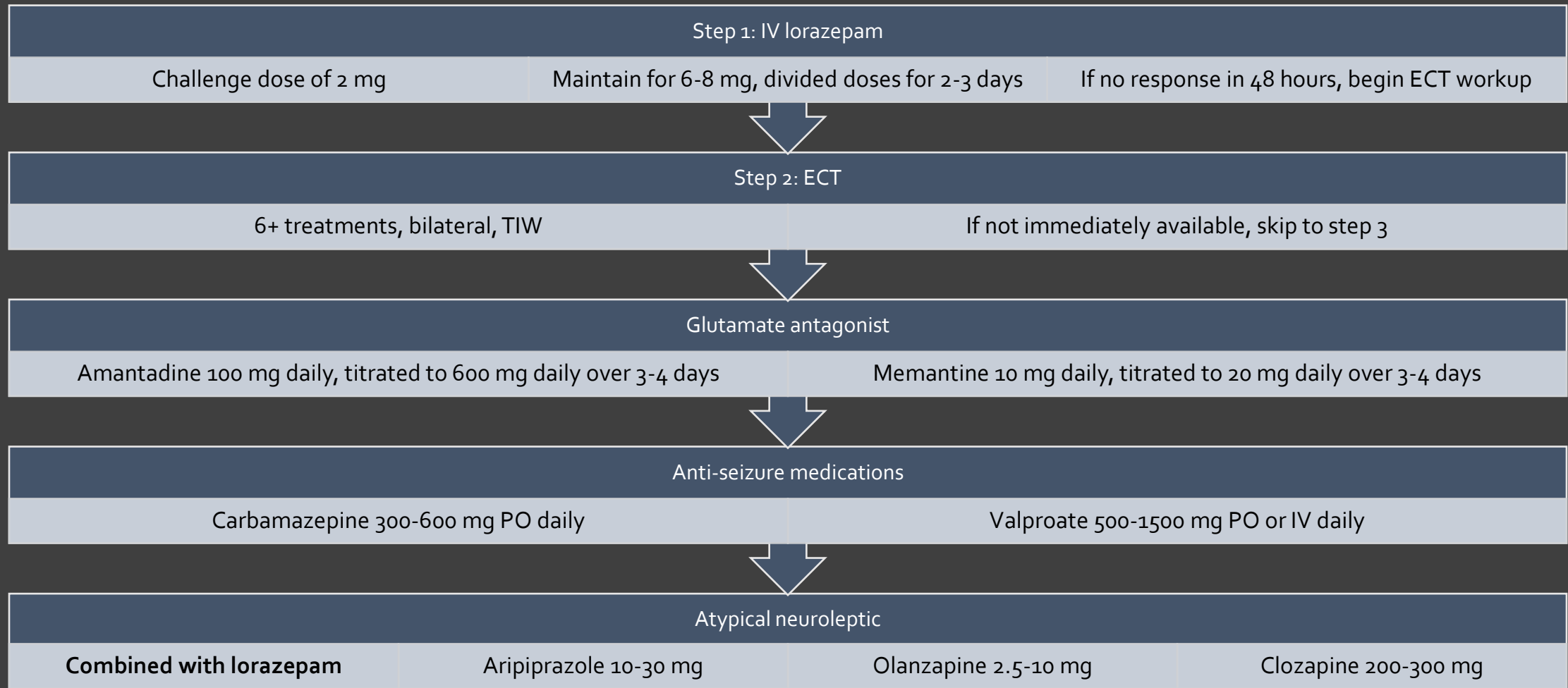
- **Overall, based on case report level of evidence**
- **Zolpidem:** Success reported on up to 10 mg QID for patients in whom lorazepam and ECT has failed by a recent review of cases
- **Dopamine agonists**
 - May be useful if likely to be neuroleptic induced, but *may worsen underlying psychosis*
 - Options include *bromocriptine, dantrolene, Sinemet, stimulants*

Treatment: Others

(Beach et al. 2017, Almeida 2021, Sienaert et al. 2014, Redon et al. 2025)

- **Overall, based on case report level of evidence**
- **Antiseizure medications:** May potentiate GABA signaling and/or treat underlying illness
- **Atypical neuroleptics** may be useful for refractory cases, particularly if underlying diagnosis is schizophrenia. Recent review of cases indicated **clozapine**, **amisulpride**, and **risperidone** may be most beneficial, although **aripiprazole** and **quetiapine** are also commonly used.
- **rTMS** targeting DLPFC and **TDCS** have been tried with anecdotal success (particularly if BZDs intolerable, ECT unavailable)

Treatment algorithm (Beach et al. 2017)



Treatment: General Management



IV fluids and nutrition if necessary; monitor UOP and labs if possible



Prophylactic anticoagulation for most, if not all (Padua score can be useful)



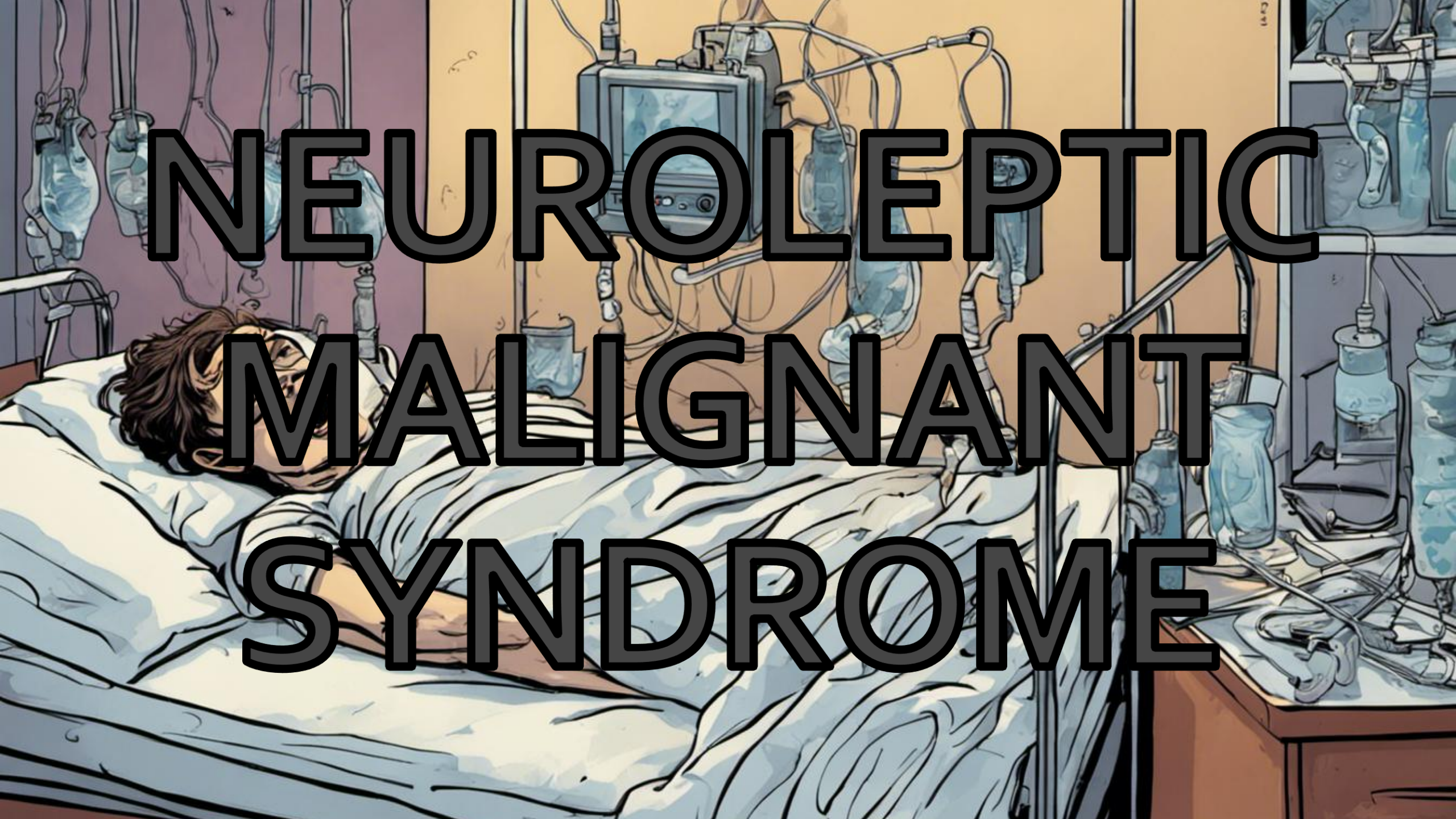
Frequent turning, barrier protection, skin exams



Discontinue high-risk agents (particularly neuroleptics), treat underlying cause

Treatment: Long-term (Brown et al. 2025, Patarayo-Rodriguez et al. 2026)

- Depends partially on underlying etiology
 - Catatonia d/t NCSE? Long-term management: ASMs
 - Catatonia d/t AE? Long-term management: immunosuppression (steroids --> rituximab)
 - Catatonia d/t SCZ or BPAD? Long-term management: mood stabilizers and neuroleptics
- Transition to maintenance phase can begin when acute treatment stabilizes and catatonia is lysed
- Once maintenance treatment established, lorazepam should be tapered very slowly to prevent relapse
 - Continued lorazepam use may be associated with reduced relapse risk
 - Antipsychotics may reduce risk of relapse within 60 days of acute catatonia but may increase risk thereafter.



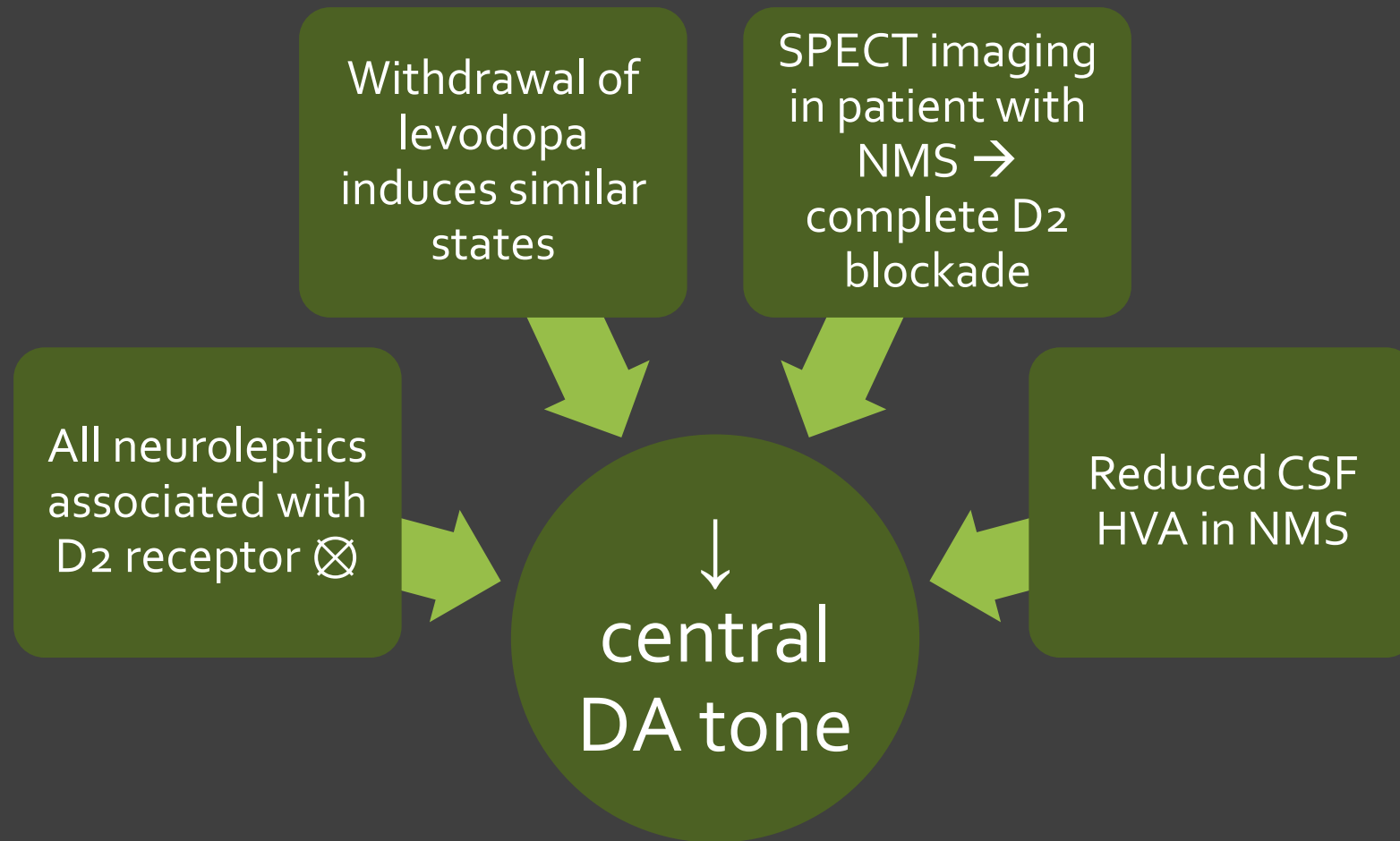
NEUROLEPTIC MALIGNANT SYNDROME

Definition and Epidemiology

(Schneider et al. 2020, Caroff and Mann 1988, Wijdicks and Ropper 2024)

- Severe, life-threatening motor disturbance with altered mentation associated with start of antipsychotic or removal of dopamine agonist (but not always—**96% within 30 days, median 4 days**)
- **0.16%** of patients treated with neuroleptics
- Generally considered to be a severe form of catatonia with a specific etiology
- Risk factors
 - High-potency neuroleptic use > low-potency/atypical
 - Multiple neuroleptics, dose dependent
 - Long-acting injectable formulations
 - History of catatonia/NMS
 - Basal ganglia disorders
 - State specific: withdrawal, dehydration, low-serum iron

Dopamine Hypothesis

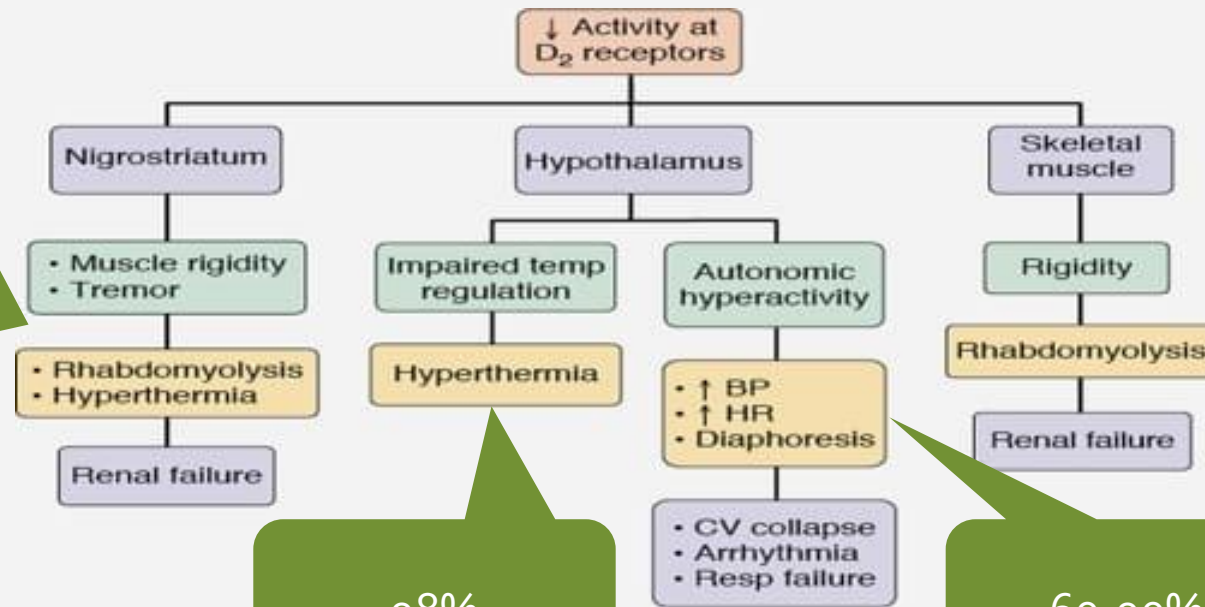


Clinical Characteristics

(Mann et al. 2003, Ozdemir et al. 2004)

7.6-9.5% mortality
even with treatment

97%
Generalized,
severe (often
unable to be
moved)



97%
encephalopathic
Very likely to meet
criteria for
catatonia

Lead Pipe Rigidity



Atypical NMS (Troller et al. 2009, Murri et al. 2015)

- Majority of cases of NMS produced by atypical antipsychotics present with “typical” NMS signs and symptoms
- However...
 - Clozapine: may be less likely to cause rigidity—but CK may still be elevated!
 - Aripiprazole: may be less likely to cause delirium and hyperthermia
- Many cases of so-called “atypical NMS” may represent non-malignant catatonia induced by antipsychotics
- Mortality of atypical NMS is 5.5%, close to typical findings

ICD Diagnostic Criteria (Gurrera et al. 2011)

Criterion	Points
Exposure to dopamine antagonist or agonist withdrawal in last 72 hours	20
Hyperthermia ($>100.4^{\circ}\text{F}$ on 2 occasions, oral)	18
Rigidity	17
Altered mental status	13
Elevated creatine phosphokinase ($>4\times$ ULN)	10
Sympathetic nervous system lability, defined by 2+ • $\uparrow$BP ($>25\%$ above baseline) • $\downarrow\uparrow$ BP (change of 20 mmHg DBP or 25 mmHg SBP in 24 hours) • Diaphoresis • Urinary incontinence	10
Tachycardia ($>25\%$ of baseline) and tachypnea ($>50\%$ above baseline)	5
Negative workup for infectious, toxic, metabolic, and neurologic causes	7

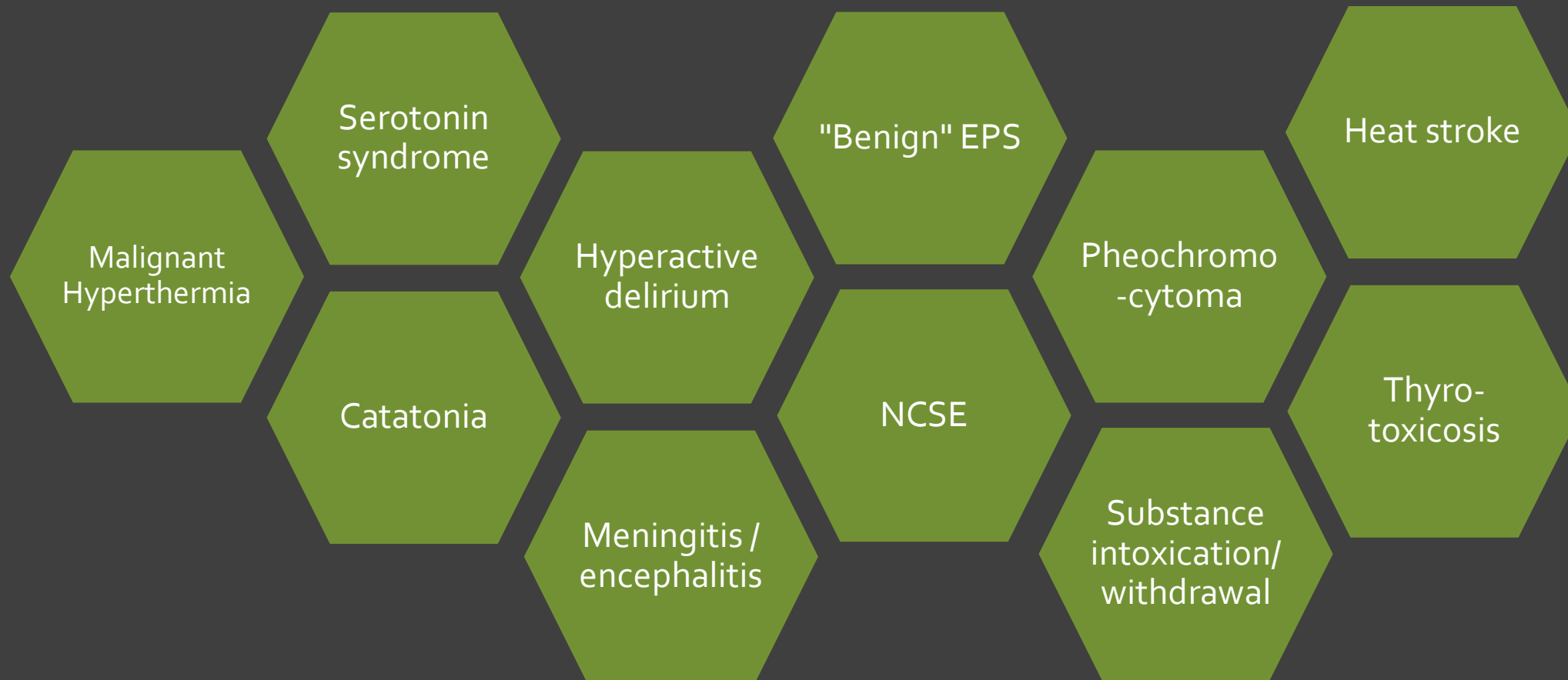
Cut-off of 74:

- 70% sensitive
- 91% specific
(*against expert opinion*)

Moderate to severe:

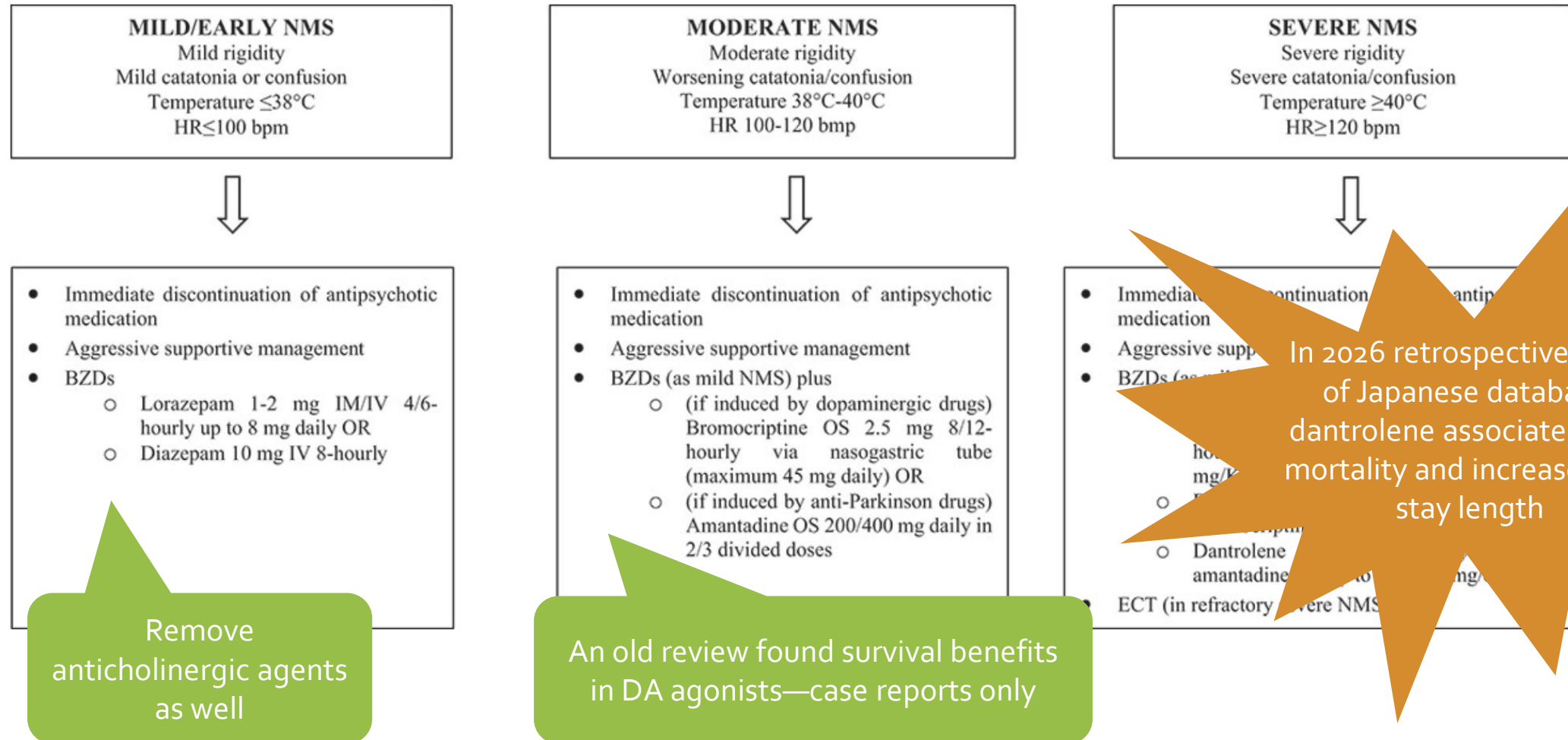
- Level of rigidity +
- $\text{HR} > 100-120$
- $\text{T} > 38-40^{\circ}\text{C}$

Differential Diagnosis



Management

(Caroff 1988, Orsolini and Volpe 2025, Sakkas et al. 1991, Chen et al. 2025, Suekene et al. 2026)



Management (Rogers 2023)

- Rechallenge? Recurrence is 30-50%!
- Wait **2 weeks** for resolution, then **gradually** titrate an **atypical** antipsychotic (ideally *clozapine*) in a **hospital**.
- Written consent (from guardian if necessary) is a must

A comic-style illustration of a man lying in a hospital bed. He has extremely exaggerated, hyper-defined muscles, particularly in his arms and legs, which are colored in a reddish-orange hue. His face is contorted in pain or distress, with his mouth wide open in a scream or shout. He is wearing a teal hospital gown. His right arm is raised, pointing his index finger upwards. The background shows a typical hospital room with a desk, a lamp, and a red wall. The text 'SEROTONIN SYNDROME' is overlaid in large, bold, grey letters with a black outline.

SEROTONIN SYNDROME

Definition and Epidemiology

(Boyer and Shannon 2005, Nguyen et al. 2017)

- Enhanced central serotonergic tone (particularly 5-HT_{1A}), likely associated with increases in dopaminergic and noradrenergic tone as well.
- Classically associated with MAOI + another serotonergic agent; now much more common in overdose or use of 3+ agents.
- 14-16% of SSRI overdoses, annual incidence (among psychiatric VA patients) of 0.07-0.09%
- Typically associated with psychotropics, but many medications are potentially serotonergic
- Commonly comorbid with other toxidromes (anticholinergic, stimulant, or NMS), confounding clinical assessment

Serotonergic Agents

Agonism

- Buspirone
- Lithium
- LSD
- L-tryptophan
- Trazodone

Decreased metabolism

- Irreversible MAOIs (phenelzine, selegiline, tranylcypromine)
- Reversible MAOIs (linezolid?)

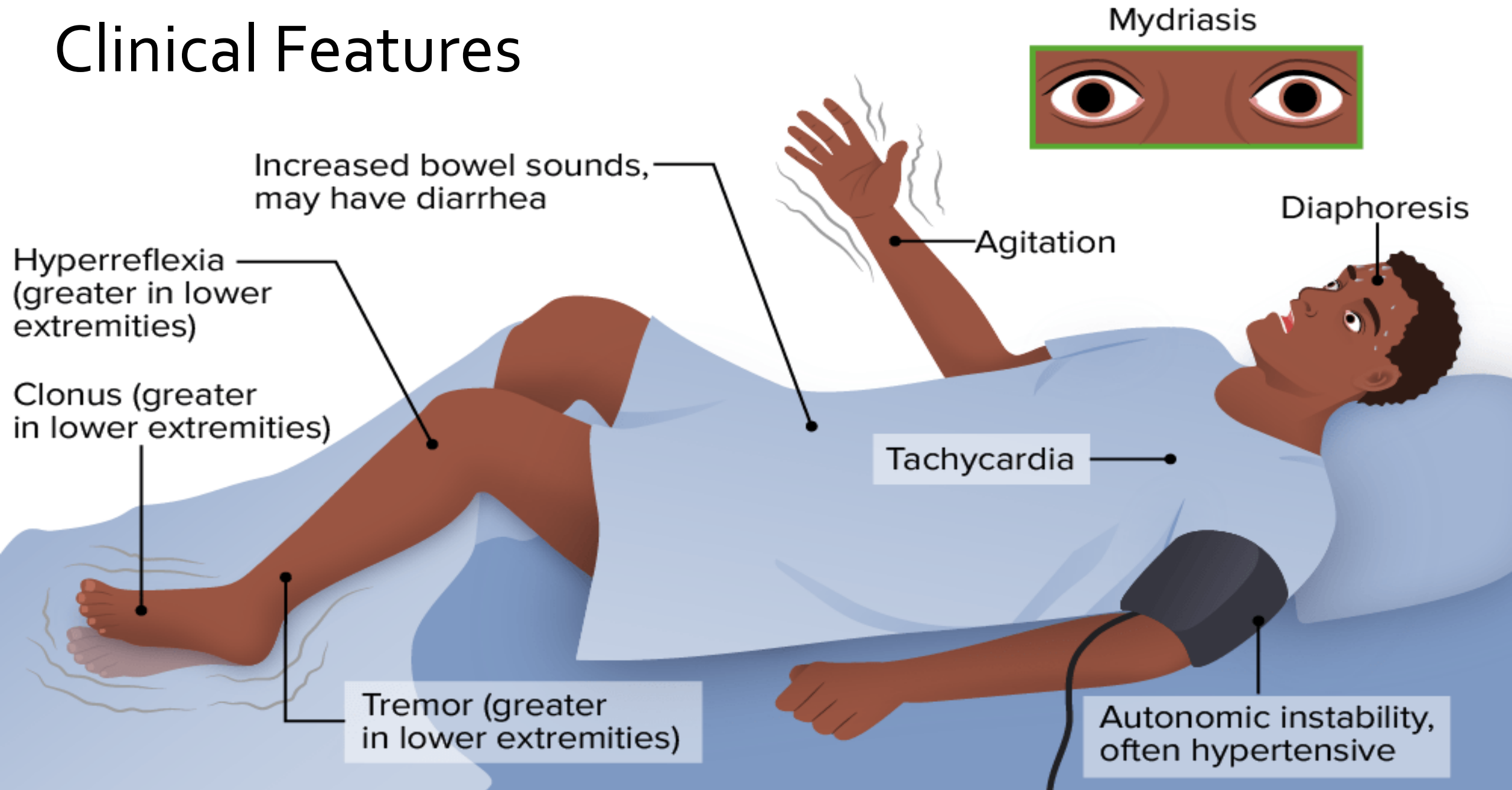
Increased release

- Amphetamines and MDMA
- Cocaine
- Fenfluramine
- Ergot alkaloids and triptans
- Mirtazapine

Decreased reuptake

- SRIs and TCAs
- Meperidine
- Tramadol
- SJW

Clinical Features



Clinical Features (Boyer et al. 2005, Scotton et al. 2019)

- No specific test: blood levels irrelevant
- WBCs ↑, CPK ↑, LFTs ↑, HCO_3^- ↓
- Severe cases can evolve to include:
 - **Disseminated intravascular coagulation (5%, but high risk of death: 67%)**
 - **Seizures (1%, but high risk of death at 39%)**
 - **Rhabdomyolysis (25%)**
 - **Myoglobinuric renal failure (5%)**
 - **Aspiration and adult respiratory distress syndrome**

Consider serotonin syndrome in...

All overdoses

All patients on >4 psychotropics

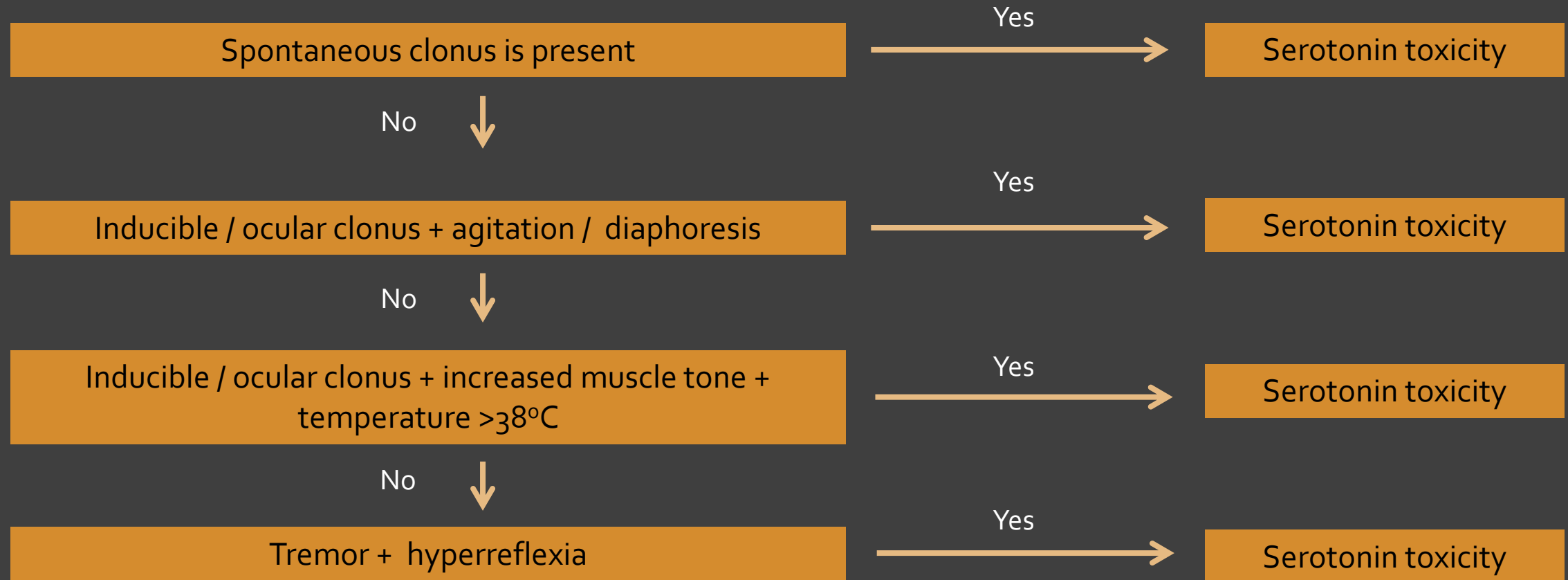
Patients with critical illness, renal dysfunction, liver dysfunction + psychotropics

Catatonic patients, particularly hyperactive

Delirious patients, particularly the twitchy ones

Hunter Criteria (Dunkley et al. 2003)

In the presence of a serotonergic agent, serotonin toxicity is diagnosed:



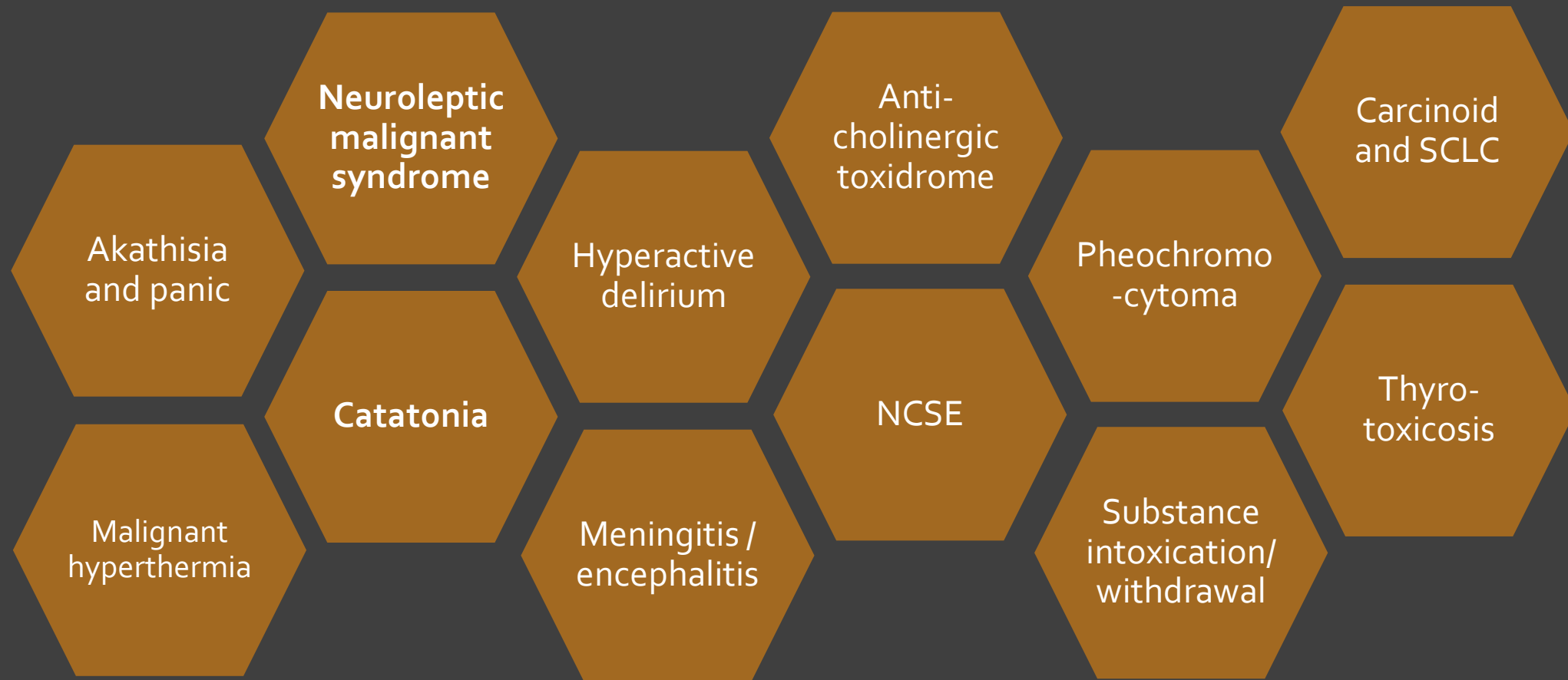
Hunter Criteria (Dunkley et al. 2003)



Hunter Criteria (Dunkley et al. 2003)



Differential Diagnosis



Management (Boyer 2005, Scotton et al. 2019)

- Discontinue the offending agent
- Often self-limited, requiring support only (including hydration and cooling, management of VS as appropriate)
- Intubation and paralysis in very severe cases not responding to above
- ASMs if seizures arise
- Avoid physical restraint if possible. Avoid neuroleptics and anticholinergics.

Management (Boyer 2005, Scotton et al. 2019, Chiew and Isbister 2025)

Benzodiazepines

- Useful for agitation and catatonic features
- Useful for hyperadrenergic state

Serotonin antagonists

- Helpful in some case reports and series. Animal data suggests a mortality benefit.
- **Cyproheptadine** 12 mg initial → 2 mg q2h until improvement → 8 mg q6h maintained then tapered
- Other agents include chlorpromazine, risperidone, ketanserin, sertindole

Alpha-2 antagonists

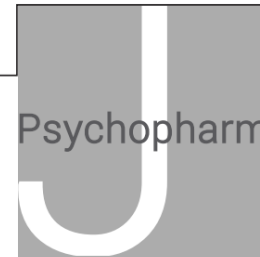
- Dexmedetomidine reportedly helpful to mitigate agitation and VS abnormalities

Suggested Reading

BAP Guidelines

Evidence-based consensus guidelines for the management of catatonia: Recommendations from the British Association for Psychopharmacology

Jonathan P Rogers^{1,2} , Mark A Oldham³, Gregory Fricchione^{4,5}, Georg Northoff⁶, Jo Ellen Wilson^{7,8}, Stephan C Mann⁹, Andrew Francis¹⁰, Angelika Wieck^{11,12}, Lee Elizabeth Wachtel^{13,14}, Glyn Lewis¹ , Sandeep Grover¹⁵, Dusan Hirjak¹⁶, Niraj Ahuja¹⁷, Michael S Zandi^{18,19}, Allan H Young^{20,2}, Kevin Fone²¹, Simon Andrews²², David Kessler²³, Tabish Saifee¹⁹, Siobhan Gee^{24,25} , David S Baldwin²⁶ and Anthony S David²⁷



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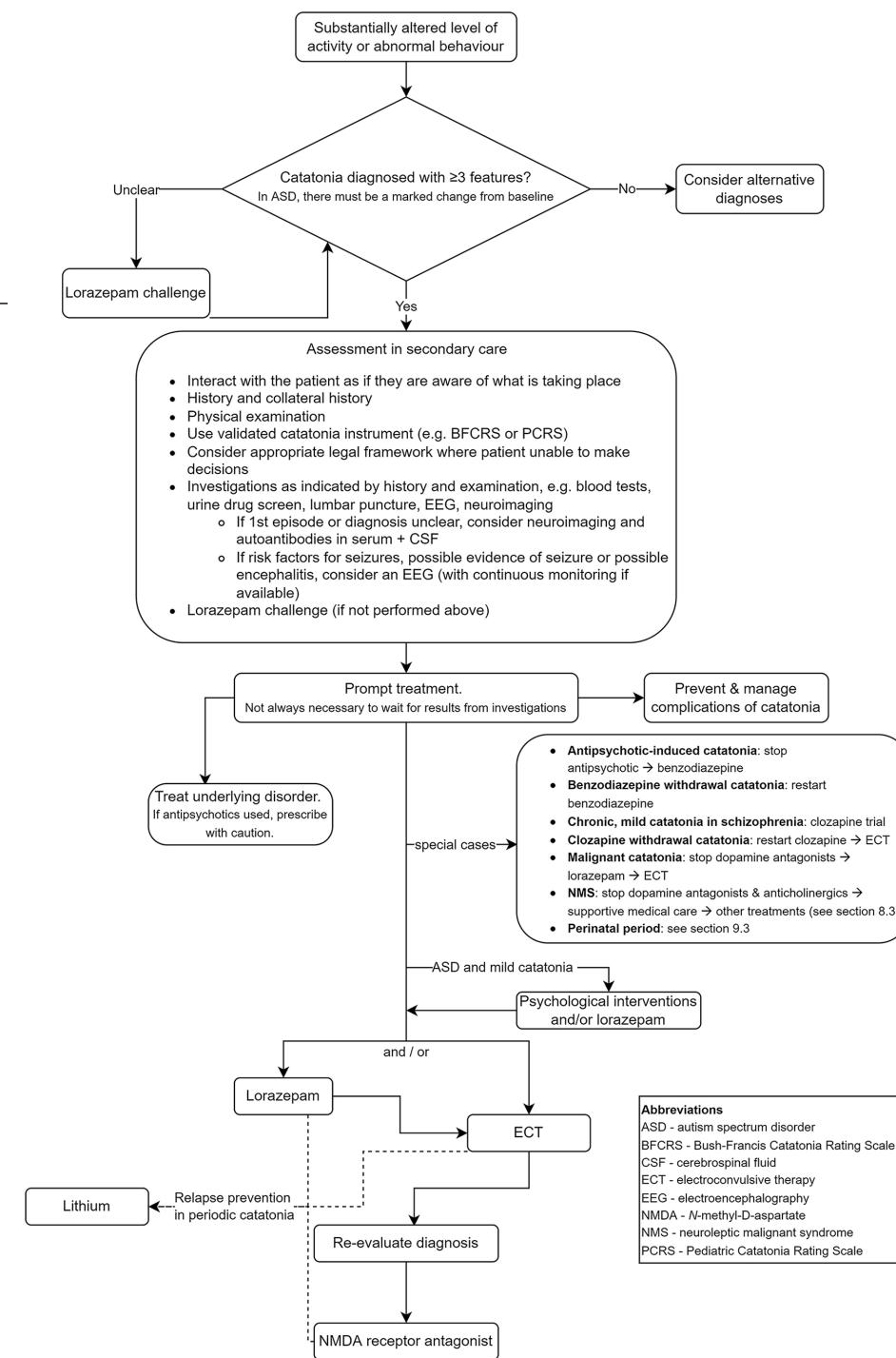


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THE MADNESS OF FEAR

A History of Catatonia



EDWARD SHORTER
MAX FINK

OXFORD

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Special Article

Catatonia: American Psychiatric Association Resource Document

Jo Ellen Wilson, M.D., Ph.D., M.P.H., Mark A. Oldham, M.D., Andrew Francis, M.D., Ph.D.,
Dina Perkey, M.D., Eric Kramer, M.D., M.P.H., Shixie Jiang, M.D., Jennifer Yoon, M.D.,
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Joseph D. Dragonetti, M.D., Avi Gerstenblith, M.D., Anne Louise Stewart, M.D.,
Jon Sole, M.D., James A. Bourgeois, O.D., M.P.A., M.D.

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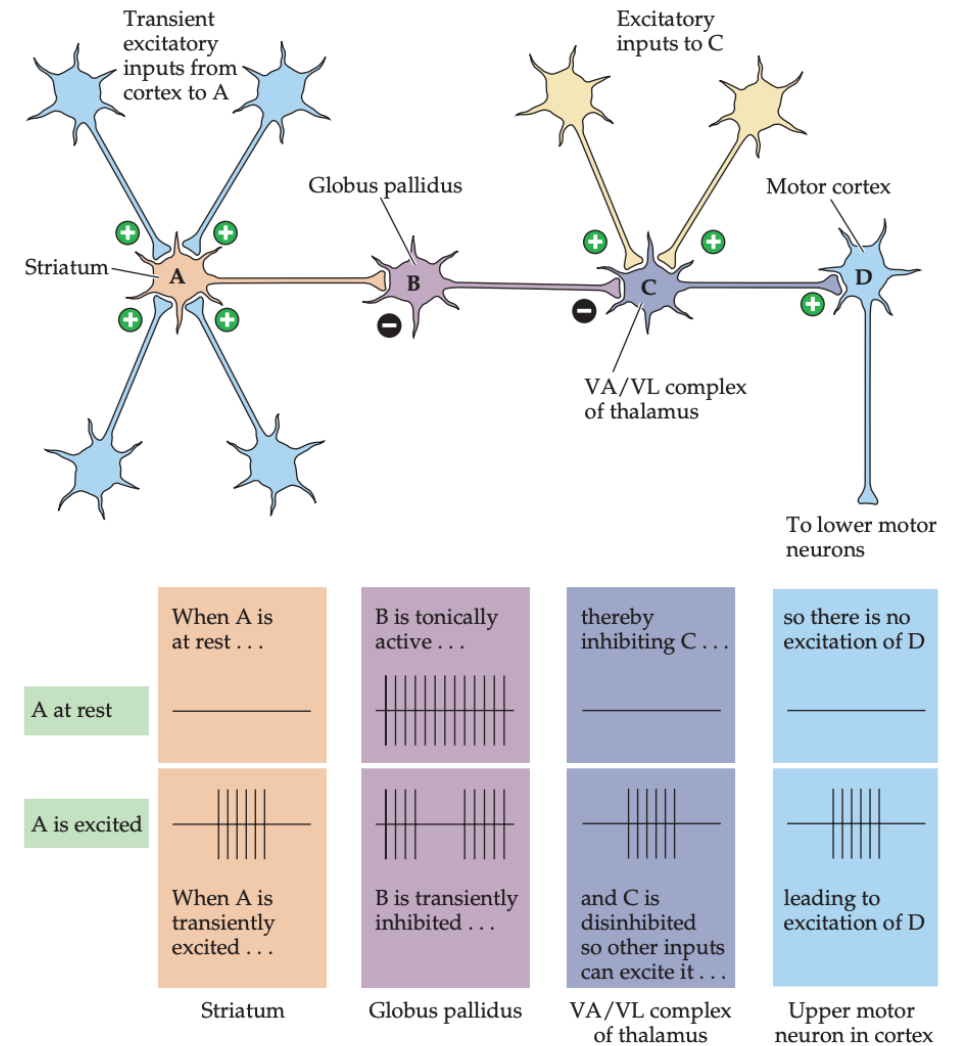
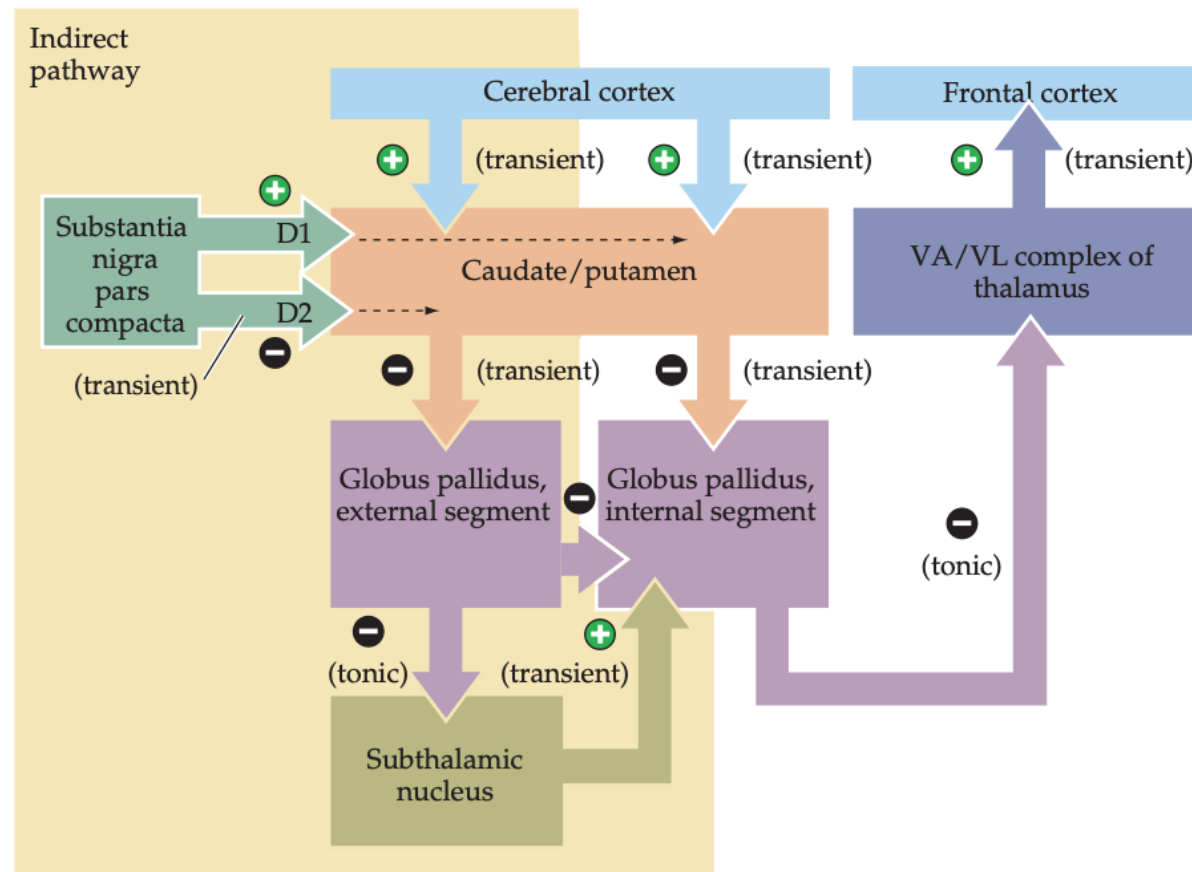
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From *Neuroscience, 3rd Ed. Ch 17: Modulation of Movement by the Basal Ganglia*

(B) Indirect and direct pathways



Case 1: Background

- **ES** was a **42-year-old left-handed man**, married with children, living locally and working as an investment banker...
- ...with a history of *pineal glial cyst s/p resection in 1996* c/b posterior fossa edema necessitating **partial cerebellar resection**, as well as (possibly) *premorbid episodes of depression and mania*...
- ...who was brought to the ED for concerns about **catatonia** in the context of *depression precipitated by allostatic overload*.

Case 1: HPI

Preceding months...

Ongoing mood instability
Depression → increased mirtazapine
Mania → discontinued mirtazapine, increased quetiapine
Depression again in context of work changes → lamotrigine added, lorazepam decreased
Ongoing difficulty to executive function

Preceding weeks...

Onset of odd behaviors, negativistic thinking
Perseveration and severe indecision
Latency of speech and thought, large amplitude movements of arms and body

ED

BFCRS 30

Stuporous and mute

Staring, grimacing, posturing

Echophenomena and stereotypies

Paratonia and grasp reflex

Ambitendency and perseveration

Tachycardia

Case 1: Workup

- CMP WNL
- CBC WNL
- TSH 1.17
- UA negative
- Stox, Utox negative
- Ammonia 35
- CK 286
- B₁₂ WNL
- EEG WNL
- MRI brain: stable cerebellar encephalomalacia
- Med Rec
 - ↑ QTP
 - + LTG

Case 1: Course

- Psychiatry consulted in ED → lorazepam 2 mg IV → improved symptoms
- Admitted to internal medicine, psychiatry following for catatonia, followed by transition to med-psych unit
- Medication changes → resolution of catatonia
 - Lorazepam 2 mg q4h → 1 mg BID by discharge
 - Discontinued lamotrigine
 - Discontinued quetiapine

Case 2

- AB was a 56-year-old woman with bipolar disorder who was admitted to the hospital after arguing with her husband.
- She was agitated in the ED, required restraint, and received haloperidol 5 mg IM atop home medications perphenazine, lithium, sertraline.
- Soon after the administration, she was noted to be **withdrawn, hypokinetic, mute** and **rigid**.
- VS were notable for tachycardia, hypothermia, and work-up demonstrated elevated CPK, generalized slowing on EEG.
- Home neuroleptics were held and she was treated with lorazepam, ultimately achieving a good response.

Case 3

- A 69-year-old woman with recurrent episodic MDD presented for follow-up reporting partial response to paroxetine 40 mg/day.
- The dose was increased to 60 mg, and buspirone 5 mg TID and trazodone 100 mg nightly were added two weeks later.
- Within 24 hours, she presented to the ED confused, diaphoretic, febrile, hyperreflexic, and mildly rigid.
- All medications discontinued and her condition gradually improved over 2 days.