Catatonia

Martin Balslev Jørgensen

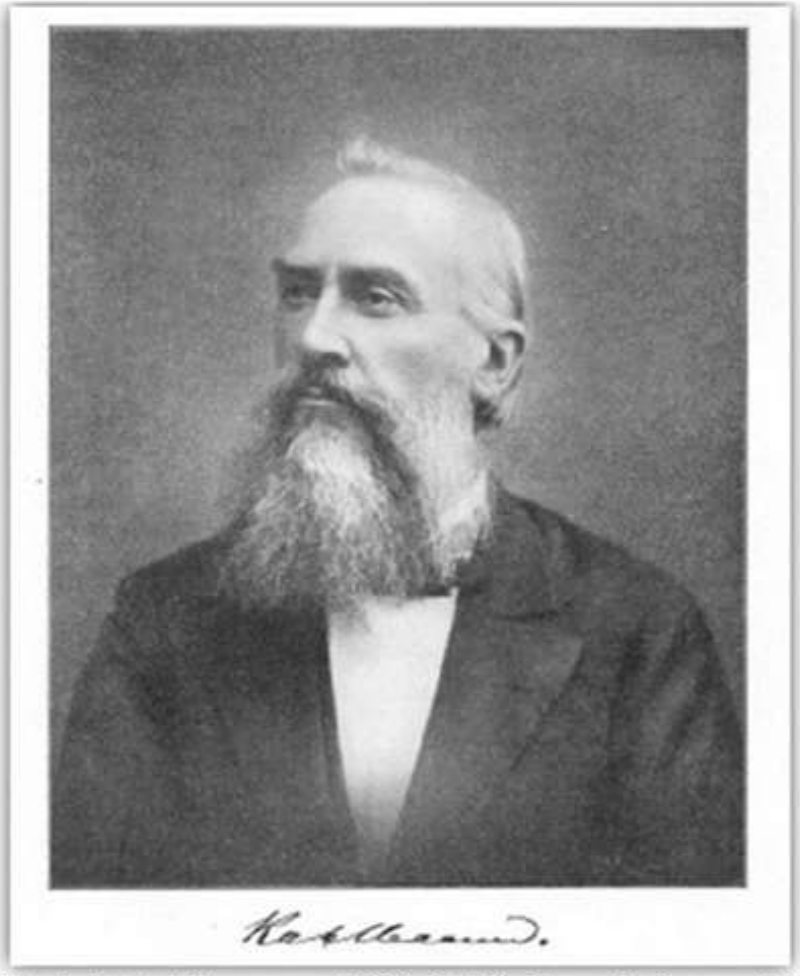
Catatonia is a syndrome of psychotic body movement and speech

- occur in the context of general medical, neurological, and psychiatric conditions as well as associated with medications and drugs of abuse

Kahlbaum

Die Katatonie oder das Spannungsirresein", 1874

Disturbance in motor functionality that represents a phase in a progressive illness that includes stages of mania, depression and psychosis



Karl Ludwig Kahlbaum, ca 1890, Public Domain in countries where copyright term is life of author plus 70 years.

Catatonia Nosology: ICD and DSM

Kraepelins perspective of catatonia as a form of schizophrenia

DSM-IV Catatonia secondary to a medical condition with a distinct code of 293.89 (10).

ICD-10 organic catatonia F06.1

DSM-V: 293.89 and ICD-11: 6A40-6A4Z

- Catatonia associated with another mental disorder (6A40)
- Catatonia induced by substances or medications (6A41)
- Catatonia, unspecified (6A4Z)

DSM-5 Catatonia is diagnosed by the presence **of three or more of the** psychomotor symptoms below:

Always associated with a mental disorder, medical condition, or unspecified:

Stupor no psycho-motor activity; not actively relating to the environment

Catalepsy passive induction of a posture held against gravity

Waxy flexibility allowing positioning by an examiner and maintaining that position

Mutism no, or very little, verbal response

Negativism: opposition or no response to instructions or external stimuli

Posturing spontaneous and active maintenance of a posture against gravity

Mannerisms odd, caricatures of normal actions

Stereotypy repetitive, abnormally frequent, non-goal-directed movements

Agitation

Grimacing keeping a fixed facial expression

Echolalia mimicking another's speech

Echopraxia mimicking another's movements.

Bush-Francis Catatonia Rating Scale

BUSH-FRANCIS CATATONIA RATING SCALE

Use presence or absence of items 1-14 for screening
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
2 = Constant motion, hyperkinetic without rest periods
3 = Full-blown catatonic excitement, endless frenzied motor activity

2. Immobility/stupor:

Extreme hypoactivity, immobile, minimally responsive to stimuli

0 = Absent
1 = Sits abnormally still, may interact briefly
2 = Virtually no interaction with external world
3 = Stuporous, non-reactive to painful stimuli

3. Mutism:

Verbally unresponsive or minimally responsive

0 = Absent
1 = Verbally unresponsive to majority of questions; incomprehensible whisper
2 = Speaks less than 20 words/ 5 min
3 = No speech

4. Staring:

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

0 = Absent
1 = Poor eye contact, repeatedly gazes less than 20 seconds between shifting of attention; decreased blinking
2 = Gaze held longer than 20 seconds, occasionally shifts attention
3 = Fixed gaze, non-reactive

5. Posturing/catalepsy:

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

0 = Absent
1 = Less than 1 minute
2 = Greater than one minute, less than 15 minutes
3 = Bizarre posture, or mundane maintained more than 15 minutes

6. Grimacing:

Maintenance of odd facial expressions

0 = Absent
1 = Less than 10 seconds
2 = Less than 1 minute
3 = Bizarre expression(s) or maintained more than 1 minute

7. Echopraxia/echolalia:

Mimicking of examiner's movements/speech

0 = Mimicking of examiner's movements/speech
1 = Occasional
2 = Frequent
3 = Constant

8. Stereotypy:

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

0 = Absent
1 = Occasional
2 = Frequent
3 = Constant

9. Mannerisms:

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

0 = Absent
1 = Occasional
2 = Frequent
3 = Constant

10. Verbigeration:

Repetition of phrases or sentences (like a scratched record).

0 = Absent
1 = Occasional
2 = Frequent
3 = Constant

11. Rigidity:

Maintenance of a rigid position despite efforts to be moved, exclude if cog-wheeling or tremor present.

0 = Absent
1 = Mild resistance
2 = Moderate
3 = Severe, cannot be repositioned

12. Negativism:

Apparently motiveless resistance to instructions or attempts to move/examine patient. Contrary behavior, does exact opposite of 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 repositioning of patient, patient offers initial resistance before allowing himself to be repositioned, similar to that of a bending candle.

0 = Absent
3 = Present

14. Withdrawal:

Refusal to eat, drink and/or make eye contact

0 = Absent
1 = Minimal PO intake/interaction for less than 1 day
2 = Minimal PO intake/interaction for more than 1 day
3 = No PO intake/interaction for 1 day or more

BUSH-FRANCIS CATATONIA RATING SCALE (CONT.)

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 only a facile explanation.

0 = Absent
1 = Occasional
2 = Frequent
3 = Constant or not restrictable

16. Automatic obedience:

Exaggerated cooperation with examiner's request or spontaneous continuation of movement requested.

0 = Absent
1 = Occasional
2 = Frequent
3 = Constant

17. Mitgehen:

"Anglepoise lamp" arm rising in response to light pressure of finger, despite instruction to the contrary.

0 = Absent
3 = Present

18. Gegenhalten:

Resistance to passive movement which is proportional to strength of the stimulus, appears automatic rather than willful.

0 = Absent
3 = Present

19. Ambitendency:

Patient appears motorically "stuck" in indecisive, hesitant movement.

0 = Absent
3 = Present

20. Grasp reflex:

Per neurological exam

0 = Absent
3 = Present

21. Perseveration:

Repeatedly returns to same topic or persists with movement.

0 = Absent
3 = Present

22. Combativeness:

Usually in an undirected manner, with no, or only a facile explanation afterwards.

0 = Absent
1 = Occasionally strikes out, low potential for injury
2 = Frequently strikes out, moderate potential for injury
3 = Serious danger to others

23. Autonomic abnormality:

Circle: temperature, BP, pulse, respiratory rate, diaphoresis

0 = Absent
1 = Abnormality of one parameter (excluding pre-existing hypertension)
2 = Abnormality of two parameters
3 = Abnormality of three or more parameters

TOTAL:_____

LETTER TO THE EDITOR

Should the term catatonia be explicitly included in the ICD-10 description of acute transient psychotic disorder F23.0?

JEANETT BAUER, METTE ØLLGAARD PEDERSEN, MARTIN BALSLEV JØRGENSEN

Bauer J, Øllgaard Pedersen M, Jørgensen MB. Should the term catatonia be explicitly included in the ICD-10 description of acute transient psychotic disorder F23.0? Nord J Psychiatry 2011;Early Online,1–2.

- The term catatonia was not applied in the description og psychomotor symptoms in acute polymorphous psychois F23.0

- A patient undoubtedly classified as suffering from ATPD according to ICD-10 (F23) and the psychomotor symptoms he exhibited were clearly those of catatonia.

- This is further supported by his response to benzodiazepines, which is known to be highly effective in catatonia especially in acute conditions. Moreover, his symptoms progressed towards a condition, which might be explained as malignant catatonia or neuroleptic malignant syndrome, conditions that are not easily separated.

Stuporous, excited and malignant catatonia

Stuporous or retarded

Kahlbaum syndrome
Delirious melancholia/depression
Akinetic mutism
Coma vigil
Benign stupor

Excited

Bell's mania
Oneirophrenia, oneiroid/oneiric state
catatonos raptus,
Delirium acutum,
Delirious mania per Kraepelin
Delirious catatonia

Malignant (+ autonomic instability and fever)

Malignant catatonia
Pernicious catatonia
Deadly catatonia
Lethal catatonia (Stauder's)

Neuroleptic malignant syndrome and serotoninergic syndrome

Prevalence

Prevalence rates of catatonia range from 6% - 38% among psychiatric inpatients

Mood disorders about 30% of cases (in particular mania or mixed states)

Of all Catatonia cases 25% due to medical condition
of which 70% are neurological
30% were associated with structural
25% CNS infection,
10% seizure disorder

Luchini et al 2015, Oldham et al 2015

BRAIN

EEG: Most catatonia tends to present with a normal EEG, but medical catatonia represents an exception to this rule. Over 80% of medical catatonia exhibit abnormal EEG findings, the most common being diffuse slowing.

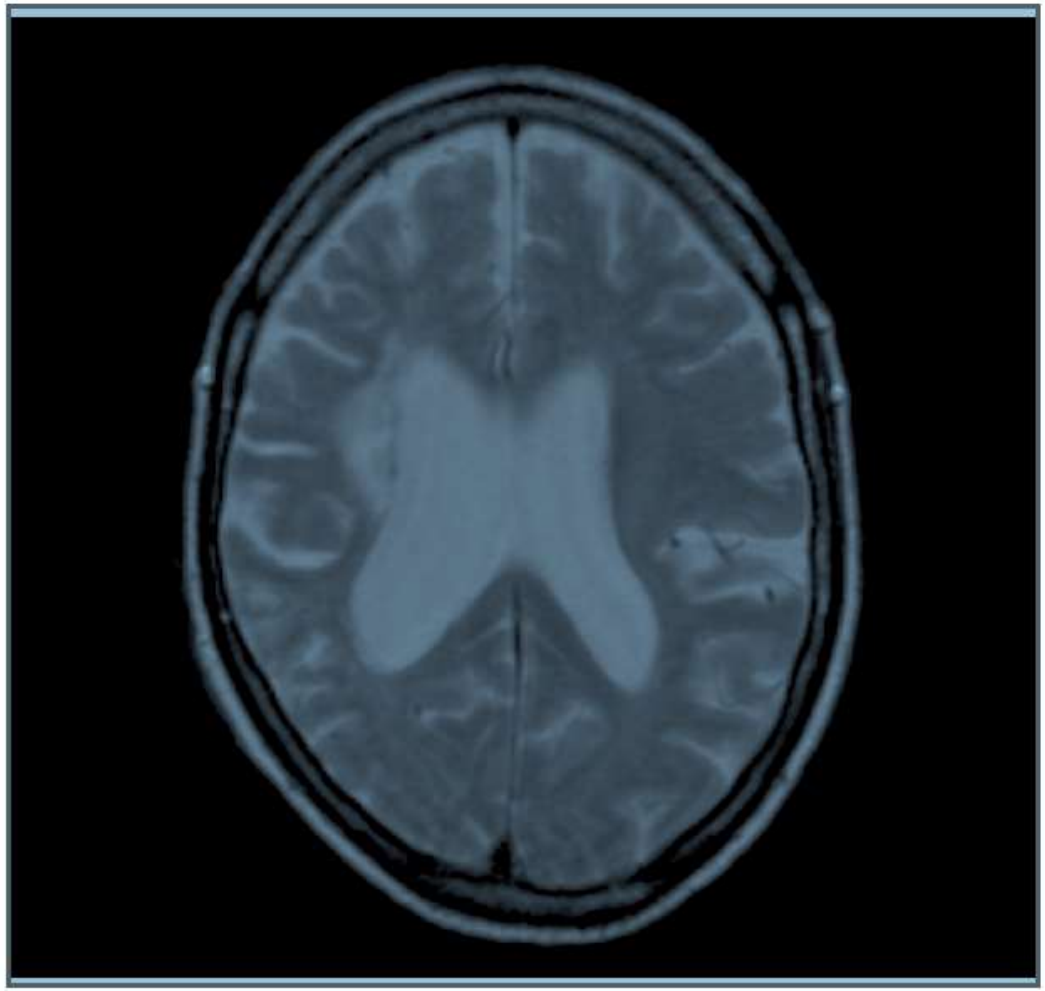
CT/MRI normal except if associated with structural damage

fMRI: disrupted sensorimotor network control during distinct functional states (Sambataro 2021).

SPECT/PET: Depends on brain tissue

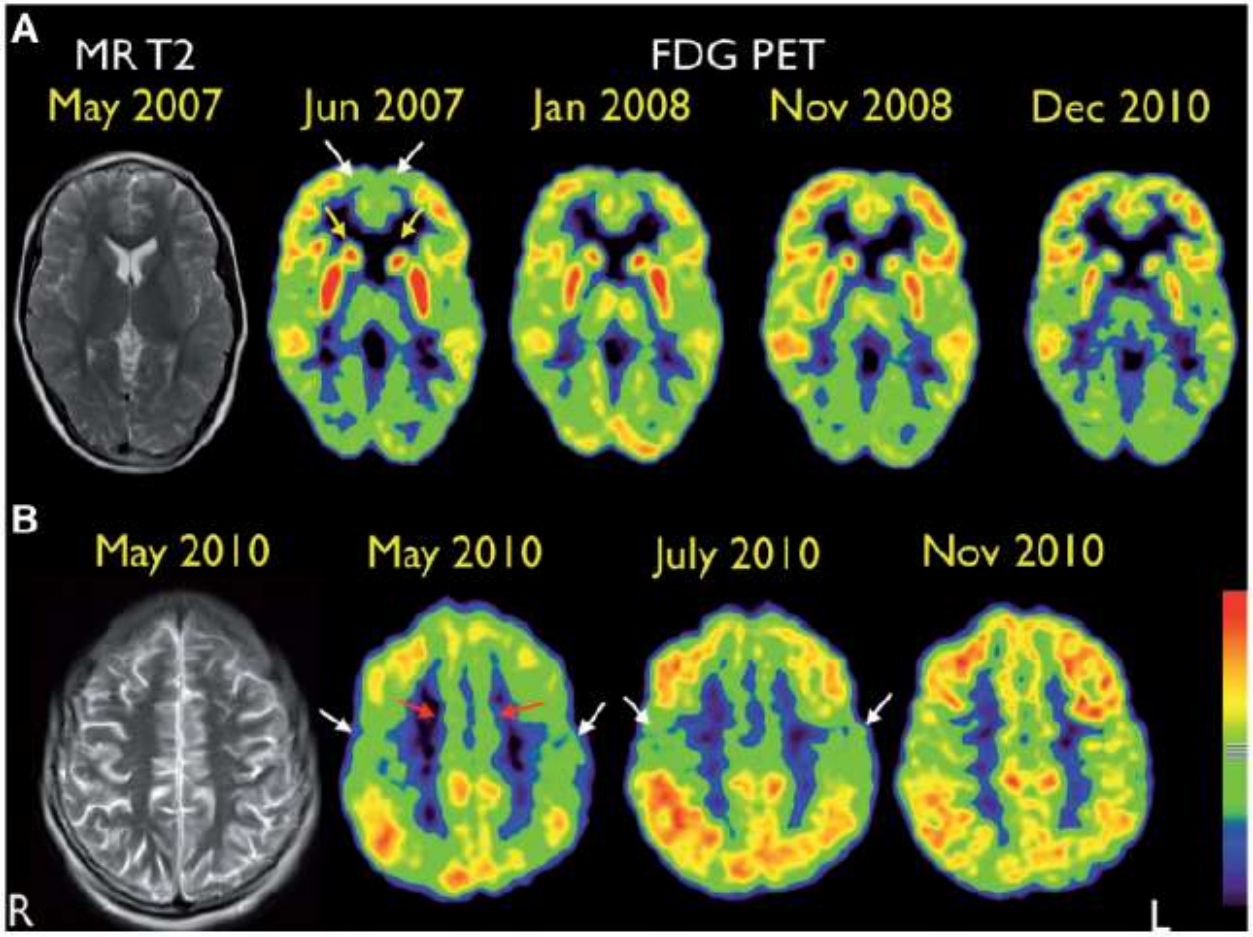
61 y male wit organic catatonia

Stroke in 2005 psychotic depression in 2006 catatonia in 2007



MR-cerebrum and SPECT-CBF: infarct seqv in right hemisphere

FIG. 1 Chronological presentation of co-registered transaxial and normal T_2-weighted MRI scans and consecutive FDG PET scans through the basal ganglia [Case 1, (**A**)] and just above the ventricles [Case 2 (**B**)]. (**A**) During psychosis (June 2007), FDG PET shows a bilateral metabolic increase in the striatum (yellow arrow) and a slight to moderate decrease in the anterior cingulate cortex (white arrow), which gradually resolves with treatment (January 2008–December 2010). (**B**) During catatonia (May and July 2010), there are marked and symmetrical reductions of metabolic activity in the mesial frontoparietal areas (red arrow), along the central sulci (white arrow) and in the occipital lobes, which shows almost complete normalization after ECT (November 2010). A similar pattern was found when comparing with a normal database [3] (see supplementary data, available at *Rheumatology* online).



Management of catatonia

1. Treat the underlying cause: Contributory medications should be avoided

2. Lorazepam: lorazepam 2 mg Lorazepam 2 mg can be scheduled thrice daily or increased in upward of 16 mg per day in divided doses catatonia related to chronic psychosis may represent a distinct clinical entity.

3. Avoid high-potency antipsychotics. They are not effective for catatonia. They can cause catatonic-like (i.e., extrapyramidal) side effects and may potentiate neuroleptic malignant syndrome. Atypical antipsychotics may improve nonmalignant catatonia but should be used with caution as an adjunct to a benzodiazepine or other catatonia-specific intervention.

4. Supportive measures: thromboembolism, pressure ulcers, contractures, nutrition and liquid balance, hyperthermia, pneumonia, oxygen

5. ECT: 90% of catatonia respond to ECT. If malignant catatonia ECT should be considered as an emergent intervention. If patients refuse to eat and are unable to provide self-care, ECT should be considered urgently.

6. Glutamate antagonists: memantine, amantadine, topiramate ? Dopamine agonist bromocriptine?

Modified from Oldham and Lee 2015

Catatonia ”organic”

Electroconvulsive Therapy for the Treatment of Organic Catatonia Due to Viral Encephalitis

Lekharah Shukla, MBBS, Janardhanan C. Narayanaswamy, MD, Srinath Gopinath, DPM, and Suresh Bada Math, MD, PGDMLE, PGDHRL

Abstract: Catatonia is a common presentation to psychiatric services in developing countries. Medical causes of catatonia are common and are difficult to treat. A 20 year old woman presented with an acute illness consisting of fever, delirium, perceptual abnormalities, and catatonic state. After trials with antiviral medications, benzodiazepines, and atypical antipsychotic medications, she was treated with 6 sessions of electroconvulsive therapy with complete recovery and no complications. Catatonia arising in the background of organic pathology can be treated on similar lines as in other psychiatric disorders. Electroconvulsive therapy can be a safe option that needs consideration in such cases after ruling out the contraindications.

Key Words: electroconvulsive therapy, catatonia, encephalitis

(*J ECT* 2012;28: e27–e28)

A computed tomographic scan of the brain revealed mild cerebral edema. The electroencephalogram demonstrated generalized slowing. Analysis of the cerebrospinal fluid leak showed 14 cells that were all monocytes, 46 mg/dL protein, and 70 mg/dL glucose. Immunoglobulin M titers of rubella and cytomegalovirus were positive. A diagnosis of viral encephalitis was made, and the patient was treated with antibiotics and antiviral medication (acyclovir) for 1 week with no improvement in the neurology ward. The patient continued to be mute, actively negativistic, and stuporous. She had rigidity, posturing, and urinary incontinence. The patient was subsequently referred to the psychiatric ward because of prominent behavioral symptoms and agitation. A diagnosis of organic catatonia was considered. A score of 11 in the Bush Francis Catatonia Rating Scale was observed. She was started on quetiapine up to 250 mg/d and

Rheumatology 2012;51:193–195
doi:10.1093/rheumatology/ker287
Advance Access publication 7 October 2011

Fluorodeoxyglucose positron emission tomography in juvenile systemic lupus erythematosus with psychiatric manifestations: relation to psychopathology and treatment response in two cases

Anders Jørgensen[1], Ian Law[2], Susan Nielsen[3] and Martin B. Jørgensen[1]

Br Med J

Typhoid catatonia responsive to ECT

WILLIAM R BREAKEY, A K KALA

British Medical Journal, 1977, 2, 357-359

Summary

Twelve patients with typhoid fever presented with a catatonic syndrome that persisted after other signs of the fever had disappeared. The syndrome was distinct from the delirium seen in typhoid fever and did not have the characteristics of an affective or schizophrenic illness. Electric convulsion therapy produced rapid and lasting improvement.

common complication of typhoid fever, and we report 12 cases seen in India.

Present series

During 1974-6, 238 patients were admitted to the Christian Medical College Hospital with typhoid fever. Of these, 45 showed gross behavioural disturbances, which in 32 cases necessitated psychiatric examination. Though most of these patients had typical delirium, 12 presented with a catatonic syndrome that persisted after the fever had resolved and their physical condition had returned to normal. These 12 patients are the subject of this report.

Wachtel 2018

REVIEW

CURRENT OPINION

Electroconvulsive therapy for self-injurious behaviour in autism spectrum disorders: recognizing catatonia is key

Lee Elizabeth Wachtel[a], Edward Shorter[b], and Max Fink[c]

KEY POINTS

- Self-injurious behaviour in ASDs is a serious clinical problem, and may be resistant to behavioural and psychopharmacological approaches. Recent reports have conceptualized some intractable self-injury within the diagnostic framework of agitated catatonia.
- Multiple reports have demonstrated a significant reduction in self-injurious behaviour in those diagnosed along the catatonia spectrum using electroconvulsive therapy.
- Electroconvulsive therapy is a therapeutic medical intervention distinct from contingent electric shock used in behavioural psychology as an aversive behavioural treatment procedure.
- Patient benefit from ECT may be profound in terms of reduction in self-injury, and improvement in overall psychosocial functioning.

Purpose of review
Self-injurious behaviour (SIB) is a devastating condition frequently encountered in autism spectrum disorders (ASDs) that can lead to dangerous tissue injury and profound psychosocial difficulty. An increasing number of reports over the past decade have demonstrated the swift and well tolerated resolution of intractable SIB with electroconvulsive therapy (ECT) when psychopharmacological and behavioural interventions are ineffective. The current article provides a review of the salient literature, including the conceptualization of repetitive self-injury along the catatonia spectrum, and further clarifies the critical distinction between ECT and contingent electric shock.

Recent findings
We searched electronically for literature regarding ECT for self-injurious behaviour from 1982 to present, as the first known report was published in 1982. Eleven reports were identified that presented ECT in the resolution of self-injury in autistic or intellectually disabled patients, and another five reports discussed such in typically developing individuals. These reports and related literature present such self-injury along the spectrum of agitated catatonia, with subsequent implications for ECT.

Summary
Intractable self-injury remains a significant challenge in ASDs, especially when patients do not respond adequately to behavioural and psychopharmacological interventions. ECT is well tolerated and efficacious treatment for catatonia, and can confer marked reduction in SIB along the agitated catatonia spectrum.

Anti NMDAr encephalitis og ECT

Anti-N-Methyl D-Aspartate Receptor Encephalitis and Electroconvulsive Therapy

Literature Review and Future Directions

Yasas Chandra Tanguturi, MBBS, MPH, Allyson Witters Cundiff, MD, Catherine Fuchs, MD*

KEYWORDS

• Electroconvulsive therapy • ECT • Anti-NMDA encephalitis
• Autoimmune encephalitis • Catatonia

KEY POINTS

- Catatonia is a syndrome, with one potential cause being an autoimmune encephalitis such as anti-N-methyl D-aspartate receptor encephalitis.
- Benzodiazepines (BZDs) are first-line treatment for symptoms of the syndrome of catatonia.
- Electroconvulsive therapy (ECT) should be considered if patients have autonomic instability or if they are not responding well to BZDs in combination with immunotherapy. The use of BZDs + ECT may be synergistic in efficacy.
- ECT is considered a safe intervention with appropriate assessment and management, although the exact mechanism of action of ECT is unclear.
- Treatment should be a multidisciplinary collaborative effort to include assessment and treatment of the autoimmune process.

Luchini et al. 2015 ECT in Catatonia 8 open observational studies including at least 10 patients with different form of catatonia

Table 1 Studies that investigate efficacy of electroconvulsive therapy in catatonia (number of patients receiving electroconvulsive therapy ≥ 10)

Ref.	Sample (n)	Patients receiving ECT n (%)	Diagnosis	Design	ECT technique	Outcome measures	Results	Variables associated with response
Unal *et al*[20], 2013	57	57 (100)	63% Mood disorders 29% psychotic disorders (including schizophrenia) 3.5% pts mental retardation 3.5% pts without psychiatric disorders	Retrospective All pts. received ECT in combination with oral BZDs	BL (bifrontal)	CGI, HDRS, YMRS, PANSS	Response = 100%	Not assessed
Tuerlings *et al*[16], 2010	34	34 (100)	59% mood disorders 77% schizophrenia and other psychotic disorders 37% somatic, toxic, post-traumatic stress disorder, mental retardation	Retrospective	Non specified	No standard diagnostic instruments or catatonia scales	58% pts treated with BZDs and/or ECT had clinically complete remission. 50% pts treated with ECT after unsuccessful medication trials recovered completely	Responders: Autonomic dysregulation Non-responders: Initial treatment with amantadine, bromocriptine and dantrolene More comorbid disorders
Van Waarde *et al*[19], 2010	27	27 (100)	48% mood disorders 44% psychotic disorder (including schizophrenia) 19% others (alcohol/substance abuse, mental retardation)	Retrospective	BL (bifronto-temporal) or UL (according to d'Elia)	Response defined as CGI < 2	Response = 59%	Responders: Younger age Autonomic dysregulation at baseline (especially higher body temperature) Daily ECT during the first treatment week Longer duration of motor and EEG seizure activity at the final ECT session Less morbidity in the year after ECT
Ravvendranathan *et al*[21], 2012	63	63 (100)	41% mood disorders, 49% psychotic disorders (including schizophrenia) 6% idiopathic catatonia	Retrospective	BL (bitemporal)	Response = complete resolutions of symptoms and/or BFCRS = 0	Response = 89%	The Authors divide the responders in faster (≤ 4 sessions) and slower (> 5 sessions) Faster responders: Lower duration of catatonia Greater severity of BFCRS Lesser electrical charge used overall Shorter duration of inpatient stay Waxy flexibility and gegenhalten. Lower responders: echophenomena
England *et al*[22], 2011	25	12 (48)	Total sample: 36% bipolar disorder 32% pts with psychosis NOS 4% depressive episode and anxiety disorder 16% schizophrenia 8% without previous psychiatric history	Retrospective	BL	BFCRS, clinical evaluation	83% pts treated with ECT definite beneficial effects > BZDs, APs, MS, ADs	Not assessed
Hatta *et al*[17], 2007	50	17 (34)	Total sample: 34% mood disorders 46% schizophrenia and other psychotic disorder 20% medical condition	Observational study I step: BZDs or ECT II step: APs (RIS or HAL) or ECT III step: CPZ or ECT IV step: ECT	BL (bitemporal)	"Partial response": disappearance of one or more catatonic symptoms; "complete response": disappearance of all catatonic symptoms	(Cumulative) ECT 100% > CPZ 68% > RIS 26% > HAL 16%	Not assessed
Dutt *et al*[23], 2011	51	42 (82)	75% psychotic disorders (including schizophrenia) 14% mood disorders 8% organic brain syndromes	Retrospective	Unspecified	Reduction of BFCRS assesses the response	Response = 100%	Not assessed
Rohland *et al*[18], 1999	22	22 (100)	59% mood disorders 32% schizophrenia and schizoaffective disorder 9% organic mental disorder	Retrospective	BL (bitemporal)	Response to ECT was assessed by not meeting Kahlbaum and Rosebush criteria for catatonia. Then, the number of single signs and symptoms prior and after ECT is another parameter	Response = 93%	Trend toward a better response in affective than psychotic pts (non statistically significant)
Medda *et al* (manuscript in preparation)	26	26 (100)	100% bipolar disorder	Observational	BL (bitemporal)	Response = CGI < 2	Response = 81%	Non-responders: Older age at onset of mood disorders Lower number of mood episodes Higher BFCRS total score at baseline Less psychotic symptoms Higher rate of past treatment with anticholinergics and dopamine agonists and lower rate of past treatment with typical antipsychotics

Retrospective chart reviews report response rates in catatonic patients ranging from 80%-100% - perhaps less in primary psychotic disorders

Eur Arch Psychiatry Clin Neurosci (2018) 268:675–687
https://doi.org/10.1007/s00406-017-0819-5

ORIGINAL PAPER

Is electroconvulsive therapy an evidence-based treatment for catatonia? A systematic review and meta-analysis

Arnaud Leroy[1] · Florian Naudet[2] · Guillaume Vaiva[1] · Andrew Francis[3] · Pierre Thomas[1] · Ali Amad[1,4]

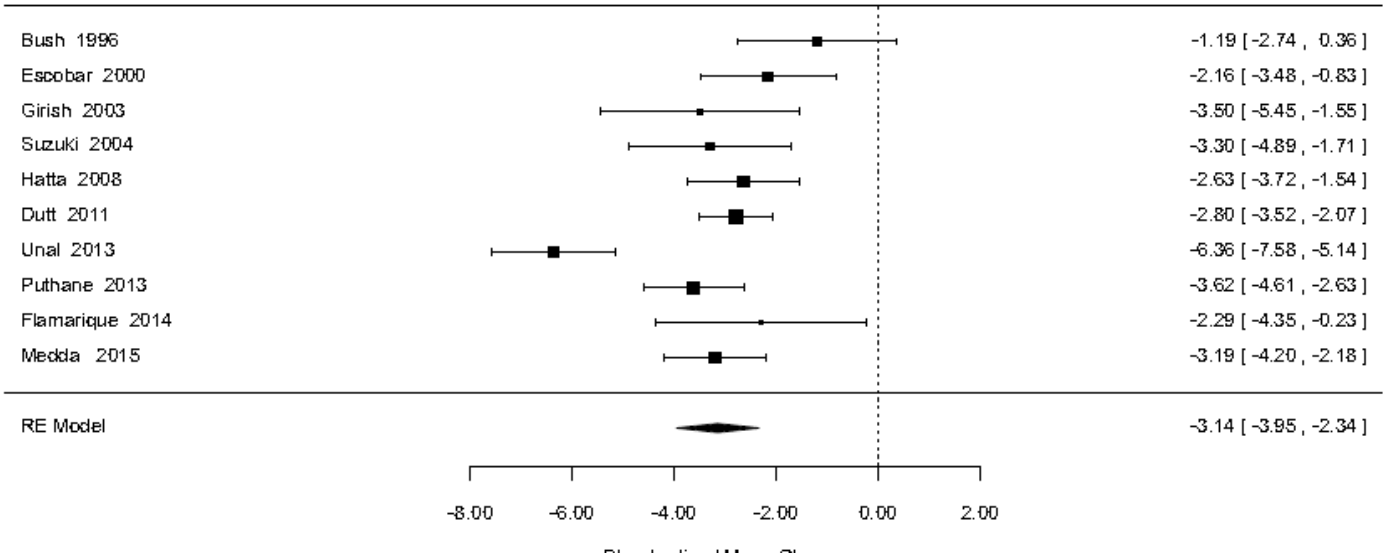
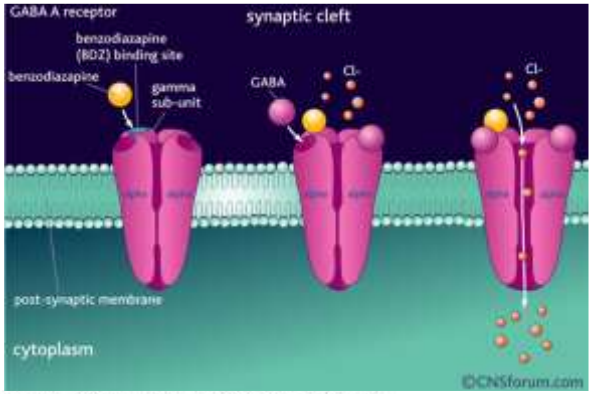


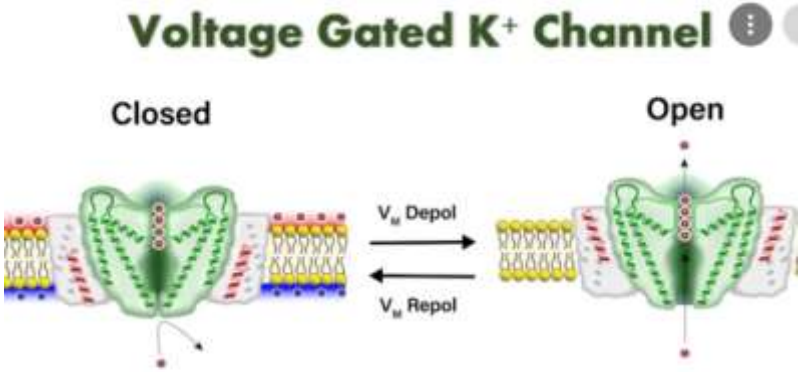
Fig. 4 Forest plot for pre-post differences in severity after ECT treatment in catatonic patients. Results are presented with 95% confidence interval. Pre-post correlation = 0.10. $I^2 = 76.6\%$, Q test: $p < 0.001$

- The results of this systematic review revealed that ECT in catatonia is still an understudied treatment and that published studies, for various reasons, generally do not satisfy rigorous criteria for efficacy (e.g., difference between ECT and placebo) or comparative effectiveness (e.g., ECT vs. an active comparator treatment modality). ….

- However, our data suggest an important, robust and consistent mprovement in catatonic symptoms after ECT across several studies

Repolarizing inhibitory membrane receptors are upregulated in ECT





Pharmacological Research Communications, Vol. 18, No. 6, 1986 587

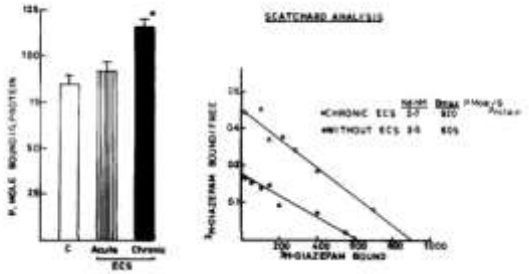
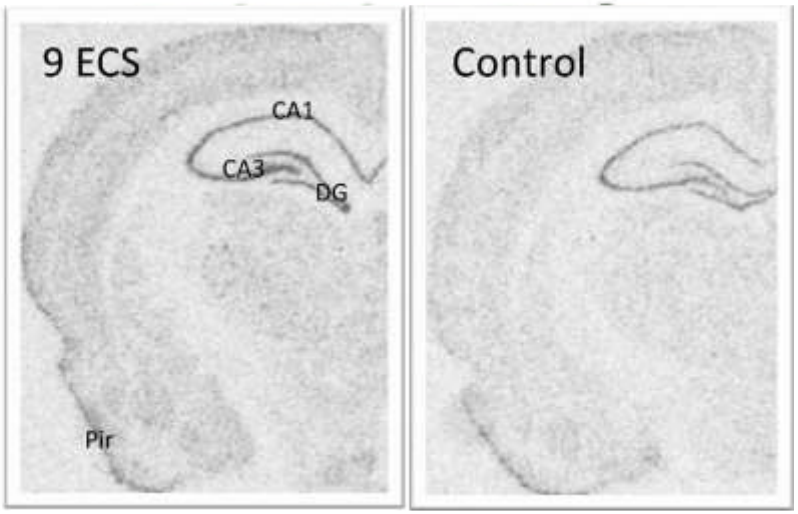


Figure 3: Shows ^{3}H-diazepam binding in control (hollow bar), acute (lined bar) and chronic (solid bar) ECS treated rats. Astrisk indicates a significant ($P < 0.01$) increase in the binding from the control group. Scatchard analysis indicates a decrease in K_D and increase in Bmax by chronic ECS treatment. Lines are drawn using linear regression analysis. The values are mean of three experiments. Detailed procedure of Scatchard analysis is given in the text.

$K_V7.2$



Gulati et al 1986

Hjæresen et al. 2012

ECT Technicalities

General consensus that bitemporal placement is the most effective

More frequent ECT sessions than in major depression and is generally given three times per week on alternating days. However, clinical urgency may necessitate daily treatments until the patient is more stable.

Succinylcholine may be avoided due to increased risk of severe hyperkalemia (more chronic cases?)

Discontinue benzodiazepine treatment just prior to ECT, whereas others recommend continuing benzodiazepines

In a study, lorazepam occasionally shortened seizure duration below the conventional minimum (25 sec motor convulsion). In these cases the stimulus energy was increased at the following ECT session. These shortened seizures did not appear to diminish the beneficial response of our patients to ECT. In all cases, the last dose of lorazepam was administered at least 12 hours prior to treatment. The short half-life of lorazepam and the absence of active metabolites may minimize possible antagonism of ECT (Greenberg and Pettinati 1993).

Fink M, Taylor MA. Catatonia: a separate category for DSM-IV? Integrative Psychiatry 1991;7:2–10.