Epilepsy & Behavior 159 (2024) 109983

Contents lists available at ScienceDirect

Epilepsy & Behavior

journal homepage: www.elsevier.com/locate/yebeh

Catatonia and epilepsy: An underappreciated relationship

Jonathan P. Rogers [a,*], Simon Shorvon [b], James Luccarelli [c,d]

[a] Division of Psychiatry, University College London, London, UK
[b] Queen Square Institute of Neurology, University College London, London, UK
[c] Department of Psychiatry, Harvard Medical School, Boston, USA
[d] Department of Psychiatry, Massachusetts General Hospital, Boston, USA

ARTICLE INFO

Keywords:
Catatonia
Epilepsy
Seizure
Electroencephalogram
Electroconvulsive therapy

ABSTRACT

Catatonia is currently conceived in the major diagnostic manuals as a syndrome with a range of possible psychiatric and general medical underlying conditions. It features diverse clinical signs, spanning motor, verbal and behavioural domains and including stupor, catalepsy, mutism, echolalia, negativism and withdrawal.

The existing literature suggests that seizure activity may underlie catatonia in approximately 2% of cases. There are three possible temporal relationships between catatonia and seizure activity: (1) ictal catatonia, in which catatonia is a presentation of non-convulsive status epilepticus; (2) postictal catatonia, in which catatonia follows a seizure, and (3) interictal catatonia, in which catatonia and seizures occur in the same individual without any clear temporal relationship between them. Electroencephalographic (EEG) abnormalities are common in catatonia, even in those cases with a presumed primary psychiatric origin, and often consist of generalised background slowing. Paradoxically, electroconvulsive therapy is an effective treatment for catatonia.

There are several converging pieces of evidence suggesting that there may be underlying seizure activity in more cases of catatonia than has hitherto been recognised, though identification of these seizures may require intracranial EEG recording.

1. Introduction

Catatonia is a neuropsychiatric disorder that has historically been viewed through multiple lenses, including that of a movement disorder, a neurodegenerative disorder, a neurodevelopmental disorder, a form of schizophrenia, and a psychomotor disorder [1–6]. A relationship to epilepsy has been suggested in a number of cases in the literature, but it has received little theoretical attention.

This review approaches this relationship from several perspectives. First, we provide a brief introduction to catatonia, before outlining the established temporal relationships in which catatonia and seizures may be comorbid. We then examine the evidence for electroencephalographic (EEG) findings in catatonia. Having reviewed the myriad ways in which catatonia and epilepsy may exhibit a rather dysfunctional relationship, we outline the paradoxical potential for iatrogenic seizures to effectively treat catatonia. We then critically examine the evidence for a new lens through which to view catatonia: that catatonia may be a seizure disorder in some patients.

To conduct the review, we searched PubMed from inception to 2024 using keywords 'catatonia' and 'epilepsy', which generated 148 articles. We identified relevant themes and then conducted further secondary searches on these themes.

2. The conventional understanding of catatonia

Catatonia as a term was coined by the German psychiatrist Kahl Ludwig Kahlbaum [7], but historical examples that likely correspond to the same phenomenon have been described since antiquity in Graeco-Roman literature [8] and in India as early as the 1st century CE [9]. Kahlbaum's aim in delineating catatonia as a distinct disorder was to emulate the successful clinico-pathological correlation that had been achieved with neurosyphilis [10].

The great psychiatric nosologist of the early 20th century Emil Kraepelin incorporated catatonia into his concept of *dementia praecox*, which was inherited by subsequent psychiatric classifications in the form of catatonic schizophrenia. It was in this form that the major diagnostic manuals – the *International Classification of Diseases* from the World Health Organization and the *Diagnostic and Statistical Manual of*

Abbreviations: ECT, electroconvulsive therapy; EEG, electroencephalogram; NMDA, N-Methyl-D-aspartate; SPECT, single-photon emission computed tomography.

* Corresponding author at: UCL Division of Psychiatry, Maple House, 149 Tottenham Court Rd, Bloomsbury, London W1T 7NF, UK.
E-mail address: jonathan.rogers@ucl.ac.uk (J.P. Rogers).

https://doi.org/10.1016/j.yebeh.2024.109983
Received 14 June 2024; Received in revised form 1 August 2024; Accepted 4 August 2024
Available online 24 August 2024

Mental Disorders from the American Psychiatric Association – encapsulated catatonia until recently [11].

Since the 1970s, however, evidence has emerged that catatonic features are not exclusive to schizophrenia and in fact occur in a wide range of psychiatric and general medical conditions [12,13]. Today, catatonia is conceived as a syndrome that may appear in a diverse selection of conditions, ranging from depression, schizophrenia and autism to autoimmune encephalitis, neurodegenerative disorders and drug-induced conditions [14–18].

What unites this syndrome is a collection of rather diverse clinical signs indicative of reduced, increased or abnormal psychomotor activity [19]. These signs may be motor (e.g. catalepsy, echopraxia, ambitendency, stupor), verbal (e.g. mutism, verbigeration, echolalia) or behavioural (e.g. staring, negativism, withdrawal) [20]. The other unifying feature is the response to treatment with benzodiazepines or electroconvulsive therapy, which is often rapid and dramatic, though not entirely ubiquitous [21].

The neurochemistry of catatonia, however, remains obscure. Neuroimaging findings have largely been diffuse and non-specific [22,23], although an intriguing result from an iomazenil single photon emission tomography (SPECT) study suggested a reduced density of GABA-A receptors in the left sensorimotor cortex [24]. However, evidence implicating the role of dopamine [25], glutamate [17] and acetylcholine [26] suggests that the pathophysiological basis is unlikely to be as simple as involving a single neurotransmitter.

3. Temporal relationships between catatonia and seizures

In a systematic review, Oldham estimated that approximately 20 % of cases of catatonia have an underlying general medical condition, of which 9.3 % are thought to be due to epilepsy [14], suggesting that approximately 2 % of all cases of catatonia are seizure-related. As is the case with epileptic psychoses [27–29], catatonia may occur in three temporal relationships to seizures: ictal catatonia, postictal catatonia and interictal catatonia, as illustrated in Fig. 1.

Ictal catatonia is a manifestation of non-convulsive status epilepticus. A systematic review identified 66 such cases with a mean age of 42 years and an approximately equal sex ratio [30]. The authors emphasised the importance of making this diagnosis, but they highlighted several difficulties in doing so. Specifically, only 38 % had any prior history of seizures, only 29 % exhibited subtle ictal phenomena during the catatonic episodes and most responded to benzodiazepines, which is diagnostically non-specific in catatonia [31,32]. Common semiology of ictal catatonia includes mutism, stupor, staring, catalepsy, rigidity and negativism [33], all of which are frequent in catatonia samples of mixed aetiology [34,35].

Postictal catatonia has been described less frequently. Catatonia has been reported as starting on the same day as the seizure [36,37] and there are sometimes multiple episodes [37,38]. EEG has been reported to be normal [37] or to show generalised background slowing [36,38]. High doses of lorazepam are sometimes required [36,37] and ECT has successfully been used [38]. The pathophysiology of postictal catatonia is not understood, but by analogy with epileptic psychosis, it could be due to ongoing seizure activity not visible on scalp EEG. Postictal behavioural arrest with features suggestive of catatonia has been induced in rats by electrically or chemically induced seizures [39].

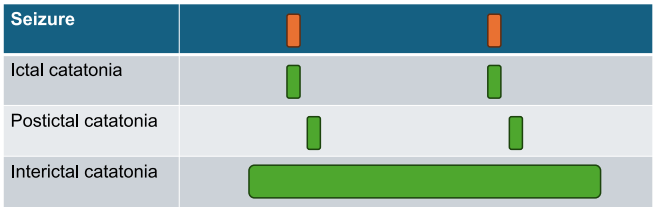

Fig. 1. Illustrative temporal relationships between catatonic episodes and seizures.

Interictal catatonia can be defined as catatonia in an individual with epilepsy without any clearcut temporal relationship between catatonic signs and seizures. Table 1 provides the prevalences of catatonia and epilepsy in various conditions in which both appear at what is likely to be a higher-than-expected prevalence. It also worth noting conditions in which seizures are directly provoked during an acute illness, such as NMDA receptor encephalitis, where seizures have been reported in 76 % and catatonia in 88 % [40]. In such conditions it is possible that the seizures and catatonia are caused by the underlying pathology and have no causal relationship to each other. The causal relationship in interictal catatonia without a clearcut precipitant is unknown. It is possible that there is a long-term effect of seizures on the risk of catatonia through neuronal damage, as has been speculated to occur in postictal psychosis [41]. However, given that there are some individuals with the conditions in Table 1 who experience catatonia without seizures, it is perhaps more likely that catatonia and seizures result from shared neurobiological dysfunction.

4. EEG findings in catatonia

EEG and cortical movement potentials in catatonia have been studied by a variety of methods [49,50]. In a systematic review of 355 studies, Hosseini et al identified EEG reports from 707 patients with catatonia [51]. This study suggested that focal abnormalities, epileptiform discharges, evidence of status epilepticus and features of limbic encephalitis – while rare in catatonia – were seen almost always in catatonia due to general medical conditions such as autoimmune encephalitis rather than in catatonia in primary psychiatric conditions. Generalised background slowing was not useful in differential diagnosis and occurred in 23 % of the primary psychiatric cases.

If the EEG is sometimes abnormal in catatonia, it is important to establish whether this is a trait or a state. That is, do EEG abnormalities represent an enduring latent vulnerability to catatonia, or are they transient phenomena that are closely linked to the clinical presentation? In catatonia secondary to an epileptic seizure, it is to be expected that EEG changes represent a state and are closely linked to the clinical presentation, but even in this scenario the evidence is not straightforward. In a series of three cases of catatonia in the context of status epilepticus, catatonic stupor persisted after electroencephalographic resolution of the seizure, subsequently resolving with either a further benzodiazepine dose or electroconvulsive therapy (ECT) [52]. In a classic study by Gjessing and colleagues, three individuals with periodic catatonia showed an increase in the alpha frequency during phases of stupor [53]. Several studies have examined cases where generalised slowing was observed during the catatonia. In one study, there were 6

Table 1
Prevalences of epilepsy and catatonia in selected conditions, illustrating high comorbidity.

Condition	Prevalence of epilepsy	Prevalence of catatonia
Autism	10 % [42] [a]	10.4 % [16] [c]
Intellectual disability	22.2 % [43] [a]	Unknown but high prevalence in some conditions causing intellectual disability
Down's syndrome	12.4 % [43] [a]	Many cases reported, particularly in Down's syndrome regression disorder [44–46]
Schizophrenia	0.35 – 7.30 % [47] [b]	9.8 % [48] [a]

[a] Meta-analytic estimate based on studies using various definitions of prevalence.
[b] Range based on findings of a narrative review using various definitions of prevalence.
[c] Meta-analytic estimate based mainly on studies using point-prevalence.

patients who had an EEG during and after catatonia, all of whom showed some improvement, mostly in generalised slowing, but there was not complete normalisation in all patients [32]. Other small reports have also described generalised slowing during catatonia (or transition to it) with normal traces before or afterwards [54,55]. Perhaps most convincingly, in one case report, investigators administered intravenous diazepam during a catatonic state with continuous EEG monitoring, observing marked diminishing of high-amplitude slow activity as the diazepam took effect [56]. However, a recent series including cases of catatonia with comorbid delirium and generalised periodic discharges who had an EEG after treatment found that the EEG of only one had normalised after clinical improvement of the catatonia [57]. Overall, the evidence on the persistence of EEG abnormalities in catatonia is inconclusive and may depend on the presence of comorbid conditions.

Beyond clinical EEG, some quantitative EEG techniques have also been employed in this field. In a study of 10 patients with catatonia, who were compared to psychiatric and healthy controls, participants were asked to extend their right index finger when they wanted to. EEG monitoring found that late *Bereitschaftspotentials* were delayed in the catatonic group compared to both control groups [49].

5. Catatonia as a seizure disorder

We have established already that catatonia – in rare instances – has been shown to be the result of a seizure. Is it possible that catatonia is in fact more often intrinsically a seizure disorder? At a first glance, given that most scalp EEGs of patients with catatonia do not show obvious seizure activity, this might seem rather implausible, but epileptic activity on intracranial EEG has sometimes been shown in the absence of a scalp EEG correlate [58].

If it is possible then that a condition may feature seizures without a clear scalp EEG correlate, is it probable in catatonia? There are several arguments that may support this proposition. Firstly, there are many examples of epileptic activity demonstrated by intracranial EEG from deep-brain regions without any scalp EEG abnormalities, and which are associated with behavioural change and psychiatric features [27]. Secondly, the semiology of catatonia can be indistinguishable from some presentations of non-convulsive status epilepticus, as some cases of ictal catatonia have demonstrated. It is not only the motor features of catatonia which are shared with non-convulsive status epilepticus, but also affective features such as extreme anxiety or fear [34,59,60]. Thirdly, both catatonia and seizures can respond rapidly to benzodiazepines – catatonia is, in fact, highly unusual among psychiatric disorders in often responding dramatically within a few minutes to intravenous lorazepam [61]. Similarly, catatonia can be precipitated by the withdrawal of antiseizure medications, such as benzodiazepines [62,63] and gabapentin [64,65]. Finally, there are a wide range of conditions in which seizures and catatonia both occur, at least raising the possibility that the pathophysiology may be similar. There are also reports of individuals with catatonia with seizures that were not readily apparent. In one case, an individual with a diagnosis of catatonic schizophrenia deteriorated, exhibiting worsening psychosis, impaired memory and stereotyped movements. While this patient had a normal routine surface EEG, continuous electroencephalographic monitoring revealed seizures lasting up to two hours, which responded to treatment with antiseizure medications [66]. A further case series of three patients with an apparently psychiatric catatonia, on which clinical seizure activity then became superimposed, has also been reported [52]. In another study, 29 patients with catatonia were followed up to ascertain how many developed seizures during the catatonic episode, finding 4 patients (13.8 %) with an electroclinical seizure diagnosis [67].

There is also some overlap in the neurobiology of catatonia and epilepsy, although there remain substantial gaps in the understanding of both. Structural imaging studies in catatonia have found alterations in white matter tracts connecting regions including the prefrontal cortex, primary motor cortex, supplementary motor area and basal ganglia [68–72]. Functional MRI has identified alterations across a wide range of brain networks in catatonia [73], while a range of theories consider abnormalities in GABA, glutamate and dopamine to be implicated [72]. Glutamatergic and GABA-ergic dysfunction are also thought to be central to the pathophysiology of epilepsy, indicating an imbalance between excitatory and inhibitory systems [74]. While epilepsy can be highly focal, generalised-onset seizures are thought to be sustained by the thalamo-cortical networks that are hypothesised to be relevant to catatonia [75,76].

What about the counterarguments? Importantly, clozapine, which is probably effective in chronic catatonia in schizophrenia [77], is the most proconvulsant antipsychotic [78,79]. Moreover, there are cases of catatonia where a psychogenic formulation seems overwhelmingly compelling: take for instance the case of a man whose catatonic episodes always occur during conflict with his parents [80], the school girl who is assaulted by a family member then chased by a wild dog whereupon she adopts a foetal position for days on end [81], or the man who develops catatonia after inadvertently amputating his hand with a malfunctioning chop saw [82]. It is hard to speculate a convincing epileptic origin for these cases.

6. Induced seizures as a treatment for catatonia

Hitherto our discussion has focussed on the potential for seizures and abnormal electrical activity to cause catatonia. However, paradoxically, one of the most effective treatments for catatonia relies on the induction of seizures. László Meduna is credited as first person to use seizures to treat psychiatric disorders in the early 1930s. Using camphor or cardiazol for seizure induction, out of the first 15 patients with schizophrenia treated by Meduna, 13 had catatonia and 5 of these improved in an era when there was no other effective treatment [83].

Chemically induced seizures were soon supplanted by electrically induced seizures in electroconvulsive therapy (ECT). The first patient treated with ECT by its inventors, Bini and Cerletti, also had catatonia [84] and since then there have been numerous observational studies supporting the use of ECT in catatonia [85,86]. Perhaps the most convincing evidence comes from a small randomised controlled trial, in which 18 inpatients with catatonia who had failed to respond to a 5-day course of lorazepam were randomised either to ECT plus an oral placebo or to sham ECT plus the oral antipsychotic medication risperidone [87]. After 3 weeks, the mean Bush-Francis Catatonia Rating Scale fell by 7 points in the risperidone group and by 12 points (down to less than 1 point) in the ECT group. ECT is a recommended treatment for catatonia in various clinical guidelines [21,88–91].

Given that electrical seizure induction is such an effective treatment for catatonia, it may seem counterintuitive to suggest that catatonia may sometimes be due to seizures. However, rather paradoxically, ECT has also been found to be an effective treatment in refractory status epilepticus with 11 of 19 patients in the literature responding at least partially [92].

7. Conclusion

Catatonia and epilepsy are epidemiologically linked, featuring in several of the same disorders. A seizure can present with a catatonic phenotype or may be followed by a catatonic episode. Paradoxically, however, pharmacologically or electrically induced seizures are effective treatments for catatonia. Surface EEG in catatonia is sometimes abnormal, and it is possible that some cases of catatonia have occult electrical seizure activity. This is not an adequate explanation for all cases of catatonia, but it is possible that underlying seizure activity accounts for more cases than those for which it is currently given credit.

Author contributions

JPR conceived the manuscript with assistance from SS and JL. JPR

drafted the manuscript, which was edited for important intellectual content by SS and JL.

Funding

JPR is funded by an NIHR Clinical Lectureship. This work was supported by the National Institute of Mental Health (T32MH112485, JL). No funders had no role in devising the content of the manuscript or in the decision to publish.

JL receives funding from Harvard Medical School Dupont Warren Fellowship and Livingston Awards, the Rappaport Foundation, and the Foundation for Prader-Willi Research. He has received equity and consulting fees from Revival Therapeutics, Inc.

CRediT authorship contribution statement

Jonathan P. Rogers: Writing – original draft, Conceptualization. **Simon Shorvon:** Writing – review & editing, Conceptualization. **James Luccarelli:** Writing – review & editing, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Wijemanne S, Jankovic J. Movement disorders in catatonia. J Neurol Neurosurg Psychiatry 2015;86:825–32. https://doi.org/10.1136/jnnp-2014-309098.
[2] Northoff G, Koch A, Wenke J, Eckert J, Böker H, Pflug B, et al. Catatonia as a psychomotor syndrome: A rating scale and extrapyramidal motor symptoms. Mov Disord 1999;14:404–16. https://doi.org/10.1002/1531-8257(199905)14:3<404::AID-MDS1004>3.0.CO;2-5.
[3] Kendler KS. The Development of Kraepelin's Mature Diagnostic Concept of Catatonic Dementia Praecox: A Close Reading of Relevant Texts. Schizophr Bull 2019. https://doi.org/10.1093/schbul/sbz101.
[4] Rogers D. Catatonia: a contemporary approach. J Neuropsychiatry Clin Neurosci 1991;3:334–40. https://doi.org/10.1176/jnp.3.3.334.
[5] Moore S, Amatya DN, Chu MM, Besterman AD. Catatonia in autism and other neurodevelopmental disabilities: a state-of-the-art review. Npj Mental Health Res 2022;1:1–10. https://doi.org/10.1038/s44184-022-00012-9.
[6] Consoli A, Raffin M, Laurent C, Bodeau N, Campion D, Amoura Z, et al. Medical and developmental risk factors of catatonia in children and adolescents: A prospective case–control study. Schizophr Res 2012;137:151–8. https://doi.org/10.1016/j.schres.2012.02.012.
[7] Kahlbaum KL. Die Katatonie : oder das Spannungsirresein, eine klinische Form psychischer Krankheit. 1874.
[8] Berrios GE. Stupor revisited. Compr Psychiatry 1981;22:466–78. https://doi.org/10.1016/0010-440X(81)90035-3.
[9] Jeste DV, del Carmen R, Lohr JB, Wyatt RJ. Did schizophrenia exist before the eighteenth century? Compr Psychiatry 1985;26:493–503. https://doi.org/10.1016/0010-440X(85)90016-1.
[10] Foucher JR, Jeanjean LC, de Billy CC, Pfuhlmann B, Clauss JME, Obrecht A, et al. The polysemous concepts of psychomotricity and catatonia: A European multi-consensus perspective. Eur Neuropsychopharmacol 2022;56:60–73. https://doi.org/10.1016/J.EURONEURO.2021.11.008.
[11] Tandon R, Heckers S, Bustillo J, Barch DM, Gaebel W, Gur RE, et al. Catatonia in DSM-5. Schizophr Res 2013;150:26–30. https://doi.org/10.1016/j.schres.2013.04.034.
[12] Abrams R, Taylor MA. Catatonia: A Prospective Clinical Study. Arch Gen Psychiatry 1976;33:579–81. https://doi.org/10.1001/ARCHPSYC.1976.01770050043006.
[13] Gelenberg AJ. The catatonic syndrome. Lancet (London, England) 1976;1:1339–41. https://doi.org/10.1016/s0140-6736(76)92669-6.
[14] Oldham MA. The Probability That Catatonia in the Hospital has a Medical Cause and the Relative Proportions of Its Causes: A Systematic Review. Psychosomatics 2018;59:333–40. https://doi.org/10.1016/j.psym.2018.04.001.
[15] Starkstein SE, Petracca G, Tesón A, Chemerinski E, Merello M, Migliorelli R, et al. Catatonia in depression: prevalence, clinical correlates, and validation of a scale. J Neurol Neurosurg Psychiatry 1996;60:326–32. https://doi.org/10.1136/jnnp.60.3.326.
[16] Vaquerizo-Serrano J, Salazar de Pablo G, Singh J, Santosh P. Catatonia in autism spectrum disorders: A systematic review and meta-analysis. Eur Psychiatry 2021. https://doi.org/10.1192/j.eurpsy.2021.2259.
[17] Rogers JP, Pollak TA, Blackman G, David AS. Catatonia and the immune system: a review. Lancet Psychiatry 2019;6:620–30. https://doi.org/10.1016/S2215-0366(19)30190-7.
[18] Ungvari GS, Leung SK, Ng FS, Cheung HK, Leung T. Schizophrenia with prominent catatonic features ('catatonic schizophrenia'): I. Demographic and clinical correlates in the chronic phase. Prog Neuropsychopharmacol Biol Psychiatry 2005;29:27–38. https://doi.org/10.1016/J.PNPBP.2004.08.007.
[19] World Health Organization. ICD-11 for Mortality and Morbidity Statistics 2018. https://icd.who.int/browse11/l-m/en (accessed February 4, 2019).
[20] Oldham MA. Describing the features of catatonia: A comparative phenotypic analysis. Schizophr Res 2022. https://doi.org/10.1016/j.schres.2022.08.002.
[21] Rogers JP, Oldham MA, Fricchione G, Northoff G, Ellen Wilson J, Mann SC, et al. Evidence-based consensus guidelines for the management of catatonia: Recommendations from the British Association for Psychopharmacology. J Psychopharmacol 2023;37:327–69. https://doi.org/10.1177/02698811231158232.
[22] Jeyaventhan R, Thanikasalam R, Mehta MA, Solmi F, Pollak TA, Nicholson TR, et al. Clinical Neuroimaging Findings in Catatonia: Neuroradiological Reports of MRI Scans of Psychiatric Inpatients With and Without Catatonia. J Neuropsychiatry Clin Neurosci 2022. https://doi.org/10.1176/appi.neuropsych.21070181.
[23] Haroche A, Rogers J, Plaze M, Gaillard R, Williams SC, Thomas P, et al. Brain imaging in catatonia: systematic review and directions for future research. Psychol Med 2020:1–13. https://doi.org/10.1017/S0033291720001853.
[24] Northoff G, Steinke R, Czervenka C, Krause R, Ulrich S, Danos P, et al. Decreased density of GABA-A receptors in the left sensorimotor cortex in akinetic catatonia: investigation of in vivo benzodiazepine receptor binding. J Neurol Neurosurg Psychiatry 1999;67:445–50. https://doi.org/10.1136/jnnp.67.4.445.
[25] Hinkle JT, Pontone GM. Reconceptualizing catatonia as "psychiatric parkinsonism". Am J Geriatr Psychiatry 2023. https://doi.org/10.1016/j.jagp.2023.07.010.
[26] Lander M, Bastiampillai T, Sareen J. Review of withdrawal catatonia: what does this reveal about clozapine? Transl Psychiatry 2018;8:139. https://doi.org/10.1038/s41398-018-0192-9.
[27] Elliott B, Joyce E, Shorvon S. Delusions, illusions and hallucinations in epilepsy: 2. Complex phenomena and psychosis Epilepsy Research 2009;85:172–86. https://doi.org/10.1016/j.eplepsyres.2009.03.017.
[28] Maguire M, Singh J, Marson A. Epilepsy and psychosis: a practical approach. Pract Neurol 2018;18:106–14. https://doi.org/10.1136/practneurol-2017-001775.
[29] Elliott B, Joyce E, Shorvon S. Delusions, illusions and hallucinations in epilepsy: 1. Elementary phenomena Epilepsy Research 2009;85:162–71. https://doi.org/10.1016/j.eplepsyres.2009.03.018.
[30] Ogyu K, Kurose S, Uchida H, Kanemoto K, Mimura M, Takeuchi H. Clinical features of catatonic non-convulsive status epilepticus: A systematic review of cases. J Psychosom Res 2021;151:110660. https://doi.org/10.1016/J.JPSYCHORES.2021.110660.
[31] Espi Forcen F, Respino M, Eloge J, Baldwin M, Burns K, Patron V, et al. Catatonia in the medically ill: Differences and parallels in etiology, phenomenology and management. Int J Psychiatry Med 2021. https://doi.org/10.1177/0091217421994078.
[32] Smith JH, Smith VD, Philbrick KL, Kumar N. Catatonic disorder due to a general medical or psychiatric condition. J Neuropsychiatry Clin Neurosci 2012;24:198–207.
[33] Silva Gadelho L, Gama MJ. Catatonia associated with epileptic seizures: A systematic review of case reports. Epilepsy Res 2022;186:107016. https://doi.org/10.1016/j.eplepsyres.2022.107016.
[34] Dawkins E, Cruden-Smith L, Carter B, Amad A, Zandi MS, Lewis G, et al. Catatonia Psychopathology and Phenomenology in a Large Dataset. Front Psych 2022:977. https://doi.org/10.3389/FPSYT.2022.886662.
[35] Grover S, Sahoo S, Chakravarty R, Chakrabarti S, Avasthi A. Comparative study of symptom profile of catatonia in patients with psychotic disorders, affective disorders and organic disorders. Asian J Psychiatr 2019;43:170–6. https://doi.org/10.1016/j.ajp.2019.05.024.
[36] Verbraeken R, Luykx JJ. Persistent catatonia following epileptic seizures: a case report and systematic literature search. BMC Psychiatry 2018;18:350. https://doi.org/10.1186/s12888-018-1935-0.
[37] Gupta M, Gupta M, Aggarwal A, Karahda A, Jindal R. Catatonia in adolescents: a case series. Int J Acad Med Pharm 2013;5:1391–4.
[38] Gunduz A, Benbir G, Bayar R. Postictal Catatonia in a Schizophrenic Patient and Electroconvulsive Treatment. J ECT 2008;24:166. https://doi.org/10.1097/YCT.0b013e318150665c.
[39] Myslobodsky MS, Mintz M. Postictal behavioral arrest in the rat: "catalepsy" or "catatonia"? Life Sci 1981;28:2287–93. https://doi.org/10.1016/0024-3205(81)90581-6.
[40] Dalmau J, Gleichman AJ, Hughes EG, Rossi JE, Peng X, Lai M, et al. Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies. The Lancet Neurology 2008;7:1091–8. https://doi.org/10.1016/S1474-4422(08)70224-2.
[41] Tarulli A, Devinsky O, Alper K. Progression of Postictal to Interictal Psychosis. Epilepsia 2001;42:1468–71. https://doi.org/10.1046/j.1528-1157.2001.10701.x.
[42] Liu X, Sun X, Sun C, Zou M, Chen Y, Huang J, et al. Prevalence of epilepsy in autism spectrum disorders: A systematic review and meta-analysis. Autism 2022;26:33–50. https://doi.org/10.1177/13623613211045029.
[43] Robertson J, Hatton C, Emerson E, Baines S. Prevalence of epilepsy among people with intellectual disabilities: A systematic review. Seizure 2015;29:46–62. https://doi.org/10.1016/j.seizure.2015.03.016.

[44] Jap SN, Ghaziuddin N. Catatonia Among Adolescents With Down Syndrome: A Review and 2 Case Reports. J ECT 2011;27:334. https://doi.org/10.1097/YCT.0b013e31821d37c6.

[45] Miles JH, Takahashi N, Muckerman J, Nowell KP, Ithman M. Catatonia in Down syndrome: systematic approach to diagnosis, treatment and outcome assessment based on a case series of seven patients. Neuropsychiatr Dis Treat 2019;15:2723–41. https://doi.org/10.2147/NDT.S210613.

[46] Ghaziuddin N, Nassiri A, Miles JH. Catatonia in Down syndrome; a treatable cause of regression. Neuropsychiatr Dis Treat 2015;11:941–9. https://doi.org/10.2147/NDT.S77307.

[47] Adachi N, Ito M. Epilepsy in patients with schizophrenia: Pathophysiology and basic treatments. Epilepsy Behav 2022;127:108520. https://doi.org/10.1016/j.yebeh.2021.108520.

[48] Solmi M, Pigato GG, Roiter B, Guaglianone A, Martini L, Fornaro M, et al. Prevalence of Catatonia and Its Moderators in Clinical Samples: Results from a Meta-analysis and Meta-regression Analysis. Schizophr Bull 2018;44:1133–50. https://doi.org/10.1093/schbul/sbx157.

[49] Northoff G, Pfennig A, Krug M, Danos P, Leschinger A, Schwarz A, et al. Delayed onset of late movement-related cortical potentials and abnormal response to lorazepam in catatonia. Schizophr Res 2000;44:193–211. https://doi.org/10.1016/S0920-9964(99)00189-9.

[50] Carroll BT, Goforth HW, Boutros NN, Anfinson TJ, Werner L. Electroencephalography in Catatonic Disorders Due to General Medical Conditions and Psychiatric Disorders. CNS Spectr 1998;3:57–61. https://doi.org/10.1017/S1092852900005551.

[51] Hosseini P, Whincup R, Devan K, Ghanem DA, Fanshawe JB, Saini A, et al. The role of the electroencephalogram (EEG) in determining the aetiology of catatonia: a systematic review and meta-analysis of diagnostic test accuracy. EClinicalMedicine 2023;56:101808. https://doi.org/10.1016/j.eclinm.2022.101808.

[52] Suzuki K, Miura N, Awata S, Ebina Y, Takano T, Honda T, et al. Epileptic Seizures Superimposed on Catatonic Stupor. Epilepsia 2006;47:793–8. https://doi.org/10.1111/J.1528-1167.2006.00528.X.

[53] Gjessing H, Jenner FA, Johannessen NB. The EEG in three cases of periodic catatonia. Br J Psychiatry J Ment Sci 1967;113:1271–82.

[54] Annell AL. Periodic catatonia in a boy of 7 years. Acta Paedopsychiatr 1963;30:48–58.

[55] Ando M, Ito K. Clinical and electroencephalographical studies on catatonia. Folia Psychiatr Neurol Jpn 1959;13:133–42. https://doi.org/10.1111/J.1440-1819.1959.TB02428.X.

[56] Iseki K, Ikeda A, Kihara T, Kawamoto Y, Mezaki T, Hanakawa T, et al. Impairment of the cortical GABAergic inhibitory system in catatonic stupor: a case report with neuroimaging. Epileptic Disord 2009;11:126–31. https://doi.org/10.1684/EPD.2009.0257.

[57] Luccarelli J, Smith JR, Fricchione G, Westover MB. Generalized Periodic Discharges Associated With Catatonia and Delirium: A Case Series. JNP 2024. https://doi.org/10.1176/appi.neuropsych.20230174.

[58] Lam AD, Deck G, Goldman A, Eskandar EN, Noebels J, Cole AJ. Silent hippocampal seizures and spikes identified by foramen ovale electrodes in Alzheimer's disease. Nat Med 2017;23:678–80. https://doi.org/10.1038/nm.4330.

[59] Northoff G, Krill W, Wenke J, Travers H. Pflug B [The subjective experience in catatonia: systematic study of 24 catatonic patients]. Psychiatr Prax 1996;23:69–73.

[60] Biraben A, Taussig D, Thomas P, Even C, Vignal JP, Scarabin JM, et al. Fear as the main feature of epileptic seizures. J Neurol Neurosurg Psychiatry 2001;70:186–91. https://doi.org/10.1136/jnnp.70.2.186.

[61] Suchandra HH, Reddi VSK, Aandi Subramaniyam B, Muliyala KP. Revisiting lorazepam challenge test: Clinical response with dose variations and utility for catatonia in a psychiatric emergency setting. Aust N Z J Psychiatry 2020;000486742096891. https://doi.org/10.1177/0004867420968915.

[62] Sidana A, Goel V. Benzodiazepine Withdrawal Catatonia: A Diagnostic Intervention. The Primary Care Companion for CNS Disorders 2022;24:41538. https://doi.org/10.4088/PCC.21CR03135.

[63] Sayles BD, Royer EG, Meyer VM, Balasanova AA. Malignant Catatonia Induced by Concurrent Opioid and Benzodiazepine Withdrawal. Prim Care Companion CNS Disord 2023. https://doi.org/10.4088/PCC.22cr03346.

[64] Rosebush PI, MacQueen GM, Mazurek MF. Catatonia Following Gabapentin Withdrawal. J Clin Psychopharmacol 1999;19:188.

[65] Henry L, Khan A, Krupka PN, Stringer B. Gabapentin Withdrawal Presenting as Catatonia and Altered Mental Status. A43. CRITICAL CARE CASE REPORTS: TOXICOLOGY AND POISONINGS. American Thoracic Society 2020:A1697–. https://doi.org/10.1164/ajrccm-conference.2020.201.1_MeetingAbstracts.A1697.

[66] Lim C, Donovan AL, Harrold GK, Freudenreich O. A Case of Undetected Seizure Disorder That Exacerbated Catatonia and Treatment-Resistant Schizophrenia: Comprehensive Evaluation Is Key. Prim Care Companion CNS Disord 2022. https://doi.org/10.4088/PCC.22cr03282.

[67] Primavera A, Fonti A, Novello P, Roccatagliata G, Cocito L. Epileptic seizures in patients with acute catatonic syndrome. J Neurol Neurosurg Psychiatry 1994;57:1419–22.

[68] Wasserthal J, Maier-Hein KH, Neher PF, Northoff G, Kubera KM, Fritze S, et al. Multiparametric mapping of white matter microstructure in catatonia. Neuropsychopharmacology : Official Publication of the American College of Neuropsychopharmacology 2020. https://doi.org/10.1038/s41386-020-0691-2.

[69] Viher PV, Stegmayer K, Federspiel A, Bohlhalter S, Wiest R, Walther S. Altered diffusion in motor white matter tracts in psychosis patients with catatonia. Schizophr Res 2020;220:210–7. https://doi.org/10.1016/J.SCHRES.2020.03.017.

[70] Dean DJ, Woodward N, Walther S, McHugo M, Armstrong K, Heckers S. Cognitive motor impairments and brain structure in schizophrenia spectrum disorder patients with a history of catatonia. Schizophr Res 2020;222:335–41. https://doi.org/10.1016/j.schres.2020.05.012.

[71] Krivoy A, Shrot S, Avrahami M, Fischel T, Weizman A, Mardor Y, et al. Brain Motor Region Diffusion Tensor Imaging in Patients with Catatonic Schizophrenia: A Case-Control Study. Isr Med Assoc J 2021;23:625–30.

[72] Hirjak D, Rogers JP, Wolf RC, Kubera KM, Fritze S, Wilson JE, et al. Catatonia Nat Rev Dis Primers 2024;10:1–18. https://doi.org/10.1038/s41572-024-00534-w.

[73] Sambataro F, Hirjak D, Fritze S, Kubera KM, Northoff G, Calhoun VD, et al. Intrinsic neural network dynamics in catatonia. Hum Brain Mapp 2021. https://doi.org/10.1002/HBM.25671.

[74] Scharfman HE. The neurobiology of epilepsy. Curr Neurol Neurosci Rep 2007;7:348–54. https://doi.org/10.1007/s11910-007-0053-z.

[75] Devinsky O, Vezzani A, O'Brien TJ, Jette N, Scheffer IE, de Curtis M, et al. Epilepsy Nat Rev Dis Primers 2018;4:1–24. https://doi.org/10.1038/nrdp.2018.24.

[76] Northoff G. What catatonia can tell us about "top-down modulation": A neuropsychiatric hypothesis. Behav Brain Sci 2002;25:555–77. https://doi.org/10.1017/S0140525X02000109.

[77] Saini A, Begum N, Matti J, Ghanem DA, Fripp L, Pollak TA, et al. Clozapine as a treatment for catatonia: A systematic review. Schizophr Res 2022;S0920–9964(22):00363. https://doi.org/10.1016/j.schres.2022.09.021.

[78] Jackson A, Seneviratne U. EEG changes in patients on antipsychotic therapy: A systematic review. Epilepsy Behav 2019;95:1–9. https://doi.org/10.1016/j.yebeh.2019.02.005.

[79] Ruffmann C, Bogliun G, Beghi E. Epileptogenic drugs: a systematic review. Expert Rev Neurother 2006;6:575–89. https://doi.org/10.1586/14737175.6.4.575.

[80] Calle D, Rivera B, Munoz San Jose A, Marin Lozano J, Bravo-Ortiz. Conversion catatonia. A case report of a rare cause of catatonia syndrome. Eur Psychiatr 2019;56. p. S470–S470.

[81] Ahmed GK, Elbeh K, Karim AA, Khedr EM. Case Report: Catatonia Associated With Post-traumatic Stress Disorder. Front Psych 2021;12:740436. https://doi.org/10.3389/FPSYT.2021.740436.

[82] North N, Bishop J, Bruce D, Boardman T. Traumatic Hand Amputation Resulting in Catatonia. Air Med J 2021;40:278–9. https://doi.org/10.1016/j.amj.2021.04.007.

[83] Baran B, Bitter I, Ungvari GS, Gazdag G. The birth of convulsive therapy revisited: A reappraisal of László Meduna's first cohort of patients. J Affect Disord 2012;136:1179–82. https://doi.org/10.1016/J.JAD.2011.11.045.

[84] Fink M. Is Catatonia a Primary Indication for ECT? J ECT 1990;6:1.

[85] Leroy A, Naudet F, Vaiva G, Francis A, Thomas P, Amad A. Is electroconvulsive therapy an evidence-based treatment for catatonia? A systematic review and meta-analysis. Eur Arch Psychiatry Clin Neurosci 2018;268:675–87. https://doi.org/10.1007/s00406-017-0819-5.

[86] Smith JR, Baldwin I, Termini KA, McGonigle T, Vandekar S, Luccarelli J. Use of ECT for Children With and Without Catatonia: A Single-Site Retrospective Analysis. J ECT 2024. https://doi.org/10.1097/YCT.0000000000000993.

[87] Girish K, Gill NS. Electroconvulsive therapy in Lorazepam non-responsive catatonia. Indian J Psychiatry 2003;45:21–5.

[88] National Institute for Health and Care Excellence. Guidance on the use of electroconvulsive therapy. 2003.

[89] American Psychiatric Association. The American Psychiatric Association Practice Guideline for The Treatment of Patients with Schizophrenia. Third Edition. Washington DC: American Psychiatric Association Publishing; 2021.

[90] Hasan A, Falkai P, Wobrock T, Lieberman J, Glenthoj B, Gattaz WF, et al. World Federation of Societies of Biological Psychiatry (WFSBP) Guidelines for Biological Treatment of Schizophrenia, Part 1: Update 2012 on the acute treatment of schizophrenia and the management of treatment resistance. World J Biol Psychiatry 2012;13:318–78. https://doi.org/10.3109/15622975.2012.696143.

[91] Denysenko L, Sica N, Penders TM, Philbrick KL, Walker A, Shaffer S, et al. Catatonia in the medically ill: Etiology, diagnosis, and treatment. The Academy of Consultation-Liaison Psychiatry Evidence-Based Medicine Subcommittee Monograph. Annals of Clinical Psychiatry : Official Journal of the American Academy of Clinical Psychiatrists 2018;30:140–55.

[92] Zeiler FA, Matuszczak M, Teitelbaum J, Gillman LM, Kazina CJ. Electroconvulsive therapy for refractory status epilepticus: A systematic review. Seizure 2016;35:23–32. https://doi.org/10.1016/j.seizure.2015.12.015.