REVIEW

Cerebellar involvement and stimulation in epilepsy

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Purpose of review
This review discusses the current state of the evidence related to the relationship between the cerebellum and epilepsy, highlighting evidence on neurostimulation of the cerebellum for treatment of epilepsy, and placing current knowledge into historical context.

Recent findings
The cerebellum plays an important role in certain epilepsy types, both as a key part of epileptic networks and an area that can give rise to seizures. Cerebellar stimulation as a potential treatment for drug-resistant epilepsy is a recurring, albeit niche, topic of interest. Over decades of intermittent, often highly limited investigations into this area of research, there are still more questions than answers. However, more recent preclinical insights point the way towards leveraging modern surgical techniques and technology in investigating cerebellar stimulation as a potential viable treatment approach to select types of epilepsy.

Summary
Cerebellar stimulation holds promise for improving seizure control in people with specific types of drug-resistant epilepsy. Future studies should leverage new preclinical data, along with modern technology, neurosurgical techniques, and clinical trial design, to help determine the optimal stimulation parameters, optimal stimulation targets, and optimal patient-selection for this promising area of investigation.

Keywords
cerebellum, neurology, neurostimulation, neurosurgery, seizures

INTRODUCTION

Epilepsy affects well over 60 million people worldwide [1]. While most people with epilepsy can be seizure-free with antiseizure medication (ASM), approximately one-third continue to experience seizures, and may be candidates for invasive treatments including neurostimulation. However, there remain significant barriers to care and limited treatment options for those with drug-resistant epilepsy [2]. A full assessment for potential invasive treatments is complex, time-consuming, and expensive. Many patients continue experiencing disabling seizures despite undergoing an invasive treatment. For this reason, the cerebellum has re-emerged as a potential target for the treatment of epilepsy.

Although historical clinical trials of cerebellar neurostimulation yielded inconsistent outcomes, recent advances in neurophysiology, functional imaging, optogenetics, neurosurgical targeting, and stimulation analysis have brought increasing clarity and promise for clinical applicability. This review briefly summarizes the relevant cerebellar neuroanatomy, evidence for the cerebellum's role in epilepsy, and both historical and contemporary animal and human cerebellar neurostimulation trials, highlighting recent contributions and discussing potential future clinical applications.

CEREBELLAR NEUROANATOMY

The cerebellum is a complex, tightly folded structure located in the posterior fossa, dorsal to the pons, medulla, and fourth ventricle. It is separated from the cerebral cortex through a thick layer of dura known as the tentorium cerebelli; its connections with the rest of the brain must travel through the brainstem, primarily through three paired cerebellar peduncles. Anatomically, the cerebellum consists of two hemispheres (subdivided into lateral and intermediate hemispheres), a midline vermis, and the flocculonodular lobe. The cerebellum is separated rostral-to-caudal into the anterior lobe (located above the primary fissure) and the posterior lobe (located below the primary fissure and above the posterior fissure). Lobes are separated further into 10 lobules, which are

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KEY POINTS

- Cerebellar stimulation for treatment of epilepsy has a long, sporadic history that raises more questions than it answers.
- Current clinical trial data is inadequate for either supporting or refuting cerebellar stimulation as a potential target for treatment of drug-resistant epilepsy.
- Recent preclinical data has helped renew interest in exploring more specific cerebellar targets for treatment of epilepsy using modern surgical techniques, technology, and clinical trial design.

then divided into folia. The flocculonodular lobe (located below the posterior fissure) consists of a midline nodule and paired lateral flocculi, with one on each side of the nodule [3].

Derived from the Latin for "little brain" due to accounting for only 10% of the brain's volume, the cerebellum contains nearly 80% of neurons in the central nervous system [4]. Histologically, it consists of the outermost cerebellar cortex and cerebellar white matter, with three paired gray matter nuclei embedded within the white matter known as the deep cerebellar nuclei (DCN). The cerebellar cortex has three highly organized cell layers: the molecular layer, the Purkinje cell layer, and granular layer, which is just superficial to cerebellar white matter. The DCN include the fastigial nucleus, medial to the cerebellar vermis; the interposed nucleus (consisting of two subnuclei, the anterior emboliform nucleus and the posterior globose nucleus), located in the intermediate hemisphere; and the dentate nucleus, located in the lateral hemisphere. The DCN have broad projections which include the brainstem, thalamus, and cerebral cortex, highlighting the cerebellum's role in diverse neurological processes [3].

The cerebellar cortex is composed of Purkinje cells and interneuron populations including granule cells, Golgi cells, basket cells, and stellate cells. Purkinje cells are the sole neuronal output, providing tonic inhibitory control on the DCN. Purkinje cells have been reported to directly project to extracerebellar locations as well, including the locus coeruleus [5]. Granule cells are the sole excitatory interneurons in the cerebellum, whereas Golgi cells, basket cells, and stellate cells are inhibitory. Granule cells (located in the granular layer) project through specialized axons known as parallel fibers onto dendrites of Purkinje cells, located in the molecular layer. The molecular layer is the most superficial, containing stellate and basket cell bodies, and is where granule cell axons synapse with Purkinje cell dendrites.

CEREBELLAR FUNCTIONAL ANATOMY

Functional divisions of the cerebellum include vestibulocerebellum (known as the archicerebellum), spinocerebellum (known as the paleocerebellum), and cerebrocerebellum (known as the neocerebellum). The vestibulocerebellum consists of the flocculonodular lobe and part of the vermis, and has roles in balance, posture, and eye movements. The spinocerebellum consists of the vermis and part of the lateral hemispheres including the fastigial and interposed nuclei, and is involved in muscle tone, sensory modulation of movement, and postural control, among others [5,6]. The cerebrocerebellum consists of the lateral cerebellar hemispheres, including the dentate nuclei and has roles in motor planning, motor learning, complex movements, speech, and cognitive functions [3,6].

The cerebellum receives afferent input through mossy fibers and climbing fibers. Mossy fibers originate from diverse regions throughout the brain, including the cerebral cortex, brainstem, and spinal cord. Mossy fibers synapse directly on DCN in addition to synapsing on excitatory granule cell interneurons and inhibitory interneurons, which modulate Purkinje cell activity. Climbing fibers originate from neurons in the inferior olivary nucleus, synapsing directly on DCN and directly with Purkinje cells [3,5].

Cerebellar output is broad and complex, with multisynaptic and reciprocal connectivity throughout the brainstem, cortex, and subcortical regions [3]. Outflow travels predominantly through the superior cerebellar peduncles, with fibers that decussate in the midbrain and synapse on numerous contralateral thalamic and brainstem nuclei. DCN contain complex cytoarchitecture with both local and projection neurons, and excitatory glutamatergic or inhibitory glycinergic and GABAergic neurons [4,5]. Fastigial nucleus efferents predominantly target the reticular formation and vestibular nuclei, though additional outputs to the thalamus have been described to underlie connections with numerous cortical regions, with over 60 distinct regions identified including motor cortex and limbic system [3–5,7,8]. Outputs of the interposed nuclei include the motor cortex through the thalamus and the magnocellular division of the red nucleus. Dentate nucleus output travels via thalamic nuclei to multiple cortical regions including frontal and parietal lobes, and the parvocellular division of the red nucleus [3–5]. Among cerebellar nuclei, the dentate nucleus receives the majority of Purkinje cell afferents and provides excitatory output to the thalamus and brainstem in addition to inhibitory outputs to the inferior olive [6]. The dentate nucleus is broadly connected, with outputs to the thalamus, basal ganglia, and neocortex, highlighting its potential role in motor, cognitive, and affective disorders.

Among its diverse connections are the default mode network (DMN) and visual networks, implying a role in thought processing, visual attention, and attention-driven motor control [6].

Historically, there has been an emphasis on the cerebellum's role in balance, posture, and motor control, and more recently, it is understood to also have an important role in nonmotor functions including language, executive function, autonomic regulation, attention, and emotion [4,5]. The cerebellum has direct connections to the amygdala, appears critical for hippocampal spatial representation, and can be functionally connected to the hippocampus during conditioning tasks [5,8,9]. The cerebellum also influences recovery from autonomic disturbances and is hypothesized to have a role in SIDS and SUDEP [4,5,10]. Recent and ongoing work involving transneuronal virus tracing, fMRI, and diffusion tensor imaging are providing important insights into cerebellar connectivity [6].

THE CEREBELLUM'S ROLE IN EPILEPSY

The cerebellum's broad and diverse connectivity implicates it in numerous neurological disorders, including seizures and epilepsy [4,5,8,9]. Across preclinical models, the cerebellum is prominent in epileptic networks, and may have an active modulatory and inhibitory role in epilepsy. Phase-locked cerebellar spiking is present in absence and TLE models; cerebellar neuronal depolarization abnormalities are present in absence and motor seizures; pharmacological disruption of cerebellar excitatory and inhibitory tone produces spontaneous seizures; genetic deletion of cerebellar P/Q channels produces absence epilepsy [4,5,7]. Similar to chronic epilepsy in humans, animal models have shown midline cerebellar volume loss, particularly in the posterior lobe [4,11,12]. Using a novel calcium imaging technique in a preclinical model of focal epilepsy, widespread cerebellar alterations were preceding both hippocampal seizures and interictal discharges [13].

While neuroanatomical changes in the cerebellum in people with chronic epilepsy have historically been attributed to ASMs (i.e. phenytoin), additional studies controlling for exposure identified cerebellar atrophy correlating to disease severity and duration, particularly in temporal lobe epilepsy [4–6]. Volume loss in the corpus medullare and posterior lobe, as well as Purkinje cell degeneration are hallmarks of epilepsy-related cerebellar atrophy [4,11]. Atrophy is associated with epilepsy duration and tonic-clonic seizure frequency, and reduced white matter volume has been associated with generalized tonic clonic and myoclonic seizures [4,14]. Decreased cerebellar gray matter, particularly in the vermis, has been associated with SUDEP and those with high SUDEP risk, compared to controls and low-risk patients [15]. This may be related to Purkinje cell injury resulting in impairment in the DCN function responsible for autonomic recovery [16].

Functional imaging has also demonstrated important findings for understanding the role of the cerebellum in epilepsy. Cerebellar crossed diaschisis is well known phenomenon in people with focal epilepsy, and ictal FDG-PET during absence seizures has demonstrated cerebellar vermis hypermetabolism with cerebellar hemisphere hypometabolism [17]. In patients with idiopathic generalized epilepsy, EEG-fMRI shows cerebellar hyperperfusion following generalized epileptiform discharges [18]. Volumetric MRI in patients with progressive myoclonus epilepsy has revealed decreased white matter fiber density, particularly in deep cerebellar nuclei (DCN) and superior cerebellar peduncles [19]. Similar analyses in familial cortical myoclonic tremor with epilepsy also identified decreased cerebellar volume and gray matter compared to both controls and patients with essential tremor [20]. Cerebellar hyperperfusion is commonly seen on ictal SPECT of focal seizures, with increasing cerebellar hyperperfusion in proportion to the time from seizure onset to radiotracer injection [21,22]. In focal to bilateral tonic-clonic seizures, cerebellar hyperperfusion changes follow bilateral propagation and then become highly significant postictally [23]. Functional connectivity studies have identified abnormalities in both temporal lobe epilepsy and generalized epilepsy [7]. In patients with mesial temporal sclerosis who undergo anterior temporal lobectomy, postoperative PET hypermetabolism in the cerebellar vermis and bilateral hemispheres is predictive of poor surgical outcomes [24].

In case reports of cerebellar lesional epilepsy, stereotyped features include hemifacial and ocular movements, as well as autonomic dysfunction including apnea and bradycardia [4]. Depth electrode sampling of cerebellar lesions have found both interictal discharges and seizure onsets when surface EEG was unrevealing [25–27]. Cerebellar lesional epilepsy is nearly always diagnosed in infancy, with a third presenting on the first day of life and over 80% before age one. It typically presents with ipsilateral hemifacial clonic or tonic seizures, which are highly resistant to ASMs, but respond well to resective surgery: patients with such lesions typically achieve long-term seizure-freedom and medication discontinuation following resection [4,27,28].

CEREBELLAR STIMULATION AS EPILEPSY TREATMENT

Modern preclinical studies support investigation into human trials. As opposed to open-loop stimulation,

closed loop stimulation has been recently explored as a more controlled method of stimulating in response to seizures or interictal activity [29–31]. Closed loop stimulation has allowed for optimization analyses, improving the ability to suppress seizures with stimulation, in contrast to open loop stimulation [32–34]. Excitation of excitatory DCN output appears critical for seizure inhibition across preclinical models, though specific DCN targets may vary between epilepsy models and seizure types [8,9,32].

There is evidence of different stimulation effects at specific targets in various models. For instance, excitation and not inhibition of fastigial nuclei reduces seizures in a mouse model of TLE [33]. Generalized spike-wave discharges have been reduced through stimulation of intermediate and lateral cerebellar nuclei, and exacerbated through inhibition of DCN [35]. Based on current preclinical data, the fastigial nucleus and medial cerebellum may be more effective for treating TLE, while the dentate nucleus and lateral cerebellum may be more effective in targeting generalized epilepsies (Fig. 1); however, it remains unclear which may be ideal for extratemporal focal, multifocal, or mixed epilepsies [4,8,51].

Data from humans on this topic predates animal models. From a historical perspective, targeting the cerebellum has been a recurring topic in the treatment of epilepsy beginning with Hughlings Jackson's first case report of seizures secondary to a cerebellar tuberculoma nearly 150 years ago [28]. Galvani and Aldini explored electrical stimulation of the ox cerebellum as early as 1798, yet advances in understanding have been slow and incompletely explored [36]. Years later, human and animal studies in the 1930s and 1940s demonstrated cerebellar stimulation can affect electroencephalography (EEG), including the abrupt termination of cobalt-induced frontal lobe seizures [37–40]. Based on this, researchers at the time thought the cerebellum may have an "active inhibitory role" in epilepsy and hypothesized that status epilepticus was at least partially related to a breakdown of cerebellar inhibition [37,41–43]. In 1965, Snider and Wetzel [44] performed the first studies of cerebellar stimulation in humans, demonstrating both "synchronizing and desynchronizing effects" on the awake EEG. Additional studies found evidence of fastigial nuclei involvement in at least some seizures, though this was not adequately controlled for epilepsy type [45].

Irving S. Cooper performed the first clinical trial of chronic cerebellar stimulation in epilepsy, utilizing an "electrode mesh" and reporting that 56% of patients experienced 50% or more reduction in seizures. The stimulation targets for these cases were reported as regions the cerebellar cortex that were not well specified, and the specific stimulation settings were also largely unreported apart from using a pulse width of 1 ms [46–48]. This was

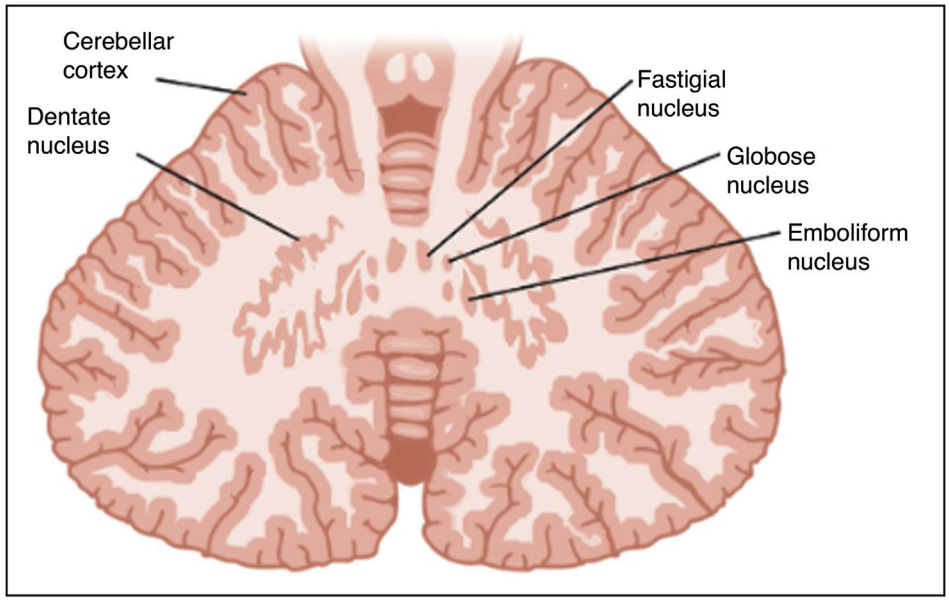

FIGURE 1. Theoretical targets of cerebellar stimulation for the treatment of epilepsy. Current preclinical data suggests the fastigial nucleus and medial cerebellum may be more effective for treating temporal lobe epilepsy; the dentate nucleus and lateral cerebellum may be more effective in targeting generalized epilepsies. Adapted with permission from Elder *et al.* 2024 [4].

followed by several small, uncontrolled, unblinded trials reporting improvements in seizure control though of unclear significance due to poor study design. In the many studies, targets included various (and often unspecified) areas of the cerebellar cortex [49–51], and one included the dentate nucleus [52]. Eventually, two controlled, blinded trials reported no benefit to seizure control, and subsequent trials of cerebellar stimulation were largely abandoned [50,53]. However, even these trials were fraught with inconsistences. The blinded controlled trial by van Buren *et al.* [50] reported no benefit, but following review, four out of five study patients had experienced dramatic seizure reduction when cerebellar stimulation was enabled [38]. A 2005 study was the most recent clinical trial of cerebellar stimulation for epilepsy, and showed that in five patients with drug-resistant epilepsy, tonic-clonic seizure was reduced by 59% within 6 months of stimulation, and in the three patients who continued stimulation up to 2 years, this reduction increased to 76% [38]. However, three of five patients experienced displacement of the lead or electrode requiring surgical repositioning, and one experienced serious infection, indicating that cerebellar electrode placement may have unique risks as compared to other regions.

Considering available clinical trial data with the (implications from the most prominent studies outlined in Table 1), there is limited information on the efficacy of cerebellar stimulation for epilepsy. This highlights the importance of this as an area for further investigation, utilization more rigorous study methods, and accounting for important patient-specific information such as seizure and epilepsy classifications, seizure frequencies, medication data, imaging data, type of stimulation, and controlling for potential placebo and implant effects [54].

CONCLUSION

The cerebellum, once viewed primarily through the lens of motor control, has re-emerged as a compelling target for epilepsy treatment. Growing evidence supports the cerebellum as an active, possibly critical component of seizure networks, with a likely role in SUDEP. Regrettably, early work in this field was largely abandoned in response to studies, that in retrospect had significant methodological and technological limitations. However, with advances in imaging, optogenetics, and closed loop stimulation, contemporary preclinical research has provided critical insights for the re-emergence of human cerebellar stimulation trials. The therapeutic effect of cerebellar stimulation is now hypothesized to be driven by excitation of DCN, achieved through either direct stimulation or indirectly through disinhibition. The fastigial nucleus and medial cerebellum may be a viable target for temporal lobe epilepsy, while the dentate nucleus and lateral cerebellum may be effective for generalized epilepsies. With modern stimulation technology and surgical techniques, the field is poised for hypothesis-driven, well designed clinical trials.

Table 1. Cerebellar stimulation clinical trials: key findings and implications

Study	Summary	Implications
Cooper *et al.* 1973, 1978 [47,48]	32 total patients with epilepsy received stimulation via cortical mesh stimulator. Uncontrolled, unblinded, and not stratified by seizure/epilepsy classification. Overall reported that 56% of patients experienced >50% seizure reduction.	Reported benefit. However, numerous concerns regarding study design and replicability.
van Buren *et al.* 1978 [50]	Enrolled five patients and compared 1-week-on vs. one-week-off cerebellar cortical stimulation intervals under blinded and double blinded conditions using a wide range of stimulation parameters.	Reported no benefit. However, upon subsequent review it was found that four of five participants had experienced dramatic reduction in seizure frequency when cerebellar stimulation was enabled over a longer period.
Wright *et al.* 1984 [53]	Enrolled 12 patients for double-blind cerebellar cortical stimulation for six months.	Reported no objective benefit could be attributed to stimulation, though 11 of 12 reported subjective benefit.
Velasco *et al.* 2005 [38]	Enrolled five patients for midline cerebellar cortical stimulation; control period of 3 mo preimplant compared to postimplant at multiple time points, with double-blind stimulation initiation protocol	Reported clinically significant improvement in seizures. However, frequent clinically significant complications (three of five patients).

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Conflicts of interest

The authors report no relevant conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest

1. Ngugi AK, Bottomley C, Kleinschmidt I, *et al.* Estimation of the burden of active and life-time epilepsy: a meta-analytic approach. Epilepsia 2010; 51:883–890.
2. Pellinen J. Treatment gaps in epilepsy. Front Epidemiol 2022; 2:976039.
3. Martin J. The cerebellum. Neuroanat. Text atlas. 5ed. 2021; Columbus, OH: McGraw Hill
4. Elder C, Kerestes R, Opal P, *et al.* The cerebellum in epilepsy. Epilepsia 2025; 66:1773–1792.
5. Streng ML, Krook-Magnuson E. The cerebellum and epilepsy. Epilepsy Behav 2021; 121:106909.
6. Benarroch E. What is the role of the dentate nucleus in normal and abnormal cerebellar function? Neurology 2024; 103: https://doi.org/10.1212/WNL.0000000000209636.
7. Herlopian A. Subcortical structures and epilepsy. Stereotact Funct Neurosurg 2025; 1–32. https://doi.org/10.1159/000548279.
8. Streng ML, Froula JM, Krook-Magnuson E. The cerebellum's understated role and influences in the epilepsies. Neurobiol Dis 2023; 183: https://doi.org/10.1016/j.nbd.2023.106160.
9. Streng ML. The bidirectional relationship between the cerebellum and seizure networks: a double-edged sword. Curr Opin Behav Sci 2023; 54: https://doi.org/10.1016/j.cobeha.2023.101327.
10. Calton MA, Howard JR, Harper RM, *et al.* The cerebellum and SIDS: disordered breathing in a mouse model of developmental cerebellar purkinje cell loss during recovery from hypercarbia. Front Neurol 2016; 7: https://doi.org/10.3389/fneur.2016.00078.
11. Kerestes R, Perry A, Vivash L, *et al.* Patterns of subregional cerebellar atrophy across epilepsy syndromes: an ENIGMA-Epilepsy study. Epilepsia 2024; 65: 1072–1091.
12. Lee Y, Rodriguez OC, Albanese C, *et al.* Divergent brain changes in two audiogenic rat strains: a voxel-based morphometry and diffusion tensor imaging comparison of the genetically epilepsy prone rat (GEPR-3) and the Wistar Audiogenic Rat (WAR). Neurobiol Dis 2018; 111:80–90.
13. Streng ML, Kottke BW, Wasserman EM, *et al.* Early and widespread cerebellar engagement during hippocampal seizures and interictal discharges. Neurobiol Dis 2025; 213: https://doi.org/10.1016/j.nbd.2025.106991.
14. Oyegbile TO, Bayless K, Dabbs K, *et al.* The nature and extent of cerebellar atrophy in chronic temporal lobe epilepsy. Epilepsia 2011; 52:698–706.
15. Allen LA, Vos SB, Kumar R, *et al.* Cerebellar, limbic, and midbrain volume alterations in sudden unexpected death in epilepsy. Epilepsia 2019; 60: 718–729.
16. Whatley BP, Winston JS, Allen LA, *et al.* Distinct patterns of brain metabolism in patients at risk of sudden unexpected death in epilepsy. Front Neurol 2021; 12: https://doi.org/10.3389/fneur.2021.623358.
17. Zuo JW, Shao XQ, Wang Q, Lv RJ. The clinical features and 18F-FDG-PET analysis of absence status epilepsy. Front Neurol 2025; 16: https://doi.org/10.3389/fneur.2025.1521842.
18. Gotman J, Grova C, Bagshaw A, *et al.* Generalized epileptic discharges show thalamocortical activation and suspension of the default state of the brain. Proc Natl Acad Sci U S A 2005; 102:15236–15240.
19. Cameron JM, Mito R, Berkovic SF, Jackson GD. Progressive myoclonus epilepsy: distinctive MRI changes in cerebellar and motor networks. Ann Clin Transl Neurol 2025; 12:1135–1143.
20. Buijink AWG, Broersma M, van der Stouwe AMM, *et al.* Cerebellar atrophy in cortical myoclonic tremor and not in hereditary essential tremor—a voxel-based morphometry study. Cerebellum 2016; 15:696–704.
21. Bohnen NI, O'Brien TJ, Mullan BP, So EL. Cerebellar changes in partial seizures: clinical correlations of quantitative SPECT and MRI analysis. Epilepsia 1998; 39:640–650.
22. Dupont P, Zaknun JJ, Maes A, *et al.* Dynamic perfusion patterns in temporal lobe epilepsy. Eur J Nucl Med Mol Imaging 2009; 36:823–830.
23. Blumenfeld H, Varghese GI, Purcaro MJ, *et al.* Cortical and subcortical networks in human secondarily generalized tonicclonic seizures. Brain 2009; 132:999–1012.
24. Chiang S, Guindani M, Yeh HJ, *et al.* A hierarchical bayesian model for the identification of PET markers associated to the prediction of surgical outcome after anterior temporal lobe resection. Front Neurosci 2017; 11: https://doi.org/10.3389/fnins.2017.00669.
25. Foit NA, van Velthoven V, Schulz R, *et al.* Lesional cerebellar epilepsy: a review of the evidence. J Neurol 2017; 264: https://doi.org/10.1007/s00415-016-8161-9.
26. Martins WA, Paglioli E, Hemb M, Palmini A. Dysplastic cerebellar epilepsy: complete seizure control following resection of a ganglioglioma. Cerebellum 2016; 15:535–541.
27. Lascano AM, Lemkaddem A, Granziera C, *et al.* Tracking the source of cerebellar epilepsy: hemifacial seizures associated with cerebellar cortical dysplasia. Epilepsy Res 2013; 105:245–249.
28. Jackson JH. Case of tumour of the middle lobe of the cerebellum: rigidity in cerebellar attitude. Br Med J 1871:2528–2529.
29. Krook-Magnuson E, Szabo GG, Armstrong C, *et al.* Cerebellar directed optogenetic intervention inhibits spontaneous hippocampal seizures in a mouse model of temporal lobe epilepsy. ENeuro 2014; 1: https://doi.org/10.1523/ENEURO.0005-14.2014.
30. Streng ML, Popa LS, Ebner TJ. Complex spike wars: a new hope. Cerebellum 2018; 17:735–746.
31. Berényi A, Belluscio M, Mao D, Buzsáki G. Closed-loop control of epilepsy by transcranial electrical stimulation. Science 2012; 337:735–737.
32. Stieve BJ, Richner TJ, Krook-Magnuson C, *et al.* Optimization of closed-loop electrical stimulation enables robust cerebellar-directed seizure control. Brain 2023; 146:91–108.
33. Streng ML, Krook-Magnuson E. Excitation, but not inhibition, of the fastigial nucleus provides powerful control over temporal lobe seizures. J Physiol 2020; 598:171–187.
34. Davis R, Gray E, Engle H, Dusnak A. Reduction of intractable seizures using cerebellar stimulation. Stereotact Funct Neurosurg 1983; 46:57–61.
35. Kros L, Eelkman Rooda OHJ, Spanke JK, *et al.* Cerebellar output controls generalized spike-and-wave discharge occurrence. Ann Neurol 2015; 77: 1027–1049.
36. Aldini G. Essai théorique et expérimental sur le galvanisme: avec une série d'expériences faites en présence des commissaires de l'Institut national de France, et en divers amphithéatres anatomiques de Londres Paris Fournier Fils 1804.
37. Dow RS, Fernández-Guardiola A, Manni E. The influence of the cerebellum on experimental epilepsy. Electroencephalogr Clin Neurophysiol 1962; 14: 383–398.
38. Velasco F, Carrillo-Ruiz JD, Brito F, *et al.* Double-blind, randomized controlled pilot study of bilateral cerebellar stimulation for treatment of intractable motor seizures. Epilepsia 2005; 46:1071–1081.
39. Cooke PM, Snider RS. Some cerebellar influences on electrically-induced cerebral seizures. Epilepsia 1955; 4C:19–28.
40. Mitra J, Snider RS. Effects of hippocampal afterdischarges on Purkinje cell activity. Epilepsia 1975; 16:235–243.
41. Hunt JR. Dyssynergia cerebellaris progressivaa chronic progressive form of cerebellar tremor. Brain 1914; 37:247–268.
42. Foerster O, Altenburger H. Elektrobiologische Vorgänge an der menschlichen Hirnrinde [Electrobiological processes in the human cerebral cortex]. Dtsch Z Nervenheilkd 1935; 135:277–288.
43. Iwata K, Snider RS. Cerebello-hippocampal influences on the electroencephalogram. Electroencephalogr Clin Neurophysiol 1959; 11:439–446.
44. Snider RS, Wetzel N. Electroencephalographic changes induced by stimulation of the cerebellum of man. Electroencephalogr Clin Neurophysiol 1965; 18:176–183.
45. Niedermeyer E, Uematsu S. Electroencephalographic recordings from deep cerebellar structures in patients with uncontrolled epileptic seizures. Electroencephalogr Clin Neurophysiol 1974; 37:355–365.
46. Cooper IS, Crighel E, Amin I. Clinical and physiological effects of stimulation of the paleocerebellum in humans. J Am Geriatr Soc 1973; 21:40–43.
47. Cooper IS. Effect of chronic stimulation of anterior cerebellum on neurological disease. Lancet 1973; 301:206.
48. Cooper IS, Upton ARM. Use of chronic cerebellar stimulation for disorders of disinhibition. Lancet 1978; 311:595–600.
49. Gilman S, Dauth GW, Tennyson VM, *et al.* Clinical, morphological, biochemical, and physiological effects of cerebellar stimulation. Funct Electr Stimul Appl Neural Prosthesis 5th Ed Hambr FT Ed New York, NY: Marcel Dekker; 1977.
50. Van Buren JM, Wood JH, Oakley J, Hambrecht F. Preliminary evaluation of cerebellar stimulation by double-blind stimulation and biological criteria in the treatment of epilepsy. J Neurosurg 1978; 48:407–416.
51. Klun B, Stojanović V, Strojnik P, *et al.* Chronic cerebellar stimulation in the treatment of epilepsy. Regul Cereb Blood Flow Metab Neurosurg 1987; 205–209.
52. Sramka M, Fritz G, Galanda M, Nádvornik P. Some observations in treatment stimulation of epilepsy. Acta Neurochir (Wien) 1976:257–262.
53. Wright GDS, McLellan DL, Brice JG. A double-blind trial of chronic cerebellar stimulation in twelve patients with severe epilepsy. J Neurol Neurosurg Psychiatry 1984; 47:769–774.
54. Kellinghaus C, Loddenkemper T. Double-blind, randomized controlled study of bilateral cerebellar stimulation. Epilepsia 2006; 47:1247.