Psychiatria Danubina, 2023; Vol. 35, Suppl. 2, pp 189-195
Conference paper
Review

HOW THE INFLAMED SKULL-BOX MIGHT RUIN THE CEREBELLO-CEREBRAL SOCIAL REPERTOIRE: IMAGING, TESTING, RESCUING WITH CURRENTS

Gottfried R. S. Treviranus
BipoSuisse, Berne, Switzerland

SUMMARY

Nascent cerebellar neuropsychiatry is rewriting complex human relations. In daily practice this sheds light on subsets of therapy-resistant patients, who feel hampered by a lack of skills in predictively presensing the trajectories to where especially their interpersonal appropriations might end up. Humans affected by "dysmetric" social phobia often lead minimal lives, strongly dislike exposition, suffer fatiguability also from immune dysfunctions, and anhedonia. In social dysmetria especially on the cerebellar cortex`s both lateralmost Crus-II (Van Overwalle) seem damaged, the left Crus-I may add agentic failure (Guell). The cerebellum, and the basal ganglia together discipline the cortex through parallel closed circuits partly interconnected through the thalamus. "Patho-trajectories" of the cere-bellum in daily care emerge through also neurological ataxiology and MRI-imaging. A uniquely pontine axonal diffusive damage to an executive-control-loop as core of "p-factor", the prime broad risk factor, points to an immune-arterial hotspot at the root of cerebellar arterial supply, as supported by the pediatric mostly inflammatory somatic "d-factor". While localizations refer back to his "phrenology", F. G. Gall's contention, that skulls, plausibly via mycobacteria, were relevant, are rehabilitated by calvario-menigeal vessels, which added the skull to the new brain logistics. Mast cells are uniquely positioned to cause superficial and deep cortical pathologies, and, en-closed in its subtentorial posterior fossa, also the cerebellum is exposed to various, often intracellular, smoldering originally dental or ORL-infections exemplifying non-neural psychiatric etiologies.

Key words: *Cerebellar Cognitive-Affective Syndrome – Disengagement syndrome – transcranial direct current stimulation – mast cell – IPAD – psycho-neuro-lympho-arterio-immunology*

* * * * *

"Indeed the world is ruled by little else. (...). Madmen in authority, who hear voices in the air, are distilling their frenzy from some academic scribbler of a few years back.",
J. M. Keynes. General Theory (...), Conclusions, V. 1935, Memorating Riccardo Delle Chiaie

IMAGI(NI)NG SOCIAL PHOBIA "PLUS"?

Social anxiety (SAD) is a cruelly common condition whereby human relations are fraught with an often long anticipated ruminative sensation of a deepseated incapacity to match the behavior of the self with the (often imagined) scrutinizing social behavior of others. Overuse of alcohol and sedatives may form a triad within the "TAM"-spectrum (Karam 2023), wherein hypomania "treats" SAD (Valença 2005). Therapists find themselves often entangled as only social referents in endless therapies, which nevertheless fail, since ingrained beliefs and negative social emotions pile up within rigid self-conceptions. While few are hyperthymic (Figure 5. V#2m65 in Treviranus 2022) most fluctuate in deep dysphoria within the mixed bipolar spectrum (Tavormina 2021, Cervone 2022). Early life victimizations (mlp92; mfr65) resist broad combined therapies, while hallucinatory dreams show vermal activation (Anderson 2002) responding to lithium. Positive feedback to own acts in SAD runs dry, increasing self-abandonement from low anticipated rewards, while the striatum shames incompetence (Becker 2017). But often, beyond inexperience, void metafears loom. Some profit from atomoxetine for their hypermind-wandering and, maybe cerebellar (Bareš 2018) spacey, slipping-focus Cognitive Disengagement Syndrome (CDS) (Frederick & Becker 2023), whereby 2% of us are burdened by the task to dissimulate severe instability of focus in intimate and, despite normal IQ, "never-career" domains. Hypoactivity and apathy arise early, after grey matter losses (Nivins & Klingberg 2023). But in schooler cohorts 2/3 symmetrically switch between CDS and not-CDS (Mayes 2023), pointing to inflammatory insults. ADHD with CDS shows more autistic and anxious traits (Ekinci 2021), while ADHD shows a dip in cerebellar marrow (Shaw 2018). Whilst in SAD much appears as "cerebral" some "persisters" describe more abstract problems, possibly, with "cerebellar" prediction and anticipation of encounters. "I can't call the car mechanic because I will not know how to reply…" (mmp79), "I can't go to the public garden because I don't know what will happen and how to react." (mlp92), "I need a structure, a path to follow…" (fma94). "I prepare carefully how to behave, but then I'm at a loss when unexpectedly…" (Vm65). But the inverse may happen: "The voices scream and mostly correctly tell me, what I have to do, while my vegetative system turns up and my eyes and skin become all red!" (Vf63), pointing to hypothalamic efferents (Zanchetti & Zoccolini 1954). Still others fit into the slighter just "clumsy" end of the quite "cerebellar" Developmental (sensorimotor) Coordination Disorder. DCD-children dislike and fail at balance, motor imagery and tasks. DCD overlaps with dyslexia,

but only DCD shows clearly disrupted sensorimotor corticocerebellar and posterior parietal corticostriatal circuits (Cignetti 2020), while dyslexia emerges as related to osmotic damage (Meisler & Gabrieli 2020) - from interstitial pushback. Here, having started out from skull infections and cerebellar MRI-imaging (Figure 1) we boldly refer to ongoing bioscience. Patients are encoded as V[gender][no19/YoB]. In-text-references being mostly polyauthored and therefore "et al." is omitted, but single authors have [1st letter of Forename] mentioned.

CEREBELLUM: WHEREBY IS IT SPECIAL?

Cerebella emerged with sharks to provide new "central patterns". The human cerebellum (Gruol 2023) harbors 80% (Herculano-Houzel 2009) of neurons its surface being 20% smaller than the big brain's through doublefolding. It combines repetive simplicity with slowly maturing sophistication, which is by far more intricate than the basic models had suggested (Hull & Regehr 2021). Each of the inhibitory, sole output generating, large Purkinje cells, stacked in its 2nd layer within 0.2-mm-zones, have their dendrites arborize like a fan into the subpial molecular layer (ML), where ML-neurons expect to brake them. The fans cut the minifoldings at right angle, receiving, via 500 "synapses", one fibre's restoring input climbing up from the inferior olivary nucleus or medulla. Interacting unipolar brush cells add lasting reactions to cerebellar transiencies and often ultrafast extracellular ephaptic modulation intervene.

CEREBELLUM: WHAT FOR?

Through the "task-dependent engagement of specific cerebellar areas" (Koziol 2014) an attention-sparing optimization occurs providing tasks with seamless fluidity, beyond internal background models. These homeostatic functions can be disrupted within three ataxiological domains (Cabaraux & Monti 2023): at the Anterior sensorimotor lobe (with lobule VIII) with dysarthria and compromised gait, posture (Dijkstra 2020), at the Posterior lobe for eye movement and vestibular balance (vermal IX-X, flocculus) or as Cerebellar Cognitive-Affective Syndrome (CCAS; Schmahmann 1998) with disturbed cognition, inappropriate also linguistic behavior, and affect. Through neuroclinical overlap (Dekeyzer 2023) psychiatric ataxiology has impressively caught up (Eccles 1967, Guell & Schmahmann 2019, 2020). New roles beyond e. g. sequenced movements have emerged through functional imaging (fMRI) as essential for attention, working memory, executive control, and learning, whereby "predictive errors" slowly shape a repertoire of ready-made realistic models (Manto & Shaikh 2020). This led to a further extension to the demanding interactions with conspecifics. From the analogy of a uniform cortex hiding a universally applicable computation the stimulating "dysmetria of thought theory" (Schmahmann 1998 ff.) was born, whereby trajectories of thoughts, or encounters were to be understood in computational terms of movement. This after an ongoing U.S.-interest in autism (Fatemi 2012, Theoharides 2023) incited a mainly Italo-Belgian network to invest in the sociocognitive cerebellum (Van Overwalle 2020). An extended mentalizing network uses cortical areas of all lobes and the amygdala which, as those of the cerebellum (or the separating tentorium), directly or at their connecting axons can suffer from many "logistical" problems like interstitial pushback, visible as "worms and bubbles" on MRI. "Mentalizing", i.e. faculties to interactively conceive mental states (ToM, Theory-of-mind, Baron-Cohen 1985, Luyten et al. 2020), through still enigmatic computations (and tests) encompasses: conceptual (false-belief), perceptual (face-based), affective (empathy), cognitive (perspective), and motoric (mirror-moves) domains, the latter linking with the anterior cerebellum (Rizzolatti 2014).

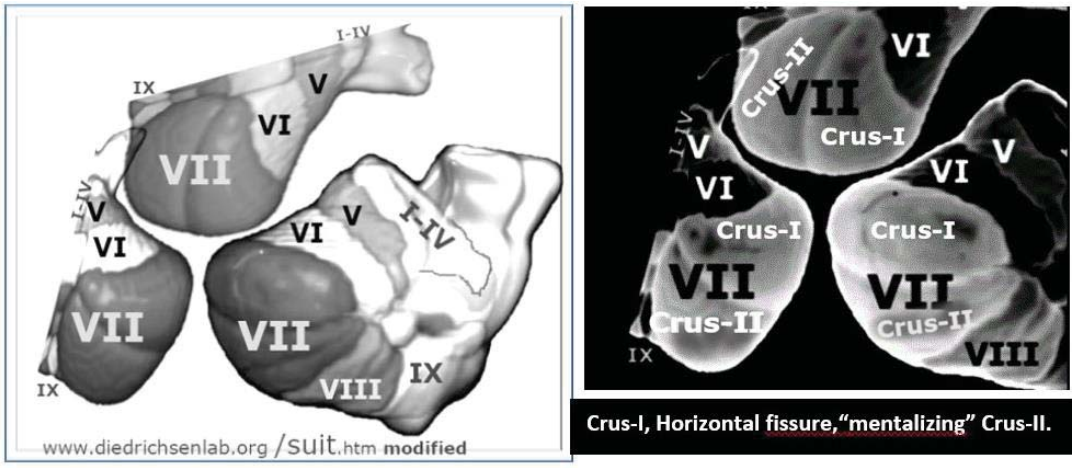


Figure 1. The 2 areas for motor control are: Anterior lobes' lobules I–V, VI partly, and VIII. Posterior lobes` 3 areas for attention/execution and default-mode are: lobule VI-Crus I; Crus II–VIIB; IX–IX. X, Flocculus e.g. not shown. Rearranged numbered view of a standard cerebellum (Diederichsen 2009)

The maturation of repertoires sustaining early ToM (Beuriat 2022) emerged as a needed for cerebrally trainable optimizations in adults, hampered by early scars (Olson 2023). Soon the posterior cerebellum moved centerfold (Van Overwalle 2011-2023). The "sequential" ToM-faculties are largely provided with the Crus-II and sustained mainly by the Crus-I (Ito 2008, Guell 2018, Schmahmann 2019). Conserved mentalizing – especially for sequential tasks – after several damages requires merely the left superior posterior cerebellum, uniquely connected to all the five frontal mentalizing areas, especially to the lateralized right ones, via a stronger thalamocortical and a still weaker corticopontine loop (Metoki 2022). A meta-analysis starting inversely from functional activations (by ToM-subtasks) revealed that those at Crus-II drove 57% of the studies involving true ToM, and even 74% adding emotional introspection (Van Overwalle 2020). Cerebellar degeneration (with cerebral shrinkage above the tentorium cerebelli and also the sphenoid sinus) caused losses in both cerebellar upper halfs, but autism just in the right-sided lower half (Clausi 2021). Delle Chiaie and the ataxia team discovered that BPAD-II patients showed a broader diminution of ToM-skills and cerebellum than BPAD-I-patients (Olivito 2022), while commonalities with schizophrenia (SCZ) appear as "pedun-culo-cerebellar" (Figure 2).

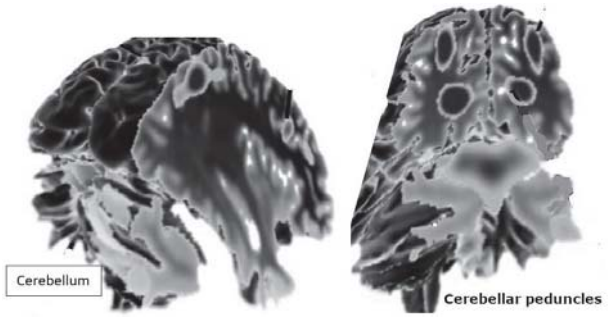


Figure 2. Common grey matter losses from SCZ and BPAD in networks (filtering individual dissimilarities) are mostly cerebellopontine ones, leaving few contrasts. Distorted, after (Sorella 2019)

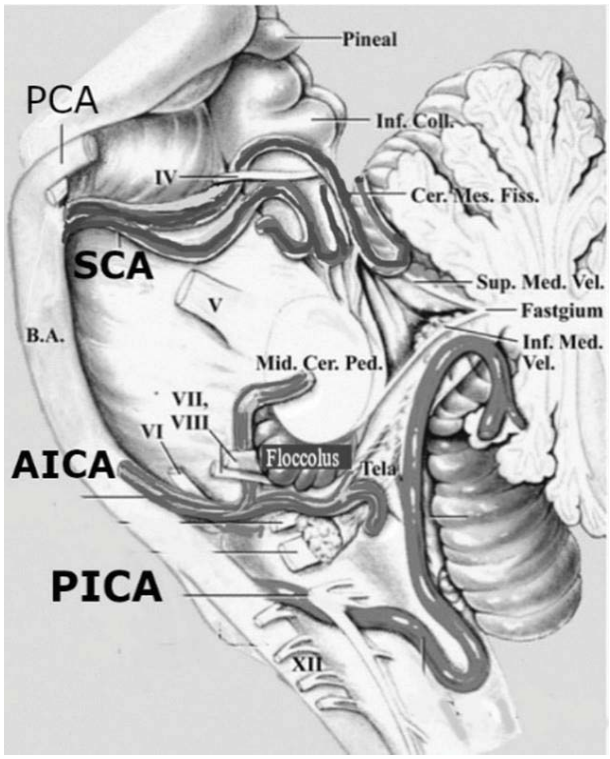
Genetic cues (Smeland 2020, Hughes 2023) and fMRI-connectomics (Matkovič 2023) converge on an emerging, also fetal, infectio-immuno-arterial paradigm (Fatemi 2005, Treviranus 2018ff., Carbia 2023), which strongly relates to the cerebellum and social cognition towards conspecifics, deteriorating in the prodromes (Porcelli 2019). The corroboration of a "d-factor" underlying both mental and physical pediatric conditions (Arrondo 2022) is an important step towards the "clinically obvious", that slight inflammations, mast cells, and infections count a lot, and meets the other broad risk "p-factor" for mental conditions (Selzam 2018), which by correlations unexpectedly (Hariri 2019) converged on unique circumscribed damages to a pontine white matter area within CPC (receiving cerebral inputs to convey, after crossing, through the MCP to the cerebellar cortex). Here the lobulus VIIb (next to Crus-I/-II; Ciapponi 2023), influencing cortical "executive control" through the CTC-limb via the SCP and ventral thalamus was linearly smaller.

CEREBELLAR BLOOD AND LYMPH FLOWS

Among the three backwards coursing cerebellar arteries the posterior one (PICA) takes off near the top of the vertebral arteries as its largest branch. Behind the medulla it dives into a sharp coronal loop followed by a sagittal one overarching the tonsilla to feed the suboccipital cerebellar cortex posteriorly (Miao 2020, Plasticsurgerykey 2023). The PICA's segment before this siphon is prone to inflammations and aneurysms, as if immune cells intruding along the adventitia should be kept off the cerebellum (Coisne 2013, Treviranus 2020), yet territorial cerebellitis of both occurs (Orman 2021). Such is suggested by the CIMURAF-model explaining the rapid reverse intramuroarterial cortical interstitial clearance of Abeta (Szentistvanyi 1984, Cserr 1974-1984, Carare 2008-2023) via the first arterial macro-bio-mechanics (Treviranus 2012, 2018). The adventitial integrin-driven migration routes along calvario-meningeal vessels, newly discovered by oncologists (Yao 2018, Winkler 2018), contribute strongly to a new also non-neural psychiatry (Segawa 2021). While the core of SCZ seems largely caused by such hallucinatory or pathomodulatory cortical events, mast cells may prove key to the "p-factor". They release (drained) vesicles damaging cytoskeletons (as probably in celiac disease) not only of oligodendrocytes through tryptase (Medic 2009), but likely also of axonal dendrites, transiently destructured during Cortical Spreading Depression (Kirov 2020, Dussor 2019). The derided premonitory localizing concepts of F. J. Gall (Finger 2010), possibly related to "bumpy" calvarial tuberculosis spreading colonized mast cells along the calvario-meningeal vessels or to clinical psychiatric associations with smoldering intracellular ORL-infections (Treviranus 2022). In cerebellitis microglial weakness adds to Purkinje cells` overstimulation (Yamamoto 2019) (Figure 3).

CEREBELLAR TDCS AT HOME?

Non Invasive Brain Stimulations (NIBS), however they might work (Singh & Kar 2016), are prime neuropsychiatric therapies. High frequency quite individual induced electric fields, the longer the more, increase many brain tissues, but not the left cerebellum (Deng 2023). The left lobulus VIIa showed more perfusion in MDD-patients, while ECT added more to VIIa and VIIIb, while reducing cortical coupling of lobules VI/VII (Depping 2018).



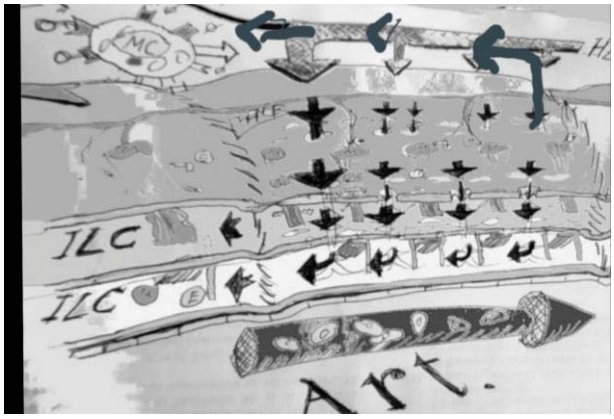


Figure 3. Cerebellar arteries: the PICA shows a biphasic, the AICA a sole loop, which seem apt to hinder adventitial of immune (mast) cells predicted by the CIMURAF-model (⬇) of muscle cells mainly providing constricting co-axial torsion segmenting the Interlaminar Compartments (ILC) into sliding chambers. Their radial channels should create adventitial reverse flows attracting Mast Cells (Treviranus 2019)

Implicit learning of sequences improves after semi-hidden repetitions in contrast to slowing hesitations from disruptions to expected sequences of a certain short length. While curve fitting or AI-prediction are being questioned (Hennings 2022), sliding configural frequency mediation counts each pattern as a discrete string inhabited by "types" (confreq in R; Treviranus 2018), "as if" by a hidden cerebellar algorithm. By recognizing repetitive sequential social patterns and updating models the Crus-I might anticipate subsequent social events offering ready-made behavioral formulas to ongoing interactions: from the plain comfort zone to mentalizing others. After posteromedial cerebellar tDCS (Ma 2023) the learning of "cognitive" sequences indeed sped up for days, while alternative "socially" conceivable perceptions of others further anteriorly (lobuli VI/VIII) did not (Figure 4).

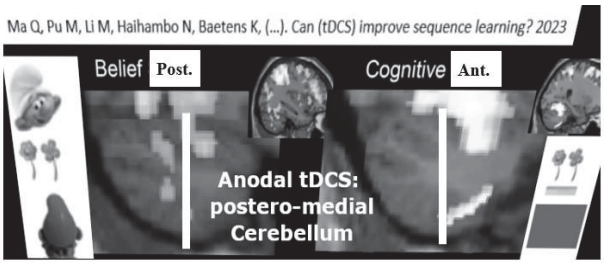


Figure 4. Serial reaction times and BOLD-signals differed depending on the mastering of series with "social" (Crus-I) or anterior "cognitive" contingencies (Ma 2023). When counting flowers (not leafs) only "Murph"'s last count while turning to them is valid. Similarly, a blue square, but not a black circle, convalidate

Different tDCS seems able to stimulate also social sequences (Catoira 2023). Perspective taking improved only after anodal tDCS on the vermis stimulated the posterior portion (Clausi 2020), which also improves semantic tasks (D'Mello 2017) and activates the right Crus-I/-II (Rice 2021). Responses to "rapid antidepressants" sustain concepts of rapid repair. Among many proposals (See Table 1 – Putative Mechanisms (…) in: Treviranus 2019), while IL-6 is dampened in serum (Belge 2020), NIB-stimulations plausibly might act by electrically flushing away cytokine dipoles like IL-1, which is crucial for the activity of pathogenic mast and other cells drawn in by a complex arterial machinery mainly active along straight segments. The paths of least electrofluidic resistance followed should show upon the new NODDI-MRI imaging, differentiating hindered from free diffusion e. g. in neurodegenerative asthma (Nair 2023). Anodal tDCS on the M1-region inhibits, whereas cerebellar stimulation facilitates corticospinal excitation (Behrangrad 2022). Anodal or cathodal tDCS in- or decrease CBF (Zheng 2011).

CEREBELLUM: SICK FROM ITS BOX?

Intracranial infections from common bacteria (Patel 2020, Douglas 2022) and ORL-infections like frontal sinusitis and otitis may spread intracranially causing abscesses, arterial damage, lymphophlebitis, and osteomyelitis (SBO) of the skull base (Nowinski & Thaung 2018). Loose periosteum attracts abscesses from otomastoiditis or favors cysts, which are more common (1.4%) in the arachnoid (AC; J. Lü 2015). ACs near the cerebellum relate to cognitive decline, psychosis and violence, when still pressurized, while depressing cortical CBF (Škarić 2021) (Figure 5).

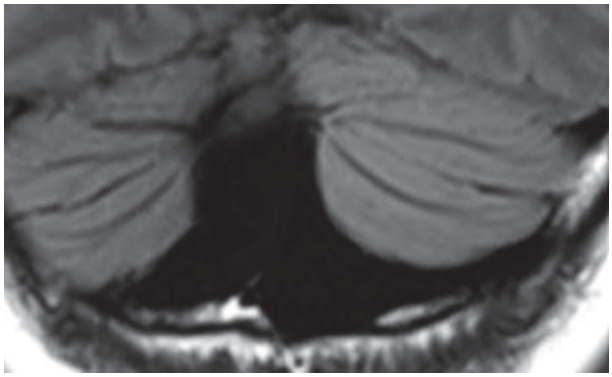
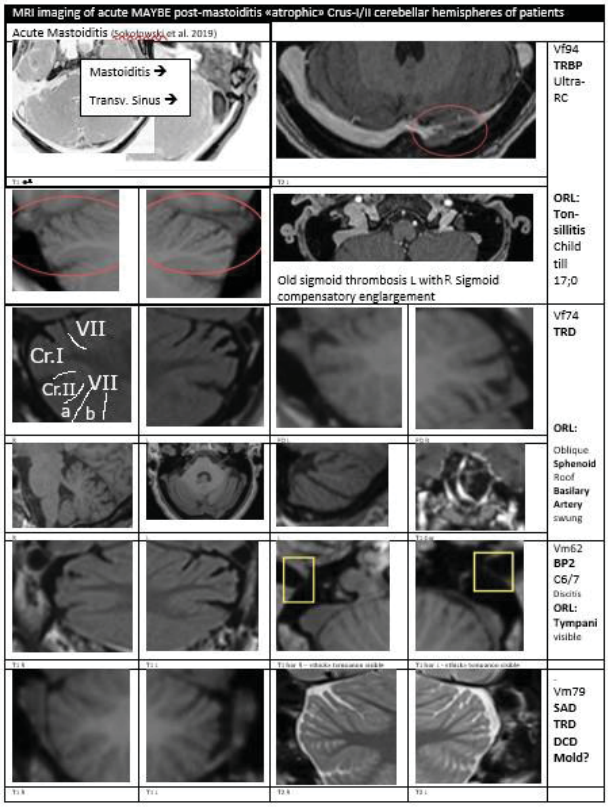


Figure 5. Vm83 "war-child" with F38, F4 PTSD, F11.7, aggressivity, arachnoid cyst

Immunesilencing subacute variants merit more consideration since calvario-meningeal vessels and easy bacterial access (Audshasai 2020) were discovered, which along adventitias also transmit processes from meningeal and upstream arteries (Treviranus 2022). Incidental mastoid opacification in 9% points to ORL-infections (Sayal 2018), which for neuropsychiatry could prove crucial. The mastoid sits anterolateral to and beneath the cerebellar hemispheres and the bony borders may yield to inflammation (Platzek 2014) (Figure 6).



TRD: Therapy Resistant Depression. SAD: Social Anxiety Disorder; DCD: Developmental Coordination Disorder

Figure 6. MRI-images of a) acute Mastoiditis (Mod. from: J. Sokołowski Acta Neurol Belg 119:432) and b) Crus-I/-II with apparent thinning and adjacent transverse venous scarring

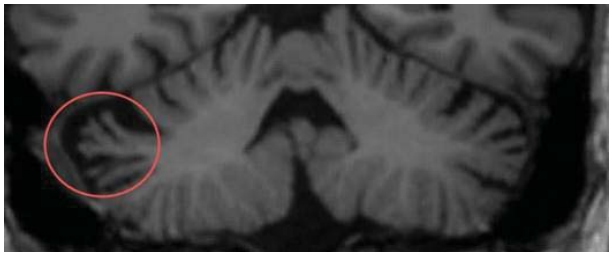


Figure 7. Social phobia and hallucinating ethilism. Cerebellum with Crus-I/-II-dystrophy possibly from bacteria leaving through lympha-tics around nearby transverse sinuses, since ethanol explains the parallel crushed CNS-metabolism (see Fig. 5 on Vm65 with plausible life long M. chelonae. Treviranus 2022) also in cerebellum (Rapp 2022). Young binge drinkers show "cerebellar" social dysfunctions while bacteria from the gut (Carbia 2023) plausibly translocate into the lymphatics and immune cells, which after crossing into the adventitial pathways plausibly reach the cerebellum also via skull and meniniges

Polymorbidity is challenging along untread paths, and ethylism a common companion (Figure 7).

Cerebellitis may well be due to coeliac disease, whereby mast cells (Frossi 2019), colonized by Candida (Renga 2019), stimulated by gliadin, plausibly liberate tryptase, destabilizing the cytoskeletons (Discepolo 2021), as when provoking CSD-migraine, cause 60% more neuropsychiatric issues (Qasim 2022).

Hyperalert microglia and Bergmann glia around PCs, preventing excitotoxicity, makes the cerebellum resilient, but vulnerable to paraneoplastic (or primary) attacks with, but maybe not through, many autoantibodies. These stem from infections out- or inside the brain, often colonizing marrow-derived immune cells. They having their antigens presented to T-cells often within the rapidly renewed, omnipresent CSF (Mitoma 2021).

CONCLUSION

Cerebellar psychiatry is rapidly expanding and merits interest from clinicians as does its partaking in the multiscale infection-immuno-medical integration involving brain's skull and arterial and other logistics.

Acknowledgements: *None.*

Conflict of interest: *None to declare.*

References

1. *Anderson CM et al.: Abnormal T2 cerebellar vermis of adults sexually abused in childhood: Psychoneuroendocrinology 2002; 27:231-44*
2. *Arrondo G et al.: mental and physical conditions in children and adolescents. Neurosci Biobehav Rev 2022; 137:104662*

3. *Audshasai T et al.: Streptococcus pneumoniae rapidly the cribriform plate to invade meninges. mBio 2022; 13:e01024222*
4. *Bareš M et al.: Consensus paper: cerebellum as a time machine. Cerebellum 2018; 18:266–86*
5. *Becker MPI et al.: ventral striatum in social anxiety disorder. Psychol Med 2017; 47:2502-2512*
6. *Behrangrad S et al.: anodal tDCS of primary M1 and cerebellum on corticospinal. Brain Struct Funct 2022; 227:2395-408*
7. *Belge JB et al.: Inflammation, hippocampal volume, and outcome following ECT in depressive. Neuropsychobiol 2020; 79:222-232*
8. *Beuriat PA et al.: cerebellum in executive, emotional, social lifespan. Behav Brain Funct 2022; 18:6*
9. *Cabaraux P & Monti M: The Three Cornerstones of the Cerebellar Syndrome. Chp.74 In Gruol DL: Essentials 2023: 469-*
10. *Carare RO et al.: Solutes, but not cells, drain from the brain parenchyma along basement membranes. Neuropathol Appl Neurobiol 2008; 34:131-44*
11. *Carbia C et al.: microbiome-gut-brain axis social cognition young binge drinkers. EBioMedicine 2023; 89:1044422*
12. *Catoira B et al.: cerebellum on social sequences: a tDCS-fMRI pilot study. Int J Clin Health Psychol 2023; 23:100373*
13. *Cervone A, D'Ostuni FP, D'Aietti E, Esposito G, Masella M & Tavormina G: Mixed states: diagnosis, assessment and diagnostic stability. Psychiatr Danub 2022; 34(Suppl. 8):S38-41*
14. *Ciapponi C et al.: cerebellum and emotional processing. Front Syst Neurosci 2023; 17:1185752*
15. *Cignetti F et al.: Intrinsic cortico-subcortical dyslexia and D Coordination D. Cereb Cortex Commun 2020; 1:tgaa011*
16. *Clausi S et al.: MRI autism and cerebellar neurodegenerative. Autism Res 2021; 14:2300-13*
17. *Coisne C et al.: T cell trafficking across the blood-brain barrier. Fluids Barriers CNS 2013; 10:7*
18. *Dekeyzer S et al.: anatomy and pathology of the cerebellum. Clin Neuroradiol 2023*
19. *Deng ZD et al.: ECT-induced volumetric brain changes common causal circuit in depression. Res Sq 2023:rs.3.rs-2925196. doi:10.21203/rs.3.rs-2925196/v1*
20. *Depping MS, Schmitgen MM: Cerebellar major depression. Front Psychiatry 2018; 9:634*
21. *Diederichsen J: www.diedrichsenlab.org/SUIT.htm, 2023*
22. *Dijkstra BW et al.: Functional neuroimaging of postural control. Neurosci Biobehav Rev 2020; 115:351–62*
23. *Discepolo V et al.: Pediatric celiac disease dendritic cell shape and actin. Int J Mol Sci 2021; 22:2708*
24. *D'Mello AM et al.: Cerebellar tDCS semantic prediction. J Neurosci 2017; 37:1604-13*
25. *Douglas JE et al.: Odontogenic sinusitis extra-sinus infectious complications. Am J Rhinol Allergy. 2022; 36:808-15*
26. *Dussor G: migraine mechanisms and therapeutic targets. Curr Opin Physiol 2019; 11:116-124*
27. *Eccles JC, Ito M, Szentágothai J: The cerebellum as a neuronal machine. Springer, Heidelberg, 1967*
28. *Ekinci O et al.: Sluggish cognitive tempo with autistic traits and anxiety disorder ADHD. Braz J Psychiatry 2021; 43:153-9*
29. *Fatemi SH (ed.): Neuropsychiatric Disorders and Infection. Taylor & Francis: Abingdon UK, 2005*
30. *Fatemi SH et al.: Consensus: cerebellum in autism. Cerebellum 2012; 11:777-807*
31. *Finger S, Boller F, Stiles A (eds.): Literature, Neurology, and Neuroscience. Progr Brain Res 2013; 206. ISBN 10: 0444633642*
32. *Finger S: Chapter 10: the birth of localization theory. Handb Clin Neurol 2010; 95:117-28*
33. *Fredrick JW & Becker SP: Cognitive Disengagement Syndrome (SCT). J Atten Disord 2023; 27:38-45*
34. *Frossi B et al.: Coeliac disease and mast cells. Int J Mol Sci 2019; 20:3400*
35. *Gruol DL et al. (eds.): Essentials of Cerebellum and Cerebellar Disorders. Springer, Cham, 2023*
36. *Guell X & Schmahmann J: Cerebellar functional anatomy. human fMRI. Cerebellum 2020; 19:1-5*
37. *Hariri AR: Cerebellum in broad risk for psychopathology. Neuron 2019; 102:17-20*
38. *Herculano-Houzel S: The human brain scaled-up primate brain. Front Hum Neurosci 2009; 3:31*
39. *Herisson F et al.: Direct vascular channels connect skull bone marrow and the brain surface enabling myeloid cell migration. Nat Neurosci 2018; 21:1209-17*
40. *Hughes DE et al.: Genetic child psychopathology fetal cerebellar development. Nat Neurosci 2023; 26:959-69*
41. *Hull C & Regehr WG: The cerebellar cortex. Annu Rev Neurosci 2022; 45:151-755*
42. *Ito M. Control of mental activities by internal models in the cerebellum. Nat Rev Neurosci. 2008; 9:304-13*
43. *Karam EG et al.: The role of affective temperaments in bipolar disorder. Eur Psychiatry 2023; 66:e37.6*
44. *Kirov SA et al.: Rapid neuronal disruption and recovery during spreading depolarization. Cereb Cortex 2020; 30:5517-5531*
45. *Koziol LF et al.: Consensus paper: the cerebellum's role in movement and cognition. Cerebellum 2014; 13:151–77*
46. *Lü J: Arachnoid membrane. Surg Radiol Anat 2015; 37:127-38*
47. *Luyten P et al.: The mentalizing approach to psychopathology. Annu Rev Clin Psychol 2020; 16:297-325*
48. *Ma Q et al.: (tDCS) of the cerebellum improve implicit social and cognitive sequence learning? Int J Clin Health Psychol 2023; 23:100355*
49. *Manto MU & Shaikh AG (eds): Predictive cerebellocerebral. Front Cell Neurosci 2019; 13:549*
50. *Matkovič A et al.: Static and dynamic functional connectomes similar information. bioRxiv 2023; 2023.01.24.525348*
51. *Mayes SD et al.: Cognitive Disengagement Syndrome autism, and insomnia in childhood predict CDS. Child Psychiatry Hum Dev 2023*
52. *Medic N et al.: Mast cell adhesion induces cytoskeletal death in oligodendrocytes. J Neuroimmunol 2010; 218:57-66*
53. *Meisler SL & Gabrieli JDE: Fiber-specific structural properties relate to reading Elife 2022; 11:e820888*
54. *Metoki A et al.: The social cerebellum: (...) connectivity. Cereb Cortex 2022; 32:987-1003*
55. *Miao HL et al.: posterior inferior cerebellar artery. Int J Med Sci 2020; 17:3005-19*
56. *Mitoma H et al.: Immune-Mediated Cerebellar Ataxias. J Mov Disord 2021; 14:10-28*
57. *Nair AK et al.: asthma on the brain: diffusion MRI, CSF biomarkers and cognitive. Brain Commun 2023; 5:fcad180*
58. *Nivins S & Klingberg T: maternal diabetes on deep grey matter ADHD. Acta Paediatr 2023; 112:1511-23*

59. *Nowinski WL & Thaung TSL: A 3D stereotactic atlas of the adult human skull base. Brain Inform 2018; 5:1*
60. *Olivito G et al.: Theory of mind cerebellar alterations in bipolar disorder 1 and 2. Front Behav Neurosci 2022; 16:971244*
61. *Olson IR et al.: cerebellum in social development. Dev Cogn Neurosci 2023; 60:101238*
62. *Orman G et al.: MRI infectious and immune-related acute cerebellitis. AJNR Am J Neuroradiol 2021; 42:2231-7*
63. *Patel K et al.: Eikenella corrodens and Streptococcus anginosus skull base osteomyelitis artery lesion. IDCases 2020; 20:e00740*
64. *Plasticsurgerykey [https://plasticsurgerykey.com/ three-cerebellar-arteries] with permission 2023*
65. *Porcelli S et al.: Social brain, social dysfunction and withdrawal. Neurosci Biobehav Rev 2019; 97:10–33*
66. *Qasim H et al.: Dysbiosis and migraine adults with celiac disease. Cureus 2022; 14:e28346*
67. *Rapp C et al.: Alcohol binge drinking decreases brain glucose metabolism rats. Metab Brain Dis 2022; 37:1901-8*
68. *Renga G et al.: Candida albicans and celiac disease. Front Immunol 2019; 10:2844*
69. *Rice LC, D'Mello AM, Stoodley CJ: Differential behavioral and neural effects of regional cerebellar tDCS. Neuroscience 2021; 462:288-302*
70. *Rizzolatti G et al.: Cortical goal-directed mirror neuron-based action. Physiol Rev 2014; 94:655-706*
71. *Sayal NR et al.: Incidental mastoid effusion diagnosed on imaging Laryngoscope 2019; 129:852-7*
72. *Schmahmann J et al.: The theory and neuroscience of cerebellar cognition. Annu Rev Neurosci 2019; 42:337–64*
73. *Schmahmann JD: Dysmetria of thought: cerebellar cognition and affect. Trends Cogn Sci 1998; 2:362-71*
74. *Segawa K et al.: A destruction model of the vascular and lymphatic emergence of psychiatric. Biology (Basel) 2021; 10:34*
75. *Selzam S et al.: A polygenic p factor for major psychiatric disorders. Transl Psychiatry 2018; 8:205*
76. *Shaw Pet al.: (...) longitudinal cerebellar development in ADHD. J Child Psychol Psychiatry 2018; 59:1114-23*
77. *Singh A & Kar SK: How ECT. Clin Psychopharmacol Neurosci 2017; 15:210-21. [www.cpn.or.kr/journal/ download.pdf.php?doi=10.9758/cpn.2017.15.3.210]*
78. *Škarić M et al.: Cognitive and psychotic symptoms infratentorial arachnoid cyst. Acta Clin Croat 2021; 60:304-308*
79. *Smeland OB: schizophrenia - rethinking pathogenesis and nosology. Nat Rev Neurol 2020; 16:366-379*
80. *Sokołowski J et al.: Skull base osteomyelitis: outcome. Acta Neurol Belg 2019; 119:431-7*
81. *Sorella S et al.: continuum hypothesis of schizophrenia and bipolar disorder. Neuroimage Clin 2019; 23:101854*
82. *Tavormina GRS: From the temperaments to the bipolar mixed states. Psychiatr Danub 2021; 33(Suppl. 9):S6-10*
83. *Treviranus GRS: Immunopsychiatry of infected ears, skulls. Psychiatr Danub 2022; 34(Suppl. 8):S265-75. www.psychiatria-danubina.com/UserDocsImages/pdf/ dnb_vol34_noSuppl%208/dnb_vol34_noSuppl%208_265.pdf*
84. *Treviranus GRS: Mast cell autocrinicity near cerebral arterial wall target of electromagnetic. Psychiatr Danub 2019; 31(Suppl. 3):S357-70. www.researchgate.net/publication/336776494*
85. *Treviranus GRS: Psychoses by attacks from subverted mast cells arterial intramural flow nasal ganglia? Psychiatr Danub 2020; 32(Suppl. 1):S93-104. www.psychiatriadanubina.com/UserDocsImages/pdf/dnb_ vol32_noSuppl%201/dnb_vol32_noSuppl%201_93.pdf*
86. *Treviranus GRS: Rescue of the appropriative "Thought-Action-Mood" anatomy and mast cells language and statistics. Psychiatr Danub 2018; 30(Suppl. 7):S620-9. www.psychiatria-danubina.com/UserDocsImages/pdf/ dnb_vol30_noSuppl%207/dnb_vol30_noSuppl%207_620.pdf*
87. *Theoharides TC: perinatal mast cell focal brain inflammation autism. J Pers Med 2021; 11:860*
88. *Valença AM et al.: Do social anxiety disorder patients belong to a bipolar spectrum subgroup? J Affect Disord 2005; 86:11-8*
89. *Van Overwalle F et al.: crus II cerebellum mentalizing and emotional self (..). Soc Cogn Affect Neurosci 2020; 15:905-28*
90. *Van Overwalle F et al.: Consensus: cerebellum and social cognition. Cerebellum 2020; 19:833-8681*
91. *Vergadi E et al.: Acute mastoiditis cerebral venous sinus thrombosis in children. Int J Pediatr Otorhinolaryngol 2021; 141:1105088*
92. *Winkler F: Leukaemia follows a blood-vessel track to enter the nervous system. Nature 2018; 560:35-36*
93. *Yamamoto M et al.: Microglia-triggered psychomotor acute cerebellar inflammation. Cell Rep 2019; 28:2923-38.e8*
94. *Yao H et al.: Leukaemia hijacks invade the central nervous system. Nature 2018; 560:55-60*
95. *Zanchetti A & Zoccolini A: Autonomic hypothalamic cerebellar stimulation. J Neurophysiol 1954; 17:475–83*
96. *Zheng X, et al.: (tDCS) on human regional cerebral blood flow. Neuroimage 2011; 58:26–33*

Correspondence:
Gottfried R.S. Treviranus, MD
BipoSuisse - Psychiatrische Praxis
Vereinsweg 11, CH 3012 Berne, Switzerland
E-mail: biposuisse@bluewin.ch