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Sesquicentennial Tribute to Emil Redlich (1866–1930), the 'Embodied Conscience of Neurology'

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Running Title:

Professor Emil Redlich (1866–1930)

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Abstract

Professor Emil Redlich (1866–1930) of the University of Vienna was born 150 years ago. Raised in a humble environment, he became an eminent researcher and neurology scholar, and succeeded in laying some of the key foundations of neuroanatomy and neuropathology. His name is linked to medical eponyms that define the dorsal root entry zone into the spinal cord, epidemic disseminated encephalomyelitis, narcolepsy, senile plaques and dementia. As its first director, he managed to organize the Maria-Theresien-Schlössel into a first-class neuropsychiatric hospital. The one attribute that his colleagues constantly recognized was his inexorable scientific demeanor, always insisting on a critical checking of the facts before formulating any hypothesis.

Key Words

History of neurology · History of neuropathology · Emil Redlich · Heinrich Obersteiner · Vienna school of neurology

Introduction

This paper pays tribute to Emil Redlich, a notable representative of the Vienna school of neurology, on the occasion of the sesquicentennial of his birth (on January 18, 1866). At the time of his death, many laudatory testimonies were given by his eminent colleagues, including Julius Wagner von Jauregg (1857–1940), Hermann Schlesinger (1866–1934), Alexander Pilcz (1871–1954), Otfrid Foerster (1873–1941), Otto Marburg (1874–1948), Constantin von Economo (1876–1931), Erwin Stransky (1877–1962) and Joseph Wilder (1895–1976) [1–9]. They all expressed the unfillable void left in the scientific base of Viennese neurology.

Despite his inclusion as one of the pioneers of neuropathology in the original 'Founders of Neurology' [9], his biography was deleted from the second (1970) edition of that compilation. Since then, not much has appeared in print in the biomedical literature on him.

Emil Redlich (fig. 1) has been considered for Vienna what Hermann Oppenheim (1858–1919) had once been for Berlin: an international representative and great medical consultant for nervous diseases [8]. Redlich had full command of the neurological literature, and strictly opposed unsubstantiated statements and vague theories [9]. An attribute that Redlich's colleagues repeatedly emphasized was his insistence on critically checking the facts [8]. Prompted by the rigorous scientific demeanor and personal honesty that epitomized him, fellow neurologists called Redlich the 'embodiment of neurological conscience' *(Die Verkörperung des neurologischen Gewissens)* [7], the 'critical conscience of the Viennese' [6] and the 'living conscience of neurology' [9].

In the only obituary that Economo ever wrote [7], he explained that, of all branches of biomedicine, it is neurological diagnosis that comes the closest to the exact sciences; whoever practices it, must almost possess the precision of a mathematician. Contemporary neurology, with its auxiliary scientific branches, represents a vast, firmly established edifice that presumes a tremendous amount of work, diligence and memory from anyone engaged in it. Economo credited Redlich as one of the foremost architects of that edifice, having laid rock-solid foundations and some of the supporting pillars, which retain their value in the test of time.

Academic Career

A native of Brünn, Moravia (today Brno, Czech Republic), Redlich was the son of Hermann and Johanna (née Wagner) Redlich [10]. He grew up in poor conditions, but forced himself through obstacles with perseverance, an iron energy and unrelenting diligence [3, 8]. Redlich entered the University of Vienna in 1883 and obtained his M.D. in 1889. In the following three years, he carried out neuroanatomical research work at the Neurological Institute of Heinrich Obersteiner (1847–1922). Obersteiner recognized Redlich's talent in neuroanatomy and neuropathology at the outset [8]. Absolute sincerity, extensive command of the literature, and a striking critical ability were the outright hallmarks of the young apprentice [6]. Redlich concomitantly worked in several departments of the *Allgemeines Krankenhaus* as an unremunerated resident *(Aspirant)* [1].

In 1892 Redlich became Assistant Physician *(Sekundararzt)* at Vienna City's *Versorgungsanstalt*, where he had the opportunity to perfect his diagnostic skills with a wealth of clinical material [3]. His colleagues observed the young physician, immersed with eagerness pursuing clinical examinations late into the night, meticulously studying each case history and taking notes by stenography [3]. Two years later, he was habilitated *(Venia legendi)* in neurology, based on the anatomical work conducted at Obersteiner's Institute, and secured a travel stipend from the Oppolzer-Stiftung that allowed him to train for a few months in Paris under Jules Déjerine (1849–1917) and Pierre Marie (1853–1940). At about that time, in June 1894, Redlich lost his elder sister, Fanny Cohn (née Redlich, 1863–1894), at the age of 30 years [10].

Upon his return to Vienna in 1895, Redlich became an Assistant to Wagner von Jauregg, for a three-year period, at the Clinic for Mental and Nervous Diseases [1, 2]. In 1897 he was habilitated in psychiatry [11]. In the fall of 1898, when the Director of the *Privatirrenanstalt Inzersdorf*, Emil Fries (1844–1898), died, Redlich took charge of that post (until 1903); he also became Chief of Nervous Diseases at the *Mariahilfer Franz-Joseph-Ambulatorium* (through 1908) [1]. In December 1901 Redlich married Amalia Rudinger (née Zuckerkandl), the younger sister of the Hungarian-Austrian anatomist Emil Zuckerkandl (1849–1910) [12].

In March 1914 Redlich was appointed Director of the *Nervenheilanstalt Maria-Theresien-Schlössel* in Vienna, a top-ranking neuropsychiatric hospital constructed and equipped based on his own suggestions and funded by the Rothschild-Stiftung. He managed and organized a model institution: this post offered Redlich an opportunity to continue his work independently and to found his own school of neurology, with an emphasis on neuropathology, and with many respected names among his alumni. Redlich knew how to present a complicated topic in ways easily understood [7], and he generously instructed young researchers in neuroanatomy during off-duty hours [4]. The institute attained international renown as a place of vital scientific research and successful medical consultation [3, 6].

With the outbreak of World War I in July 1914, the facility was used as a military hospital; Redlich was assisted in surgery by Anton von Frisch (1849–1917) and in neuropsychiatry by Johann Paul Karplus (1866–1936) [1].

Redlich resided and kept his private practice at Schlösselgasse 15 in Vienna's Eighth District. He became one of the most prominent neurologists on Viennese soil, attracting patients from far away. He possessed excellent diagnostic skills and enjoyed great popularity among colleagues; his patients relied on their physician with confidence [3, 7]. His practice grew such that he could lead a carefree existence [1]. At the Medical Faculty of Vienna University, Redlich was appointed Assistant Professor (1900), Associate Professor (1906) and full Professor (1922) [1, 5, 6, 9, 13].

Emil Redlich died in Vienna on June 7, 1930. A special session was held at the Society for Psychiatry and Neurology on June 17, 1930, with Wagner von Jauregg delivering the commemorative speech [1]. Josef Gerstmann (1887–1969) succeeded Redlich as Director of the *Maria-Theresien-*

Schlössel, until his forced emigration to the United States on June 14, 1938 [14–16]. It was during that period (1935–1936) that Gerstmann, together with neuropathologists Ernst Sträussler (1872–1959) and Ilya Scheinker (1902–1954), described the familial neurodegenerative disease that today bears their names [16].

In their tributes, Viennese neurologists underlined Redlich's good human nature, citing the *Odes* of Horace (*integer vitae scelerisque purus*, 'flawless in life and pure of sin') [8], and *Persa* of Plautus (*nomen est omen*, 'the name speaks for itself') [7]. Foerster [5] concluded his homage to Redlich on September 18, 1930 at the 20th Annual Meeting of the Society of German Neurologists (on the executive board of which Redlich had served as a member) with the comment, 'As long as there will be a neurological science, Redlich will live on'. A modest man, Redlich never accepted the offer that the title of Court Councillor *(Hofrat)* be conferred upon him [9, 13]. An outlet from the mundane was his love for the arts, including painting, music, and art history [4, 9].

After the *Anschluss*, Amalia ('Amal') Redlich was deported to the Łódź (Litzmannstadt) Ghetto in German-occupied Poland on October 23, 1941; she died in the Holocaust [10].

Scientific Works

There is hardly an area of neurology that Redlich did not touch [7]. Wilder [17] compiled an index of 138 scientific papers from Redlich's pen, published between 1891 and 1930, spanning across diverse themes. (Wilder had worked for many years at the *Maria-Theresien-Schlössel* under Redlich, whom he revered greatly; following the death of his mentor in 1930, Wilder became Associate Director of the institution). The actual number of Redlich's works exceeds 140, if one includes the synopses and chapters in manuals and textbooks and the clinicopathological presentations in the proceedings of medical conferences.

Nearly all of Redlich's scientific works are important; some of them constitute an inextricable part of the growth of neurology and psychiatry [8]. His writings reveal, again and again, his yearning to illuminate and unveil the physical basis of nervous and mental diseases [7].

In neuroanatomy, Redlich made important contributions, including studies on the cingulum [18] and the inferior longitudinal fasciculus [19]. At the suggestion of Wagner von Jauregg [3], he conducted extirpation experiments in cats (fig. 2) to study motor pathways [20, 21]. Meyer [22] credits Redlich [20] with being the first researcher to trace, in cats, postcentral fibers running in the corticospinal tract to the gracile and cuneate nuclei, an observation subsequently confirmed by other investigators and extended to additional sensory nuclei of the tegmentum and spinal cord.

In a comparative study, Redlich, with Obersteiner, conducted ablation-degeneration experiments [23] and differentiated the subcallosal stratum ('bundle of Muratoff') from a stratum zonale that lies adjacent to the caudate nucleus, and also from the fronto-occipital fasciculus (fig. 2). Nonetheless, while they considered the latter to be a 'reticular cortico-caudate bundle', modern tract-tracing experiments have confirmed Déjerine's original thesis that the fronto-occipital fasciculus actually

represents a long cortical association fiber tract, which as a matter of fact connects dorsolateral premotor and prefrontal areas to parieto-occipital cortical regions [24]. We also know today that the bundle of Muratoff connects the occipital, parietal, temporal, frontal lobe, and the cingulate gyrus, to the caudate nucleus, being a strictly corticostriatal bundle; the fronto-occipital fasciculus only contributes a few fibers to the bundle of Muratoff, which end in the striatum [24].

Redlich possessed a tedious method of clinico-pathological correlation, combining anatomy, histology, pathophysiology, and therapy. With pathologist Alexander Kolisko (1857–1918), they published a manual with brain schemes [25] for neuropathologists to draw autopsy finds (fig. 3).

Most of Redlich's clinical papers are single-authored. His occasional co-authors include H. Obersteiner, D. Kaufmann, G. Bonvicini, A. Schüller, C. von Economo, O. Pötzl, L. Hess, O. Binswanger, E. Lazar, J.P. Karplus, and J. Wilder.

His first publication [26], coming from Obersteiner's Institute, dealt with the degeneration of the dorsal columns of the spinal cord of vascular etiology. His second paper was on the genesis of *corpora amylacea* in the CNS, and one of his last papers also dealt with the occurrence of *corpora amylacea* in neurons of the substantia nigra in postencephalitic Parkinsonism [8, 17].

Redlich published one of the earliest cases of complete alexia without agraphia, which he documented with macroscopic and histological autopsy results [27, 28]; it had only been preceded by the reports of Edmund Landolt in 1888 and Jules Déjerine in 1892 [29]. Redlich demonstrated, in his 64-year old patient, a generalized atrophy of the brain, with lesions of the left calcarine sulcus, lingual and fusiform cortex, splenium, posterior thalamus, and inferior longitudinal fasciculus.

He contributed a paper 'On the clarification of paralysis type in cerebral hemiplegia' [30] to the *Festschrift* for Richard von Krafft-Ebing (1840–1902) on the occasion of the 30-year anniversary of the latter's professorship. He carried out innovative work and made valuable contributions to virtually every chapter of neurology, and some in psychiatry, the more outstanding of which relate to idiopathic and postencephalitic Parkinsonism [31], mental disturbances in meningitis [32], tumors of the CNS [33, 34], acute disseminated encephalomyelitis and transverse myelitis [35], and tetany [36]. In addition, he published on syringomyelia, tabes, metalues and progressive paralysis, diabetic hemiplegia, acute infantile poliomyelitis, infantile gigantism, disturbances of the pupillary and corneal reflex, and auditory hallucinations [17]. At the 1901 Congress for Internal Medicine in Berlin he presented, alongside the great Ernst von Leyden (1832–1910) and Adolf von Strümpell (1853–1925), a report on acute myelitis, and already at that time, emphasized its special character and difficulties in its pathological-anatomical diagnosis [6]. From *Maria-Theresien-Schlössel*, Redlich reported two clinical cases of pituitary tumors with reference to Simmonds hypophyseal cachexia [37].

Redlich investigated the neuroses early on, as he always held the view that their anatomical basis would provide leads to many questions. He tried to show that what was called neurosis, i.e. a disease without anatomical substrate, was actually a well-defined organic disease, by revealing more of its neuropathological underpinnings [38]. He further reported on brain changes in psychoses [39].

The study of multiple sclerosis and its pathology, as well as its relation to psychiatry and congenital syphilis, also occupied Redlich's mind (fig. 4) at a time when little was understood about the disease. Redlich [40] actually associated the insult as a toxin or microorganism induced primary demyelination, with the inflammation and the vascular changes being secondary [41]. With Economo, they presented histological findings from a young woman with multiple sclerosis with psychosis in March 1909 at the Society for Psychiatry and Neurology in Vienna [42]. At the 3rd Annual Meeting of the Society of German Neurologists, held in Vienna in September 1909, Redlich and Economo presented findings from several autopsies, including a case of meningitis with a tubercle specimen showing plasma cells in metachromasia, and a case clinically diagnosed as multiple sclerosis, but proven upon pathological examination to be a combined degeneration of the cerebellum, pons and red nucleus [43]. Between 1914 and 1919, Redlich became occupied with war neurology [3, 17] and studied grenade explosion wounds, gunshot injuries of the peripheral nerves, traumatic neuroses, and epilepsy after head injuries [44] (fig. 3).

By studying six cases of pontine and cerebellar encephalitis in young men [45, 46], Redlich drew in a precise and concise manner its clinico-pathological picture (fig. 4). Cases in the literature involving the midbrain and hindbrain had been rare. He classified them into two groups based on their clinical course: an acute form, comprising the majority of cases and resulting from acute infections, that eventually ended in improvement or recovery; and a subacute form, with a course of weeks to months, that ended either in recovery or exitus. Scanning speech, difficulties in swallowing, cerebellar ataxia, and adiadochokinesia were accompanied by hemihypesthesia and crossed hemiplegia; sensory disturbances were rare, but cranial nerves III, V, VI, VII or VIII were occasionally damaged. In a note added in proof, Redlich [45] noted a similarity of his cases to poliomyelitis, and also to certain atypical forms of encephalitis lethargica [47]. Around the 1920s, multiple types of epidemic encephalitides were observed, including Redlich's cases, Albrecht's encephalomyelitis migrans, the Japanese epidemics, and acute forms of multiple sclerosis; Economo made the distinction among all those entities before the International Neurological Congress, held in Berne, in what would be his last scientific presentation [48].

Redlich's neuropathological works and their diversity were consistently crowned with success. He turned to the theme of epilepsy in 1900, making key advances [6, 8], such as the discovery of senile plaques in epilepsy of the elderly [49]. Epilepsy was the theme that Redlich revisited repeatedly for the rest of his life [44, 50, 51], publishing, altogether, 30 papers on its pathogenesis, etiology, prognosis, treatment, laterality [52] and left-handedness [53], CSF analyses [54, 55], as well as radiological findings, the implication of alcoholism, pituitary tumors, malaria, and migraine [17]. In an extensive report, with Binswanger, before the Society of German Neurologists, he examined the question of epilepsy; a yet another long report, which found him alongside Bumke in another annual meeting of the Society, won him great acclaim [6].

Neurological Eponyms

Redlich's discoveries led to eponyms in neuroanatomy, neurology and neuropathology.

Redlich-Obersteiner Zone

An early study by Redlich from Obersteiner's Institute was on 'The dorsal roots of the spinal cord and the pathological anatomy of tabes dorsalis' [56], where he discussed the normal structure of the dorsal roots and dorsal spinal columns, and the involvement of the dorsal columns and behavior of the grey matter in tabes dorsalis (fig. 5). He continued to carry out those studies with Obersteiner [57], culminating in a comprehensive monograph on 'The pathology of tabetic dorsal column disease' [58]. Altogether, Redlich published a dozen works on tabes. From that work derives the eponym 'Redlich-Obersteiner zone' or 'root entry zone of Obersteiner and Redlich', meaning the entrance point of the dorsal roots into the spinal cord. Redlich and Obersteiner [57, 58] postulated that tabetic degeneration of the dorsal spinal columns begins in the dorsal roots at the point where the root becomes a central tract, i.e. the *locus minoris resistentiae* [6, 59].

Flatau-Redlich Disease or Redlich-Flatau Syndrome

'Redlich syndrome' or 'Flatau-Redlich disease' or 'Redlich-Flatau syndrome' denotes a form of epidemic disseminated encephalomyelitis with scattered lesions across the brain and spinal cord, with a chronic course and mild symptoms, including paresthesias [13, 60]. The condition was independently described by Redlich in Vienna [61] and by Edward Flatau (1868–1932) in Warsaw; Flatau attributed a viral cause to it [62].

Gélineau-Redlich Narcolepsy

The issue of narcolepsy occupied Redlich from 1915 onwards [63, 64]. This sleep disorder has been called 'Gélineau-Redlich narcolepsy' or 'morbus Gélineau-Redlich'. Redlich [65] favored a strict use of the term 'narcolepsy' to include cataplexy and sleep attacks as the core signs [66]. He used the terms *plötzlicher* or *affektiver Tonusverlust* for the sudden loss of muscle tone (or what is now termed 'cataplexy'), and *Körperschlaf* for body sleep [67, 68], and further added hypnagogic hallucinations to its symptoms [68]. Redlich [65] was also the first author to associate narcolepsy with traumatic brain injury [69]. Subsequently, in 1926, the Australian-British neurologist William J. Adie (1886–1935) pinpointed narcolepsy as a specific disease, and brought this nosological entity to general recognition [68, 70]. Redlich's 'Epilegomena on the question of narcolepsy' [71] appeared posthumously, after an important manuscript edited by his pupil Joseph Wilder.

Redlich-Fischer Senile Plaques

It is widely acknowledged that Redlich [72] demonstrated plaques in the brain of patients with senile dementia eight years before Alzheimer described his patient Auguste Deter in 1906 [73–75].

Redlich [72] showed extensive formation of plaques in the cerebral cortex, using carmine red staining (fig. 4). He referred to the condition as 'miliary sclerosis' (from the foci the size of millet seeds) and suggested that the lesions were most likely glial in origin; he raised the question whether they were a primary event or a sequel of the neuronal degeneration, which has been a long-lasting debate [76].

The first account of senile plaques, under the designation *amas ronds*, is credited to Blocq and Marinesco [77], who, working at La Salpêtrière, observed them in the brain of an elderly patient with epilepsy, six years prior to Redlich. Oskar Fischer (1876–1942) in Prague confirmed the diagnostic value of 'miliary necrosis' in senile dementia in 1907; the actual term 'senile plaque' was coined by the Polish neurologist Teofil Simchowicz (1879–1957) in 1911 [78, 79].

In the first half of the 20th century, the eponym 'Redlich-Fischer plaques' was used in Europe [80] and the Americas [81, 82] as a synonym for senile plaques. Vianna [81] traced the evolution of the histological picture of senile dementia from the Alzheimer neurofibrillary changes in the hippocampus to the Redlich-Fischer plaques in the cortex; subsequently, plaques develop in Ammon's horn, and finally neurofibrillary changes take place in the cortex. Divry [80] developed his own opinion that plaques were local deposits of hyaline amyloid material in the cerebral cortex. Soniat [82] found senile plaques to be more numerous in the frontal cortex and Ammon's horn, and microglia was attracted by the remnants of neurons, axons, dendrites, and neurofibrils in the necrobiotic focus, forming part of the plaque.

Redlich-Alzheimer Disease or Marinesco-Redlich-Alzheimer Disease

The doctoral thesis of Roland Tumbelaka (1880–1946) from the University of Utrecht is considered an important monograph on Alzheimer disease [83, 84]. Tumbelaka acknowledged Redlich's precedence in observing pathological cortical changes in two cases [72] eight years before Alzheimer's first paper. Redlich pointed out that, before his time, Obersteiner, Blocq and Marinesco were aware of the existence of such plaques. Moreover, Redlich mentioned homogeneous little spots on the wall of small cerebral vessels. The new point that Alzheimer established with his 1906 study was the intracellular fibrillary changes, to which his name is correctly attached. Tumbelaka argued that, 'If we wish to give a name to the whole clinico-histological picture of this disease, we ought to call it *Redlich-Alzheimer disease*' [83, 84].

Klaas Herman Bouman (1874–1947) in Holland defined 'Marinesco-Redlich-Alzheimer disease' as a form of cerebral parenchymatous involution, characterized by the presence of plaques, fibril degeneration, and gyral atrophy; he conjectured the presence of a substance 'foreign to the brain tissue', a double refractive fatty acid bearing a distinct relationship to the *corpora amylacea*, that mobilized the microglia [85].

Conclusion

In summary, Redlich's impact on today's neurology extends over anatomical concepts, such as the dorsal root entry zone into the normal spinal cord, as well as the clinical and pathological manifestation of disorders, including epidemic disseminated encephalomyelitis, narcolepsy, and senile dementia.

Disclosure Statement

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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Figure captions

Fig. 1 Professor Emil Redlich. Photo by Max Schneider, Vienna. Courtesy: Bildarchiv, Institut für Geschichte der Medizin, Medizinische Universität Wien, Austria. Used by permission and protected by Copyright Law. Copying, redistribution or retransmission without the authors' express written permission is prohibited. Signature from Stransky [8], superimposed digitally.

Fig. 2 Examples from Redlich's anatomical studies.
Upper composite: extirpation experiments in cats [21]. Left half of the plate: **5** Sacral spinal cord after hemisection at the level of the upper cervical cord (the lesion affected the entire left half of the spinal cord and also the anterior funiculus and part of the ventral horn on the right side); *fma*, anterior marginal fasciculus. **6-8** Destruction of pulvinar, anterior tectum and lateral part of the crus cerebri and resulting secondary degeneration. **6** Semischematic drawing of the extent of the lesion *(L)*. **7** Section through the cerebral peduncle. **8** Section through the upper half of the pons. *Pu*, pulvinar; *Pp*, crus cerebri; *Cm*, mamillary body; *Qa*, superior colliculi; *Qp*, inferior colliculi; *TB*, degeneration of temporopontine fibers; *MHK*, degeneration of Meynert's tegmental decussation; *VD*, degeneration of anterior funiculus; *Py*, pyramidal tract; *PyS*, lateral corticospinal tract; *Vd*, descending root of the trigeminal. Right half of the plate: Degeneration following destruction of the left half of the pons (the trigeminal was also sectioned). **13** Section through the uppermost part of the medulla oblongata. *Py*, pyramidal tract; *SD*, bundle entering the intermediolateral fasciculus; *VD*, bundle entering the ventral column; *Cr*, restiform body (slight degeneration related to accidental injury); *Vsp*, spinal trigeminal root. **14** Section through the pyramidal decussation. **15** Section through the upper cervical cord. *VD*, degeneration of anterior funiculus; *PyS*, degeneration of lateral corticospinal tract; *fil*, intermediolateral fasciculus. **16** Portion of the anterior funiculus and ventral horn with degenerated collaterals *(fma)*; *vC*, anterior commissure with individual degenerated fibers.
Bottom row: **a, b** Coronal and **c** horizontal section from the brain of a 5-month old child, from the study by Obersteiner and Redlich on the subcallosal stratum and the fronto-occipital fasciculus [23]. *Ccl*, corpus callosum; *Cr*, corona radiata; *Nc*, caudate nucleus; *rcc*, fronto-occipital fasciculus ('reticular cortico-caudate bundle'); *Ssc*, subcallosal stratum; *Sznc*, stratum zonale of caudate nucleus; *Th*, optical thalamus; *Al*, alveus; *Fli*, inferior longitudinal fasciculus; *Ro*, optic radiation; *V*, ventricle; *F*, frontal lobe; *O*, occipital lobe.

Fig. 3 Upper composite: Kolisko and Redlich's *Schemata* of the human brain for use by pathologists [25], which were sold in sets of 25, 50, 100 or 500 plates; convexity (upper) and median surface (lower) of human cerebral hemisphere.
Bottom row: Three cases of head injuries manifesting with epileptic attacks [44]. **a** Trauma at the left temple in a 34-year old reserve officer, accompanied by right hemiparesis, hemihypesthesia, dysphasia, dysgraphia, dyslexia, right-side Jacksonian seizures and loss of consciousness; the autopsy showed a large hematoma in the region of the left parietal lobe with rapture into the lateral ventricle, extending into the entire ventricular system through the foramen of Magendie, and with the subarachnoid space of the cerebrum and cerebellum filled with liquid and dried blood. **b** A 25-year old soldier with a gunshot wound to the left side of the neck, presenting with left hemiparesis, hemihypesthesia, and seizures on the left side (mouth and thumb). **c** A 44-year old farmer with

injury to the right side of the skull from a rifle stock, impression fracture of the right parietal and temporal bones, and general epileptic attack beginning on the left side of the body.

Fig. 4 a, b Foci of demyelination in multiple sclerosis [40]. **a** Section from the lumbar spinal cord with sclerotic plaques on both sides of the anterior median fissure. **b** Bilateral degeneration of the lateral corticospinal tract. **c-f** Autopsy findings in one of the five cases of encephalitis of the pons and cerebellum described by Redlich [45]. **c, d** Macroscopic findings in a 20-year old man with encephalitis, Weigert method; **c** rarefaction and gliosis at the level of the tectum, with the ventral pons spared; **d** asymmetric lesions in the midbrain involvong the red nucleus and the oculomotor nucleus, but relatively sparing the substantia nigra. **e-f** Histological findings in the cerebellum of the same patient; **e** section stain with the Marchi method (osmium tetroxide and potassium chlorate); *m*, molecular layer; *k*, granule cell layer; *h*, focus with very abundant fatty granule cells; **f** section away from the encephalitic foci, stained with picric acid and acid fuchsin (van Gieson method); *M*, molecular layer; *K*, granule cell layer; *Gl*, swollen glial cells; *iG*, infiltration of a blood vessel; *iGw*, white matter infiltration by glia. **g, h** Senile dementia; histological documentation of cortical senile plaques (‘miliary sclerosis’) by Redlich [72].

Fig. 5 a-h Spinal cord sections in tabes cases of varying severity [56]. **a** Moderate case, lumbar enlargement; **b** lighter case, cervical enlargement; **c** same case, middle thoracic cord; **d** severe case, upper cervical cord; **e** moderately severe case, upper cervical cord; **f** case of cervical tabes, cervical enlargement; **g** tabes with uneven involvement of the two sides, upper cervical cord; **h** same case, lumbar spinal cord.
i-l The ‘Redlich-Obersteiner zone’ in normal and tabetic spinal cord [57]. **i** Longitudinal section through the normal cervical enlargement, eosin stain; a striking constriction is seen at the dorsal root, into which the pia mater is intercalated; nerve fibers come together at this point; the outer layer of the cord continues into the dorsal root and becomes compacted and narrower at the constriction. **j** Cross section through the normal cervical enlargement, Pál stain; the dorsal root displays a slight constriction laterally, at the level of the pia; on the lateral side of the root one sees fine fibers coming together and radiating into the tract of Lissauer. **k** Longitudinal section from the lower thoracic cord in tabes, Pál stain; in the extramedullary part of the dorsal roots one still discerns some, mostly degenerated, nerve fibers; beyond the constriction, which in one root is clearly seen, normal fibers are absent altogether, from the intramedullary part as well; beneath the root a blood vessel appears in cross section; at the lowest part of the preparation the section turns into the lateral column. **l** Longitudinal section from the cervical cord in a case of tabes at a modest stage of advancement, alum-hematoxylin and eosin stain; the drawing shows inflammatory changes in the pia, consisting of connective tissue hyperplasia and cell infiltrates; at the dorsal root the cross section becomes thicker, and vessels appear infiltrated by cells.