Károly Schaffer and his school: The birth of biological psychiatry in Hungary, 1890–1940

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Abstract

In the first third of the twentieth century, neuropathology seemed to offer the key to unlock the causes of psychiatric illness. Among the top centers devoted to the microscopic anatomy of the brain was that of Károly Schaffer in Budapest. Schaffer, a pioneer in the histopathology of Tay–Sachs–Schaffer disease, was also a charismatic teacher, bringing forth a school of investigators in psychopathology. Among them was László Meduna, who originated convulsive therapy. Despite the importance of the Schaffer school, it is almost unknown outside of Hungary, largely the result of the introduction of neurophysiological, neurochemical and molecular genetic methods that distracted attention away from histopathological contributions in psychiatry after the Second World War. The microscopic study of the brain and its diseases seemed increasingly less important.

The present biographical account of Károly Schaffer and his school seeks to bring this important story in the early history of biological psychiatry to a wider audience and explain why it has since been forgotten.

Keywords

Schaffer; Károly; Neuropathology; Neurohistology; Psychiatry in Hungary; Meduna; Ladislaus (László); Convulsive therapy

1. Introduction

Psychiatry often seems driven more by technology than by science. At the beginning of its history in the early nineteenth century, psychiatry was driven by the kind of macroscopic brain pathology seen in neurosyphilis. After the development of new stains in the last third of the nineteenth century, psychiatry was pushed forward by neurohistology: the search for

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lesions under the microscope. In the 1930s and 1940s, Hans Berger's electroencephalograph became the technology du jour. And after the 1940s, neurochemistry started lashing things forward, to say nothing of the study of neuroendocrines with radioimmune assays, and of brain imaging and genetics. New technologies mean that new diseases come into the spotlight more or less willy nilly. We're like the storied drunk searching for his keys under the streetlight: "Why there?" "Because that's where the light is."

We present a chapter from one of psychiatry's neurohistological moments in the early twentieth century, a moment in Hungary that is largely unknown. During the first third of the twentieth century, Károly Schaffer – or Karl as it appears on his publications – was the leader of the Hungarian school of histological neuropathology that made Budapest one of the top centers worldwide dedicated to explaining mental symptoms as disorders of microscopic brain anatomy. The Schaffer school developed important new knowledge of hereditary nervous illnesses and produced students who on their own made historic contributions to clinical psychiatry, such as László, or Ladislaus, Meduna, the originator of convulsive therapy. Yet Schaffer and his students remain unheralded outside of Hungary, where from 1925 to 1935 he was professor of psychiatry in Budapest, and active until his death in 1939 (when he died, two articles were lying on his desk ready for publication) [21]. The story is a reminder that the microscope and brain-based studies once contributed valuable information in psychiatry.

In some centers, neurohistology still flourishes within psychiatry. Yet on the whole, the subject seems very remote to psychiatry today, and indeed is sooner taught in departments of pathology. The situation was different early in the twentieth century, when important research institutes in Berlin, Munich, Frankfurt and Vienna developed greater understanding of hereditary brain diseases. Schaffer and his school – including such figures as Dezs Miskolczy, István Környey, Kálmán Sánta, Lajos Angyal, and Béla Hechst-Horányi – were internationally recognized within this context.

Schaffer inhabited a world drenched in German. He was born in 1864 in Vienna as his father, a noted artisan-sculptor, was working on a contract there [20]. Schaffer's brother Frigyes always spoke to their father in German, although Károly himself – all the while having excellent German – spoke Hungarian in the family and identified himself as Magyar rather than German [Baran B, Gazdag G, Interview with grandson Attila Bogsch, Budapest, 2006].

Schaffer's exposure to German neuroscience began as a medical student in Budapest in the mid-1880s. Because the summer semester ended a month earlier than in Vienna, Schaffer was able to attend the lectures of the leading neuroanatomist Theodor Meynert [13]. After graduating in medicine in 1888, Schaffer worked in the laboratory of Károly Laufenauer, then extraordinary professor of psychiatry (he had inaugurated the chair of psychiatry in 1882). Laufenauer, a student of Ferenc Schwartz, a founder of Hungarian psychiatry, had studied with Meynert and Charcot; his main medical writings appeared in German journals. At the time that Schaffer graduated, Ern Jendrassik, the Hungarian neurologist, had just finished his post-doctoral habilitation and would shortly begin his lectures in neurology at a special chair established for him at the university. Jendrassik's medical writings were also

mainly in German, capped by his chapter on hereditary diseases for Lewandowsky's textbook in 1911 [8]. Jendrassik also described a simple maneuver to reinforce the patellar reflex – “Jendrassik hand grip (hooking the fingers of both hands inside each other, and powerful outward tugging with both hands)...is useful maneuver in eliciting the knee jerks” [24].

Schaffer later gave the title of *Hirnpathologische Beiträge* (Contributions to Brain Pathology) to the bound collection of reprints begun in 1912 of his and his pupils' and colleagues' work, mainly from the German journal *Zeitschrift für die Gesamte Neurologie und Psychiatrie* (Combined Journal of Neurology and Psychiatry). The nineteen volumes sat prominently on the shelves of the Institute of Brain Histology, which, after 1925, was part of the university psychiatric clinic. So, the influence of German scholars and the German language upon Hungarian psychiatry was overwhelming [15]. For Hungarians of that generation – born in the Austro-Hungarian Empire – German was a mother tongue among educated people; English had limited importance.

Around age 12, while at the technical high school (Realschule), Schaffer decided to devote himself to anatomical studies. During the science class, the teacher placed a thin slice of potato on the glass plate of a microscope. As Schaffer related in his unpublished manuscript autobiography, “The teacher said that [if we looked through the lens] we would see the so-called starch particles in the potato. When... I saw the circular, rainbow-colored light of these particles, it felt as if an earthquake had just occurred inside me; I saw a thus far totally unknown world, a world which [until then] was hidden from my eyes.” Schaffer vowed “that I would spend the rest of my life in the company of a microscope!” [Schaffer K, typescript autobiography, Manuscript Archives of Hungarian Academy of Sciences, Budapest, Deák B, trans.]. Since his parents were unable to finance this pastime, Schaffer realized that he would have to study medicine, although he felt no intrinsic interest in the subject, still less in psychiatry (later saying that hospital psychiatry offered absolutely no charms for him) [32]. On his own, he developed the knowledge of Latin that he needed for medical-school admission (given that medical students were usually admitted from the Gymnasium, not the Realschule), and in 1882 began his medical studies.

He later wrote, “As a first year student, I was immediately absorbed by the subject of anatomy. When I attended [classes in] physiology as a second-year student, I became most interested in histology.” Schaffer had inherited, or learned, some of his father's graphic talent, and showed himself a master of drawing the structures he saw through the microscope.

Schaffer was drawn to neuropathology through Laufenauer's lectures, which he audited in his fourth year of medical school. Laufenauer had made important contributions to the neuropathology of epilepsy, chorea gravis, and catatonia, among other subjects. So Schaffer was thoroughly implanted within the world of international research.

Schaffer made a surprising discovery as a medical student. “I was able to examine the central nervous system of a woman who had died of rabies in our section, and – still as a fourth-year student – I got to publish my findings in the most distinguished German

specialized journal, *Archiv für Psychiatrie [und Nervenkrankheiten]*.” In 1888, Schaffer discovered that the clinical symptoms of rabies could be linked anatomically to central nervous lesions [32]. In a second article in 1890, he reported that the maximal damage to the spinal cord corresponded to the site of the bite, that the virus reaches the spinal cord from the bite wound along the path of the nerves, and that the virus creates the central nervous symptoms as it spreads up the spinal cord [34]. This was a promising academic beginning.

Between 1889 and 1895, Schaffer worked as a teaching assistant, or assistant professor, in the Department of Psychiatry, usually referred to as “the Clinic.” A word of background: The chair of psychiatry at Péter Pázmány University in Budapest had been founded in 1882, and Laufenauer had been its first chairman. But there was no dedicated building for the department (one was opened only in 1908), so the department of psychiatry functioned in the observational unit of the St. Roch Hospital, not far from the University. In 1889, this unit was “deported,” as Schaffer put it, to a much farther and uncomfortable site, and the audience for psychiatry decreased. Professor Sándor Korányi, chief of internal medicine, helped Laufenauer by giving him one ward with six beds for patients with neurological disorders (especially for research into hypnosis). This small unit was referred to as the “klinikoid,” which means “department-like” [20]. Academic psychiatry at the Budapest university was not an imposing specialty.

In 1893, Schaffer acquired the academic title “private professor of neuropathology and neurology,” which qualified him to lecture, though without a salary. He started accumulating an audience that would later swell to large numbers as excitement about the microscopic study of the pathology of the nervous system enveloped the Budapest medical community. In the 1880s and early -90s, under the influence of Jean-Martin Charcot, psychiatrists became much interested in hysteria. Laufenauer, who had studied with Charcot, undertook experiments with hypnotic suggestion, drawing Schaffer into the project. Schaffer discovered that under the influence of hypnotism certain reflex phenomena occurred; he wrote the experiments up in his first book, *Suggestion and Reflex*, published by Fischer in Jena in 1895 [35]. The experience led Schaffer to become an active hypnotherapist, and since all of his hospital and university appointments until the First World War were without pay, Schaffer, somewhat improbably for someone whose life ambition was to be an anatomist, earned his living in the private practice of psychiatry with hypnosis as a tool in the treatment of his patients [47].

In 1891, Schaffer visited the German histologist Carl Weigert in Frankfurt, who was introducing stains for identifying different tissue types. Schaffer formed a lifelong friendship with Weigert’s assistant Ludwig Edinger, and returned to Budapest more familiar with staining techniques, especially Weigert’s procedure for staining myelin. Schaffer began a histological study of the normal hippocampus, which until then had not been studied with the precision that the new stains permitted [30]. This was the second big project he undertook at the Laufenauer Clinic. The article was widely noticed, and Santiago Ramón Y Cajal, the Nobel-Prize-winning Spanish histologist, and Rudolf von Kölliker, professor of anatomy at Würzburg, cited it [21]. Finally, in 1893, Schaffer established the morphological difference between the axon of a neuron and its dendrites using Nissl’s new stain [33].

In 1895, Schaffer left the Laufenauer Clinic, apparently in the normal course of events to make room for the next cohort of young clinicians, and accepted two hospital appointments: as physician on the psychiatry and neurology wards (160 and 50 beds, respectively) of the Elizabeth Almshouse Hospital, and as neurologist at Count Albert Apponyi's Polyclinic. Schaffer now entered into his life's work, the study of inherited illnesses of the central nervous system [Schaffer, autobiography]. Indeed, he had accepted the Almshouse Hospital appointment, which otherwise offered few charms, for precisely the same reason that Charcot had signed onto the Salpêtrière, that great Parisian warehouse of chronic illness in women, because he would have the chance to follow patients from the clinic to the autopsy table.

At the Almshouse Hospital, Schaffer set up a laboratory in a space little larger than a broom closet, equipping it at his own expense. This little laboratory turned into a virtual scientific greenhouse for the next generation of Hungarian psychiatrists and neuropathologists, and at one point or another Artúr Sarbó, László Epstein, Rezső Bálint, Sándor Ferenczi (the psychoanalyst!), Ernst Frey, László Balassa and Pál Ranschburg (the pediatric psychiatrist who succeeded Schaffer at the Polyclinic) worked there. The little laboratory acquired a microtome and a microcamera in 1899, when Schaffer was asked to attend the World Exposition in Paris and received a government grant to display the brain sections he could now produce [20].

A string of findings fundamental to neuroanatomy followed. Using Golgi's technique, in 1897 Schaffer demonstrated the abundance of nerve cells with short axons in certain parts of the cortex [43]. There were several other findings of this nature [7]. Most importantly, in 1904 Schaffer was among the first to demonstrate empirically (alas published in Hungarian!) that the neuron was an independent anatomical unit and only contiguously connected to the other cells of the central nervous system [29]. Neuron theory had previously surfaced in the writings of Wilhelm His and Auguste Forel. It was H. W. G. Waldeyer-Hartz who in 1891 created the term "neuron" [48], but Schaffer was among the first to offer a precise definition.

Between 1899 and 1904, Schaffer was asked by director Gyula Grósz of the Children's Hospital to do post-mortem studies on six infant victims of Tay-Sachs disease. Known then as among the disorders called "familial amaurotic idiocy," children were born or became blind with an inherited central nervous disease involving the accumulation of the GM₂ ganglioside in neurons (they lacked the enzyme beta-hexosaminidase). Until that point, the disease had been rare in Hungary, and its upswing was probably due to the rapid in-flux in those years of Ashkenazic Jews from the Russian Empire. Two Anglo-Saxon physicians had already described the distinctive retinal and clinical findings, making clear that this was a disease *sui generis*, or at least belonged to a family of specific inherited diseases. In a series of reports that began in 1902 and continued for the next twenty-five years, Schaffer described the histopathological anatomy of the infantile form, which became known to German and American authors as "Tay-Sachs-Schaffer disease" [40,38].

Schaffer's most important discovery in 1905 was that the basic pathology of Tay-Sachs disease involved a neuronal swelling. His beautiful schematic hand-drawings using Weigert's myelin stain were actually much clearer than microphotographs. He said, "The

striking characteristic is that the nerve cells of the entire central nervous system are affected.” He attributed the disease to the group of “Aufbrauchkrankheiten,” or wasting diseases, that his friend Ludwig Edinger had postulated [44]. The following year Ernst Sträussler at Arnold Pick’s Clinic in Prague called this ballooning characteristic of a number of hereditary nervous diseases [46]. This was the first of the lipidoses to be identified, initially known as “Schaffer cell degeneration” [22]. But the concept of lipidoses has melded so much into the inherited knowledge of medicine that Schaffer’s priority has been forgotten.

In Budapest in those days, Jendrassik’s concept of “heredo-degeneration” was very much discussed. Jendrassik meant familial nervous diseases that manifested themselves sometime after birth [2]. Schaffer extended the notion to any familial neurological disease with a defined microscopic pathology, and identified the common morphological features of these illnesses. Tay–Sachs is a heredodegenerative illness par excellence; from it, Schaffer developed a theory that such diseases developed from the embryonic ectodermal layer and manifested an “electivity” in pathological anatomy (“Schaffer’s triad,” now forgotten). In a lecture to the Hungarian Academy of Sciences in 1914, Schaffer explained Tay–Sachs as “an illness of the germinative layers” [41,42].

In the belle époque, Hungary bounced with research in neuroscience, but Schaffer’s achievements in histopathology made him the star in that firmament. In 1899, he received the title of professor without a chair. When Laufenauer died suddenly in 1901 and the chairmanship of psychiatry became vacant, Schaffer sought the appointment, which went instead to Ern Emil Moravcsik, known for his work on hallucinations [22], and for coining the term “epileptic euthymopraxia.”

In 1903, a group of colleagues suggested that a personal professorship together with laboratory be created for Schaffer, and after years of dithering, the Ministry acted on this proposal in 1912, making Schaffer an extraordinary professor with a small department, the Institute of Brain Histology. An international commission of similar institutes conferred upon it the sobriquet “Interacademic.” Modeled on institutes in Vienna and Zurich, “interacademic” meant interdisciplinary, combining psychiatry and histopathology. But in Budapest, it did not include physiology and biochemistry. One reason Schaffer’s work on heredodegeneration failed to have an international impact was that he had no other “wet” bench sciences to underpin it. For example, the nature of the swelling of the neurons in Tay–Sachs was identified in 1939 by Ernst Klenk, a medical physiologist at the University of Cologne, as a lipid, which Klenk further characterized in 1942 as a ganglioside [9,10]. Closer collaboration in Budapest might have produced this result earlier. Instead, progress at the new Institute was measured in the accumulation of thousands and thousands of slides that in the “almost chapel-like stillness” filled the cabinets [21].

In 1919, Schaffer became full professor of psychopathology and neuropathology. When in 1925 he finally obtained the professorship of psychiatry, the Institute became the Department of Brain Histology of the Clinic.

When Moravcsik died in 1924, Schaffer clearly was the prime candidate to replace him. Was the Medical Faculty uneasy about making a candidate whose major achievements lay in histology the professor of psychiatry? Not at all. “Considering that most mental illnesses are incurable,” said the Faculty of Medicine to the Ministry, “psychiatry is a field which is... of secondary importance only.” A psychiatry chair focused on microscopy seemed quite reasonable [Faculty of Medicine, Council Meeting, February 10, 1925, minutes and annexed documents, vols. 51 and 52, Semmelweis University, Budapest, Faculty of Medicine Archives].

The more exact story is as follows: Schaffer was so devoted to his work that he repeatedly refused the chair, giving way only when one of the barons of the Faculty of Medicine urged him to take it on “in the national interest” [47]. But when in January 1925 he finally accepted, it was with conditions: All the equipment and the staff of the Institute for Brain Histology were to be integrated into the Department of Psychiatry, and a new paid assistant professor position was to be created for one of his assistants. The Faculty agreed to move the equipment of the Institute to the Clinic and to create a new assistant professor’s position. But they did not want to integrate the whole Institute (which was a department of the university) into the Department of Psychiatry, because they wanted to transform it into a department of forensic psychiatry. So only one of his preconditions was refused by the Faculty. Yet Schaffer would not compromise. He said he would apply directly to the Ministry. At the end, it was not Schaffer but his colleagues at the Faculty who wrote a letter to the Ministry asking for permission for Schaffer’s preconditions, instead of creating a new department for forensic psychiatry. It was a mark of the esteem in which Schaffer and his conception of psychiatry were now held, that the Ministry consented [Faculty of Medicine, Council Meetings, October 1924–February 1925, vols. 50–52, Semmelweis University].

With the appointment of Schaffer in Budapest, neurology became integrated with psychiatry, as opposed to its earlier integration with internal medicine. This “marriage” of the two disciplines lasted in Budapest for fifty years, when neurology and psychiatry were again separated.

Schaffer’s work in the 1920s was dominated by proving his concept of heredodegeneration, diseases designated today as “inherited metabolic diseases of the nervous system,” was correct. In 1926, he published a synthesis pulling these highly diverse disorders together on a histologic basis [36]. His views were little accepted, and the concept of heredodegenerative diseases has largely passed from usage.

In European neuropathology in the 1920s, there was much interest in neuroglia, the support cells in the brain whose function is even today imperfectly understood. In this area, Schaffer made important contributions, arguing in 1918 that the glia were capable of undergoing an “Eigenerkrankung,” or autonomous disorder, proliferating and then dying, independent of external diseases [31]. In 1920, Pio Del Rio-Hortega described microglia, and Schaffer and one of his students (Lehóczky) went on to postulate that the microglia are metabolically active and have more than a support function [39].

The main impact of Schaffer's glia research was probably domestic: In 1927, László Meduna, who had just finished his training under Schaffer at the Clinic, published on "the histopathology of the microglia," quite curious about the relationship between hypertrophy, hyperplasia, and cell death of the glia [16]. Meduna, who saw much epilepsy in the Clinic's Out-patient Service, then became interested in the relationship between epilepsy and schizophrenia, intrigued by an observation in 1929 of Gyula Nyír and Albin Jablonszky at the Royal Hungarian State Asylum at Lipótmező that, "in the cases of schizophrenia that were comorbid with epilepsy, the epileptic attacks very often completely disappeared or only seldom occurred" [26]. This was the first intimation of an antagonism between seizures and psychosis. Meduna asked himself if schizophrenia, a disorder considered untreatable by the Schaffer school because it arose in the ectodermal germ plasm of the fetus, could somehow be relieved by inducing convulsions.

Meduna had one further piece of evidence for such an antagonism that he did not commit to print: histopathological studies that showed a proliferation of the glia in epilepsy, their dramatic cell death in schizophrenia. Miskolczy had shown him privately a collection of autopsied schizophrenic brains in which the "glia reaction" was missing. Béla Hechst-Horányi at the Clinic had been working on the histopathology of schizophrenia and had also noted the absence of a glia reaction in the disorder [5,6]. As for the histopathology of epilepsy, Meduna was already quite familiar with the increases in glia, having studied cell changes in idiopathic ("genuine") epilepsy [17]. Moreover, as Meduna said in his unpublished autobiography, "I persuaded a surgeon to open the skull of these [epilepsy] patients and to cut the primary ill focus of their brains." Under the microscope, Meduna found the predicted hyperplasia of the glia [Meduna L, typescript autobiography, Meduna Collection, University of Illinois Archives, Champaign-Urbana, IL]. He first did animal experiments to see if it was safe to treat guinea pigs with a convulsive agent (camphor) [18]. Then, in January 1934, he induced therapeutic convulsions in patients sick with dementia praecox, and at the beginning tried to treat patients with every form of the illness [19]. This was the beginning of convulsive therapy, and of all the achievements of Schaffer and his school, it is this discovery that has had the greatest impact on world psychiatry. In 1938, Ugo Cerletti demonstrated the induction of seizures with electricity rather than chemical agents, and electroconvulsive therapy became the treatment of choice for melancholia, catatonia and mania, still in use after more than seventy years [45].

When Meduna presented Schaffer with a draft copy of this report on "curing" schizophrenia by giving the patients epilepsy, the Master, as he was called, was not pleased. Meduna wrote, "He called me a swindler, a humbug, a cheat, every bad name he could think of. How did I dare to claim that I cured schizophrenia, an endogenous, hereditary disease." Schaffer told Meduna, "If you dare to publish this paper, I will disown you. And if ever anybody asks me whether you were my pupil, I will deny it and your name will never be mentioned in my presence" [Meduna, autobiography]. (In retrospect, Schaffer was partly right, because neither convulsive treatment nor any other offers a cure for schizophrenia, while at the beginning Meduna was quite optimistic about a "cure" [Heilung].)

Yet Schaffer must have been impressed by the wave of international attention that Meduna's work received, for he evidently cancelled the expulsion from the garden. In his manuscript

autobiography, written in 1936, Schaffer mentioned Meduna, along with Richter and Miskolczy, as among his most important students (because of Meduna's studies of "glia degeneration"). In 1936, at Schaffer's wish, Meduna became the first recipient of the "Schaffer Medal," lecturing to the Hungarian Society of Neurologists and Psychiatrists on "the convulsive treatment of schizophrenia" [Váradi G, "The history of the Schaffer medal" (2006)].

As the 1920s wore on, Schaffer could not get beyond heredodegeneration. He became tedious on the subject of his theory of hereditary nervous diseases, neglecting, as István Környey said, other issues in the professional literature of the day. "In his writings [after 1925] there is much repetition; it is therefore no wonder that among the international readership only those directly involved in the debate paid attention, and with that, the credit Schaffer deserved for the discovery [of the lipidoses] became soon forgotten" [12]. (One bears in mind, of course, that between Környey and Schaffer there was little love lost.) The circumstances following World War II also contributed to the decline of Schaffer's concept, as hereditary degeneration, and genetics in general, was viewed with reservations because of its abuse in Nazi Germany.

Schaffer's personality might have contributed in some measure to this dogmatic insistence on his own correctness. He is best described as a totally-focused workaholic. When, for example, the family went on vacation at the seaside spa of Lovran, as his grandson Attila Bogsch recalled, "Arriving at the hotel [from the station] in the hotel car, [Schaffer] closed the door of his room and began to work." His family would thereafter occasionally glimpse him going for a walk. Schaffer had few pleasures in life outside of his work; he would confine his evening meal to "a single soft-boiled egg eaten with salt," and endure "as the tortures of hell" sitting still for the measuring of a new suit after his wife had determined that the previous one had become threadbare. Every morning his wife counted out for him the streetcar fare for his trip to his office [Bogsch, interview]. This is not the portrait of a man with a mobility of spirit. Said Ludo Van Bogaert, who in the 1930s as a young Belgian neuroscientist visited Schaffer's laboratory, any discussion of Schaffer's ideas became "painful very quickly. It was this mentality and this need for his ideas to be integrally accepted that were, for the most part, at the origin of the quarrels that he had with the German histopathologists" [47]. (Schaffer was involved in the 1920s in various debates with Walther Spielmeier in Munich and Max Bielschowsky in Berlin that have now passed from memory [11,4].)

One of Schaffer's close students, István Környey, later a professor of neurology, psychiatry and neurosurgery at several universities, said "He was reserved, austere, stern, had a deaf ear for jokes and small talk." Schaffer socialized little or not at all. "As to music," said Környey, "Schaffer's silver-stained sections were his 'visual concerts'" [14]. Max Nonne, the Hamburg neurologist, summed up, in a kind of involuntary irony, the driven nature of this lifestyle in a tribute to Schaffer on his seventieth birthday: "His life was wonderful, because it was struggle and work" [25].

Schaffer was obliged by law to retire in 1935 after reaching age 70, though he remained in office until László Benedek's arrival in the autumn of 1936. As the infirmities of age

gathered, he had to give up laboratory research and seems to have undergone a clinical depression, though this is not readily discussed among his admirers [27]. It is, however, possible that Schaffer was not depressed but rather merely upset because of his succession: He had campaigned for his favorite student Dezső Miskolczy, who at the time was chairman of psychiatry in Szeged University, to get the chair. When Benedek received it instead, it was for Schaffer a disaster! [Faculty of Medicine Minutes, 1935, vol. 63.] Also, speaking against the depression hypothesis is that Schaffer completed two books in 1936 and 1938 after his retirement.

In any event, he recovered from this moroseness as he returned to one of his favorite subjects, the relationship between the structure of the brain and talent. Schaffer believed that he had discovered the anatomical basis of genius and his morphological observations in this area have never really been followed up on [37].

What is Schaffer's legacy? It was unhelpful that, as we have seen, László Benedek, a student of Károly Lechner, professor of psychiatry at the University of Kolozsvár, became his successor. Benedek used, as Meduna put it, "iron and fire against many representatives of the morphological school of Schaffer" [Meduna, autobiography]. There was a great debate between Benedek and Schaffer about eugenics: Benedek advocated legislation for eliminating the eugenically unfit, and Schaffer was the only leader of psychiatry to oppose it, in several publications between 1924 and 1937 [2]. One biographer concluded, "It is [Schaffer's] merit that no forced eugenic measures were introduced in Hungary in the 1930s" [14]. So Benedek did not exactly hold high the torch of Schaffer's legacy.

Science dug away at Schaffer's concept that the microglia, disorders of which played a central role in inherited nervous pathology, were of ectodermal origin; indeed, Schaffer's student Kálmán Sántha, later a pioneer of neurosurgery in Hungary, made one of the first breaches into this doctrine [28]. The war and subsequent tumult introduced great discontinuities into Hungarian society and academic life, as almost all previous written records relating to psychiatry have vanished.

Even before the war, Schaffer's achievements began to be overlooked. The numbers of cases of hereditary degeneration that Schaffer had studied were small and he wasn't able to identify "all the combinations in which diseases of the various systems occurred," as Környey put it [14].

In 1940, his student Hugó Richter summed up the core of Schaffer's views: that all inherited nervous diseases have "as a common basis an inborn weakness of the protoplasm of the nerve cell, a pathological tendency characteristic of the hyaloplasm that causes metabolic disturbances visible at the microscopic level" [27]. Science today has moved past this vision, yet as a unitary concept in the 1930s, it stimulated much research on the biological basis of psychiatric illness that kept the field of psychiatry focused on the brain. In the heyday of psychoanalysis in the decades following the Second World War, this vision was momentarily lost. But it has emerged with renewed vigor to drive forward biological psychiatry in our own time. The name Károly Schaffer has, however, been lost.

After the War, Schaffer's school faded from sight at an international level, although in Hungary its graduates continued to play leading roles. There were several years when Schaffer's students held three of the four neuropsychiatry chairs in Hungary: Béla Horányi in Budapest, Kálmán Sántha in Debrecen, István Környey in Pécs, and Dezső Miskolczy in Maros-vásárhely, Romania. (Miskolczy's concept, articulated in 1933 when he still was the professor of psychiatry at Szeged, had some impact on postwar scholarship: that only humans can have schizophrenia because only they among the creatures have brains with higher-order components capable of schizophrenic "decerebration" [23,1].) Yet outside of Hungary only elderly neuropathologists would recall the name Schaffer, and Dorland's Medical Dictionary passes in silence over him [3].

2. Conclusion

Why did a school of such accomplishment pass so quickly from memory? The reasons range from the circumstantial to the most fundamental issues in the unfolding of the neurosciences.

1. The Hungarians lost most of their international connections because of the Iron Curtain: even correspondence with western colleagues was suspect and could be prohibited. Given that few people outside of Hungary read Magyar, to have an impact the members of the Schaffer school had to publish in an internationally-understood language. From the late-nineteenth century, when Hungarian psychiatry attained international recognition, to the Second World War, that language was German. Most Hungarian academic psychiatrists also spoke English, and two of Schaffer's fellows even worked in the United States (Sántha with Penfield in Montreal and Környey with Peet and Baeley in Ann Arbor); yet English was of secondary importance at that time.
2. The Schaffer school itself withered in Hungary. Horányi, Benedek's successor, lost the chair of psychiatry and neurology in 1950 when he was alleged to be an enemy of the People's Democracy. Gyula Nyirő, the new professor of psychiatry, had little time for Schaffer's work and sought to combine Jaspersian psychopathology with the approach of his teacher Lechner, who was interested in reflex theory [Ban T, personal communication, May 7, 2006]. Sántha lost his chair in Debrecen under similar circumstances in 1951. Both were rehabilitated, but Sántha died in 1956, and when Horányi returned to teach in Budapest he found two combined departments of neurology and psychiatry, simply splitting the building. After his death, the First Department of Neurology and Psychiatry became the Department of Neurology, the Second the Department of Psychiatry.
3. After the Second World War, neuroscience fundamentally changed its orientation from neurohistology – information gained by looking through a microscope – to electrophysiology, and beyond that to neurochemistry. This began in the 1930s with Alfred Zeller's discovery in Basel of the enzymes involved in amine metabolism [49]. After the war, biological psychiatry and neuroscience became biochemical subjects. Neurohistology ceased to make a big contribution, and suddenly Schaffer's approach was dated.

4. Finally, the triumph of psychoanalysis within clinical psychiatry rendered suspect – indeed odious – the assumptions of biological psychiatry that situated mental symptoms within the structure and chemistry of the brain. The analysts mocked knowledge gained with the microscope and believed that only psychoanalytic therapy would lay bare the “roots” of mental symptoms in unconscious conflict. To this, there was no possible response from the biological psychiatrists, including the microscopists, as it involved a paradigm shift, a political coup d’état. Midst this tumult, who could recall the morphological school of Hungarian psychiatry?

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