Sigmund Freud

Sigmund Freud's life has been the subject of several well-known biographies as well as a brief autobiography (Clark, 1980; Gay, 1988; Jones, 1961; Freud, 1935). Although his productive career in neurology has been eclipsed by the magnitude of his contributions to psychiatry (Jelliffe, 1937), Freud's neurological training and discoveries are well recognized and his work on cerebral palsy has recently been briefly presented in American pediatric literature (Accardo, 1982).

Freud (1856–1939) (figure 1) was born in Moravia (later part of Czechoslovakia) and moved to Vienna with his family at the age of four. He was at the top of his class at the gymnasium for seven years and, by his own account, enjoyed special privileges (Freud, 1935). His father, Jacob Freud, also seemed to have recognized his son's brilliance; Ronald Clark relates that the medical interests of Freud's sisters were sacrificed to preserve the quiet environment that the young Freud required for his studies (Clark, 1980). In addition, Freud's father allowed him to choose a profession on the basis of his own inclinations. Although initially attracted to law, Freud was drawn toward natural science by then recent theories of Darwin and decided to become a medical student “after hearing Goethe's beautiful essay on Nature read aloud at a popular lecture” (Freud, 1935). Although Freud registered in the University of Vienna's medical department in 1873, he initially pursued a broad curriculum with a particular interest in philosophy. In part because of the breadth of his interests, he required eight instead of the usual five years to graduate. In 1877, under the tutelage of the comparative anatomist Carl Claus, Freud presented a paper on the reproductive physiology of the eel (Clark, 1980).

Between 1877 and 1883, Freud worked in Ernst Brücke's Institute of Physiology. Brücke was one of the founders of a movement which began in the 1840s and has been designated the Helmholtz School of Medicine. The other founders of this movement were Emil du Bois-Reymond, Carl Ludwig, and Hermann Helmholtz. These four men and their students dominated German
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physiology during the last half of the nineteenth century (Amacher, 1965). While at the Institute, Freud devoted his attention to the phylogenetic evolution of the nervous system of the lamprey eel and crayfish, a subject in which interest had been spurred by the controversy surrounding the recent publication of Darwin's The Origin of Species (Clark, 1980). He developed a technique for staining nervous tissue with gold chloride; this led to a publication in the German medical literature as well as one in the journal Brain (Jones, 1961).

Freud's neuropathological interests subsequently shifted to the medulla oblongata. His methods of studying the medulla made use of a recent discovery by Paul Flechsig: various tracts in the central nervous system have independent timetables of myelination. By studying serial sections of fetal brains, he was able to make observations on the course of tracts that would have been lost in the "inextricable pictures of cross sections, which permit hardly more than a superficial topographical survey" when comparable adult material was examined (Jones, 1961, 137). Freud's investigations utilized the brains of experimental animals and also included human material. It seems likely that this early work, which led to three publications, contributed to his interest in cerebral palsy and was in part responsible for the emphasis that he placed on prenatal factors in its genesis.

During the time he worked with Brucke, Freud was "decidedly negligent" in pursuing his medical studies and did not receive his degree as Doctor of Medicine until 1881. He states in his autobiography that "the various branches of medicine proper, apart from psychiatry, had no attraction for me" (Freud, 1935). Although Freud enjoyed success in the laboratory and was designated for promotion when a suitable position became available, Brucke urged him to pursue a career in clinical medicine in order to better his financial situation (Freud, 1935). Freud spent his next three years at the Vienna General Hospital, rotating through the surgical services and later through the medicine department of Dr. Hermann Nothnagel, who was to become the editor of the Handbuch to which Freud contributed his monograph on infantile cerebral paralyses approximately fifteen years later. His experience at the Vienna General Hospital included five months in Theodor Meynert's department of psychiatry (Clark, 1980).

Freud transferred to the department of nervous diseases in 1884. Over the next few years he published a number of clinical reports. Freud observed that the fame of my diagnosis and of the post mortem confirmation brought me an influx of American physicians, to whom I lectured upon the patients in my department in a sort of pidgin-English. I was able to localize the site of lesion in the medulla oblongata so accurately that the pathological anatomic had no further information to add; I was the first person in Vienna to send case for autopsy with a diagnosis of polyneuritis acuta (Freud, 1935, 20).

Freud read the account of Theodor Aschenbrandt on the stimulation effects of cocaine and began to experiment with the drug and encourage its use by friends in other branches of medicine. Although Freud made the observation that cocaine had anesthetic properties and wrote a monograph on the history of cocaine and its potential applications in medicine, "the decisive experiments upon animals' eyes" were made by his friend Carl Koller to whom Freud had introduced the drug (Freud, 1935; Jones, 1961).

In 1885 Freud received an appointment as privatdozent (university lecturer) for neuropathology, and this enabled him to secure a travelling fellowship to Paris, where he worked under Jean Martin Charcot for six months. The main result of this segment of Freud's education was to stimulate an interest in hysteria, which would ultimately lead to the methods and theory of psychoanalysis. Freud planned to carry out pathological studies of children's brains while at the Salpêtrière. However, his letters indicate that he abandoned his plans to pursue laboratory work during his sojourn in Paris (Clark, 1980).

On his return trip to Vienna, Freud "stopped for a few weeks in Berlin, in order to gain a little knowledge of the general disorders
of childhood” in the clinic of Adolf Baginsky (Freud, 1935, 24). In a letter to his fiancée, he described his reaction to pediatric practice: “As long as their brains are free of disease, these little creatures are really charming and so touching when they suffer . . . I think I would find my way about in a children’s practice in no time” (Clark, 1980, 76). Over the next several years, as the result of work done at the Kassowitz Institute in Vienna, he published several monographs dealing with unilateral and bilateral cerebral paralyses in children.

Freud had resigned his position at the Vienna General Hospital prior to leaving for his fellowship in Paris. On his return to Vienna in 1886, he entered practice as a specialist in nervous diseases. His views on hysteria brought him into conflict with Meynert, led to his exclusion from the Laboratory of Cerebral Anatomy, and perhaps accelerated the development of an independent approach to mental illness (Freud, 1935). Freud’s major neurological publications after 1890 came at a time when he was already laying the foundations of modern psychiatry. Studies on Hysteria, which was written in collaboration with Josef Breuer, was published in 1895 and The Interpretation of Dreams in 1900.

His monograph on aphasia, Zur Auffassung der Aphasien, eine Kritische Studie (On Aphasia: A Critical Study), published in 1891 as the result of an invitation to contribute to a medical encyclopedia, reflected a mastery of the theories of Carl Wernicke and Ludwig Lichtheim as well as a broad understanding of neurology and neuropathology. Freud meticulously reviewed the accepted categories of aphasia, revealed the limitations of current classifications, and also suggested that attempts to localize various aspects of language function appear inadequate to explain some forms of aphasia. Recognition that anatomic distribution of cerebral lesions does not always correlate predictably with clinical manifestations is also a theme to which Freud returned to in his book Die Infantile Cerebralblähung. In his book On Aphasia, Freud introduced the term agnosia to neurology and proposed the category of agnostic aphasia in which the language disturbance resulted from failure of object recognition. He redefined the term asymbolia, which had previously been used by Karl Finkelnburg, to mean a disturbance in the association between word concept and object concept. Freud traced his own views of language to the writings of John Stuart Mill, which he had translated years earlier (Freud, 1953).

Freud’s writings on cerebral palsy spanned a ten-year period beginning in 1889 when he was the first to report on the presence of visual field deficits in children with cerebral palsy (Freud, 1889). One of his best known works, in collaboration with Oscar Rie, was a 220-page monograph entitled Klinische Studie über die halbseitige Cerebralblähung der Kinder (Clinical Study on the Unilateral Cerebral Paralysis of Children), which was printed in 1891. This extensive study of the literature and collection of case reports was of interest because Freud was the first to focus on the occurrence of hemiatrophy on the affected paralysed side and also helped establish the choreoathetotic form of cerebral palsy as a distinct subtype of this disorder. Two years later a shorter monograph, further elaborating on the clinicopathological aspects of cerebral palsy, particularly in relation to Little’s disease, was published. Freud also wrote a paper on the same subject, which appeared in the Revue neurologique (1893b). During the next four years Freud consolidated and revised these efforts into one of the most important works ever written on this subject, Die Infantile Cerebralblähung.

Freud’s Die Infantile Cerebralblähung (Infantile Cerebral Paralysis) was published in 1897 for Nothnagel’s Handbuch der Allgemeinen und Speziellen Therapie. In his letters, Freud expressed some frustration over the necessity of producing this work on cerebral paralysis at a time when he was already preoccupied with the neuroses: “I am fully occupied with the children’s paralyses, in which I am not the least interested” (Accardo, 1982, 453).

Infantile Cerebral Paralysis was an exhaustive work divided into eleven chapters. The first seven chapters include a discussion of nosological considerations, an extensive review of the literature, detailed discussions of diplegic and hemiplegic forms, and an exhaustive compilation of the pathological anatomy of the disorder. The eighth chapter deals with the classification of various types of cerebral paralysis on the basis of their underlying pathology and etiological factors. In the chapter on pathological physiology, Freud focuses on the relationship between the distribution of pathological changes and clinical symptomatology. Chapter Nine reviews familial and hereditary forms of infantile cerebral paralysis while the following chapter examines the relation of epilepsy and
mental impairment ("idiocy") to the other symptoms of the disorder. The brief final chapter discusses differential diagnosis and therapy.

Freud began with a pathological rather than a clinical definition of the disorder:

*I actually advocate that this term be applied even to cases in which paralysis is completely absent or where the disease consists merely of a periodic recurrence of convulsions (epilepsy). This infantile cerebral paralysis is merely a contrived term of our nosographic classification, a label referring to a group of pathological cases... It would be desirable to replace this term by another not conveying such a definite, inadequate image.* (Freud, 1968, 17)

Throughout his text, Freud’s use of the general term “infantile cerebral paralysis” approximates the modern pediatric neurologist’s phrase “nonprogressive encephalopathy.” Freud amply justifies his desire for a looser interpretation by frequently pointing out the variability of clinical and pathological manifestations in patients with childhood encephalopathies and the lack of a predictable relation between pathologic features and clinical manifestations.

Freud was confronted with the task not only of cogently presenting his own schema of classification but also of justifying his departure from the classifications proposed by many who preceded him. Several themes recur throughout his work. He demonstrated transitional forms between types of cerebral paralysis, considered by other authors to be distinct in their clinical presentation; showed the lack of an exclusive relation between etiological factors and the type of deficit; and illustrated that the recognized deficit may evolve, diminish, appear, or disappear as a function of the patient’s age. He made many pertinent observations concerning the relation of certain forms of motor deficit to other manifestations such as mental impairment (“idiocy”), aphasia, and visual disturbance. He appeared to achieve the balanced goal of arguing forcefully for his own views without engaging in excessive criticism of those propounded by previous authors.

Freud accorded particular importance to the views of William Little concerning the importance of various parturitional factors including premature birth. He disputed in several places the controversial view of Adolf Strümpell that encephalitis is the major factor responsible for infantile cerebral paralysis (Freud, 1968). Freud acknowledged that about one-half of all “noncongenital forms” of hemiplegic infantile cerebral paralysis are of unknown etiology and later stated that in his own case material, an etiology could not be identified in 34 percent of patients with cerebral diplegia (Freud, 1968). While accepting the term “Little’s Disease,” he did so with reservation and not as a synonym for “infantile cerebral paralysis.” Rather, he restricted its use to diplegic cases in which factors related to delivery appeared to be of predominant importance. He suggested that a broader use of the term “would only serve to obscure Little’s contribution in regard to the etiology of birth paralysis” (Freud, 1968, 260). He dealt at some length with the troublesome observation that many children experiencing obstetric difficulties did not develop infantile cerebral paralysis and surmised that

*Little’s etiology cannot in all cases exist without the simultaneous effort of congenital factors. Furthermore, one has to consider that the anomaly of the birth process, rather than being the causal etiological factor, may itself be the consequence of the real prenatal etiology.* (Freud, 1968, 142)

Freud individually discussed various manifestations of nonprogressive encephalopathy. A recurring theme is that the severity of one neurological manifestation did not reliably predict the severity or even the existence of another. However, he did note that “one should add to the peculiarities of choreatic paresis the fact of occurrence of aphasia, atrophy, epilepsy and slight degree of mental impairment” (Freud, 1897). He explained these observations by reference to a single autopsied case, reported by Jean Landouzy, who demonstrated a calcified lenticular lesion (Freud, 1968, 109).

Freud did not feel compelled to explain the pathologic or physiologic basis for certain manifestations of cerebral paralysis. In some cases, he examined the hypotheses that had been propounded, found them deficient, and acknowledged that a satisfactory alternative is not apparent:

*The explanation of such trophic disturbances (growth arrest) following cerebral affections with a complete absence or only a trace of paralytic symptoms has not been made so far. The assumption of “trophic centers” in the brain is merely the value of prejudiced circumvention of facts.* (Freud, 1968, 129)

Freud noted that he was the first to report the association of
homonymous hemianopsia with hemiplegic cerebral palsy (Freud, 1889; 1897, 89). He elaborated on the difficulty of detecting visual field defects in children. In reviewing the relative concomitants of "infantile cerebral paralysis", he made a forceful argument for the extension of the term to include a broad range of neurological deficits:

Such symptoms as hemianesthesia and hemianopsia are decisive in the interpretation of the concept of "hemiplegic cerebral paralysis" by forcing us to go beyond the literal meaning of the term and to eliminate the last possibility of considering this frequently occurring disorder a systemic disease limited to the gray motor cortex. The localization of the lesion cannot be the essential factor of infantile cerebral palsy. This is further confirmed by pathological anatomy, which exhibits in hemiplegic cerebral paralysis the same lesions in various other parts of the cortex or of the brain that, if they occurred in the motor regions, would also create hemiplegia. Consequently, it would be quite correct to attribute isolated hemianopsia to hemiplegic cerebral paralysis as long as the other clinical instances (etiology, course of disease, complications) show conformity. (Freud, 1968, 92)

In Infantile Cerebral Paralysis, Freud dealt with practical considerations in childhood language disorders but made little reference to the concepts in his earlier monograph, On Aphasia. He distinguished between acquired aphasia ("as a disturbance of already acquired speaking ability") and developmental language delay (in which "we are not justified to look at the disturbance as a focal symptom because delayed development of speech of children does occur so frequently as a symptom of poor cerebral development") (Freud, 1968, 92). He supported the views of previous authors, including Jean Louis Cotard, that aphasia in patients with hemiplegic cerebral palsy is not "a permanent symptom" and noted that aphasic disturbances in children are not uncommonly associated with left rather than right hemiparesis (Freud, 1968, 94). He summarized the conclusions of Max Bernhardt concerning aphasia in association with cerebral paralysis and expressed his agreement with the following conclusions that are generally accepted today: (1) Aphasia appears often at an early age; (2) The right hemisphere is often able to substitute for the left in language function; (3) Motor (expressive) aphasia is the most common type in childhood (Freud, 1968, 95).

Freud's chapter on familial and hereditary forms of infantile cerebral palsy included a detailed description of several degenerative disorders, including Pelizaeus-Merzbacher disease. On the one hand he appeared willing to include such conditions under the umbrella of cerebral paralyses, but he implied acceptance of the view that the great majority of cases of cerebral paralysis in childhood have a nonprogressive etiology: "It is, therefore, more probable that the forms of infantile cerebral paralysis we are considering actually are diseases with accidental etiology" (Freud, 1968, 318). In this context, Freud appears to use the term "accidental" in a broad sense to refer to disease processes that are not of genetic origin.

Freud's discussion of the pathology of infantile cerebral paralysis reviewed numerous entities that he divided into three categories: intrauterine forms, birth paralyses, and forms acquired postnatally (Freud, 1968, 159). He noted that neurological deficit may arise not only from injury to a portion of the nervous system early under development but also from injury at an earlier stage when development of a portion of the brain may "fail to materialize" (Freud, 1968, 175).

Freud brought coherence to the classification of infantile cerebral paralysis. He recognized that a broad range of etiological and pathological factors were associated with cerebral palsy and emphasized that lesions outside the motor system but otherwise directly to those which produce paralysis may cause disturbances of language and sensory function. He acknowledged that in a substantial proportion of patients, the etiology remained obscure even after careful evaluation. He recognized that obstetric factors did not account for all cases of infantile cerebral paralysis and accepted the term "Little's Disease" as appropriate for only one subgroup of infantile cerebral diplegia.

Freud's transition from the world of neurology to that of psychiatry is perhaps reflected in one of the last passages in the chapter devoted to "Epilepsy and Idiocy":

The condition underlying idiocy must be more specific. It must be related to a localized disturbance, although localization must not be used here in the literal sense, such as pertaining to a definite anatomical region or a histological layer of the brain, but in the sense that the injury affects one or the other of the complex apparatuses [sic] that subserve mental functions. Any more definite
conditions are completely unknown, just as we are completely in the dark in regard to the structure of the mental apparatus. (Freud, 1968, 339)

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**REFERENCES**


NIKOLAUS FRIEDREICH

NIKOLAUS FRIEDREICH (figure 1) was born into a medical family in Würzburg, Germany, on July 31, 1825. His father had been Professor of General Pathology at Würzburg. His grandfather, Nikolaus Anton Friedreich, was a director of the medicine clinic at Würzburg and was apparently interested in the nervous system, having described acute unilateral facial palsy in 1781, twenty-three years before Bell’s description (Bird, 1977). Friedreich’s education began in 1832. He obtained most of his undergraduate and medical training in Würzburg and worked initially with Carl Friedrich von Marcus, a blind clinician, and then with Rudolf Albert von Kolliker (1817-1905) in 1847 and 1848 (Clarke and O’Malley, 1968). However, his major mentor was Rudolph Virchow who held the chair of pathological anatomy in Würzburg and was a political activist for a time at the barricades during the revolution of 1848. In 1849 Friedreich joined the faculty in Würzburg. He presented his first paper on November 1, 1851, describing a case of lymphangiectasia of the penis. In 1853 he completed his private docent thesis on “Contributions to the Study of Intracranial Tumors.” In 1854 he presented a series of papers on pneumothorax, emphysema, and typhus. In the summer of 1856, Virchow was called to Berlin and Friedreich succeeded to his chair in 1857 at the age of thirty-one. Virchow’s training provided Friedreich with a skill, which was developing in medicine at the time, of applying pathology to clinical disorders. Friedreich was a consummate clinician, especially interested in physical diagnosis. After only a year in Virchow’s chair, Friedreich became director of the medical clinics at Germany’s oldest university at Heidelberg. At the time, this school was rivaled only by those in Berlin and Vienna.

Friedreich was proficient in all areas of medicine, but his main interest was the nervous system. Many of his important contributions involved pediatric patients, most notably those with ataxia and disease and ataxia. He wrote an elaborate monograph on progressive muscular atrophy (Friedreich, 1873). This work, regarded by contemporaries as his greatest (“Nikolaus Friedreich,” 1888), was