dard to conceal the identity of the real family. This came from the Greek *kallos* (beauty) and *kakos* (bad). This succinctly summarized the essence of the book: the good Kallikaks and the bad Kallikaks. Goddard's field worker, Elizabeth Kite, was able to trace Deborah's ancestors back several generations to Martin Kallikak. In a dalliance with a feebleminded tavern girl, he had a son, nicknamed "old horror." Of his son's 300 descendants, 65% were feebleminded and became paupers, criminals, prostitutes, drunkards, and all forms of "social pests." Later, Martin Kallikak married into one of the best families in the state. The 496 descendants of this marriage contained nothing but those of "good representative citizenship": doctors, lawyers, judges, educators, and other respectable citizens.

This was the essence of the study. In its time it was widely hailed as a prime study of the effect of good genes and bad genes on individuals and society and was one of the most quoted works of the eugenics movement. Through extensive detective work, David Smith was able to identify the true Kallikaks and track them down for re-evaluation. At times he asks of himself: "Why devote time and effort to a 75-year-old study that is now widely acknowledged as invalid?" His answer: "It is, perhaps more than anything else, an illustration of the power of a social myth. Goddard found the characters that he could make fit the tale." Smith proceeds to demonstrate just how flawed the original work was. Hundreds of diagnoses of feeblemindedness were made on flimsy evidence and recollections of others by his medically and psychologically untrained field worker. Many of the conclusions about the mental capabilities of individuals begged the fact: if they were on the bad side of the family, they must be feebleminded.

The problem was not so much the defects of the study, but the uses to which it was put in the name of being correct. It played an important role in the passage of compulsory sterilization laws in 30 states and the Immigration Restriction Act of 1924, which was responsible for limiting the number of Jewish immigrants who were attempting to flee Germany. The book was translated and widely distributed in Germany in the mid-1930s. Shortly after the Nazi's came to power, one of the book's prime champions, Harry Laughlin, was given an honorary doctor's degree by the University of Heidelberg.

Davis concludes, "I do not believe that Henry Goddard was a sinister man intent upon doing harm to the poor, the foreign-born, the uneducated, or people with different racial or religious backgrounds. He was as much a product of a powerful idea as he was the creator of a social myth. He took the idea, cast it with characters, and embellished it with stories of what he wanted to be true of the characters—he saw what he wanted to see in the Kallikaks."

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The Hereditary Ataxias and Related Disorders. By A. E. Harding. New York, Churchill Livingstone, Inc., 1984. Pp. 256. $56.00.

This book opens with the sentence, "The term ataxia literally means disorder or confusion." This is probably one of the most profound summarizing statements about this subject that I have read and it is of great help in clearing the confusion to have them all discussed in one monograph. Appropriately one-fifth of the book is devoted to Friedreich's ataxia (FA). One hundred percent of typical FA patients have gait ataxia,

progression of symptoms, lower limb areflexia, sensory loss, and muscle weakness, and 96% show extensor plantar responses and pes cavus. This allows a distinct separation of "atypical FA" into a category of "early onset cerebellar ataxia with retained tendon reflexes." This is much clearer than the more fuzzy concept that some FA patients may have normal tendon reflexes. A second one-fifth is devoted to the autosomal dominant cerebellar ataxias of late onset, that is, olivopontocerebellar atrophies types I, III, IV, and V. Although post-mortum examinations often solve our problems in clinical diagnosis, in the dominant ataxias, different members of one family can have different pathological features.

The classification of the ataxias that Hardwig uses is as follows: (1) congenital disorders of unknown etiology, (2) ataxia with known metabolic or other cause, (3) ataxia of unknown etiology (A, onset before 20 years; B, onset after 20 years), and (4) the hereditary spastic paraplegias. Most of these categories have subtypes with associated features such as hypogonadism, retinal degeneration, deafness, or retardation to provide a clinically useful scheme for identifying a given case.

This monograph will be quite useful to those who often find themselves needing to make sense of this difficult group of patients.

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