probably is handled best by reassurances of hair regrowth. When involvement becomes more extensive, however, a careful explanation is wiser.

Treatment of alopecia areata is unsatisfactory. The long-term systemic administration of corticosteroids has proved impracticable because of side effects and because of relapse on discontinuance. However, exceptions are observed occasionally, although spontaneous remission cannot be excluded. Intralesional injection of dilute solutions of triamcinolone acetonide causes hair to regrow in many instances, but unfortunately it does not prevent new plaque formation or relapse. Judgment of all regimens should consider the overwhelming tendency for regrowth.

Most provocative in Muller and Winkelmann's study was the suggestion that patients in whom alopecia areata develops are more subject to diseases of hypersensitivity, notably atopic dermatitis and asthma and autoimmune disorders such as collagen disease and chronic thyroiditis (Hashimoto's disease). As the investigators point out, the meaning of this increase in associative incidence is unknown, but in all these diseases familial incidence is significant, relapse is common, and a background of hypersensitivity is likely. Further investigation of alopecia areata—and especially of its relationship to hypersensitivity—may prove an interesting chapter in dermatological research.

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2. Greenberg, S.I.: Alopecia Areata: Psychiatric Survey, *Arch Derm (Chicago)* 72:454-457, 1955.

JAMES B. HERRICK (1861-1954)

The "foremost clinical cardiologist of the Midwest in his day" was a true son of the prairie whose love for teaching, native ability, and clinical curiosity earned for him this mythical title. James was born in Oak Park, Ill, a community to which his maternal grandfather traveled overland in a covered wagon, having migrated from England to the United States. He attended the Oak Park High School and the Rock River Seminary at Mount Morris, Ill; liberal arts training was received at the University of Michigan, where he came under the influence of M. C. Tyler, colonial historian and student of Chaucer. This influence has been cited as an important force in Herrick's literary interests, medical and nonmedical, in his professional years.[1] Herrick returned to Illinois, taught school at Peoria and Oak Park, and began the part-time study of medicine at Rush Medical College in 1885, graduating MD in 1888. He served an internship at the Cook County Hospital, followed by private practice in Chicago and teaching at the Presbyterian Hospital, in academic affiliation with his alma mater. He engaged in general practice for a time, but his interests and skill, supplemented by exposure to some of the outstanding medical teachers in central Europe, seemed most suited to a consulting practice in internal medicine. And this was his destiny—to work particularly in diseases of the cardiovascular system. A description of sickle cell anemia and the clinical conditions of coronary vascular disease are his two outstanding contributions to the medical literature.

A case report of sickling in a Negro was published in the *Archives of Internal Medicine*, although the name "sickle cell anemia" was not proposed until more than a decade later. The clinical correlation of the physiology and the morbid anatomy of the coronary vessels was published in this *Journal* slightly more than 50 years ago. Herrick published, in all, more than 30 communications in the *Journal of the American Medical Association* or the *Archives of Internal Medicine*. The particulars of the sickle cell findings included a six-year history of a patient from Grenada, West Indies, whose symptoms began at the age of ten. During adolescence, the patient complained of weakness, palpitation, and shortness of breath, and suffered from icterus and a purulent otitis. When Herrick saw the patient first at the age of 20, he was described as being well developed, but several stigmata of sickle cell anemia were recorded. The observations included glandular adenopathy, deeply pigmented patches of leukodermatitis on the chest and abdomen, scars on the legs, icteric sclera, and pale mucous membranes. A soft systolic murmur was heard over the base of the heart and a faint systolic murmur at the apex. Neither the spleen nor the liver could be palpated. Examination of the blood revealed a red blood cell count of 2,570,000/cu mm and a white blood cell count of 40,000/cu mm. The color index was 0.78. Microscopic examination of the blood was described as follows.[2]

> The red corpuscles varied much in size, many microcytes being seen and some macrocytes. Polychromatophilia was present. Nucleated reds were numerous, 74 being seen in a count of 200 leukocytes, there being about 5,000 to the c.mm. The shape of the reds was very irregular, but what especially attracted attention was the large number of thin, elongated, sickle-shaped and crescent-shaped forms. These were seen in fresh specimens, no matter in what way the blood was spread on the slide and they were seen also in specimens fixed by heat, by alcohol and ether, and stained with the Ehrlich triacid stain as well as with control stains. They were not seen in specimens of blood taken at the same time from other individuals and prepared under exactly similar conditions. They were surely not artefacts, nor were they any form of parasite. In staining reactions they were exactly like their neighbors, the ordinary red corpuscles, though many took the stain heavily. In a few of the elongated forms a nucleus was seen. In the fresh specimen where there was a slight current in the blood before it had become entirely quiet, all of the red corpuscles, the elongated forms as well as those of ordinary form, seemed to be unusually pliable and flexible, bending and twisting in a remarkable manner as they bumped against each other or crowded through a narrow space and seeming almost rubber-like in their elastic resumption of the former shape. One received the impression that the flattened red discs might by reason of unusual pliability be rolled up as it were into a long narrow bundle. Once or twice I saw a corpuscle of ordinary

form turn in such a way as to be seen on edge, when its appearance was suggestive of these peculiar forms.

With supportive treatment and the use of syrup of iodid of iron, the patient improved. The blood at the time of discharge showed a red blood cell count of 3,900,000/cu mm, a white blood cell count of 15,000/cu mm, and the hemoglobin, 58% of the normal. Nucleated red blood cells and the tendency to an unusual cresent configuration of the red blood cells persisted in smaller numbers. Two years later the patient was seen by Dr. Ernest E. Irons, who reported an effusion in the left knee-joint and attributed it to physical trauma. The morphologic findings in the blood persisted. When the case was reported by Herrick, he recognized the unusual hematologic changes but admitted that a definite diagnosis could not be made. It remained for Huck, Sydensticker, and others to identify the entity as a familial malady among Negroes and for Pauling and associates, by electrophoresis, to identify an abnormal hemoglobin (hemoglobin S). This abnormal moiety was found to be characteristic of hemoglobin of patients with sickle cell anemia, thereby establishing the general principle that a molecular abnormality in a single protein may be responsible for a disease entity.[3]

Although by no means the first to describe coronary heart disease, Herrick was vitally interested in this subject and gave one of the best descriptions of overt coronary occlusion and angina pectoris early in this century.[4] In the lead article in this *Journal* for December 7, 1912, he reviewed briefly the literature of the preceding 100 years on the clinical and pathological observations in relation to the physiological and anatomical knowledge of the coronary arteries.[5]

No one at all familiar with the clinical, pathologic or experimental features of cardiac disease can question the importance of the coronaries. The influence of sclerosis of these vessels in the way of producing anemic necrosis and fibrosis of the myocardium, with such possible results as aneurysm, rupture or dilatation of the heart, is well known. So also is the relation of the coronaries to many cases of angina pectoris, and to cardiac disturbances rather indefinitely classed as chronic myocarditis, cardiac irregularities, etc. It must be admitted, also, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved.

But there are reasons for believing that even large branches of the coronary arteries may be occluded—at times acutely occluded—without resulting death, at least without death in the immediate future. Even the main trunk may at times be obstructed and the patient live. It is the object of this paper to present a few facts along this line, and particularly to describe some of the clinical manifestations of sudden yet not immediately fatal cases of coronary obstruction.

Before presenting the clinical features of coronary obstruction, it may be well to consider certain facts that go to prove that sudden obstruction is not necessarily fatal. Such proof is afforded by a study of the anatomy of the normal as well as of the diseased heart, by animal experiment and by bedside experience.

Attempts to group these cases of coronary obstruction according to clinical manifestations must be more or less unsatisfactory, yet, imperfect as the groups are, the cases may be roughly classified.

One group will include cases in which death is sudden, seemingly instantaneous and perhaps painless.

A second group includes those cases in which the attack is anginal, the pain severe, the shock profound and death follows in a few minutes or several minutes at the most.

In a third group may be placed non-fatal cases with mild symptoms. Slight anginal attacks without the ordinary causes (such as walking), perhaps some of the stitch pains in the precordia, may well be due to obstruction of small coronary twigs.

In a fourth group are the cases in which the symptoms are severe, are distinctive enough to enable them to be recognized as cardiac, and in which the accident is usually fatal, but not immediately, and perhaps not necessarily so.

Other clinical conditions discussed by Herrick included typhoid fever, hemophilia of the newborn (his first published communication following graduation), rupture of the urinary bladder, malaria, ainhum, pneumonia, polymyositis, peptic ulcer, lymphatic leukemia, chronic nephritis, ulcerative endocarditis, pulmonary tuberculosis, and rheumatoid arthritis. The respect that he enjoyed as a teacher and physician is reflected in the professional and nonmedical positions for which he was chosen. These were working positions in most instances, for he was willing to allocate a portion of his industry where it would be most effective. Herrick served as president of the following organizations: the Chicago Pathological Society, the Chicago Society of Internal Medicine, the Institute of Medicine of Chicago, the Association of American Physicians, the Society of Medical History of Chicago, and the American Heart Association. He was a lecturer of the Harvey Society of New York in 1931. The honorary master of arts in 1907 and the honorary degree of doctor of laws in 1932 were conferred upon him by the University of Michigan. The Association of American Physicians awarded him the George M. Kober medal in 1930 and the American Medical Association the Distinguished Service medal in 1939, the second to be awarded in its history.

In the humanities, a love for Chaucer was insatiable. At the age of 70, before the Chicago Literary Club, he discussed this subject, an indication of a persistent interest in literature and composition. Herrick will be remembered by his students, associates, and patients for his warmth of spirit and clinical acumen at the bedside. History will remember him for the description of sickling in the Negro and the clinical correlation of the form and function, normal and abnormal, of the coronary vessels.

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