

MD REMEMBERS: The Discovery of Hansen's Bacillus

■ A century ago Norwegian physician Dr. Gerhard Armauer Hansen reported to the medical society of Christiania (Oslo) his discovery in every leprous tubercle of "small staff-like bodies, much resembling bacteria." Although he was unsuccessful in infecting cats and rabbits with these bodies he thought they were the etiologic agents of leprosy and hence suggested that a chronic disease might be caused by a microorganism eight years before Robert Koch discovered the tubercle bacillus in 1882.

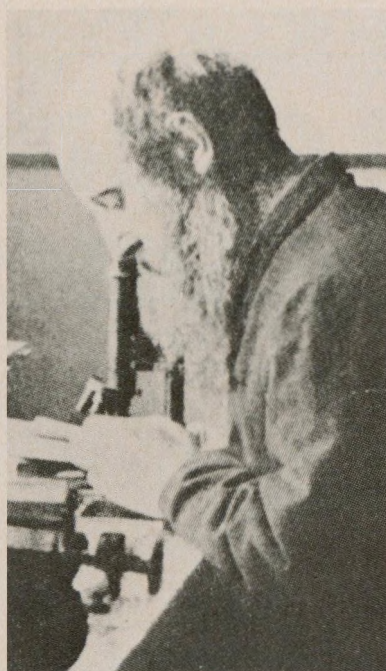
Hansen, born in Bergen in 1841, worked his way through medical school teaching at a girl's school and substituting for the anatomy demonstrator. He received his degree with honors from the University of Christiania in 1866 and following an internship in the national hospital served as physician to a fishing community in the Lofoten Islands. In 1868, when he became an assistant in the St. Jorgan Hospital in Bergen, more than one percent of the population in the city and its environs suffered from leprosy.

At that time one of the many physicians who believed that the disease was hereditary was Hansen's chief and father-in-law, Dr. Daniel Cornelius Danielssen. The two scientists had heated disagreements on this subject, when Hansen concluded from epidemiologic studies that the disease must be caused by a specific agent. In 1870 with a travel grant from the University of Bergen he went abroad to study histopathologic methods in Bonn and Vienna and later observed the rod-shaped acid-fast organisms that Albert Ludwig Neisser first stained successfully in 1879. The following year Hansen named the organism, now classified as a Mycobacterium *Bacillus leprae*, but in later years the pathogen bore his eponym.

Hansen's efforts to culture the bacillus and transmit the disease to animals failed, and when he inoculated the eye of a woman who had neural leprosy with material from a patient's cutaneous nodule without the experimental subject's consent, he lost his hospital post. Since

he was allowed to keep his position as leprosy medical officer for all of Norway, he helped to bring about a law that required the precautionary isolation of leprosy patients from their families.

A student of marine fauna, Hansen wrote a book on Darwinism and



Dr. Gerhard Armauer Hansen

delivered many lectures on the theory of evolution. In his later years he suffered from heart disease and in February, 1912 while visiting the fishing town of Floro on Norway's western coast he died in the home of a friend. His only child, a son who studied medicine, became chief of Bergen's tuberculosis hospital in 1929.

Modern Studies. Recent research on Hansen's disease was reviewed at the 10th International Leprosy Congress in Bergen last summer by more than 1000 participants from 75 countries. More than 65 papers presented at the meeting dealt with immunologic studies of leprosy which is recognized as an immune deficiency disorder as well as an infectious disease. Patients with lepromatous leprosy often lack all cellular immunity, have no skin reaction to lepromin but have high levels of circulating antibody to the

antigen and carry huge numbers of macrophages loaded with bacilli that the phagocytes are unable to destroy.

In an immunotherapy trial Dr. Ward E. Bullock of the University of Kentucky and associates induced weak systemic hypersensitivity in six of nine lepromatous patients by administering sensitized lymphocytes or transfer factor from subjects with a high degree of delayed hypersensitivity to lepromin. A more successful approach made by Dr. Soo Duk Lim of the University of Korea and Dr. Robert A. Good was the infusion of massive doses of lymphocytes from unmatched donors in an effort to induce a limited graft versus host reaction in 14 lepromatous patients. The weekly infusions, for periods up to 16 weeks, helped all these patients who had failed to respond to other forms of treatment. Following immunotherapy they showed softer, smaller external lesions and fewer bacilli in their tissues.

At the Bergen meeting clinicians expressed cautious optimism about two drugs that have been recently introduced in leprosy therapy. Clofazamine was found effective in sulfone-resistant cases and in severe lepra reactions, and the antibiotic rifampicin was reported to destroy *M. leprae* more rapidly than any other drug. Studies by Dr. K. Prabhakaran's group at the Garville leprosy center, indicating that DOPA is essential for the organisms, may explain their predilection for nerves, skin and other sites with free DOPA, and they may also lead to the development of chemotherapeutic agents that inhibit the utilization of this compound.

Recent research by Dr. Tore Gødal and an associate at the Addis Ababa Hansen Institute indicates that leprosy seems to be more infectious than its prevalence indicates, and subclinical infections commonly follow exposure to the organisms. Using lymphocyte transformations to determine immune responses, these investigators found a positive response in a fourth of the subjects who had lived in an endemic area for over a year.