ARNING'S OBSERVATIONS

Even to those who are aware that it is possible to demonstrate bacilli in the active macular lesions of neural leprosy, it will perhaps be something of a surprise to learn how long ago this demonstration was first made. It was fifty years ago (about 1885-86) that Professor Arning, of Hamburg, then engaged in the study of leprosy in Hawaii, tried to solve the question "why no bacilli could be found in the skin lesions of anesthetic leprosy, though in the nodular form of the disease every pinprick revealed them in numbers." He ultimately found them, though in very small numbers, in the nerves leading to the macules [*Virchow's Archivs* **97** (1884) 170], though he himself gives credit to "Babes, Gerlach and mainly Lie for learning where and how to find them in the leprides." At the time Arning reported his findings he coined and used the term lepride, to distinguish these lesions from the lepromata of the cutaneous type, and pointed out a histological difference between these varieties of lesions, namely, that the lepromata typically show a subepidermal zone free from bacilli while in the leprides this barrier is not respected.

A few years later (1889), as Arning himself states in a letter published in this issue (p. 102), he encountered a case with necrosis of nerves and concluded that this change was due to leprosy and

not to concurrent or secondary tuberculosis. Simultaneously other workers reported tuberculosis-like changes in leprosy, and time has brought practically unanimous support to the view that the tuberculoid lesions of leprosy are due to that disease.

Arning held that the fundamental differences between the "anesthetic" and "nodular" forms of leprosy are due to "the reciprocal state of the host and the invader," and he followed up this matter at the time of the Bergen leprosy conference (1909) when the principles of immunity and allergy had become better established. More recently (1921) he elaborated on the subject in a paper entitled "Syphilis und Lepra, eine Parallel," in which he pointed out that the difference between the two types results solely from reciprocal influence and power of aggression or resistence of the bacillus and its victim. With regard to one of these elements, variability of the pathogenicity, nothing certain is known, of course. Wide differences exist between different strains of the bacillus of tuberculosis, but it requires a susceptible experimental animal to determine anything on that score. The other part of this view, that the differences between the types and varieties of leprosy are due to differences of resistance and reaction on the part of the host, has general acceptance today.

If, after negative inoculation experiments (with rabbits rather than guinea-pigs which are now known to be much more susceptible to human tuberculosis) Arning was in error in concluding that certain atypical tuberculosis-like lesions of the viscera which he found at autopsy were also due to leprosy, there can be little surprise and certainly no discredit, considering the state of knowledge of that day when the tubercle bacillus had but just been discovered. The experience of various workers, as indicated in the comments on the letter referred to, do not offer much to support Arning's view, though they do not positively refute it. There is a possibility that treatment may modify the course of the disease sufficiently to prevent the formation of such visceral lesions, but not all of the cases that now come to autopsy have had effective treatment, and the tuberculoid lesions would be expected only in relatively resistant cases anyhow. However, it must be said that today, fifty years after Arning's work in Hawaii was done, the occurrence of tuberculoid lesions of leprosy elsewhere than in skin and nerve remains to be established.