splitting of the elastic lamina. As, however, processes of degeneration were wanting, he refused to call it arteriosclerosis. He suggests that these vessels might subsequently show arteriosclerotic change. From his observations we can only conclude that the kidney lesions have advanced with greater rapidity than those in the intima of the renal vessels, and his cases illustrate the point we wish to make that the narrowly defined form of arteriosclerosis as given Jores is not an essential factor in bringing out subsequent interstitial nephritis.

Roth described 3 cases of chronic interstitial nephritis without arteriosclerosis. In the kidneys, however, endarteritis was present in the small arteries. The cases were of relatively young individuals, and all of them had definite chronic or recurrent heart and arterial diseases. Yet with it all neither Jores nor his pupil sees any direct relationship insofar as a common causative factor is concerned in the simultaneous and progressive lesions in these three organs. These authors lay much stress on the finding of a single sclerosed arteriole or the mildest beginning of intimal degeneration as indicative of the influence of arteriosclerosis upon the kidney. No recognition is given to the fact expressed in their own cases that the fibrosis of the kidney was markedly advanced, and in the late stages of contraction, while the arteriosclerosis was only beginning. We can in no way follow the conclusion of this author as illustrated by his own cases that the chronic interstitial nephritis was the result of the early endarteritis demonstrated.

In the admirable work of Councilman (1897) the part played by the inflammatory process in bringing about the interstitial lesions of the cortex of the kidney was well demonstrated. In part, the cases studied included some of scarlet fever, diphtheria, pneumonia, and other infections, and the lesions described were of the nature of diffuse non-suppurative interstitial nephritis or types of glomerulonephritis. Of the latter, two forms were distinguished: a non-suppurative exudative form and a proliferative type. No clear distinction can be made between the etiological factors present in these two types, and it would seem that both may arise from the same causative factor. At the time of carrying out his work, bacteriological methods were not available to make a distinction between the various forms of streptococci, and we find the author speaking of the organisms isolated from cases of heart disease as pneumococci. I believe we will be correct in interpreting these results as indicating the presence of the Streptococcus viridans group. These organisms were found in cases of glomerulonephritis in large percentage, but the author's descriptions of the lesions indicate a transition between the glomerulonephritis and the diffuse, interstitial type. The work of Wagner bears out these findings, particularly in indicating the importance of the inflammatory process of scarlet fever and other infections in bringing about permanent interstitial change.