in which there is apt to be an 'ascending' infection with the colon bacillus. We must then conclude that the infection is a 'descending' one by way of the blood stream. That the presence of the colon bacillus is to be explained as a terminal infection or a post mortem overgrowth, I do not believe, for it is easy to eliminate cases of this kind as I did very freely, for the differences are quite distinctive. In ante-mortem terminal infections, the germs are largely in the capillaries, often forming large plugs, and consist of large fat bacilli, short bacilli, or sometimes diplococci, but always much larger and staining more deeply than the diplococcus forms I describe. Further, there is no evidence of inflammatory reaction about these large bacteria, while in the case of the diplococcus, they are enclosed by an inflammatory round-celled infiltration. Neither is it a post-mortem growth, for in this case, the germs are in the superficial cortical layers, and are always much larger and different in appearance and staining powers. Such germs can be seen with an ordinary No. 7 objective, while the diplococcus requires the 1-12th oil immersion at least, or better the 1-18th. Then again, the diplococci are always very few in number, perhaps only five or six in a section.

It is almost impossible to get perfectly normal kidneys in the post mortem room, but I have examined a few for diplococci in which microscopically the tissue showed no abnormality. In 10 such sections, 7 showed no germs; three showed rare diplococci similar to those in the nephritis cases, but on further examination I found that in one case there had been a hernia operation, and there was an acute local enteritis; in the second there had been a gastrotomy performed, and there was local peritonitis; and in the third a spina bifida had been removed. Thus in two there could have been infection from the intestinal tract.

That the process in chronic nephritis with productive inflammation is due to an embolic infection, is strongly supported by the histological features in the sections I have studied. The lesions in the chronic forms are identical with those in the acute interstitial as to their anatomical distribution.

In the great majority of the acute interstitial and acute mixed varieties, the areas of round-celled infiltration are to be found around the glomeruli or around the afferent vessels, and interlobular arterioles exactly as would be expected in an embolic infection. The same holds good for the chronic cases. In the arterio-sclerotic type, that the infiltration and proliferation is mostly confined to vascular districts needs only to be mentioned. In the early stages of the chronic diffuse nephritis one sees the inflammatory exudation in the same way about the afferent blood vessels, associated with connective tissue hyperplasia. The cells of the Bowman's capsules proliferate causing atrophy and hyaline degeneration of the glomerular tuft, or we get small fibrous patches about the vessels between the contorted tubules.