lesion in the case has been gastric and has been followed by infection of the abdominal lymphatic system and the portal area.

From all the animals which I killed, I was more fortunate than my predecessors in gaining a characteristic micro-organism. This may have been due to the fact that I employed a somewhat different method of gaining my cultures. Instead of taking the media and inoculating on the spot, all I employed was a series of sterilised glass pipettes in which I collected relatively large amounts of the juices of the various organs ; ascitic fluid, blood, etc., and then when back in my temporary laboratory, either upon that or the following day, I inoculated my media. By this means, constantly from the lymph juice of the abdominal glands and from the liver juice or bile and more rarely from other organs and fluids, I obtained in each case growths of a characteristic micro-organism ; small, polymorphous, at times appearing as a diplococcus and at others as a diplobacillus which by its polymorphous character gave me a considerable amount of trouble, until I found that employing the same broth tube, at the end of 24 hours I obtained the one form, at the end of 48, the other. Further study showed me that this micro-organism was in reality a short bacillus with polar staining, in this resembling to some extent the micro-organisms of hæmorrhagic septicæmia in the lower animals, but unlike them, possessing a slight capsule. I was able to grow this upon all the ordinary media of the laboratory. Into the character of this micro-organism I will not here further enter, beyond stating that I found it pathogenic for rabbits, guinea pigs and mice, rabbits dying in from 15 to 35 days, guinea pigs in from 30 to 35 on the average.

The characteristic features of this disease—the ascites without jaundice, the gastric and intestinal disturbance and the condition of the liver—led me seriously to consider the points of similarity between the course and symptoms of these cases and those present in portal cirrhosis in man, and though it may seem a small matter, I was especially struck by the fact that the first post-mortem which I performed upon a case of atrophic cirrhosis upon my return from Nova Scotia in 1895, presented the same gelatinous œdema of the mesenteries and intestinal walls which was so prominent a feature in the Pictou cattle disease.

Thus on and off for the last three years my attention has been directed towards this possibility of discovering bacteria in ordinary progressive portal cirrhosis. Upon three occasions I have thought that I have gained specific micro-organisms. In two, unfortunately, the growth became contaminated with the colon bacillus, and as this occurred on the eve of my vacation I was unable to continue the