is formed which cannot absorb the most fluid exudate and it consequently becomes encapsuled and remains.

I recall one instance of an encysted effusion of the left side lying between the diaphragm and the lung, in which the connective tissue wall measured a centimetre in thickness. The fluid was pale, straw-colored slime and quite free from pus, but evidently represented the ultimate results of autolytic change. When, however, the fluid is more completely absorbed, ther. we may get calcification of the remaining exudate or even, as a final result, ossification. The specimen which I pass around and which was obtained in the dissecting room this winter, is a good example.

I recall also an interesting autopsy which I made on an asylum patient in which, at the bottom of the right pleural sac, was a large finger-like mass of bone, all that was left of an old empyema, but beneath the diaphragm of the same side were a mass of old adhesions in the centre of which lay the right kidney, a mere remnant, whilst the left kidney was enormously hypertrophied. The whole history of this case was clear at the autopsy, yet, it had occurred so many years before that no clinical record of it could be obtained.

Of very great interest from the standpoint of general pathology is the question of how a sero-fibrinous exudate becomes purulent. We must recognize, of course, that primarily this may be due to the form of infection. The question has quite recently been studied experimentally at the Rockefeller Institute by Opie. Opie studies the changes which take place in sterile pleural exudates which had been produced either by the injection of aleuronat or turpentine. As a result of his studies he has shown that the breaking down and self-digestion (autolysis) of an exudate, whether in the pleura or in an abscess, is due to two enzymes, at first to one derived from the polymorphonuclear leucocyte, which he calls leucoprotease, and which on acts in an alkaline solution, and succeeding this an enzyme derived from the large mononuclear cells, which he calls lymphoprotease, which will only act in the presence of an acid. In the early stage of an experimental exudate the leucoprotease predominates, in the later stage the lymphoprotease. Both these enzymes are inhibited by substances present in the blood serum, and Opie has found that in a sero-fibrinous fluid in experimental pleuritis there are practically all the inhibiting substances of the serum. We see, therefore, that in a sero-fibrinous exudate there are two influences at work—one, the enzymes dissolving and digesting the fibrin, the other the serum stopping this digestion. If the digestion of the fibrin is delayed, organization of it takes place, connective tissue invades it and adhesions are formed. If the serum inhibition is slight and diminishes, then autolysis becomes more active and not only do these enzymes act upon the formed elements of