from apparent rotations and dislocations of coils of intestine; from strangulations, internal and external; and a few times from intussusception. I have seeen many cases of what I supposed to be typhlitis and peri-typhlitis giving rise to severe local, but very rarely to general, peritonitis. Twice I have seen it caused, and frightfully violent in its course, by ulceration and perforation of the appendix virmiformis from lodgment of a foreign body; in one the foreign body being a kernel of wheat, and in the other a hard, half-cooked bean. I have seen a severe local peritonitis caused by the irritation and extension, without penetration, of an acute destructive ulceration of the external tissues in the right groin, causing first adhesion of intestine to abdominal walls; then, through a series of years, repeated and wearing attacks of colic from obstruction; and at last general peritonitis and death. I believe too, some authors to the contrary notwithstanding, that I have seen peritonitis occur as an idiopathic, primary disease, from exposure to cold, from wet, and from great fatigue, just as from such causes inflammation of other serous membranes may arise.

But I have never yet seen peritonitis caused by perforating ulcer of the stomach, nor by perforating typhoid or scrofulous ulcer, nor by perforating ulcer of any of the urinary passages or bladder. I have never seen it from rupture of the gallbladder, or impaction of gallstones and ulceration and perforation of the common duct. I have never seen it from the bursting of hepatic or peri-nephritic, or any other abscesses into the peritoneum. Indeed, with the single exception of one exceedingly violent attack of peritonitis, which I once saw, from the accidental bursting of a distended ovarian sac, and the extravasation of its contents into the cavity, I never saw inflammation of the peritoneum caused by the pouring into its cavity of any of the diseased or healthy fluids of the living body—always excepting, of course, traumatic cases from penetrating wounds. Now there may be very little in all this of importance enough to relate it; and yet, with the almost uniform testimony of authors to these last as recognized and not infrequent causes of peritonitis, it struck me that my experience, or want of it, might be worth mentioning, and more especially for the sake of bringing out that of others. The second point of my experience concerning which I propose to speak has to do with pathological processes, and with the progress and course of some of the severe and alarming cases of general acute peritonitis, which survive the first work of the disease, to linger through a few weeks more of suffering, and then die, or to make, through a long and tedious confinement, a more or less perfect recovery. It relates to the disposition made in some such cases of the effused products of inflammation, and instead of being at variance with books, may serve in some trifling degree to illustrate their teaching. We know that in such severe acute cases there is often immense effusion of turbid, flocculent, whey-like fluid, in which may usually be found flakes and soft masses of coagulated fibrin, in greater or less abundance; and that this yellow fibrinous deposit not only floats about to some extent loosely in the thin fluid, but also gravitates to dependent positions, and is found accumulated in the pelvis, and along the course of the spine, and especially in the folds and duplicatures of the mesentery. We know, too, that if such cases do not terminate early by death, such exudations cannot remain long without undergoing important change. We know, also, that if the patient is to recover, such change will *usually* consist of more or less rapid absorption of the effusions; first, absorption, perhaps rapidly, of the fluid portions, and then, much more slowly, of the solid.

But there are cases where the patient does not die early, and lingers on, in which this process of absorption either does not take place at all, or in which it may be hindered or arrested at any stage of its progress. In such cases as these it may happen, if no attempt at absorption has taken place, that the great and long continued pressure upon the weakened tissues may cause ulceration and perforation of the peritoneum at some portion or other of its surface, and allow the pouring forth of the accumulated effusion. Or, supposing the thin, more watery, portions to have been absorbed, and that the heavier and more solid remain, then these, through the formation of adhesions, or through agglutinations between coils of intestine, become limited and enclosed, *i.e.*, capsulated. In this manner are formed those hard, cake-like tumours which are sometimes felt in the abdomen after acute severe peritonitis has passed into the chronic stage; tumours which, in some of the various metamorphoses which they undergo, may suppurate, and at last, by a process of ulceration and perforation, find an exit for their contents. The possibility of this event, and its actual occurrence, are mentioned by most authors. Baudelacque, in his large work upon puerperal peritonitis, makes repeated allusion to it, and cites several instances from various sources. I have, however, supposed that it must be, after all, comparatively rare, and have alluded to the subject