

was not a sign of plastic exudation or adhesion anywhere. In two cases of inguinal hernia the omentum appeared indifferent and unaffected; in one there was well-marked localized subacute peritonitis after incomplete reduction of the hernia; in the others the great omentum did not extend below the umbilicus, although there was plastic exudation around the operation-wound. A case of chronic cholecystitis, leading to empyema of the gall-bladder and perforative peritonitis (again a Murphy button case), showed neither old nor recent adhesions of the omentum. Doubtless I might multiply examples. I have, however, given you enough to show that the omentum does not by any means necessarily send out processes to cover over and adhere to inflamed abdominal areas. It is not merely a question of the quality of the inflammation that determines the adhesions. Here are at least a few examples in which plastic and adhesive exudation might easily have been set up—only the omentum did not manage to find its way to the injured area, and as a consequence no adhesions developed. There is no such protective sending out or wandering or chemiotaxis of portions of the omentum to cover over and shield areas of injury and inflammation.

It cannot be urged that the omentum broods like a beneficent Providence over the abdominal contents, descending to minister help where it is needed and to arrest the spread of harmful inflammation. For in the first place, as I have just remarked, there are inflammations and inflammations—where there is very acute disturbance, or where, again, as in typhoid and the later stages of exhausting illness, the reactive power of the organism has sunk to a very low ebb, a serous rather than a leukocytic and fibrinous exudation obtains; and where this is the case there can be no adhesions, even though the omentum be lying over the area of injury.