lungs empyema will constantly be noted to contain a pure culture of one or other coccus or a mixture of both.

Kiener, however, is rather upsetting in the statement founded on examination of a good quantity of material post mortem, that he had never seen staphylococci in a meta-pneumonic pleurisy before pleurotomy or rupture of a bronchus and that in autopsies following the exitus lethalis in infected broncho-pneumonia, he had found diplo and streptococci in the pleura, pericardium, endocardium and meninges and never staphylococcus, even though he had been able at times to demonstrate it in the lung. He finds staphylococci, in short, most frequently in cases of tuberculous pyopneumothorax. As cited above, he has seen cases of serous effusion with staphylococci which recovered without evident pus formation. Prinz Ludwig-Ferdinand, of Bavaria, had two similar cases under his observation at Munich.

Probably no two recorded cases of empyema run exactly the same course. The condition is but seldom unaccompanied by some more or less disturbing element—its very proximity to heart and lungs is bound to react on these organs, so that life itself is threatened. A large exudate of pneumococcal pus in the left pleura may be more difficult of treatment and exhibit more alarming symptoms than a much smaller streptococcal one in the right, although, in a general way, streptococcal pus or the toxins absorbed is more offensive to the organism than the pneumococcal variety. In average cases, however, we are justified I think, in relying to a considerable extent on the bacteriological report for the formation of our prognosis and selection of a line of treatment. Whether from antecedent causes or inate virulence the pneumococcal variety appears the most benign. The streptococcal empyema is the most sudden and critical in onset, the staphylococcal most prone to relapse and irregular in progress towards recovery, while the tubercular variety, though at times easy to suppress, in cases of local tuberculous pleuritis is most baffling, even hopeless in advanced phthisis or other grave visceral lesion. The morbid anatomy also differs to no small degree according to the nature of the bacillus observed—whether by coincidence or not. For example, in the local tuberculous pleuritis there is a great thickening of the two pleurae; fibrin is thrown out and becomes vascularised; the tubercles remaining in its deeper layers. The pneumococcal and streptococcal pus is almost without exception reported as being agglutinated, pervaded by masses of fibrin of larger or smaller size; while in marked distinction lies the staphylococcal pus, of thin watery aspect, not walled off by any masses of adhering fibrin and apparently acting as a peptonising ferment