marks the lessened space for breath and rusty sputa the extreme congestion in the lung. There is little pain, but there is much fever and distress, and the patient has a burning skin and patches of redness on the malæ. On the seventeeth day, or later, a crisis occurs; the temperature drops suddenly, the physical signs alter, the dulness lessens, and a coarse crepitation replaces the fine. In an incredibly short time there is convalesence, and the lung-signs are normal. In this case no one can doubt that the lung-alveoli are blocked by exudation so thoroughly that, in fact, no tissue in the lung is pervious to air except the larger bronchi. But there is speedy resolution; the plastic exudation readily liquefies, resolves, and is absorbed and expectorated. It is plainly not the *extent*, but the *nature* of the lung deposit which is dangerous, and it is equally plain that the alveoli, although blocked and filled up, have not been injured, for we find that the recovery after such an attack is perfect, and the integrity and elasticity of their walls is complete. There is no ulceration of lung at all in the case, and the organ returns to a condition sound and pervious, with free elasticity, a perfect double circulation, and all functions unimpaired. Where, then, is the difference between this sthenic pneumonia and the alveolar disease which I have described as leading to phthisis, nay, as so often destructive of the lung and of the patient? Doubtless it is found in the nature of that diseased product which will not liquefy nor resolve, but preciptates destructive ulcerations of the surrounding tissues. And let us go a step further back, and ask why is not the product of disease a healthy, removable product? why is it low in organization, liable to degenerative change, but not liable to such a complete and rapid form of degeneration as would remove it from its dangerous impaction in the lung? We are compelled here to seek an antecedent cause, which is higher up in the chain of morbid events, and we say, here was a "constitution," or an hereditary predisposition, which caused this inflammatory block in a portion of one lung to be of infinitely greater danger than the inflammatory block of a whole lung in another individual. I wish I could explain this to you; but here are the facts, and they are hard of interpretation, and we are driven to obscure terms like "diathesis" to cover our ignorance. But do not mistake me; up to this point all is clear, but behind it lies the field for future advances, and perhaps a lessened mortality.

Look at a case of unresolved pneumonia, and you see "phthisis." Such a case has generally not been sthenic, the temperature has not been excessive, and there have been variations looking like recovery. The dulness has not been complete, but in patches and the locality of these patches has partially changed; one has cleared up, only to be replaced by another. The rusty sputa are rarely seen. I have said the pyrexia was less marked, but the fever changes its character into a slow remittent with the diurnal variation of phthisis; low temperature in the morning, and 103° in the evening. At the end of two or three months the case has not cleared up, and your patient is weak, emaciated, and has night-sweats. His lung (one lung) may be dull in parts at the base, much more rarely at the apex or in the middle near the root of the lung, and not only is the breath-sound tubular, but there is crepitus here and there. The medical attendant is alarmed, and with reason, and says he would gladly have exchanged such a case for a true active pneumonia with much fever and high temperature, and complete block of a whole lung. In this opinion he is right. He will ask you anxiously in consultation if this be tubercle. I care not for names. It is a deposit in the lung which will not resolve, and which threatens to destroy the alveolar walls and to give rise to the train of symptoms which indicate ulcerated lung-tissue. I would have you carefully note the small portion of lung engaged as compared with a sthenhic pneumonia, the deficiency in the resolution of the local disease, and the passage of pyrexial fever into hectic. Now we have brought our comparison of cases and our reasoning on them to this point, that the difference between the unresolved pneumonia and the catarrhal block of the alveoli or the old localised deposit of tubercle—call it which you will—is in the nature of the product extended into and around the alveoli, and not in the extent of lung engaged. For in the first case you have a commencing