tion you find the chest-walls slightly flattened to the second or third rib, lessened expansion, slight dulness, and deficient breath-sounds. There may be a whiffy character of respiration, and slightly increased vocal resonance. What is this case? I believe it to be one of *severe alveolar catarrh:* the vessels have been blocked by large granular cells which have undergone fatty degeneration and been expectorated. But the walls of the alveoli have been damaged and collapsed, and a portion of lung is permanently blocked. This is not a tubercular case, and your patient may remain for years with the same physical signs, and without extension or even renewal of disease. We meet with them every day in practice. They are delicate, but perhaps have no cough. Yet they should live with care, for they carry with them a liability. Either a second attack may occur in a different part of the lung, or the old nidus of disease in the apex may break up, and the degenerated product be carried into the circulation, and the patient be inoculated with morbid matter, resulting in a second deposit, with accompanying hectic and all the history of phthisis. The above symptoms may also, no doubt, approach insidiously, and with premonitory conditions obscure but intelligible to the observant; and this early stage has been much dwelt on. I have long believed that there are symptoms before there are physical signs, a systemic or constitutional condition before any local disease exists; and a very serious question arises here. Do the whole train of symptoms in phthisis, the hectic irritative fever, sweating, waste, and exhaustion, only depend on a localised lung disease, of which they are the reflection, and which stands to them in the relation of cause to effect, just as the diseased joint gives rise to suppurative hectic? or is there a primary constitutional disorder, of which the local disease is only a sequence, expression, and result? Would a healthy person ever have such an alveolar catarrh as I have described, resulting simply from a neglected cold, and without any previous disorder of health? It is true that by far the most important agent in precipitating lung disease is inflammation; but is the inflammation itself idiopathic, or has it arisen out of a previous condition of blood or tissues which have impressed on it a stamp and form, and made it not quite what we call a healthy inflammation? For there is a healthy inflammation, as you know, and is this it? "Certainly not," you reply, "the products of healthy inflammation are temporary, plastic, removable, not permanent, ill-vitalised, degenerative, like these blocked alveoli." Then why is it so? We all go through our severe colds and outlive them. Whence this insidious filling of the lung with epithelium granules, tubercle—what you will? or this acute localised patch of deposit after a few weeks' fever, which will not organise, nor resolve, but remains to degenerate, ulcerate, waste? It is true that the fever in phthisis is generally a measure of the irritation of the lung, but is there no fever premonitory and leading the way to these lung alterations? I must answer in the words of Latham: "Pulmonary consumption is only a fragment of a great constitutional malady, which it belongs to a higher discipline than any mere skill in auscultation rightly to comprehend." And as regards premonitory symptoms, I ask you to regard with much anxiety and grave care *the union of subfebrile symptoms with progressive waste of the body.* Here is danger without any physical signs, but if the latter be superadded you have lung disease, and localised lung disease with fever is catarrhal pneumonia, tubercle, hyperplasia of adenoid—what you will, but, above all, it is phthisis.

In studying such a case you must exclude several causes which might mislead you. Emaciation may be due to dyspepsia, and cachexia from syphilis, diabetes, chronic abscess, joint affections, and fever, as well as other alterations of health. In all cases the rule should be to regard moderate fever of the remittent kind and wasting of the body with great suspicion.

Let us just consider the progress of a healthy sthenic pneumonia in contrast. Your patient, hitherto healthy, has been exposed to cold, generally rather severely, and after sweating. He shivers, and has an immediate rise of temperature to 103° or 104°. This is followed by moderate but rapidly-increasing dulness over one lung from the base upwards even to the apex. A fine crepitus succeeds, and the breath and voice-sounds become tubular. Dyspnœa