tory of consumption), and if your patient preserve a healthy digestive system the stage of the disease is unimportant. Yet let us briefly examine these stages, which have their use and must be retained for purposes of study and accurate description, but do not mistake their meaning. They refer only to a state of lung, and not to a state of health. The *first* is the period of deposit or thickening of the lung, the *second* that of softening or disintegration, and the third that of *cavity*. The *first stage* of established disease of the lung—recognizable, that is, by physical signs denoting alterations in its structure—means such a block of a portion of the organ as shall render it less pervious to air. Hence the natural resonance which it gives on percussion is lost, the *intensity* of the air-sound as it enters is diminished, the *character* of the breath-sound is altered by the changed elasticity of the alveoli and minute tubes, and the air leaving the lung on *expiration* gives a more prolonged tone. The natural resonance of the voice and cough in the bronchial tubes is intensified and more directly conveyed to the ear, because the elastic and air-containing tissue of the lung is replaced by a solid medium which is a better conductor. It is obvious that this condition may be due to several causes either within or without the alveoli which have become closed up and impervious to air. An ordinary catarrhal pneumonia, resulting in proliferation of epithelial growth, will produce such an *intra-alveolar* block. The ultimate vesicles of the lung are filled up, the elasticity of their containing walls is destroyed, and what is called vesicular breathing is at an end, the entering air, when it reaches the ultimate bronchioles, being unable to penetrate further. Thus it is that the soft, gentle sound of pure respiration, which we hear in a healthy lung, is lost, and if the lung-block be considerable the breath-sounds are of necessity bronchial, being, in truth, only formed in the tubes, and not in the ultimate vesicles. It is safe to say that a large proportion of the cases which end in phthisis, or ulcerated lungs, begin in this way, by catarrhal products blocking the alveoli of a portion of the lung; and if you were to cut down on this diseased spot, you would not find the grey, semi-transparent granules of Bayle, but the products of an inflammation. They have the same clinical history, but such alterations in the lung are not tuberculous, although you will find in practice that if not speedily liquefied and expectorated they either become caseous, and soften, breaking down the alveolar walls and ulcerating the lung-tissues, or, in certain cases, undergo the cretaceous transformation and become obsolescent. But, again, this pulmonary block may be *outside* the alveoli, and in that interstitial tissue which is spread through the lung—peribronchial and perivascular—that is, surrounding the vessels and bronchi, the *adenoid* or lymphatic tissue described by Dr. Burdon-Sanderson. It has been well demonstrated that it is capable of overgrowth from irritation, and in such circumstances it ceases to be the fine soft bed in which vessels are contained, and hardens, thickens, and compresses both the bronchi and alveoli, causing the collapse of the latter. The vessels are also enwrapped, as it were, lessened in calibre, and the circulation, both of air and blood, interrupted. In a later stage, as I shall show you, this process ends in producing what is called fibroid alterations, which both harden and contract the lung. This first stage of phthisis, then, consists in a block of the lung; and two causes can produce it—the intra-alveolar plugging by the products of inflammation, and the extra-alveolar pressure by the natural interlobular tissue becoming thickened. A third cause is more rarely found in what are called "dust" cases—the dust of coal-mines, factories, and potteries becoming impacted in the alveoli, and mechanically blocking the lung; and this is invariably accompanied by an overgrowth of the adenoid and fibrous tissues, and all such cases, when chronic, belong to the class of fibroid phthisis. Let us consider these causes of lung-block clinically. You may not be called to see the first approaches of such a case, but very often indeed you will find a patient with this history and physical condition of a part of one lung, generally the apex. He will tell you that he has had cough after taking cold some weeks or months previously. A feverish cold, with pain in one subclavicular region; cough, slight greyish starchy expectoration, some emaciation, and generally depressed health. On examina-