the general head of "Cases Arising from Inflammation," gives several instances where apparently the phthisis had its direct origin in either acute or chronic pleurisy. In children, though pleurisy has generally a favourable termination, and they appear sometimes in a wonderful way to outgrow completely the subsequent contraction, yet in them the disease, more frequently than in the adult, terminates in empyœma, with its accompanying train of fresh dangers. To-night, therefore, I propose very briefly, to lay before your notice some of the lesions which I have occasionally observed to follow pleurisy, and will hope that, in the discussion which may follow, I may elicit from other members their opinion as to how far my observations agree with their own.

These lesions may be arranged, I think, in three  $\frac{1}{2}$  in. on deep inspiration, chiefly on right side. groups, as follows:

1st. Those which result simply from the inability of the lung to expand, owing to the fibrous adhesions. 2nd. Those which are due to the formation of new

growth through the lung. 3rd. Those due to purulent absorption, after the pleurisy has become an empyœma.

I am aware that many consider my first-class a myth, and deny entirely that any evil results follow from this inexpansive condition. Austin Flint, referring to adhesions, says, "however extensive, they occasion no appreciable disturbance of respiration. They are in fact innocuous, and perhaps confer exemption from recurrence of plcuritis on the same side, nor do they give rise to any distinctive physical signs."

Certainly a prior reasoning would not lead us to imagine it to be an advantage thus to have a lung done permanently up in splints, and I cannot say that my observations would lead me to agree with his statement, for I think, if we notice carefully, we shall find two different conditions brought about, according as the adhesions on the one hand are partial or merely local bands; or, on the other hand, the lung is universally adherent.

These local adhesions or bands interfere comparatively slightly with the movement of the chest wall, but prevent altogether a certain portion of the lung tissue from expanding, especially the alveoli towards the upper portions of the lung, and at the back near the spine. As a consequence, we must in time get a compensatory emphysœma of those portions which can expand. It is condition of things which I have several times thought I saw in the out-patient room. The following is an example from an in-patient.

Brompton Hospital March 6th, 1877. No family predisposition; habits steady; eight years ago suffered from pleurisy on left side; was three weeks in bed; six weeks ill altogether; thought he quite recovered from the illness; following winter suffered from cough, which, though better during the summer, grew more troublesome each succeeding winter. For the last two years has never been quite rid of it.

On admission complained of cough, not very severe, accompanied by frothy expectoration; pains through chest, chiefly left side, and some shortness of breath. He was a short built man, fairly nourished

Measurements of chest:

Above nipple: R.  $16\frac{1}{4}$ ; L.  $15\frac{3}{4}$ ,  $\frac{1}{4}$  in. movement

Below nipple: K.  $15\frac{1}{2}$ ; L.  $15\frac{2}{4}$ , in movement, a on deep inspiration, chiefly on right side.

Physical Signs .- Left: percussion resisting anteriorly, dull to 2nd rib; respiratory murmur very deficient, and slight bronchial click occasionally above; expiration much prolonged below. somewhat hyper-resonant; respiration harsh; 2nd sound accentuated over pulmonary.

He remained six weeks in the hospital, and was sent out with his physical signs much about the same, but his general condition much improved. In this case I would trace the beginning of his trouble to the attack of pleurisy, eight years previously, which probably left some strong adhesions towards the middle or upper part of the left lung. This prevented the air-cells above in great measure from expanding, and gave rise to an emphysœmatous condition of the lung below, and this again in turn, owing to deficient aeration would cause obstruction to the circulation, as evidenced by accentuation of 2nd sound over pulmonary artery, and a congested state of the lining membrane of the bronchi and alveoli, as shown by the frequent bronchitic symptoms.

Some may object to a case like this, that the pleuritic adhesions are the sequence, not the antecedeut, of the emphyœma. As Dr. Douglas Powell has pointed out, when a portion of lung becomes damaged in texture by disease, it ceases to follow accurately the expansive movements of the chest wall. A certain gliding or rubbing motion takes place between the two normally corresponding pleural layers at this point, friction, local pleurities, and adhesion result. No doubt this is the order of things occasionally, bringing about a precisely similar state in the end; George B-, bricklayer, æt. 46, admitted into but, in the case I have quoted, the definite attack of