A NEGLECTED FACTOR IN THE ETIOLOGY OF LUNG COLLAPSE IN EMPYEMA.

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Although the causation of lung collapse subsequent to prolonged inflammatory implication of the pleural sac is not a subject about which much obscurity exists, yet we cannot help thinking that the relative importance of the factors concerned in its production has not been hitherto sufficiently defined and emphasised.

Before examining this subject, however, it will be necessary to state that although the points to which we particularly refer may be applicable in a variable degree to every case of purulent accumulation in the pleura, yet it is to those instances of uncomplicated general inflammation of the sac that we especially propose to direct attention.

Suppose we take a case in which the serous exudation has given place to empyema, and that one of the usual methods of drainage, by trochar and cannula, intercostal incision, or resection of rib with insertion of tube, has been applied thereto. Under the most favourable circumstances that may possibly succeed the procedure, we almost invariably find a considerable diminution of the space previously occupied by the lung of the affected side, a consequence generally ascribed to the organisation and subsequent contraction of the fibrinous envelope which the inflammatory process has left on the visceral aspect of the pleura. Although this may be, in the majority of cases, the final cause of the loss of lung area, yet it is not the first or most important element in bringing about the undesirable result of collapse. Where the drainage of an empyema is accomplished by one of the methods mentioned, the withdrawal of the purulent contents is accomplished at a heavy cost, inasmuch as there is no efficient prevention to the entry of air into the sac; and although the importance of avoiding it has long been recognised, and striven against by such various but imperfect methods as those of Benjamin Bell, Easton, Potain, and others, the difficulties to be surmounted in endeavouring to prevent it are so great that its occurrence has been regarded by some writers with a fatalistic complacency as inevitable. But to this fact more than any other is due the lung collapse which we find follows its occurrence. An
examination of the mechanical and physical aspects of the case will prove this.

When the action of the inspiratory apparatus establishes negative pressure within the thorax it is practically impossible to prevent the entry of air to some degree through the parietal opening. This air is not, of course, expelled when expiration takes place, because from its own intrinsic weight the lung tends to spread out at the base, and to fall away from the chest wall at the dome, thereby tending to close the parietal opening (which we presume will be situated not higher than the fifth intercostal space and in or about the mid-axillary line), and consequently cutting off the means of exit of the imprisoned air. As a result, we find that after a time this air begins to accumulate at the apex of the pleural cavity, and is, furthermore, reinforced by the gases which are evolved when decomposition of the pus sets in below. After a time the quantity of air increases to such an extent that it becomes quite impossible for the lung to approach the chest wall in this situation, and up to a certain degree the compression goes on until the lung capacity is considerably curtailed. This condition of things gives rise not only to compression of the lung, which after a time becomes bound down in its diminished area by the fibrinous coat upon it, but it also favours the continuation of the inflammatory process by preventing the approximation of the pleural layers and the obliteration of the peccant cavity. Now it cannot be denied for a moment that the causes which have prevailed to prevent any solution of this difficulty are the mechanical obstacles which debar the possibility of securing efficient drainage, coupled with the fact that at the same time the ingress of air is prevented. And although it may be well-nigh impossible to exclude air entirely after intercostal incision has been effected, yet we think that it might be greatly lessened by due care in the selection of drainage appliances; while, furthermore—and this is the point we wish particularly to enforce—greater attention should be bestowed on the imprisoned air with a view to its removal from the pleural sac.

To this end we would urge that where physical signs admit of no doubt as to the presence of air to any considerable degree in this situation, its occasional removal by means of a Dieulafoy's aspirator is imperatively demanded.

The method to be adopted should be exactly similar to that applied when dealing with a serous infusion, except that the needle will require to be inserted in the second or third intercostal space, and that no receptacle will be required under the delivery tube, inasmuch as it is air and not fluid which is being removed.
The quantity to be extracted will vary, of course, with the amount accumulated, combined with the degree to which organisation of the fibrinous coating on the lung has advanced and permits of expansion of the lung. It is obvious that the aspiration of air should be adopted as early as possible after the signs of the condition declare themselves, for the occurrence of organisation of the lymphoid deposit on the lung to any great extent will render the operation not only more distressing to the patient, but also less likely to be followed by the result aimed at.

If early action on these principles be taken in a case, it will generally be found that five or six charges of an ordinary Dieulafoy syringe may be pumped out before the patient begins to feel a sense of tightness and oppression in the thorax. These are the signs which should guide the operator as to the moment when withdrawal of the air should be suspended.

By the undelayed application of this method the likelihood of adhesion between the layers of the pleura is greatly increased; the respiratory distress is lessened, and the ultimate loss of lung area is considerably diminished.