Argyll Robertson sign, may be found associated with loss of tendon reflexes in patients with a benign disorder that has nothing to do with syphilis whatsoever.

Note.—After this was written, Mr. Foster Moore kindly allowed me to read the typescript of his paper on the non-luetic Argyll Robertson pupil, which he read at the recent ophthalmological congress. In one of his fifteen patients with this abnormal reaction of the pupil, the knee-jerks were absent: in another they were obtained with reinforcement only.

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ACUTE MASSIVE COLLAPSE OF THE LUNG FOLLOWING LIPIODOL INJECTION

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In view of the more general use of lipiodol injection of the bronchial tree as an aid to the diagnosis of pulmonary disease and of the increasing importance which collapse of the lung is assuming in the etiology of acute pulmonary conditions, this case is of special interest.

The patient, a healthy-looking young man, aged 18, was sent to us because of a pain in the side, which he had had for two months and which his doctor suspected as a pleurisy. The pain was sufficiently severe to prevent him breathing deeply. There were no physical signs except feeble breath sounds. He was admitted to hospital for further investigation to exclude the possibility of fibrosis with bronchiectasis. A simple skiagram of the chest showed no abnormality. A lipiodol injection of the bronchial tree was done by the crico-thyroid route. To suit the convenience of the hospital this procedure was carried out on the *x*-ray table. The following points are to be noted:

1. The injection was done on the *x*-ray table, the film being in position, and the time from the injection to record of the skiagram was considerably less than five minutes, probably only three minutes.
2. At the time of the collapse the patient showed only slight respiratory distress. The physical signs present were diminished movement, slight impairment of percussion note, and a considerable diminution of the air entry over the affected side of the chest.
3. The speed with which recovery took place according to the skiagraphic records.

Jacobaeus, Selander, and Westermarck,¹ in their paper on acute massive collapse of the lung, record four such cases in which the injection of lipiodol was followed by collapse of the lung. These were from a series of eight patients with normal bronchi whom the authors were injecting for purposes of investigation, and it is somewhat astonishing that they should have found so high a proportion in whom the injection was followed by massive collapse.

In our case the bronchi were normal, but in a previous investigation on the subject of dry haemorrhagic bronchiectasis we occasionally injected lipiodol into a patient with normal bronchi, yet this is the first case of massive collapse from this cause that we have seen. In a series of many hundreds of lipiodol examinations for many varying pulmonary conditions at Victoria Park Hospital no such case of massive collapse has occurred.

A point of considerable interest is the length of time between the injection and the collapse. In Jacobaeus's cases

ten to fifteen minutes elapsed between the injection and the skiagrams. In our case the time between the injection and the record by skiagram was probably not more than three minutes. That is to say, the collapse was practically immediate. Coryllos, Birnbaum,² and also Lee, Radwin, Tucker, and Pendergrass³ produced massive pulmonary collapse in animals by plugging a bronchus with a balloon. The collapse was not complete till six hours later. The latter authors also obstructed a bronchus with a solution of gum arabic, causing complete collapse in four hours. The time taken for the collapse to disappear in Jacobaeus's cases was from two and a half to nine hours; in our case there was considerable improvement in half an hour, and complete recovery in twelve hours.

ETIOLOGY OF MASSIVE COLLAPSE

Morison,⁴ in an instructive article reviewing massive collapse, groups the four main theories of etiology as follows:

1. Failure of the respiratory power of the lung playing an active part (Pasteur).
2. The mucus plug theory (Jackson, Lee, and others).
3. Reflex spasm of muscles of the bronchioles (Bradford, Sante, and others).
4. Inflammation of the crus and one-half of the diaphragm or of pleural membrane covering it (Charlton Briscoe).

Clinical experience goes to show that Group 1 probably has least influence in the production of the condition. A mucus plug, acting either as a complete obstruction or as a check valve (in which case the air may be exhausted much more rapidly), may indeed be a factor in the causation of post-operative collapse, but cannot cause this instantaneous collapse.

Reflex spasm, involving the walls of the bronchioles, is the only way in which the lung could be so rapidly emptied of its air. Jacobaeus put forward this view in explanation of his four cases following lipiodol injection, concluding that spasm of the bronchi and bronchioles squeezed out most of the air in the bronchial system, leaving only the alveolar air to be absorbed. In support of this view he claimed that in two cases of his the bronchi were seen to be distinctly smaller in the state of collapse than later when the collapse had passed off. This scarcely seems a fair contention, as one would expect that in a collapsed lung the bronchi would naturally be smaller.

Sante⁵ incorporates the first three views in his theory that the condition is due to simultaneous inhibition of the cough reflex by some toxic or reflex stimulus, with an impairment of the respiratory muscles, caused either by a defensive immobilization or by paralysis from a toxic neuritis. This permits secretion to accumulate and block the bronchus, atelectasis resulting.

In this explanation the mucus plug is the final cause of the atelectasis. As has already been recorded, an atelectasis produced by a mucus plug in animal experiments required four to six hours to develop, but in these animals the inhibition of the cough reflex and the impairment of the respiratory muscles were not present. In cases of immediate collapse, we would suggest that it is the inhibition of the cough reflex and the impairment of the respiratory muscles which produce a reflex spasm of the bronchi and bronchioles (as suggested by Jacobaeus), thus actively expelling the air from the bronchial system, while the mucus plug acts as a check valve, not allowing air to re-enter.

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