LORD BROCK *ET AL.*: CASE OF LATE PULMONARY EMBOLECTOMY

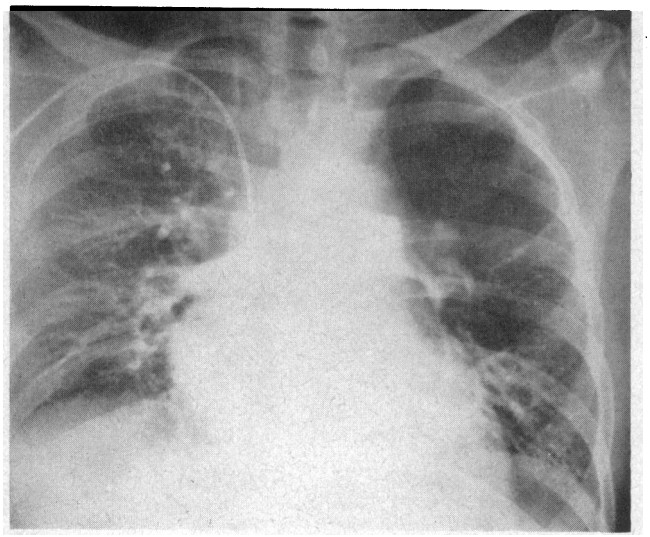

FIG. 1.—Angiocardiogram to show occlusion of main left pulmonary artery by an embolus. The small amount of lower lobe filling may be via bronchopulmonary anastomoses. A few small arterial branches in right lung are also not filled.

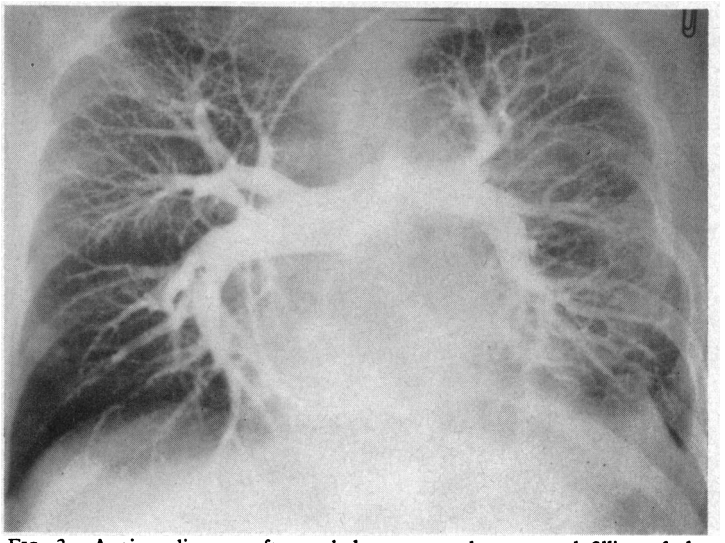

FIG. 2.—Angiocardiogram after embolectomy to show normal filling of the pulmonary arterial tree.

N. L. BROWSE *ET AL.*: MANAGEMENT OF PULMONARY EMBOLI

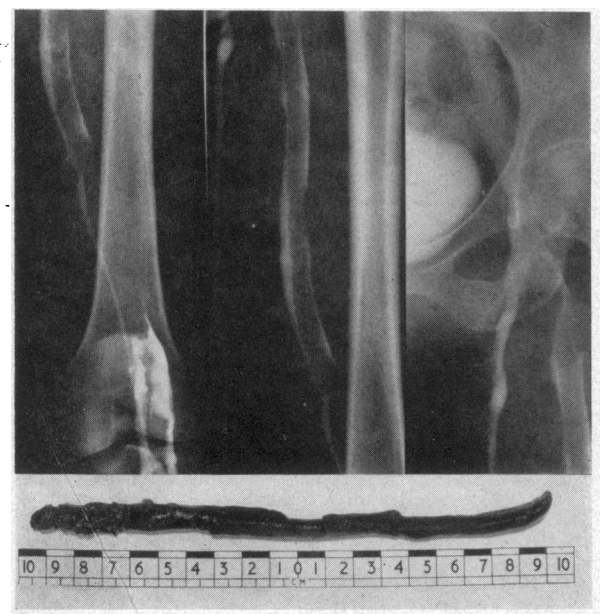

FIG. 1

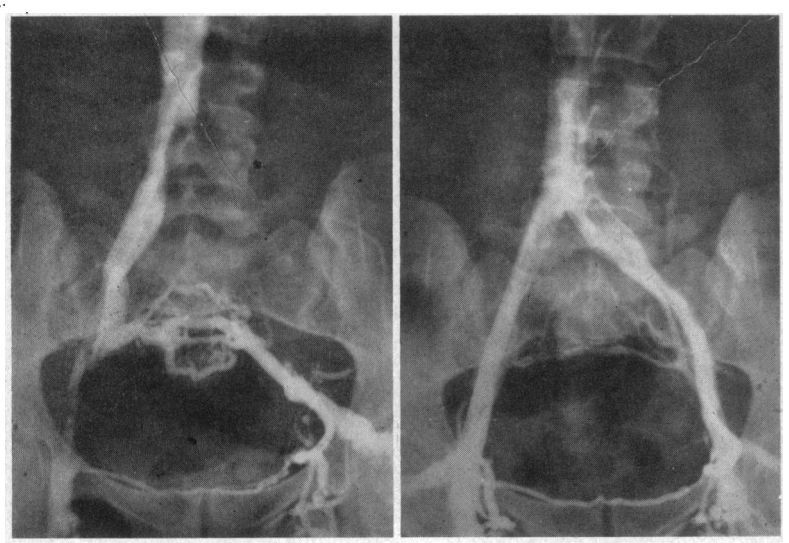

FIG. 2

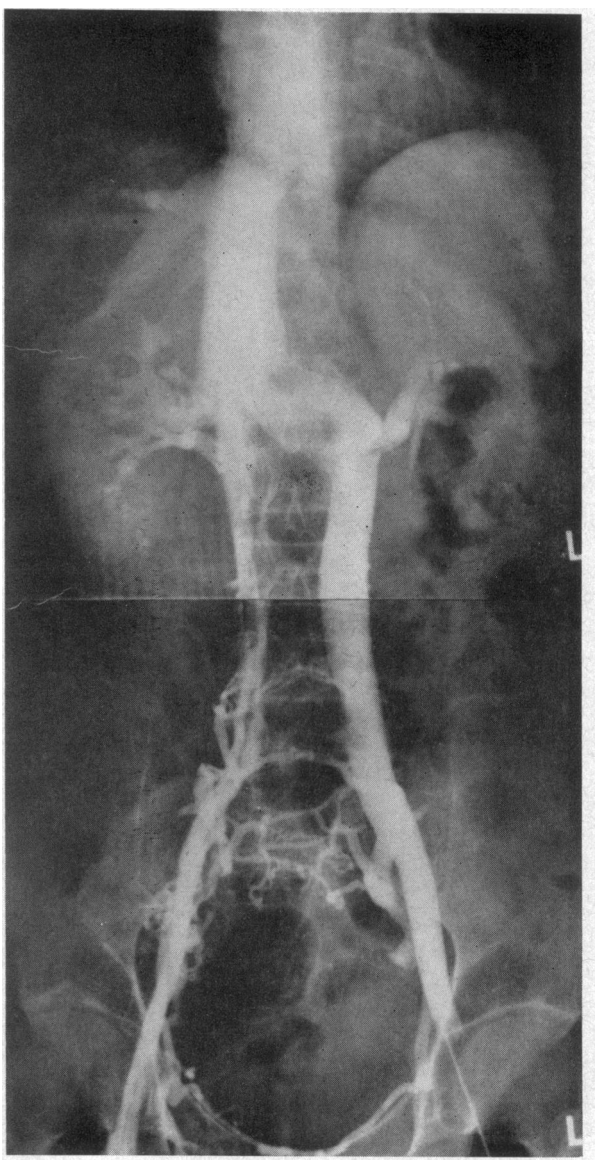

FIG. 3

Medical Memoranda

Case of Late Pulmonary Embolectomy

[WITH SPECIAL PLATE FACING PAGE 573]

Brit. med. J., 1967, 4, 598–599

The recent reawakened interest in pulmonary embolectomy has chiefly directed attention to operation as a definitive, unhurried procedure, in contrast to the more tempestuous "rush and grab" of the original Trendelenburg operation. The reason for reporting this case is fourfold: (1) it illustrates the importance of diagnosis; (2) it shows that if one pulmonary artery is affected a quite simple operation suffices; (3) it shows that late operation can be successful, indeed may be obligatory; and (4) that an embolus which does not cause early death may not be removed by lysis but can remain to cause permanent harm.

CASE REPORT

A man aged 50 had a low backache for five months, followed by pain down the outside of the left leg and foot which was said to be due to prolapse of a vertebral disc. He received various treatments, including a corset.

Three weeks after this and 17 weeks before being referred for his lung condition he had pain, swelling, redness, and pitting oedema of the left leg lasting 10 days. During this time he was mobile. Three weeks later—that is, 14 weeks before being referred—he suffered the sudden onset of pain in the left side of the chest, worse on breathing and moving. Dyspnoea became severe and oxygen was needed. He also coughed up a little blood. Over the next three weeks his breathing improved and he was then comparatively well for two months.

One month before referral he had a sudden onset of dyspnoea and palpitations while in the toilet. Two weeks later he was sitting reading when he had a sudden onset of severe left-sided chest pain with palpitations, dyspnoea, coldness, and sweating. He also coughed up some blood.

He was admitted to hospital with a diagnosis of "pleurisy" and was treated with antibiotics. The pleuritic pain improved but dyspnoea remained.

On 20 December he was seen by one of us (H. N.) for the first time, and a diagnosis of pulmonary embolism was made from the story and the radiological appearance of a poor vascular pattern in the left lung. This suggested embolism of the left pulmonary artery; an angiocardiogram taken by Dr. Chafizadeh and Dr. Massin, of Teheran, confirmed this (Special Plate, Fig. 1).

The plain radiographs of November 1966 showed evidence of bilateral pulmonary embolism. The radiographs of 26 December showed improvement in the patchy opacities previously in the right lung.

The angiocardiogram of 29 December showed an apparent dilatation of the pulmonary trunk and of its branches. The left pulmonary artery had a gross filling defect with no evidence of contrast medium entering the left upper lobe and very little in the left lower lobe. The appearances on the right side suggested partial occlusion of some branches of the upper lobe and poor and late filling of branches of the lower lobe and perhaps the middle lobe. It appeared as if several smaller emboli had lodged in smaller branches of the right pulmonary artery.

The patient was flown to London and admitted to the Brompton Hospital on 7 January 1967. He presented the clinical features of moderate pulmonary hypertension with normal jugular venous pressure. The pulse was regular; blood pressure was 90/60 mm Hg. The E.C.G. showed P-pulmonale and QS in lead III that might indicate an old posterior myocardial infarct. A quiet systolic murmur could be heard over the right upper lobe.

It seemed certain that the greater part of the left lung was deprived of its arterial blood flow by an old embolus. The patchy lesions on the right side did not look suitable for operation, and so it was decided to deal with the left pulmonary artery as a definitive procedure without the use of heart–lung bypass.

OPERATION

Thrombo-embolectomy of left pulmonary artery was done on 16 January, anaesthesia being conducted by Dr. Ruth Mansfield. Left posterolateral thoracotomy was made through the bed of the fifth rib. The upper lobe was densely adherent over the anterior segment and slightly adherent around this. The very adherent area corresponded with a pyramidal infarct about 4 cm. across in the anterior segment.

The left pulmonary artery was dissected from its origin down into the interlobar fissure. The appearance corresponded with that seen on angiocardiography. The first part of the artery was patent and the block was sudden and complete with extensions of thrombus or thromboembolism into the upper lobe branches.

The artery was clamped proximally and controlled distally by a rubber loop. It was opened by a longitudinal incision from above the highest upper lobe branch to the level of the apical lower branch. The obstruction consisted of old firm clot with a little recent thrombus. The extensions into the upper lobe branches were teased out with the aid of a small curette. The apical lower branch was similarly cleared and also the basal branches so far as was possible. The more distal branches still felt solid and it was not possible to secure any more thrombus by "milking" the lobe. After removal of clot the intima looked smooth and healthy; a weak heparin solution was injected into the distal branches. Previously a systemic dose of 1 mg./kg. had been given.

Sufficient back bleeding occurred from distal branches to indicate that total thrombosis had not occurred. It was presumed that distal patency had been ensured by the bronchopulmonary anastomosis.

The incision in the artery was closed, and when the clamp and snares were released very satisfactory and encouraging pulsation was present in the artery and in its branches.

Inspection of the thrombus showed almost uniform old adherent yellow clot in which it was not possible to identify any coiled embolus. Some of the portions extracted from the branches were redder and conical. Through an oversight pressures were not taken in the pulmonary arterial system.

Recovery from the operation was uneventful, and the chest radiograph showed greatly improved lung vascularity. Both objectively and subjectively he lost his dyspnoea.

An angiocardiogram taken 14 days after operation by Dr. G. H. Miller showed a striking restoration of the arterial pattern of the left lung (Special Plate, Fig. 2), in great contrast to the preoperative state.

The improvement and well-being continued up to the time of writing. He had no signs of pulmonary hypertension.

COMMENT

This case shows several important features as were outlined in the introductory remarks. The long overlooked diagnosis was confirmed by angiocardiography. In spite of the four months that had elapsed since embolism it was still possible to perform completely successful thromboendarterectomy. The process had not caused occlusion of the distal arterial branches; these had presumably been kept patent by the minor inflow of blood from bronchopulmonary anastomotic vessels.

It is doubtless true that if death does not occur from pulmonary embolism, clots may be removed by a process of lysis and the patency of the vessels may be restored. This case shows that this cannot be relied on to happen, and if severe secondary pulmonary hypertension is to be avoided with its attendant dyspnoea and disability, removal of embolus and clot may be necessary. Presumably whether a particular embolus may be dissolved by lysis or not will depend on its age. A recent soft embolus is more likely to be lysed than an older, rigid, and possibly partly organized one.

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Hypoglycaemic Coma Occurring during Treatment with Chlorpromazine and Orphenadrine

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Hypoglycaemia may occur with several drugs in addition to those used in the treatment of diabetes. This report describes the findings in a patient presenting in hypoglycaemic coma during treatment with chlorpromazine and orphenadrine. No such association appears to have been reported previously.

CASE REPORT

A 53-year-old married woman was admitted to hospital in coma. For some years she had been under psychiatric care, and during the previous two months had been treated with chlorpromazine 100 mg. four times a day, together with trifluoperazine (Stelazine). One week before her admission the latter drug was replaced by orphenadrine (Disipal) 50 mg. three times a day. Three days later she began to have "attacks" of excessive sweating with dryness and paraesthesiae of the mouth and tongue. On the night of admission, though feeling normal on retiring to bed, she was found later to be groaning and unrousable. She was receiving no other drugs, was only a moderate smoker, and drank no alcohol.

On examination she was sweating profusely and was deeply comatose. Slow roving movements of the eyes were observed, but the pupils responded to light. There was no papilloedema or neck stiffness and Kernig's sign was negative. Both sides of the body moved slightly in response to painful stimuli; the tone of the limbs was reduced, but the tendon reflexes were all present. The plantar responses were extensor. The pulse rate was 76 and regular; the blood pressure was 150/90. Otherwise the examination showed nothing abnormal.

A clinical diagnosis of hypoglycaemic coma was made by the house-officer on duty (J. G.). The patient was given 20 ml. of glucose (25% w/v) intravenously, after which she quickly recovered consciousness. The blood sugar before treatment was 30 mg./100 ml. (potassium ferricyanide method, normal fasting level 65–100 mg./100 ml.), and after intravenous glucose was 57 mg./100 ml.

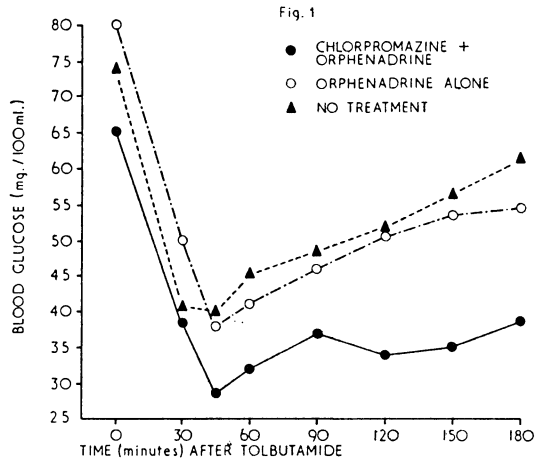
Chlorpromazine and orphenadrine treatment was continued, and the following night she had another hypoglycaemic coma, during which the blood sugar fell to 44 mg./100 ml. Recovery was rapid after 20 ml. of glucose (50% w/v) intravenously.

Investigations.—The peripheral blood count, erythrocyte sedimentation rate, urinalysis, and *x*-ray films of the chest and skull showed no abnormality. All tests of liver function were normal. The serum protein-bound iodine was 4.8 μg./100 ml. The 24-hour urinary excretion of 17-ketosteroids was 10·3 (7.2) mg., of 17-ketogenic steroids 3.2 (8·2) mg., and of 17-hydroxycorticosteroids 5.9 (11.2) mg. (The values in parentheses are repeat estimations while the patient was receiving orphenadrine alone.)

CARBOHYDRATE HOMOEOSTASIS

Investigations were carried out during and after stopping treatment with the two drugs.

(a) During Treatment with Chlorpromazine and Orphenadrine.—The fasting blood sugar was 55–65 mg./100 ml., and the patient had a further episode of hypoglycaemic coma (see above) at this time. After tolbutamide (1 g. intravenously) the blood glucose (glucose oxidase method) fell from 65 to 29 mg. at 45 minutes, and slowly rose to 34 and 38 mg./100 ml. at two and three hours respectively (Fig. 1). The fasting level of plasma insulin (measured by radioimmunoassay) was 9 μU/ml. During the tolbutamide test the plasma levels of insulin were 14 μU at 30 min., 17 μU at 45 min., 11 μU at 60 min., and 12 μU at 90 min. (all levels being within normal limits). After oral glucose (50 g.) the blood glucose



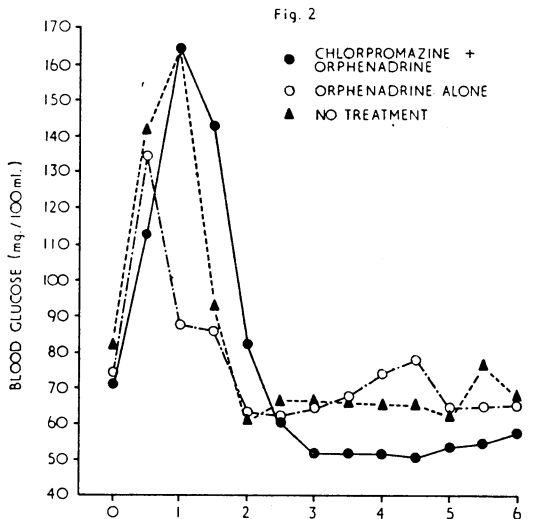


FIG. 1.—Effects of tolbutamide (1 g.) given intravenously on blood glucose levels. FIG. 2.—Six-hour glucose-tolerance tests. (Glucose estimated by the glucose oxidase methods in both instances.)

rose to 162 mg. at one hour, fell to 52 mg. at three hours, and remained below 60 mg./100 ml. for the remainder of the six-hour test (Fig. 2) The patient was starved for 72 hours, during which time the blood glucose fell to 42–44 mg./100 ml., but no hypoglycaemic symptoms developed. The response to glucagon (1 mg. intramuscularly) was normal. The blood glucose rose by 35 mg./100 ml. during the first 30 minutes, and thereafter slowly fell to a minimum value of 50 mg./100 ml. at four hours.