water. (Just recently we have had a solution prepared containing 1/300 gr. instead of 1/200 gr. of scopolamine with the idea in mind of reducing the incidence of tachycardia but since the dosage of gr. 1/200 of scopolamine should have only partial bearing on the degree of tachycardia, and the ephedrine also has some bearing on its production, it is doubtful if this variation in the scopolamine ratio will make any major change in the incidence of this finding. However, it did appear to help in three cases in which we have used the lower amount and if combined with the very important factor of slow injection it may be possible to further reduce the incidence of this mild, and harmless in the majority of cases, tachycardia. We would not like to decrease the proportion of ephedrine due to its important advantages in the solution.)

3. It was noted that psychic sedation was obtained in about 10 minutes, and maximum analgesic action in about 20 minutes, with varying degrees of amnesia being an important accompaniment to this type of basal anæsthesia.

4. It must be finally re-emphasized that for best results the solution should be given slowly and under such circumstances severe effects are practically unknown.

5. Although such may have occurred, so far as I know there has not been one case of morphine sensitivity reported in the literature with the use of this type of intravenous basal anæsthesia.

ANEURYSM OF THE PULMONARY ARTERY

M. G. Israels, M.D., F.R.C.P.[C.]
Regina, Sask.

TWO comprehensive reviews of the literature by McGavock and Boyd in 1939 and Deterling and Claggett in 1946 show that true aneurysm of the pulmonary artery is of rare occurrence. Among 35,757 consecutive autopsies by three groups of workers none was encountered. Deterling and Claggett reviewing the material at Mayo Clinic from 1910-1946 found only one case in 17,545 autopsies. They report that from the year 1785 to 1946 only six aneurysms of the pulmonary artery were found in 4,126 cases of various aneurysms occurring in the thorax.

The term aneurysm is not indicative of simple dilatation of the vessel, but must entail some destruction or damage to one or more coats of its wall. Hence the chief criterion of proof lies in autopsied cases.

Oftentimes outspoken clinical findings may indicate the diagnosis ante-mortem, although symptomatically there is nothing that would distinguish it from other forms of heart disease. The possibility of confusing it with other organic lesions involving heart or lung is great and the diagnostic criteria lie mostly with the roentgen-ray appearance, and their accurate interpretation.

In contrast to aortic aneurysms, pulmonary aneurysm occurs at an earlier age and has an equal sex distribution, as opposed to predominantly male occurrence of aortic aneurysm of approximately 6:1. The majority of recorded cases show 85% involving the main trunk. In 10% the pulmonary trunk and both main trunks were involved. The commonest type occurring in the trunks is the saccular and most often involves the left branch.

Etiology. — Numerous conditions can cause dilatation of the pulmonary artery: congenital anomalies, such as inter-auricular septal defect, patent ductus arteriosus, unequal division of the truncus arteriosus, pulmonic stenosis, anomalies of the pulmonary valves. Pulmonary hypertension due to emphysema, fibrosis, etc., or disease of the finer pulmonary arterioles, due to pulmonary arteriosclerosis or associated with mitral stenosis. Diseases that directly affect the arterial walls such as syphilis, atheroma and mycotic infection. Congenital aneurysms have also been recorded.

Non-specific arteriosclerosis has been recorded in at least 23% of cases.

Clinical symptoms.—Early in the disease these are not distinctive enough to separate them from any other form of heart disease. There may be palpitation, increasing exertional dyspnœa, cough and chest pain which may be precordial in location. Cyanosis can occur especially in the presence of a congenital lesion. Œdema is usually absent, and can occur transiently or termi-

nally. The presence of blood-streaked sputum or gross hæmoptysis has been reported.

Physical findings.—The outstanding feature on physical examination is the presence of a rough systolic murmur in the second or third left interspace. This may or may not be accompanied by a thrill. There is usually no transmission of the murmur, and it may not be accentuated in the presence of thrombus within the aneurysmal sac. Right-sided enlargement is almost always found in well developed cases.

The radiological findings are the most diagnostic in recognizing the condition. In the antero-posterior direction there is marked bulging of the pulmonary shadow, and right-sided cardiac enlargement. In the right anterior oblique view the aneurysmal shadow encroaches on the retrosternal space. This is seen when the right main branch is involved. A hilar pattern which is sharply defined, particularly on the right, is a valuable sign in the x-ray diagnosis. This is often mistaken for bronchogenic carcinoma. The presence of Pezzi's sign (hilar dance) on fluoroscopy is helpful, but occurs also in patent ductus arteriosus and inter-auricular septal defect. In the differential diagnosis aortic aneurysm, patent ductus arteriosus and patent inter-auricular septal defect are the most difficult to rule out.

The following case involving the trunk and two main branches of the pulmonary artery is the first recorded in Regina in a period covering fifteen years and 2,462 autopsies.

CASE REPORT

S.B., white, female, aged 57 years, first came under observation on September 30, 1938, complaining chiefly of dyspnœa, vertigo and fainting spells. Two weeks before she had a fainting spell of a major type. Apart from her weakness and dyspnœa she had no other complaints. Her general health had always been good until two years ago when she first noticed increasing weakness, and breathlessness on mild exertion. There was no history of rheumatic fever or hypertension, and the patient was never cyanosed. There was no cough or expectoration of blood-streaked sputum. She had no chest pain.

The physical examination was essentially negative except for the cardiac findings which showed a grade iii rough systolic murmur over the second left interspace. There was no transmission of the murmur, and no palpable thrill. Clinically the heart was normal in size, blood pressure 120/80 and lung fields clear. Remainder of the examination was negative.

The teleroentgenogram showed demarcated masses in the right pulmonary root and one of greater density in the left root with fullness in the region of the pulmonary conus and left atrium. The radiologist's report suggested lymphoma in both hilums. The fluoroscopy showed marked pulsations over the pulmonic area, but no marked "hilar-dance" was noticed.

Laboratory tests showed negative serum Wassermann, red blood cells 5,500,000, Hb. 115%, urinalysis negative.

She was discharged after several days and carried on fairly well until March 31, 1942, when she was admitted to hospital with severe epistaxis. Her intervening history was not significant except for the fact that her dyspnœa was increasing, and in the past week she claimed she could not walk 100 feet. She complained of greater weakness. Her appetite was good, she slept well and had no nocturnal dyspnœa.

The clinical examination showed her weight to be 130 pounds (she weighed 124 pounds in 1938). Her general appearance did not indicate severe illness. Her colour was good. The examination of the head and neck was negative. There was no palpable thyroid or any cervical adenopathy.

The heart was grossly enlarged and there was a marked systolic murmur over the second left interspace. The rhythm was regular and blood pressure was 130/90. The lung fields were clear. The abdominal examination was negative, and there was no clubbing. The blood showed a Hgb. of 95%, red blood cells 4,870,000, white blood cells 6,650, normal differential.

A repeated chest film showed the cardiac diameters increased considerably over the initial film in 1938, a more prominent pulmonary conus, and sharply demarcated shadows in both hila. These masses now showed calcification within them.

She was seen again on May 5, 1944, with progressive symptoms of dyspnœa and weakness and repeat examination showed no essential change in the physical findings as of April, 1942. She died suddenly on May 8, 1944.

AUTOPSY REPORT

The heart weighed 410 grams and lay freely in its pericardial cavity. The right auricle appeared to be dilated so that its capacity was increased about 50%, while moderate dilatation of the right ventricle was noted also. *In situ* the pulmonary conus appeared to be very large. The left ventricular wall measured 1.3 cm. in average thickness, while the right measured up to 1.3 cm. The mitral valve orifice measured 9.7 cm. in circumference, the aortic 7.2 cm. There was a little chronic sclerotic thickening of the free edges, and basal calcification was seen in the aortic valve cusps. The markedly dilated pulmonary valve measured 12 cm. in circumference, while the tricuspid measured 12.3 cm.

The coronary arteries were freely patent throughout and their intimal surfaces possessed slight amounts of elevated golden-yellow atherosclerotic plaquing. A supernumerary right coronary artery was present.

The aorta was slightly lax and redundant and its intimal surface possessed marked amounts of elevated golden-yellow and pearly white atherosclerotic plaquing. In the arch of the aorta a little linear grooving of the intimal surface was seen.

Pulmonary arteries.—As noted previously the pulmonary valve measured 12 cm. in circumference. The pulmonary conus was also markedly dilated and at its widest point measured 15 cm. in circumference. From the valve orifice to the bifurcation into right and left pulmonary trunks, the pulmonary artery measured 6 cm. in length. Both the right and the left pulmonary arteries were markedly dilated so that a firm area was found in the hilus of the left lung and a similar area was found in the hilus of the right although this one extended laterally for a greater distance than the left. The orifice of the left pulmonary artery measured 5.5 cm. in diameter while that of the right measured 5 cm. The left pulmonary artery had an aneurysmal dilatation of its main trunk. This was saccular in shape and measured 7 cm. in length and 3.5 cm. in average thickness. The dilatation stopped rather abruptly in the secondary divisions of the artery. It contained a large mass of golden-yellow and pale reddish-brown clot which measured 2 cm. in average thickness although it tapered as one proceeded into the distal portion of the aneurysm. Some of the clot contained granular calcific material. For the most part the surface was smooth although it was split to expose a granular greyish-cream surface. The presence of this clot had markedly stenosed the lumen of the vessel so that its calibre measured approximately

1/3 the diameter of the vessel although this was still considerably larger than the normal pulmonary artery.

The aneurysm in the right pulmonary artery was essentially similar although it measured 8 cm. in length and 3.7 cm. in average diameter.

The left lung weighed 280 gm. and lay freely in its pleural cavity. The outer surface was a bluish-purple and reddish-purple in colour while the cut surfaces was similarly coloured. A little red frothy watery fluid material could be expressed from the surface with pressure.

The mucosal surfaces of the bronchi were a reddish-brown in colour and a little slightly viscid mucoid material was attached to the walls.

Distal to the aneurysm of the main division of the pulmonary artery there was slight to moderate amount of pulmonary atherosclerosis.

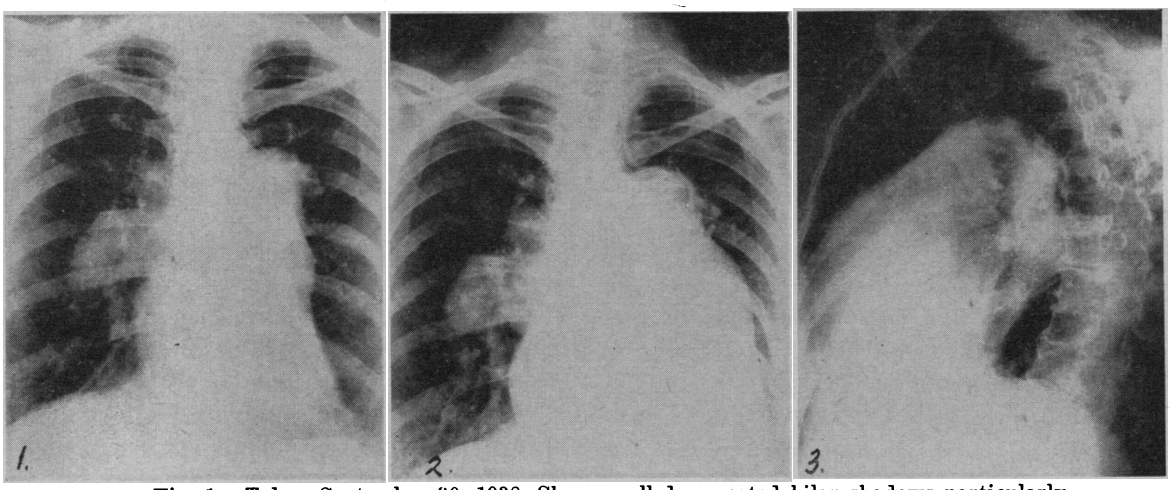

Fig. 1.—Taken September 30, 1938. Shows well demarcated hilar shadows particularly on the right. A prominent pulmonary conus. No gross cardiac enlargement. **Fig. 2.**—May 5, 1944. Showing generalized cardiac enlargement, well demarcated shadow at right hilum, prominent pulmonary conus showing calcified areas. **Fig. 3.**—Left lateral, May 5, 1944, showing encroachment of right ventricle and enlarged pulmonary conus on the retro-sternal space.

The right lung weighed 400 gm. and was similar in all respects to the left on a slightly more intense scale. The autopsy findings otherwise were not remarkable.

Microscopically, sections of the right ventricle revealed considerable hypertrophy of the myofibrils and many of them possessed smudgy and hyperchromatic nuclei. There was slight to moderate coronary arteriosclerosis. The fibres from the left ventricle were noticeably smaller than those of the right, but they too possessed hyperchromatic nuclei with a little increase of lipochrome at their poles. Throughout there was moderate fatty degeneration.

There was marked atherosclerosis of the aorta.

Sections of the aneurysms showed a considerable amount of atherosclerotic plaquing which in some areas had destroyed the media by pressure atrophy. Where the media was not too compressed there appeared to be diminution in the elastic tissue present. A considerable amount of thrombotic material was present in the aneurysms but these possessed a lining of endothelium. Evidence of recent hæmorrhage was noted in all of the atheromatous plaques and in some a moderate amount of calcific material was noted.

Sections of the lungs revealed marked pulmonary arteriosclerosis so that the calibre of the lumina was markedly diminished. The venules were also moderately sclerosed. In some sections small emboli which resembled the material found in the aneurysms were seen in some of the terminal divisions of the pulmonary artery although no infarct had been produced. One scarred and partially hyalinized area was encountered and this was thought to be a small organized infarct. Throughout the lungs there was considerable vascular congestion and moderate amounts of serous transudate with a few heart-failure cells intermingled were also seen.

This was a case of pulmonary aneurysm arteriosclerotic in origin. In retrospect the outspoken radiological findings of clear-cut hilar masses which first suggested the possibility of lymphomatous tumour, together with the cardiac silhouette, are practically diagnostic of the lesion.

Of all the criteria the x-ray should make the diagnosis in most cases.

In unilateral cases ligation of the pulmonary artery could be carried out with subsequent pneumonectomy. Dr. Claggett attempted ligation in his reported case but due to the size of the vessel and advanced arteriosclerosis this was not feasible and was abandoned.

REFERENCES

1. BOYD, L. J. AND MCGAVACK, T. H.: *Am. Heart J.*, 18: 562, 1939.
2. DETERLING, R. A. JR. AND CLAGGETT, O. T.: *Am. Heart J.*, 34: 475, 1947.

THE CAST SYNDROME.—Application of a body cast may initiate acute gastric dilatation leading to intractable vomiting and death. The mechanism is not certain but presumably is due to pressure effect of the cast. While such a catastrophe is not common it should be borne in mind and prompt treatment carried out in all patients with body casts who develop persistent vomiting. Such treatment should consist of immediate gastric intubation, removal of the cast, if necessary, and strict regulation of body fluids.—Dorph, M. H., *New England J. Med.*, **243**: 440, 1950.