- Bull, G. M., Joekes, A. M., and Lowe, K. G. (1949). *Ibid.*, 2, 229.
- Dérot, M., and Bernier, J. J. (1950). *Bull. Soc. méd. Hôp. Paris*, 66, 205.
- , Legrain, M., and Pignard, P. (1950). *Sem. Hôp. Paris*, 26, 3246.
- Dock, W., and Frank, N. R. (1950). *Amer. Heart J.*, 40, 638.
- Ellis, A. (1942). *Lancet*, 1, 1.
- Frank, H. A., Seligman, A. M., and Fine, J. (1946). *J. Amer. med. Ass.*, 130, 703.
- Hoff, H. E., Smith, P. K., and Winkler, A. W. (1941). *J. clin. Invest.*, 20, 607.
- Humphrey, J. H., and Jones, F. Avery (1947). *Clin. Sci.*, 6, 173.
- Krauss, E. (1926). *Dtsch. Arch. klin. Med.*, 150, 13.
- MacLean, H. (1919). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 43.
- Merrill, J. P., Smith, S., Callahan, E. J., and Thorn, G. W. (1950). *J. clin. Invest.*, 29, 425.
- Mudge, G. H., and Vislocky, K. (1949). *Ibid.*, 28, 482.
- Reifenstein, E. C., Albright, F., and Wells, S. L. (1945). *J. clin. Endocrinol.*, 5, 367.
- Richter, A. B. (1935-6). *Ann. intern. Med.*, 9, 1057.
- Rudebeck, J. (1946). *Acta med. scand.*, Suppl. 173.
- Strauss, M. B. (1948). *New Engl. J. Med.*, 239, 693.
- Tarail, R. (1948). *Amer. J. Med.*, 5, 828.

THE CLINICAL DIAGNOSIS OF DISSECTING ANEURYSM OF THE AORTA

BY

O. D. BERESFORD, M.D., M.R.C.P.

Medical Registrar, Southmead Hospital, Bristol

An ante-mortem diagnosis of dissecting aneurysm of the aorta was rarely made before the publication of Shennan's excellent monograph in 1934. Although described as long ago as 1728 by Nicholls, established as a pathological entity by Peacock in 1843, and correctly diagnosed during life by Swaine and Lathan (1856), the correct ante-mortem diagnosis had been recorded only seven times prior to 1934. By 1939 Rogers was able to collect from the literature 24 cases in which the diagnosis had been made during life, and since then many such reports have appeared. As pointed out by Willius and Cragg, failure to make the diagnosis is due to (1) the relative infrequency of the condition; (2) the absence of a characteristic syndrome; (3) the limitations of special diagnostic adjuncts; and (4) almost universal failure to bear the diagnosis in mind. To this may be added the extremely ill condition of many of the patients, precluding detailed clinical examination. A correct diagnosis is important if only to enable the clinician to give a more accurate prognosis. Furthermore, as the symptoms often suggest a surgical condition, a correct diagnosis will prevent unnecessary surgical procedures.

Because they are fairly representative of the way in which the condition presents, the following cases are considered worth recording. The correct ante-mortem diagnosis was made in two and suspected in three others.

Case 1

A man aged 64 was in good health until the day of admission, when he complained of sudden severe pain in the chest and neck, which came on as he was straining at stool. He described the pain as constricting and very severe. Shortly after the onset of the pain he found that his right leg had become useless and numb. By the time of admission, three hours later, the pain in his chest had improved but the right leg was still numb and painful. There was no previous history of serious illness or any symptoms suggesting hypertension.

On admission the patient, who was shocked, was lying flat in bed and not short of breath. The pulse in the right arm was 60 a minute, regular, and of fair volume. The apex beat was difficult to locate, but the heart sounds appeared normal. The blood pressure in the right arm was 110/70. There was no voluntary movement of the right leg, and the pulse was absent in the right femoral, popliteal, and dorsalis pedis arteries. Patchy areas of anaesthesia could be demonstrated. The pulses in the left leg and left arm were normal.

Eight hours after admission the pain in the chest recurred. At this time, although the pulses in the right leg were still absent, the leg felt normal to the patient, voluntary movement had returned, and the patches of anaesthesia were no longer found. The blood pressure in the right arm was 105/80. Twelve hours after admission it was noted that the left radial pulse was absent. Twenty-four hours after admission the mental condition of the patient was noted to be strange; he failed to recognize people and was in a state of stupor. Detailed neurological examination was not carried out, as he was so ill. The electrocardiogram was normal. He died 36 hours after admission.

Post-mortem examination showed a dissecting aneurysm of the aorta which began a short distance above the aortic valve, where there was a transverse rupture of the intima. It extended throughout the entire length of the aorta and for varying distances into both common carotids, the left subclavian, and the left axillary arteries. The lumen of these smaller vessels was almost obliterated by the accumulation of blood in the wall. The aneurysm had leaked into the mediastinum and also into the pericardium, where there was a large amount of blood clot. Histological examination of the aorta showed no evidence of cystic medionecrosis.

Comment.—The clinical picture was that of a coronary occlusion, associated with loss of voluntary movement in one leg and absent femoral pulsation, which had come on within a few minutes of the chest pain. Dissecting aneurysm appeared the most probable diagnosis, and this was confirmed by the normal E.C.G. and subsequent disappearance of the left radial pulse. Although pulsation was not found in the right femoral artery at any time during life, post-mortem examination showed dissection had stopped at the bifurcation of the aorta and the loss of pulse must have been due to spasm, possibly caused by interference with the sympathetic nerve supply.

Case 2

A woman aged 74 was in good health until the day before admission, when she developed severe pain in the chest and neck while doing housework. At the same time she noticed weakness of the right and slight weakness of the left leg. Sensation in the right leg was absent and she sustained a severe burn from a hot-water bottle, of which she was quite unaware at the time. The severe pain in her chest lasted for one and a half hours and normal power returned to the legs within a few hours. Examination some six hours after the onset revealed slight cardiac enlargement but no murmurs. On admission one day later the patient had no complaints. Examination revealed a difference in the blood pressure in the two arms (right 165/70, left 110/60). The heart was enlarged to the left and a loud diastolic murmur could be heard in the aortic area. All other pulses were present and no abnormal signs could be demonstrated in the legs. The electrocardiogram was normal. A radiograph of the chest showed an enlarged heart. Thirty-six hours after admission the patient collapsed and died. Permission for post-mortem examination was refused.

Comment.—Although post-mortem confirmation was not obtained in this case, the features were so similar to those of Case 1 that a diagnosis of dissecting aneurysm was made with confidence. The appearance

of a diastolic murmur overnight, the temporary anaesthesia of the leg with the history of severe chest pain, and the normal E.C.G. could not be accounted for by any other diagnosis.

Case 3

A man aged 67 had been in good health until the day of admission, when he complained of a "choking feeling," which was associated with pain in the right arm and right lower leg. One and a half hours after the onset it was noted that pulsation was absent below the right elbow, and the right forearm and hand were cold and pale. Sensation was normal and there was a good pulse as far as the lower brachial artery. A good pulse was found in the right femoral artery but none below this. The right foot and leg were cold and pale. The pulse was regular, the heart sounds were normal, and no murmurs could be heard. The blood pressure was 100/60. It was thought that emboli had lodged in the right brachial and right femoral arteries, and operation, with embolectomy in view, was decided upon.

Three and a half hours after the onset, under general anaesthesia, the right brachial artery was explored and seen to be in spasm; no embolus could be found. During the operation pulsation returned to the brachial artery, and on giving his attention to the femoral artery the surgeon found that this, too, had recommenced to pulsate. The next day the patient vomited some bright-red blood, complained of abdominal pain, and passed a large fluid bloody stool. He became mentally confused and died 30 hours after the onset of his illness.

Post-mortem examination showed the heart to be normal apart from some senile thickening of the aortic and mitral valves. At a point 2 cm. above the aortic valve there was a transverse tear in the intima communicating with a dissecting aneurysm which extended throughout the whole length of the aorta and for varying distances into all its main branches, including the renal, superior and inferior mesenteric, coeliac axis, and common iliac arteries. In the right brachial artery the dissection stopped just short of the site of the arteriotomy. The stomach contained several hundred millilitres of blood-stained fluid and showed an area of haemorrhagic infarction on the lesser curvature. Similar small infarcts were seen in the colon. Histological examination of the aorta revealed the typical picture of cystic mediocrosis of the aorta.

Comment.—Vascular phenomena were the predominating feature in this case. As in Case 1 it is probable that the spasm of the brachial artery was due to stimulation of the sympathetic nerves in the adventitia by the dissecting process. Similar spasm of the gastric and mesenteric arteries may have been responsible for the mucosal infarctions of the alimentary tract. The diagnosis of dissecting aneurysm was considered subsequent to operation.

Case 4

A man aged 69 was admitted to hospital on February 2, 1949, with a history that three hours previously, while straining at stool, he had developed severe epigastric pain, followed 10 minutes later by agonizing pain in the left groin, after which he found the left lower limb "dead."

On admission the left femoral and left external iliac pulses could not be felt. The pulses on the right side were present and pulsation could be felt in the abdominal aorta. The left thigh and the upper half of the left leg were warm, but the foot and ankle were cold and the whole limb congested in a patchy fashion and slightly cyanotic. The blood pressure was 140/90. Auricular fibrillation was present, and in view of this it was thought probable that an embolus from the left auricular appendix had lodged at the bifurcation of the aorta.

At operation two hours after admission the left common iliac and the left femoral arteries were exposed. The vessels appeared normal, and on arteriotomy a continuous flow of

blood was observed. During the next 12 days the colour of the left limb varied from a patchy subcutaneous cyanosis to pallor, but the limb was usually warm and there was no loss of sensation. On February 17, after an illness lasting 15 days, the patient had a severe haematemesis, from which he died.

Post-mortem examination showed an enlarged heart weighing 540 g. Both right and left ventricles were hypertrophied and all the chambers were dilated. The aorta showed flecks of atheroma throughout but was otherwise normal, except in the lower abdominal portion, where a large dissecting aneurysm extended from just below the origin of the renal arteries to the bifurcation of the aorta. The left common iliac artery was almost completely occluded by pressure from the dissecting aneurysm, and occlusion was made complete by thrombosis of the vessel, which extended as far as the operation site. Death was due to a massive intragastric haemorrhage from a chronic gastric ulcer. Histological examination of the aorta showed no evidence of mediocrosis, and the cause of the dissecting aneurysm was not clear. There was some endothelialization of the aneurysm cavity.

Comment.—Death in this case was not due to the dissecting aneurysm, and it is probable that healing would have taken place had the haematemesis not occurred. The diagnosis of dissecting aneurysm was considered after the operation in this case.

Case 5

A man aged 66 gave a 10-years history of dyspepsia and pain after food, suggestive of peptic ulceration. Apart from this he had been well until the day of admission, when he developed severe pain in the chest immediately after an exciting game of billiards. He was semiconscious for about 10 minutes and extremely short of breath. He also complained of numbness of the right hand and arm, especially along the medial border, which came on a few minutes after the pain in the chest.

On examination he was cyanosed, cold and clammy, and extremely restless. The pulse was irregular and could not be palpated at the right wrist. The blood pressure could not be recorded on the right arm, but on the left it was 65/0. The heart did not appear to be enlarged, the sounds were normal, and a soft systolic murmur could be heard in the mitral area. No other signs could be demonstrated on admission, but the next day a pericardial friction rub was heard. The patient was still cyanosed and sweating, and later in the day a further attack of substernal pain occurred, and at this time an aortic diastolic murmur was noted. Two days after admission he complained of suprapubic pain and died shortly afterwards. Electrocardiographic examination showed a depressed S-T segment in Leads I and II, and an inverted T wave in Lead I.

Post-mortem examination showed considerable cardiac enlargement, left ventricular hypertrophy, and dilatation of the right side of the heart. The anterior wall of the left ventricle was darker in colour than the posterior wall and appeared to be congested. At a point 3 cm. above the sinuses of Valsalva there was a horizontal tear extending through the media and intima, involving almost the whole circumference. From the tear a dissecting aneurysm extended downwards to the root of the aorta and surrounded the origin of the left coronary artery. Dissection had extended into the first part of the left coronary artery, and the lumen had been compressed to the point of occlusion. The adventitia of the remainder of the aorta had been stripped from the media and blood had leaked out in the region of the bifurcation of the aorta and in one or two places along its course. The dissecting aneurysm had ruptured back into the aorta at the bifurcation, making a transverse slit 18 mm. long. Immediately above the bifurcation of the aorta was a fusiform aneurysm extending upwards for a distance of some 10 cm. The left kidney was rather swollen and congested and the left renal artery was narrowed

by the dissecting aneurysm. There was a chronic duodenal ulcer 15 mm. from the pylorus. Histological examination of the aorta showed evidence of medionecrosis.

Case 6

A man aged 63 was well until two hours before admission, when he complained of severe abdominal pain, which came on suddenly while he was bending down. The pain was mainly in the epigastrium but was also severe in the upper lumbar region. There was no history suggestive of previous hypertension. On examination there was a marked degree of shock, but the pulse volume was good and the rhythm regular. The heart was enlarged to the left and the blood pressure was 205/180. There was generalized abdominal tenderness and rigidity. The liver dullness was not diminished. A diagnosis of perforated peptic ulcer was made, and treatment consisted of intravenous glucose and continuous gastric suction. An x-ray picture of the chest showed a prominent aortic knuckle, and the E.C.G. left axis deviation. The patient's condition steadily deteriorated, and he died three days after admission.

Post-mortem examination showed an enlarged heart with considerable left ventricular hypertrophy. The aortic valve was bicuspid and extensively calcified. The aorta was healthy as far as the origin of the left subclavian artery. Near its beginning this artery had a transverse split in the intima involving the greater part of the circumference. This communicated with a dissecting aneurysm which extended throughout the remainder of the aorta and also into the proximal part of the left common iliac artery. Rupture had occurred into the mediastinum and there was a large amount of blood in the left pleural cavity. The retroperitoneal tissues were extensively infiltrated with blood. Histological examination of the aorta revealed some atheroma. There was no evidence of medionecrosis, but the amount of destruction was so great that this condition might well have been obscured if present.

Comment.—A diagnosis of perforated peptic ulcer was originally made in this case, but the absence of previous indigestion, the severity of the pain in the back, the extreme degree of shock, and the presence of hypertension suggested dissecting aneurysm, and this was regarded as the probable diagnosis after the patient's admission to hospital.

Case 7

A man aged 70 was admitted to hospital with a history of sudden onset of severe precordial pain three hours previously. The pain had come on while he was sitting in a chair, and did not radiate. He had suffered from hypertension for many years. Examination on admission revealed a shocked patient with a blood pressure of 220/140. Pre-systolic triple rhythm was audible between the apex beat and the sternum. The day after admission there was a transient period of mental confusion. On the third day of his illness the temperature was 100° F. (37.8° C.) and the leucocyte count 11,000 per c.mm. (90% polymorphs). One week after admission he collapsed after using a bedpan, and died shortly afterwards.

Post-mortem examination showed the pericardium to be tense and bulging and full of blood clot. The heart was enlarged and weighed 550 g. There was great hypertrophy of the left ventricle. The aorta showed a moderate degree of atheroma throughout. Six centimetres above the aortic valve there was a vertical tear in the intima which communicated with a dissecting aneurysm that extended along the aorta to a point 3 cm. above the origin of the renal arteries. The aneurysm had ruptured externally by a ragged tear 3 cm. in length just above the right auricular appendix. This had given rise to the fatal haemopericardium. Histological examination of the aorta revealed the typical picture of medionecrosis.

Comment.—A confident diagnosis of coronary occlusion was made in this case and, in the absence of electro-

cardiographic examination (not done owing to technical difficulty), there seemed no reason to doubt its accuracy. This case illustrates how closely the condition may simulate a coronary occlusion.

Case 8

A man aged 68 had been well until a few hours before admission, when he complained of upper abdominal pain and nausea. On examination he was restless, cold, and sweating. Some upper abdominal tenderness and rigidity were present. The heart sounds were normal; there were no murmurs, and the blood pressure was 240/130. Examination of the chest revealed some impairment to percussion at both bases. He had had a stroke one year previously. The patient's condition gradually deteriorated, and he died 24 hours after admission.

Post-mortem examination showed the pericardium to contain about 400 ml. of blood. The heart weighed 440 g. and, apart from some atheromatous thickening of the mitral valve, appeared normal. The left ventricle was not hypertrophied. The aorta showed extensive atheroma throughout, which was most marked in the abdominal portion. At the convexity of the arch of the aorta there was a longitudinal tear in the intima. This communicated with a dissecting aneurysm which extended downwards as far as the origin of the coeliac axis. It extended beneath the adventitia to the root of the aorta, where the blood had also tracked beneath the epicardium of the right auricle to the adventitia of the pulmonary artery. From this region leakage had occurred into the pericardium. Histological examination of the aorta showed the inner five-sixths of the media to be healthy but stripped from the outer layers by the extravasated blood. The adventitia showed small collections of inflammatory cells. There was no definite evidence of medionecrosis.

Comment.—This case presented with abdominal symptoms, and the diagnosis of perforated peptic ulcer was considered. The general condition of the patient prevented operative treatment and he gradually succumbed.

Case 9

A man aged 67 was admitted to hospital as a case of impending uraemia. He had a long history of fatigue and failing vision, and a few days before admission became somewhat drowsy. On examination he showed signs of uraemia. The blood pressure was 205/140, the heart was enlarged to the left, and the second aortic sound was accentuated. The urine contained a considerable amount of albumin. Under supervision the patient's condition improved somewhat, but 10 weeks after admission he collapsed and died quite suddenly.

Post-mortem examination showed an enlarged heart (weight 515 g.) with marked hypertrophy of the left ventricle. Apart from some fibrous thickening at the base of the aortic valve cusps it appeared normal. The aorta showed a moderate degree of atheroma, and a dissecting aneurysm extended from the level of the fourth pair of intercostal arteries to the origin of the coeliac axis. Rupture of the aneurysm had occurred anteriorly. Histological examination of the aorta showed large plaques of atheroma, with some groups of atheromatoid foam cells deep in the media. There was considerable fibrosis of the media deep to the plaques but no evidence of medionecrosis.

Comment.—In this case the dissecting aneurysm was a terminal event in a patient who was seriously ill with uraemia. No particular signs or symptoms were present which suggested the diagnosis.

Case 10

A woman aged 58 gave a history of effort dyspnoea for five years, and had been known to be hypertensive for two years. Three weeks before admission her dyspnoea and

tightness of the chest became much worse. The day before admission she developed tremor of the left arm, and the left eye deviated outwards. On examination she was dyspnoeic and cyanosed. The pulse was regular and the tension and pressure were raised. The heart was enlarged to the left, the heart sounds were normal, and there were no murmurs. The blood pressure was 195/125. There was a left external strabismus, but the central nervous system was otherwise normal. The percussion note was impaired and the air entry at the left base was poor. The patient's condition gradually deteriorated and she died 48 hours after admission.

Post-mortem examination showed an old rheumatic lesion of the heart with scarring of the mitral and aortic valves. The left ventricle was considerably hypertrophied. The pericardium contained a large amount of blood as a result of haemorrhage from a ragged tear in the intrapericardial portion of the aorta. A transverse tear was found in the intima of the aorta 10 cm. above the aortic valve. A saccular aneurysm 2.5 cm. in diameter and lined by an old laminated clot was present just beyond the origin of the left common carotid artery. In the intima of the lower part of the descending thoracic aorta was a longitudinal slit 0.8 cm. long, communicating with a dissecting aneurysm which involved the remainder of the descending aorta and passed for a distance of 2 cm. into the right common iliac artery, the lumen of which it rejoined by a smooth round ostium 7 mm. in diameter. Histological examination of the aorta showed some intimal atheroma and patchy areas of medionecrosis in the neighbourhood of both the saccular and dissecting aneurysms.

Comment.—The absence of pain in dissecting aneurysm is by no means uncommon (Wood *et al.*, 1932). The other point of interest in this case was the associated saccular aneurysm. Davies (1941) suggested that there might be a relationship between the two conditions.

Case 11

A woman aged 71 gave a history of dyspnoea and chest pain which had come on suddenly 18 days previously. The dyspnoea had steadily become worse and had been associated with an irregular pyrexia up to 100° F. (37.8° C.). A diagnosis of unresolved pneumonia had been made. Examination on admission showed the signs of a left-sided pleural effusion. Aspiration on several occasions yielded almost pure blood. After a week in hospital her condition was obviously deteriorating and she developed signs of congestive heart failure. One month after the onset of her illness she collapsed and died suddenly. While in hospital her temperature had varied between 99° and 101° F. (37.2° and 38.3° C.).

Post-mortem examination revealed only slight hypertrophy of the left ventricle. The arch of the aorta showed naked-eye changes very suggestive of syphilis. (Histological examination disproved this.) Immediately beyond the origin of the left subclavian artery was a recent ragged transverse tear 2.5 cm. long, communicating with a dissecting aneurysm which surrounded the arch of the aorta and extended to just beyond the coeliac axis. Rupture had occurred into the left pleural sac, which was full of fluid and clotted blood. Histological examination of the aorta showed atheroma, with fibrosis of the adjacent media. There was a little interruption of the medial elastica in some places, but no evidence of syphilis or medionecrosis.

Comment.—This illness closely resembled a pneumonic process. The pyrexia, dyspnoea, and chest pain did not suggest dissecting aneurysm, and the aspiration of blood led the clinicians to suspect an underlying neoplasm.

Discussion

Dissecting aneurysm is said to be found once in every 400–500 necropsies (McGeachy and Paullin, 1937), but the true incidence may be higher, as this does not include

cases in which the process has healed. Weiss *et al.* (1940) thought that one in ten cases recovers and dies from other causes. About 80% of cases occur in subjects over the age of 50. The condition in young subjects has been studied by Schnitker and Bayer (1944), who stress the association with pregnancy or with a congenital narrowing of the aorta. 65% of cases occur in males. Hypertension is often present, but opinions differ about the exact frequency. Some authors state that 80% have pre-existing hypertension (Willius and Cragg, 1941). In this series five of the eleven cases had evidence of hypertension.

Baer and Goldburgh (1948) considered that a radial pulse was absent in only 2.8% of cases and a femoral pulse in only 7%, but it seems probable that peripheral vascular disturbances would be found much more often if all accessible pulses were examined at frequent intervals.

While various clinical syndromes may be met with in this condition, it is important to stress that it is often a combination of two or more of these syndromes that will arouse suspicion of the true diagnosis. Thus in a case presenting as coronary occlusion the finding of diminished or absent pulsation in a peripheral vessel should call for a reconsideration of the diagnosis, as should the finding of peripheral sensory loss in a case of "perforated ulcer."

Pathological investigations are not of much assistance in diagnosis, though electrocardiography may exclude alternative cardiac diagnoses. Nor is any active treatment likely to be of use, and adequate sedation seems the most reasonable course.

The average time of survival from the onset of symptoms is four to seven days. The occurrence of a quiescent stage after a dramatic onset should not dissuade the physician from giving a guarded prognosis. In a proportion of cases death may be too sudden for ante-mortem diagnosis. Of Shennan's 300 collected cases, 51 died shortly after the onset of symptoms.

Summary

The clinical features of 11 cases of dissecting aneurysm of the aorta are described, together with the pathological findings in 10 of the cases.

The varied clinical syndromes that may result from this condition are briefly reviewed.

Peripheral vascular disturbances are considered to be of great help in the diagnosis of the condition and probably occur in a higher proportion of cases than is usually thought.

My thanks are due to Dr. N. J. Brown, pathologist, Southmead Hospital, for the abstracts of the post-mortem findings in these cases, to Professor T. F. Hewer for helpful criticism and advice, and to the various clinicians for permission to publish the cases.

REFERENCES

- Baer, S., and Goldburgh, H. L. (1948). *Amer. Heart J.*, **35**, 198.
 Davies, D. H. (1941). *Brit. Heart J.*, **3**, 166.
 McGeachy, T. E., and Paullin, J. E. (1937). *J. Amer. med. Ass.*, **108**, 1690.
 Nicholls, F. (1728). *Phil. Trans.*, **B**, **35**, 440.
 Peacock, T. B. (1843). *Edinb. med. J.*, **60**, 276.
 Rogers, H. (1939). *Amer. Heart J.*, **18**, 67.
 Schnitker, M. A., and Bayer, C. A. (1944). *Ann. intern. Med.*, **20**, 486.
 Shennan, T. (1934). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 193.
 Swaine, K., and Lathan, P. M. (1856). *Trans. path. Soc. Lond.*, **7**, 106.
 Weiss, S., Kinney, T. D., and Maher, M. M. (1940). *Amer. J. med. Sci.*, **200**, 192.
 Willius, F. A., and Cragg, R. W. (1941). *Proc. Mayo Clin.*, **16**, 41.
 Wood, F. C., Pendergrass, E. P., and Ostrum, H. W. (1932). *Amer. J. Roentgenol.*, **28**, 437.