A comparison of the blood loss in the various groups is seen in Table II. Martin and Dumoulin found that 89.2% of their intravenous ergometrine series had losses of from 0 to 9 oz. (256 ml.). With the use of 0.5 mg. of ergometrine plus hyaluronidase intramuscularly in 1,748 cases, 85.4% had losses of from 0 to 9 oz. (256 ml.).

Length of Third Stage of Labour.—Use of intramuscular ergometrine and hyaluronidase shortens the length of the third stage as shown in Table III. In the control series 22.2% were delivered of the placenta in less than 10 minutes, while in the ergometrine and hyaluronidase series 51% completed stage III in less than 10 minutes.

Discussion

Timing is most important. Placental expression should be carried out as soon as possible. The midwife—frequently acting alone in the delivery—is unable to express the placenta quickly because she has the baby to look after immediately it is born. Nevertheless, stage III is still shorter than before intramuscular ergometrine and hyaluronidase came into use at the moment of crowning or delivery of the foetal head.

The death rate from delivery complicated by retained placenta and other post-partum haemorrhage has been supplied by the Registrar-General (Table IV), based on the 1948 International List.

TABLE IV

	No. of Deaths	Rate per 1,000 Live Births
1949	90	0.12
1950	38	0.05
1951	54	0.08
1952	39	0.06

Any technique, therefore, which will help in reducing the incidence of post-partum haemorrhage rate must inevitably reduce the risk of maternal death.

The experience of Martin and Dumoulin, confirmed by Vartan (1953), has been reaffirmed by Professor Wright, of the University of Otago, New Zealand (personal communication), who halved his post-partum haemorrhage rate in one year and did not increase his manual removal rate. Wright gives intravenous ergometrine with the delivery of the anterior shoulder.

Summary

Intramuscular ergometrine, 0.5 mg., plus hyaluronidase was given in 1,748 cases by midwives, acting alone at the delivery, as soon as the foetal head was crowned or delivered. In addition 0.25 mg., plus hyaluronidase, was given at the same point of timing in a further 254 cases, making 2,002 cases in all.

The results are compared with 747 control cases. The use of ergometrine by midwives in this way has shown that (1) the post-partum haemorrhage rate is reduced from 6.4% to 0.9%; (2) the manual removal rate is not appreciably influenced—1.6% for control cases and 1.1% for the ergometrine and hyaluronidase series; (3) blood loss is lessened; and (4) stage III is shortened.

A plea is made for the adoption by midwives of the technique outlined.

The hyaluronidase used was "hyalase" in ampoules as a sterile freeze-dried powder of 1,000 Benger units.

My thanks are due to the matron, Miss O. M. Hutson, and her sisters and staff midwives, who were entirely responsible for all the deliveries reviewed.

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DISSECTING ANEURYSM OF THE AORTA

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King George II one day "was found fallen on the ground, speechless and motionless." He had died of a dissecting aneurysm of the aorta, and his post-mortem examination provides us with the first description of this condition (Nicholls, 1761). Shennan (1934) reviewed all the published cases in a comprehensive monograph, and analysed 300 proved at necropsy: of these, only six were diagnosed during life. As dissecting aneurysms are still but rarely diagnosed clinically, the following contrasting cases are of interest.

Case 1

A dock labourer aged 36, strong and active, was suddenly seized by an excruciating tearing pain while walking down the companion ladder of a ship. The pain was mainly in the back of the chest, and traversed through to the front, and thence up the neck into the head. He collapsed at the foot of the ladder and could scarcely breathe. He was carried into hospital, where he found that the pain was aggravated by lying flat and partially relieved by sitting up.

Examination showed a very pale and collapsed man, with a cold and clammy skin. His pulse was regular, rate 50 a minute, and the radial pulses were of equal volume. The blood pressure was 95/75 in both arms. The heart sounds were heard faintly in all areas, with no added sounds or murmurs. There were no other abnormal physical signs.

The patient continued to suffer very severe pain, which was now primarily retrosternal, and radiated into his back and down into the epigastrium and abdomen. He remained pale and collapsed, but was talkative and seemed to improve with rest in bed. Four hours later he had a regular pulse of 36. The radial pulses were distinctly unequal, the right being smaller in volume and more anacrotic. The blood pressure in the right arm was 120/60; in the left arm it was 135/70. The temporal, carotid, and femoral pulses were all equal. The cardiac impulse became more forcible and the heart sounds quite loud and clear. There was no increased area of cardiac dullness to percussion or tracheal tug. There was some epigastric tenderness but no rigidity.

The urine contained a moderate amount of albumin but no blood. Radiography of the chest showed normal lung fields. The postero-anterior view showed widening of the supracardiac shadow and some widening of the arch of the aorta. The left lateral view showed obvious dilatation of the descending arch of the aorta. The electrocardiogram (E.C.G.) showed a slow regular sinus rhythm, with normal conduction time. There was a deep Q wave in the chest lead CR₇, and notching of the QRS complex in lead CR₄. The T wave was inverted in CR₁, but full and erect in all other limb and chest leads. There was no Pardee sign.

Twelve hours later the patient again collapsed suddenly, and neither radial pulse was palpable. The apical rate was 148 a minute. A fresh E.C.G. showed sinus tachycardia, with no evidence of cardiac infarction. The patient improved slightly after the subcutaneous injection of morphine $\frac{1}{4}$ gr. (15 mg.), but later collapsed again, and died.

At post-mortem examination (Dr. D. Teare) the body was well nourished, with a long recent dissecting aneurysm of the thoracic aorta. There was a horizontal break in the intima, about 1 in. (2.5 cm.) long and 1 in. (2.5 cm.) above the aortic cusps. The dissection had travelled distally as far as the bifurcation of the abdominal aorta, and had ruptured proximally into the pericardium, which contained about 16 oz. (455 ml.) of blood. There was partial occlusion by the dissection of the root of the superior mesenteric artery, with recent infarction of the small intes-

tine and copious blood-stained mucus in the bowel, stomach, and oesophagus. There was no evidence of syphilis or of atheroma. The dissecting aneurysm was due to idiopathic cystic medial necrosis of the aorta.

Case 2

A blacksmith aged 58, tough and burly, and weighing 15 st. (95 kg.), had worked his usual 10-hour day, and was sitting quietly in the bus on his way home when he "blacked out." He had no headache, no premonitory aura, and no pain at all. He awoke in the ambulance, feeling very faint, and discovered that he had been incontinent of much faeces but not incontinent of urine.

On examination he was rational and conversational and in no discomfort. He walked in from the ambulance. His skin was moist and cold. He was very pale, with a sluggish peripheral circulation and hypostatic engorgement of his limbs. The pulses were all impalpable. The apex beat could not be felt, and the heart sounds were very faint. There were no other abnormal physical signs.

The urine contained a large volume of albumin, and the stools contained blood-stained mucus. The E.C.G. showed a rather slow but regular sinus rhythm, with no impaired conduction. There was pronounced depression of the ST segment in leads I and CR₇, and also to a less extent in leads II and CR₄. The E.C.G. was indicative of marked left ventricular preponderance, and there was no evidence of cardiac infarction.

The patient remained in this condition of extreme shock and was kept on absolute bed rest and given subcutaneous injections of morphine, $\frac{1}{4}$ gr. (15 mg.) every four hours when awake. After 36 hours he recovered partially, and his circulation improved considerably. All his pulses became easily palpable and equal. His blood pressure in both arms was 130/95. His heart sounds became loud in all areas of the praecordium, with a rough systolic murmur, maximal at the aortic area and transmitted down into the apex and up into the neck. There were no additional heart sounds and no diastolic murmurs. He remained completely free from pain at all times. He was dyspnoeic while lying down, and rather distressed by the dyspnoea. He had a very poor urinary output from the onset and passed only 20 oz. (570 ml.) in 72 hours. The sclera became yellow. On the fifth day he lapsed into a coma, and died after 12 hours.

Post-mortem examination (Dr. L. Steingold) showed a well-nourished, slightly jaundiced man with a recent dissecting aneurysm of the ascending aorta. There was a transverse tear in the endothelium, about 4 cm. long and 2 cm. above the aortic valve, in the right posterior wall. The blood had formed a saccular aneurysm about 6 cm. in diameter at this point, and had dissected the layers of the aorta distally for 8 cm. and proximally into the pericardium, which contained a large quantity of blood. A small tear was found in the adventitia just above the aortic valve. The aortic valve was moderately stenosed, with calcified and distorted cusps. There was no cardiac infarction. Marked systemic atherosclerosis, with calcification and ulceration of the abdominal aorta, was present. There was no evidence of syphilis. The dissecting aneurysm was probably due to atherosclerosis.

Comment

Dissecting aneurysm is a sudden, dramatic, and not uncommon condition. In San Francisco it occurred in 1 in 430 (0.23%) routine hospital necropsies, and in 1 in 89 (1.1%) coroners' necropsies (Mote and Carr, 1942). In London Dr. Donald Teare has personally performed necropsies on 451 cases of dissecting aneurysm in about 32,000 coroners' cases: this gives an incidence of 1 in 70 (1.4%) (Teare, 1953, personal communication). This surprising figure shows that it is a much more common condition than is usually believed. In spite of this high post-mortem incidence, very few cases are diagnosed during life, because of the rapidity with which they are often fatal, and because of a common lack of clinical suspicion.

Indeed, it has been stated in textbooks that "the condition cannot be diagnosed with any certainty before death, and no treatment is of any avail" (Handfield-Jones, 1948). This is untrue. It can be diagnosed, and then supportive measures may be applied and exploratory laparotomy or embolectomy or equally harmful treatments may be avoided.

The most frequent misdiagnosis and the most difficult differential diagnosis is cardiac infarction. Here the different age-distribution may be helpful; for only 1.5% of cardiac infarcts are found under the age of 40 years (Glendy, Levine, and White, 1937), whereas dissecting aneurysm is comparatively common in the young and healthy, and 24% of these cases occur under the age of 40; while of those found in women of child-bearing years, one-half are associated with pregnancy (Schnitker and Bayer, 1944). Dissecting aneurysms can, however, be distinguished confidently from cardiac infarction in many instances by careful clinical examination alone, and the diagnosis, once made, may be supported by evidence from the electrocardiogram and the chest radiographs. In the postero-anterior view there is deformity of the supracardiac shadow, with widening of the aorta and sometimes an arcuate excrescence from it, or occasionally a shadow visible along the innominate artery. In the left lateral view the typical dilatation of the arch and the descending aorta is best seen (Wood, Pendergrass, and Ostrum, 1932).

The most disastrous misdiagnoses are the surgical. Thus of those cases which present with pain, about 60% develop epigastric pain of some severity during the course of the disease (Baer and Goldburgh, 1948). These may simulate an acute abdominal emergency, and be diagnosed as perforated peptic ulcer. But the location of the pain at the time of onset and the presence of cardiovascular signs should differentiate a dissecting aneurysm. Several cases have been reported recently in which embolectomy has been attempted from the common iliac, the femoral, or the brachial arteries (Fraenkel and Neil, 1950; Beresford, 1951). But here again the initial location of the pain in the chest or back, and the presence of cardiac murmurs, should distinguish it from a saddle embolus of the aortic bifurcation.

It is not commonly realized that over 50% of cases of dissecting aneurysm are completely and entirely painless throughout their course (Baer and Goldburgh, 1948). Patients present commonly with syncope, or they may be found in coma. They are all very collapsed, and have greatly lowered blood pressure. They are often grossly dyspnoeic, and may be much distressed. They may have neurological signs in the limbs, and these cases may easily be misdiagnosed as cerebral embolism unless the cardiovascular system is carefully examined.

Little definite is known about the natural history of those cases of dissecting aneurysm which survive the initial attack, largely because a correct diagnosis is so rare. These two cases are reported as typical of the two commonest clinical syndromes found—those which present with excruciating pain, and those which present with syncope and no pain.

Summary

Two cases of dissecting aneurysm of the thoracic aorta are reported, and the clinical diagnosis of this condition is discussed.

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