nervous system may have a bearing on the production of "functional hyperinsulinism." Cori (1931) in an extensive review concluded that vagal impulses play a part in the finer regulation of the insulin production, and Cannon *et al.* (1924) have shown that nervous control of the adrenal medulla via the splanchnic nerves ensures a rapid adrenaline response.

It can well be appreciated how the continuance of abnormal stress in a susceptible individual may give rise to abnormality or instability in the control of these opposing impulses by the hypothalamic region. The terminal stages of the glucose-tolerance test is illustrative of this.

Summary

A case of "reactive hyperinsulinism," manifest by an excessive post-prandial fall of the blood-sugar level, is described. Points in the differential diagnosis from hyperinsulinism due to tumour of the pancreatic islets are discussed.

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TRAUMATIC RUPTURE OF THE AORTA

BY

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The following case of rupture of the aorta due to a blow on the epigastrium presents clinical and pathological features interesting enough to be placed on record.

Clinical History

The patient was a farmer, aged 37, who was described by his relatives as being in robust health. He had no relevant medical history, but his family doctor had observed that he had a very high threshold for pain. On Jan. 20, 1944, he went ploughing at 1 p.m., and at 3 p.m. left the field to call at a neighbouring farmhouse for a drink of water. He there complained that shortly before that time his plough had struck a stone, and the plough handle, rearing up, had hit him somewhere in the region of the praecordium. The blow had shaken him badly, and from statements he made to various people it appears that immediately after the accident he felt very ill, owing presumably to shock; but he soon recovered, continued to plough till about 4 p.m., and made no further reference to the accident. He returned home at 5 p.m., when he complained of pain in the left side of the chest radiating through to his back. It was observed that he was cold, clammy, and pale, and he had to go to bed. He was examined later the same evening by his doctor, who found that his temperature was 100° F. and that friction could be heard in the cardiac area. The patient made no reference to the accident, and his doctor was in complete ignorance of the incident till after the man died. He passed a restless night because of chest pain, and vomited three times. Next morning the pyrexia had disappeared, and though he still complained of pain he did not seem to be particularly ill. His condition remained much the same till late afternoon, when he died suddenly within a few minutes of sitting up in bed to drink a cup of tea.

Post-mortem Findings

The deceased was well built and there was no external evidence of injury. No bones were broken. There was an area of bruising in the areolar tissue of the anterior mediastinum, and the bruising extended up the right side of the neck as far as the level of the larynx in the line of the great vessels and sterno-mastoid muscle. The pericardial sac was distended with blood, which had infiltrated

the wall, particularly in the region of the base of the heart. On the posterior wall of the first part of the aorta there were three tears in the vessel—one just above the valve cusps, a second at the beginning of the arch, and a third midway between the first two. The upper tear was half an inch and the others each a quarter of an inch in length. All the tears were oblique to the transverse axis of the vessel, but were not parallel to one another. The aorta was not dilated, and the only abnormality was some early atheroma, which appeared as small patches hardly elevated from the general level of the intima and free from calcification and ulceration. The heart weighed 28 oz. (793.5 g.), but the hypertrophy was evenly distributed throughout all the chambers; there was no dilatation; all the valves were competent and their cusps healthy. The heart muscle was of excellent tone and free from all sign of disease, both on gross examination and microscopically, but the individual fibres were hypertrophied. The coronary vessels were capacious, and on the left one there were a few small patches of early atheroma, but quite insufficient to interfere with the blood supply. The right coronary artery was healthy.

In the peritoneal cavity there was a little slightly blood-stained serum. On the posterior abdominal wall some haemorrhage could be seen shining through the peritoneum in the region of the pancreas. The left half of the body and tail of that organ were haemorrhagic, but no fat necrosis was anywhere observed. The other structures in this cavity were uninjured. The only abdominal disease found was an early hydronephrosis of the left kidney, and, though the entire urinary tract was dissected, no satisfactory explanation was discovered for this abnormality. There was no sign of chronic nephritis or any significant abnormality in other structures.

Comment

Contusion and rupture of the pancreas are relatively rare injuries. Vance (1928) quotes Gall to the effect that they occur in 4.4% of all visceral injuries, and most other authors agree with this figure. The pancreas lies in such a position that it could only be injured, in the absence of an open wound, by some hard object coming forcibly in contact with the epigastrium and crushing the gland against the body of the second lumbar vertebra. It is possible, however, that the view of Boesch (1921) is correct—namely, that the gland may frequently be injured without the condition being recognized during life. This case tends to support that view, as the patient made no complaint referable to the abdomen and was neither rigid nor tender in that region. Furthermore, the fact that fat necrosis was not present indicates that the pancreas was contused and not ruptured, and it seems likely that the condition might have resolved and remained unrecognized had the patient not sustained other more serious injuries which proved fatal.

It is clear, then, that the handle of the plough struck him a deep, powerful blow in the epigastrium. There were no injuries of any kind to the thoracic wall in the shape either of subcutaneous or muscular bruises or of fractures of the ribs or sternum, and it is almost inconceivable that a relatively sharp object like a plough handle could strike that region with enough force to produce the internal injuries described without leaving some mark on the soft tissues or the thoracic cage. It follows, therefore, that the damage to the aorta must have been produced indirectly by transmission of the force through the diaphragm. This thesis is supported by the fact that the anterior mediastinal tissues were contused in the absence of more superficial damage, the bleeding being no doubt due to the rupture of small vessels consequent upon the sudden dislocation of the pericardial sac. The heart is suspended in the pericardium by the aorta and superior vena cava, but is given basal fixation by the inferior vena cava and the structures of the lung roots. Its potential mobility is strictly limited in all directions, and it seems unlikely that rupture of the aorta alone could occur, due purely to displacement of the heart and traction on the vessels, by a force applied to the diaphragmatic surface of the heart from the abdomen.

Ruptures of the aorta are relatively frequent, but when they occur it is nearly always as a result of crush injuries, in which the osseous structures of the thoracic wall may or may not be fractured, depending largely on their elasticity. The tear is usually transverse, and is located just above the aortic valve cusps. It is due to a force thrusting the heart downwards and tearing it from its points of suspension. In these cases, as a rule, the tear is complete and the outcome immediately fatal. Partial ruptures—which become complete later—are more uncommon, and may arise in the absence of trauma if the

requisite predisposing factors are present. Peery (1942) has recorded the finding of healed partial tears in the first part of the aorta. According to him, partial tears are usually encountered in the ascending portion of the arch, are usually transverse or oblique, and are rarely longitudinal. An essential predisposing factor, in Peery's opinion, is severe hypertension of long standing, and he is not certain that there need be any abnormality in the wall of the vessel. Microscopical sections from the aorta taken near the lesion, but not including it, usually do not show any recognizable abnormality. Arteriosclerosis is probably not a cause for this condition, since the tears seldom occur through an atheromatous plaque or ulcer. The precipitating factor in most cases is exertion, which, by raising the already elevated blood pressure, exceeds the pressure the aortic wall can stand, and rupture occurs.

Another predisposing condition is the presence of coarctation of the aorta, in which the vessel tears below the constriction, presumably owing to the increased pressure in that part of the artery (Moragues, Moore, and Rossen, 1942).

A few cases of partial rupture of the aorta have been recorded in which trauma was the immediate cause. In Heller's (1904) case the partial tear was due to a heavy burden falling on a man's chest. In the case reported by Oppenheim (1918) clinical manifestations followed a blow by a falling piece of granite. Nordlander (1925) and Oppenheim (1918) both referred to the occurrence of ruptured aorta in aviators falling from a high altitude.

The present case is unusual, if not unique, because of the method of production of the damage to the vessel. This man had a grossly hypertrophied heart, and, though his blood pressure was never recorded and there were no symptoms or other post-mortem changes to suggest it, he probably suffered from essential hypertension, though the fact that he had followed a strenuous occupation may have been partly responsible for the hypertrophy. There is no such lesion now recognized as idiopathic cardiac hypertrophy in an adult, and, in the opinion of Kugel and Stoloff (1933), it appears doubtful whether there is such a condition in infants and young children, as some workers maintain. The heart, as stated, weighed 793.5 g. According to Aschoff (1924) the normal heart weight in men is up to 355 g., while Parkinson (1936) quotes Evans to the effect that the upper limit is 390 g. By either of those standards this heart was grossly hypertrophied, and though many hearts weighing more have been reported (Strong and Munroe, 1940; Rosenow and Smith, 1939; and Royster, Lisa, and Carroll, 1941) some other factor in addition to essential hypertension has usually been present to account for the hypertrophy. The mechanism of production of the aortic lesions therefore seems to have been the increased pressure in that vessel. The sudden compression of the heart must have further raised the already high pressure in the left ventricle and aorta to such a degree that the vessel wall was incapable of withstanding it, and partial rupture ensued.

The clinical symptoms in cases of partial rupture of the aorta are always indefinite, and the literature does not seem to record a case in which this condition has been diagnosed ante mortem. According to Peery (1942), "the most common symptom is a choking sensation or a feeling of suffocation. Dyspnoea may come on suddenly and continue for some time. Pain is also a frequent symptom, and may be stabbing or tearing, although pain is less frequent and less severe when dissection supervenes immediately. Occasionally cough, haemoptysis, and fever are the presenting symptoms, and may be associated with pain of pleuritic type. The most important physical finding is undoubtedly the presence of heart murmurs. These may be either systolic or diastolic, and are usually best heard in the aortic area. The systolic murmur is as a rule harsh, and is probably due to vibration, in the blood current, of the abrupt shelf-like edge of the tear, or to eddying of blood over the small pouch between the lips of the tear. In a considerable proportion of cases, however, dissection of the aorta occurs days, weeks, or months later, with the usual manifestations of the condition. In other cases the clinical picture becomes one of aortic regurgitation with congestive heart failure." In the present case there was no immediate pain referable to the thorax, and two hours elapsed before pain started. Probably during that time dissection was not occurring

and the acute pain in the left side of the chest radiating through to the back became manifest when dissection through the media began to develop. Final rupture 26 hours after the original injury is in keeping with other cases reported and, as in a proportion of these, it occurred after some mild exertion—in this case sitting up in bed.

It is interesting to speculate what the outcome might have been if the patient had stopped work after the injury and gone to bed. Perhaps the intimal tears might have healed, as happened in the cases reported (Peery, 1942).

Many cases have been recorded in which hypertension has been associated with a unilateral renal lesion of some sort, and hydronephrosis is one of the conditions mentioned. Baggenstoss and Barker (1941) have reviewed the literature and reported a series of their own cases. Their series is short and, they admit, insufficient to draw statistical conclusions in the case of hydronephrosis, but they nevertheless tend to the opinion that this condition does not appear to influence the onset of hypertension. This aspect of the case is mentioned merely for the sake of completeness, in view of the work of Goldblatt (1940) and of Wilson and Byrom (1939) on the relationship between unilateral renal ischaemia and hypertension.

Summary

The clinical and pathological features of a case of rupture of the aorta due to a blow on the epigastrium are discussed.

Gross cardiac hypertrophy and essential hypertension are considered as factors in the mechanism of production of the aortic lesion.

The aetiology and symptomatology of partial rupture of the aorta are reviewed.

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A FATAL CASE OF PURPURA AFTER SULPHAPYRIDINE

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An opportunity recently occurred of making observations on a case of acute purpura occurring during the course of sulphapyridine therapy for pneumonia. As we have found records of only 18 cases in the literature, this seems to be one of the rarer complications of sulphonamide administration. For this reason we report the essential details of the case.

Case Report

On Oct. 12, 1943, a publican aged 52 became ill with a febrile condition diagnosed as pneumonia. Sulphapyridine was administered outside hospital, 30 g. being given in seven days. The immediate response was satisfactory, but five days after cessation of treatment a rise of temperature gave cause for anxiety. Recrudescence of pneumonia or empyema was considered. Sulphapyridine therapy was restarted, 4½ g. being given during the next five days. The malaise and pyrexia, however, persisted. It was noted that the temperature rose after each dose of sulphapyridine. On Oct. 28 there was slight

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