may be restless, but in the later stages becomes weak and comatose. A mass is often palpable in the loin. Death may occur with dramatic suddenness.

The three principal aspects for discussion are the cause of the haemorrhage, the cause of death, and the treatment, if any.

Cause of the Haemorrhage.—It has been suggested that trauma during labour or delivery is the factor responsible for massive unilateral adrenal haemorrhage (Mitchell-Nelson, 1950). Cases in adults have been reported in which haemorrhagic cysts of the adrenal have followed trauma. However, it is difficult to understand how the adrenal glands, which are unusually well protected from external trauma, could suffer during normal labour or could be damaged during delivery without external signs of injury. Moreover, in our two cases delivery was quite straightforward.

Venous thrombosis, a fairly common occurrence in the neonatal period, could be a causal factor, but cases of adrenal vein thrombosis without gross haemorrhage are not rare. Moreover, in Case 2 the adrenal veins were found to be empty. It is conceivable that massive haemorrhage into the adrenal gland could be part of the picture of haemorrhagic disease in the newborn which may produce bleeding from one site only. Prothrombin estimations were not done in these cases, and no definite conclusion is possible concerning this suggested cause. A further possibility which several authors have entertained is that there is excessive degeneration of the foetal cortex. It would seem strange if this tendency were manifest on one side only; the adrenal gland on the opposite side in Case 2 appeared to be normal.

Cause of Death.—Acute adrenal insufficiency has been suggested as the immediate cause of death, but this seems to be unlikely. In the neonatal period the medullary and the chromaffin tissue are situated principally outside the adrenal gland, and the gland itself consists largely of involuting foetal cortex (von Reuss, 1920). Furthermore, Zuelzer (private communication) describes a case in which a child died when 8 days old, both adrenals having been destroyed by haemorrhage. He believes that the haemorrhage had occurred at birth, and that the child had lived for eight days virtually without adrenals. In the fatal cases so far reported the severity of the haemorrhage seems to have been a sufficient cause of death—particularly when there has been rupture of the haemorrhagic mass into the peritoneal cavity. The clinical picture is also in keeping with the diagnosis of severe internal bleeding.

Treatment.—Mild cases in which the haemorrhage is self-limited will need no treatment—in fact, many will pass unrecognized. In others the bleeding is so rapid and profuse that the infant is dead before effective measures can be started. In cases of intermediate degree early recognition of the condition and the administration of blood transfusion may be life-saving.

Summary

Two cases of unilateral neonatal haemorrhage of the adrenal gland are reported and the symptomatology is described.

The aetiology of the haemorrhage and the cause of death are discussed.

It seems probable that some of the more severe cases might be saved by early and vigorous efforts to deal with internal haemorrhage.

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ARTERIAL SPASM DUE TO AN INTRAVENOUS INFUSION

BY

M. SUTTON, M.B., B.S.

Late House-surgeon, Royal Northern Hospital, London, N.7

The following case provides an example of an unusual danger that may be encountered in the common surgical procedure of "putting up a drip."

Case Report

A bricklayer aged 52 was admitted to hospital with frequency and dysuria attributable to a myoadenoma of the prostate. He was otherwise quite healthy and no abnormalities were detected in his cardiovascular system. His blood pressure was 120/90, and his blood urea 29 mg. per 100 ml. Transvesical prostatectomy was performed (Mr. Roche), the anaesthetic being thiopentone, nitrous oxide, trichloroethylene, and D-tubocurarine chloride. On return from the theatre the patient was pale and his skin was moist. His pulse rate was 120 and blood pressure 105/75. The foot of the bed was raised on blocks, and it was decided to administer an intravenous saline infusion. The left median cubital vein was selected. French's needle from the transfusion set pierced the skin and vein wall as one, and entered the vein smoothly without any manipulation or withdrawal being necessary. Normal saline was infused at the rate of 60 drops a minute. The needle and tube were fixed in position by strapping and the whole arm was loosely bandaged to a splint. When the apparatus had been set up completely there appeared to be nothing untoward occurring, and no difference in the colour of the left hand and that of the rest of the body was observed. The patient was not in any way restless.

One and a quarter hours later it was noticed that the left hand and forearm were pale and cold, and the tips of the fingers of the left hand were slightly cyanosed; also, the intravenous drip had stopped. It had not run into the tissues. Neither the radial nor the ulnar pulse was palpable. The brachial artery was pulsating strongly down to a point about $\frac{1}{2}$ in. (1.9 cm.) proximal to the site of the intravenous infusion, but distal to this it could not be felt. Pressure on the palm of the left hand produced a blanching which persisted for about three minutes. Auscultation of the heart revealed no abnormality except that of the tachycardia (120 a minute) that was present. There appeared to be no source of origin for an embolus, but it did seem possible that the intravenous infusion was a traumatic factor, and so a diagnosis was made (later confirmed by operation) of spasm of the brachial artery. On disconnecting the infusion apparatus it was noticed that a small clot obstructed the terminal orifice of the needle. A radiant-heat chamber was placed over the patient in order to encourage cutaneous vasodilatation.

The pulse not having returned in 15 minutes, the patient was taken to the operating theatre. He had not regained consciousness from the anaesthetic given that afternoon, and anaesthesia was continued with cyclopropane and oxygen. Procaine, 4 ml., was injected subcutaneously into the left antecubital fossa. A 3-in. (7.6-cm.) longitudinal incision was made in the antecubital fossa over the line of the brachial artery. A portion of the median cubital vein $1\frac{1}{2}$ in. (3.8 cm.) long was resected; it included the site of the infusion. There was no evidence at operation that saline had leaked out into the tissues. The bicipital aponeurosis was incised and the brachial artery exposed. Bleeding during this dissection was almost completely absent even though no tourniquet was being used. The brachial artery was narrow and pulseless. The vasa vasorum were empty, making the wall of the vessel appear white. There was a small red circular area about $\frac{1}{4}$ in. (3 mm.) in diameter situated in the wall of the brachial artery almost underlying that part of the median cubital vein into which the infusion had been given.

As the artery was being freed from adjacent tissues by gentle dissection with Lahey swabs it started pulsating, but this was minimal and just perceptible at the wrist. A solution of 1 in 80 papaverine sulphate was applied to the surface of the artery in the form of drops. The artery visibly dilated and pulsation became stronger. The vasa vasorum filled and the surface of the vessel became pink. At this stage the pulse was readily perceptible at the wrist and colour returned to the hand and forearm. The papaverine was then washed off with saline and the skin sutured directly without any attempt being made to close the incision in the bicipital aponeurosis. Cotton-wool was loosely bound around the wound and the patient was returned to the ward.

The resected specimen of the median cubital vein was examined. There was a puncture wound in the superficial part of the vein through which the infusion had been given, but the deep surface showed no signs of trauma or any indication that the needle had transixed the vein and so possibly impinged upon the artery.

The next day the patient felt quite well apart from "soreness" of the tip of his left index finger. There was an area of brownish-black discoloration, about $\frac{1}{4}$ in. (6 mm.) in diameter, at the tip of the left index finger. This was slightly tender to touch. There were no other relics of the arterial spasm that had occurred a few hours before.

Comment

Spasm of the main artery of a limb is a well-recognized condition which may follow trauma to the arterial wall. This trauma may be caused by the passage of shrapnel or bullets close to the artery, as described in the cases of Küttner and Baruch (1920), or may occur during an operation in which there has been some manipulation of an artery. It may follow supracondylar fractures of the humerus, and Griffiths (1940) has brought much clinical and operative evidence to show the importance of this phenomenon in the development of Volkmann's ischaemic contracture. Griffiths quotes a case in which a tourniquet was accidentally left in place for six hours; 18 hours later, the symptoms of ischaemia persisting, the limb was explored and the femoral artery was found to be in a state of spasm. It has also been known to occur as a complication of arteriography, but most of these cases are instances of true primary thrombosis (G. J. Hadfield, 1952, personal communication).

The case reported here is interesting because the trauma to the artery, if existent at all, must have been minimal. The induction of the infusion appeared to be smooth. The patient was not restless, and therefore it is unlikely that he moved his own arm during the infusion and so traumatized the artery. The operation revealed no signs that the artery had been injured, apart from the small area of redness in its wall, and this could possibly have been an artifact occurring as a result of freeing the artery by dissection. It is interesting to speculate whether the presence of saline, at a lower temperature than that of the blood, passing through a vein close to an artery could have any adverse effect on the arterial wall.

Kinmonth (1952) has shown that in rabbits a gradual fall in blood pressure is associated with a parallel shrinking of large arteries. If we can postulate this state of affairs in man, then in post-operative shock, a condition associated with a fall in blood pressure, it is possible that there is an increased susceptibility to spasm of large arteries. This may apply to the case described above. The spontaneous termination of the intravenous infusion may be explained on the basis of venous stasis due to an impaired arterial supply leading to an impaired venous return.

This case is also of interest because it shows that, in common with many previously reported cases of arterial spasm (Cohen, 1940), mere exploration and freeing of the vessel was enough to restore some degree of blood flow to the limb.

The treatment employed in this case—namely, the local application of papaverine sulphate—is of interest in view

of the experiments recently carried out by Kinmonth (1952). Various smooth-muscle-relaxing agents were tested for their ability to relieve arterial spasm. It was shown that papaverine was the most consistently successful in producing relaxation, which did not retrogress when the drug was removed.

I wish to thank Mr. A. E. Roche for permission to publish details of the case. I am also indebted to Mr. G. J. Hadfield for helpful advice and to Mr. J. C. B. Serjeant, who operated for the arterial spasm.

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PHAECHROMOCYTOMA WITH FATAL MYOCARDIAL INFARCTION IN A MAN AGED 22

BY

W. M. PRIEST, M.D., M.R.C.P.

Physician, No. 14 South Warwickshire Group

Phaeochromocytoma is being reported with increasing frequency; about 194 cases were recorded up to 1950 (Walton, 1950), and four more have been described in the *British Medical Journal* alone in the past year (Swan, 1951; Crowther, 1951; Pantridge and Burrows, 1951). Nor is myocardial infarction in young persons rare enough to merit the report of a single case, several series having been published since 1937 (Scott, 1938; French and Dock, 1944; Newman, 1946, 1951). Myocardial infarction in a young hypertensive subject with phaeochromocytoma might occasionally be expected to occur, but I have been able to find only two references to this combination (Goldenberg *et al.*, 1947; Wilkins *et al.*, 1950).

The following case is remarkable for the short period of disability, the patient having remained at work, apart from two brief intervals (one for hospital investigation), until ten days before death; the degree of cardiac enlargement; and the evidence of multiple myocardial infarcts, old and recent.

Case History

The patient, a farm-worker aged 22, had been subject to severe headaches for two years. While fielding at a cricket match he collapsed, with burning substernal pain, and was taken home, where he remained in the care of his doctor for four days. The pain subsided in a few hours, but on the fourth day he showed evidence of left ventricular failure. Orthopnoea and cyanosis were present, the pulse was 130, the apex beat was in the sixth space in the mid-axillary line, and there was presystolic gallop. Basal rales were heard and the blood pressure was 136/120.

On his admission to hospital an x-ray film of the chest, sitting, showed gross cardiac enlargement and some pulmonary congestion (Fig. 1); and an E.C.G. showed the changes of a posterior infarction. He improved for four days; there was no further pain, the dyspnoea and cyanosis had subsided, the pulse rate fell, and the gallop became inaudible. He died abruptly on the fifth day after admission, the tenth after his collapse on the cricket field. Death was instantaneous and not associated with unusual movement or effort.

Earlier History.—He had lived and worked on a farm all his life, and, apart from what were described as occasional "sick headaches" in adolescence, had had good health.