and the gastro-intestinal tract was normal. In the right iliac fossa was a large mass of tumour tissue which was infiltrating the psoas muscle. The right ureter ran through this mass. Section through the tumour showed it to be composed of white tumour tissue, showing areas of necrosis at places. The tumour tissue had a radiate appearance, which started from a point in the ureter just above where it crossed the pelvic brim. In an upward direction the wall of the ureter was involved to a point 3 cm. from the pelvis, and in a downward direction to very near the opening of the bladder. In spite of the extensive ureteric involvement in an upward and downward direction from the primary focus there was no obstruction of the tube, and the renal pelvis showed no dilatation. The kidney on the affected side contained two nodules of tumour tissue in its substance, each slightly smaller than a pea (see Plate). The left kidney was normal. The para-aortic glands were fairly extensively involved by secondary tumour growth. The fourth and fifth lumbar vertebrae also showed invasion by tumour tissue.

Sections of the ureter and kidney were examined histologically. The tumour was found to involve the whole thickness of the ureter and to spread for some distance outside this at its point of maximum growth; but even here the remains of circular muscle and fibrous tissue were present, thus preserving the tube-like appearance. The mucous membrane was replaced by ulcerated tumour tissue. The tumour cells were rather small and were spheroidal in shape. Here and there a multinucleated cell was apparent. The tumour had a definite alveolar arrangement and was obviously a papillary type of carcinoma, resembling that got in the renal pelvis.

Commentary

The case described presents some interesting features:

1. *The Absence of Obstruction of the Ureter.*—A tumour growing outside the ureter and pressing on the tube very readily causes obstruction with secondary hydro-nephrosis. A primary carcinoma of the ureter apparently in many cases does not do this (McCown, 1930). In our case signs of obstruction were absent or slight. Examination of the specimen readily showed why this was so. The tumour was seen to be growing outwards in a radiating manner from its focus of origin, and in this way appeared actually to hold the tube open instead of obstructing it. Moreover, a carcinoma growing from outside the ureter compresses it for a time before actually infiltrating it, whereas a primary carcinoma of the tube early ulcerates into its lumen, and necrotic cells on the surface are liable to be washed down with the efflux of urine. The growth of tumour for some distance upwards and downwards along the ureter should also be noted.

2. *The Absence of Urinary Symptoms.*—This absence of symptoms is well known, and in many of the cases reported in the literature the diagnosis of renal disease was not suspected before the necropsy (McCown, 1930). This lack of symptoms is associated in many cases with the absence of obstruction of the tube. The absence of even haematuria was a notable feature in the present case. This has also been noted by other writers (Wising and Blix, 1878; Adler, 1905; van Capellen, 1916; Hektoen, 1896; Toupet and Guéniot, 1898).

Summary

The case of carcinoma of the ureter showing some interesting features is described, and others in the literature are summarized.

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SPONTANEOUS SUBARACHNOID HAEMORRHAGE

REPORT OF A CASE

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WITH

NOTES ON POST-MORTEM FINDINGS

BY

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(WITH SPECIAL PLATE)

Rupture of a congenital aneurysm has been recognized as an important and comparatively frequent cause of spontaneous subarachnoid haemorrhages, especially in young non-syphilitics. The subject has been studied in detail by Eppinger (1887), Fearnside (1916), and several other workers. Forbus (1930) investigated the histological aspect in particular, and offered certain explanations on developmental basis.

The following case is described chiefly because a detailed post-mortem examination was available and the histological studies showed certain differences in the structural changes from those described by Forbus.

Clinical History

A man aged 30, a hotel servant, developed a severe headache overnight. The next morning he felt giddy and fell down in a semi-comatose condition in the lavatory, when he was brought to the hospital.

On admission (March 27, 1936) the patient was dazed, but answered simple questions in a slow thick voice. He complained of a severe headache, but otherwise felt well, though he had a sense of impending disaster.

On examination he was seen to be well nourished, with no marks of injury; and material from gastric lavage showed no poison. Breathing was rapid and deep, and there was a definite rigidity of the neck and exaggeration of deep reflexes. Kernig's phenomena and the plantar reflex were equivocal, and abdominal reflexes were present. Lumbar puncture brought out fluid under tension, uniformly mixed with blood. No clot was found on keeping; the supernatant fluid had a golden-yellow colour. The pulse was slow—52 per minute—and full, and the blood pressure 143/100. Heart sounds were normal.

Laboratory Examination.—The urine showed a fair amount of albumin, but no casts; no red blood cells; no pus cells. Blood culture revealed no organisms. The blood count was normal; there were no malarial parasites, and Widal, Wassermann, and Kahn reactions were negative. The cerebrospinal

fluid showed no disproportionate increase in leucocytes; on culture no organisms were found.

For the next two days the patient appeared to improve, and the pupils began to react briskly to light. On March 30 he suddenly became comatose and died.

Necropsy and Histological Findings

The body was well built and well nourished, and no scar could be seen on the penis. On examination the right lung showed a small subapical scar. The right kidney was enlarged to about three times the normal size, due to hydro-nephrotic dilatation of the pelvis and calices, which contained about 120 c.cm. of urine. The left kidney was normal.

The heart was normal, though the aorta contained insignificant streaks of atheroma.

Examination of the brain showed that the veins in the dura were tense. On opening, extensive subarachnoid haemorrhage was seen, the blood clots being thickest near the optic chiasm. The median side of the left frontal lobe was extensively ploughed up by diffuse haemorrhage and felt boggy. Both the internal carotids were smaller than normal. The anterior communicating artery formed a broad band from the lower surface of which a minute branch was given off to the right frontal lobe. At the junction of this vessel with the band was a pea-sized dilatation with thin translucent walls. This had burst, leaving a round opening the size of a pin's head towards the left anterior region. On the left middle cerebral artery a similar unruptured thin-walled dilatation was also found at the beginning of a small branch.

On opening the brain the interventricular septum and both the basal ganglia were seen to be extensively broken up by blood clots, and some clots appeared in the ventricles. The Wassermann test with post-mortem serum proved negative.

Sections were taken from both the ruptured and unruptured aneurysm, and from areas where several brain vessels branched. The aneurysm showed a part of the vessel with normal muscular coat and an intact internal elastic lamina. Towards the aneurysmal sac the internal elastic lamina was absent. There was no gradual thinning and no fraying out of the ends. The intima was thickened and the muscular coat showed definite fibrosis. The muscular coat rapidly thinned out, and the wall of the sac proper was formed by inelastic connective tissue only. The distinction between the three coats was lost. The endothelial lining was intact and no thrombus had formed. Serial sections of the sac failed to show any relation of the origin of the aneurysm with the branch of the vessel.

Section of the unruptured aneurysm revealed a distinct dilatation of the vessel at the beginning of the vascular branch, but the defect in the muscular coat was some distance from the origin of the branch. The muscular coat was well preserved at the origin. The muscular wall opposite to the origin of this branch was also markedly dimpled. The intima showed a definite compensatory hypertrophy and the internal elastic lamina was frayed out. Sections of other vessels also demonstrated these irregularly distributed medial defects and patches of intimal thickenings at many places, but the internal elastic lamina was intact and no dilatations were present.

The cortical and striatal regions did not reveal any definite abnormalities of the smaller vessels. Sections were also taken from the meninges to exclude the possibility of meningo-vascular syphilis; but no cell infiltrations or vascular changes were seen. The coronary branches did not show any atheromatous changes or any medial defects.

Commentary

The thickening of the intima is the only finding which suggests an arteriosclerotic origin of the aneurysm. The early age and the absence of arteriosclerosis in other vessels does not necessarily exclude the possibility. A closer study of a large number of brain vessels does reveal a solitary case of arteriosclerosis of cerebral vessels in comparatively young persons, but histological studies

in these cases distinctly show patchy fibrosis and hyaline degeneration in media with well-marked fragmentation of the elastic lamina. The adventitia is generally uniformly thickened and the intima shows irregular thickening and hyaline degeneration. Though the vessel becomes tortuous and a beaded appearance is produced, sections do not demonstrate complete loss of musculature as in congenital or syphilitic aneurysms. Thrombosis precedes a rupture by a considerable interval.

The occurrence of congenital aneurysms is explained by Forbus on embryological grounds as being the result of a congenital weakness of vessel walls near the origin of branches. This is not always borne out, as in this case and in some others studied. The medial defects are very often irregularly distributed. Forbus also found that in many cases with defects in cerebral vessels similar latent defects occurred in other vessels, an observation supported by Duguid. The coronaries were studied in the present case, but no medial defects were seen.

These findings suggest that medial defects do not form a complete explanation of congenital aneurysms. The internal elastic lamina can maintain the integrity of the cerebral vessels in the presence of such defects. The embryological explanation of the occurrence of medial defects is also not always borne out. The defects may occur independently some distance from the site of branching. Further histological studies are therefore necessary before congenital aneurysms can be accounted for satisfactorily.

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RE-EXPANSION OF THE ATELECTATIC LOWER LOBE AND DISAPPEARANCE OF BRONCHIECTASIS

BY

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(WITH SPECIAL PLATE)

In discussion of the factors producing bronchial dilatation it is customary to mention the weakened, inflamed bronchial walls; the pressure of retained secretions; the expiratory strain of coughing; and the possible factor of traction on the bronchial walls by intrapulmonary fibrosis.

Mechanism of Production of Atelectatic Bronchiectasis

Frequently no mention is made of the constant factor of the normal elastic tension of the lung. This factor, which is measurable by the intrapleural pressure, is greatest in full inspiration and least in full expiration. It is obviously likely to lead to pathological dilatation when the bronchial walls are inflamed and weakened. In the presence of an atelectasis this force is greatly increased on the affected side, as has been shown by the very high negative intrapleural pressure on that side. Such pressures as -432 mm. of water have been recorded by Habliston (1928) in massive collapse of a lung. On the bronchi in the collapsed lower lobes, which have no expansile alveolar "buffer" to protect them from this external suction, there is exerted a powerful and sustained dilating force. Findlay (1935) has described the process of bronchial dilatation in these cases as "compensatory,"

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FIG. 1.—The unruptured aneurysm of the middle cerebral artery. Inset: diagram of appearance.

FIG. 2.—Photomicrograph of the aneurysm.

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FIG. 1.—April 15, 1937. Skiagram showing the triangular shadow cast by the atelectatic left lower lobe (contact print).

FIG. 2.—May 4, 1937. Contact print of skiagram showing marked cylindrical dilatation of the bronchi in the left lower lobe which is still largely collapsed. (Note the crowding together of bronchi.)

FIG. 3.—August 20, 1937. Contact print showing that during the past three months, in which the left lower lobe has remained expanded, its bronchi have been of normal calibre.