that it may be insidious in its onset, transient in its course, and complete in its resolution. It is also suggested that in some cases it may be a late primary tuberculous manifestation and analogous to pleural effusion.

The difficulty in proving the aetiology is stressed and the value of pneumopericardium in allowing the heart outline to be visualized is noted.

Guinea-pigs inoculated with pericardial fluid may survive for many months before showing signs of disease, and much longer than the conventional six weeks is required before passing judgment.

Gross pericardial thickening may develop in a relatively short space of time, and this relationship with Pick's disease is considered.

All cases of pericardial effusion with a small heart and absence of rheumatic manifestations should be considered as potentially tuberculous unless proved otherwise.

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MEDULLARY SUPRARENAL CHROMAFFINOMA PRODUCING MALIGNANT HYPERTENSION

BY

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The following case of medullary suprarenal chromaffinoma (phaeochromocytoma), with presenting features of intermittent attacks of paroxysmal hypertension and vasomotor crises, ending in malignant hypertension with uraemia as the cause of death, seems worthy of record.

Case History

A married woman aged 38 was admitted to hospital on Oct. 13, 1944. She complained of cough with sputum of several years' duration, and attacks of dyspnoea, chiefly at night. These usually started with a feeling of discomfort in the lower abdomen, followed by nausea and shivering, severe palpitation, sensation of constriction in the chest, profuse perspiration, pain behind the eyes, and marked tremor of the hands. The patient was kept under close observation and was examined during and between attacks, which occurred about every third day. The following is a brief description of a typical one.

The attack was induced by the patient's turning on the right side whilst lying in bed. She became restless, trembled, was very excited and talkative, and sweated so profusely that her underclothing was drenched with perspiration. There was pronounced tachypnoea and some dyspnoea and pallor, with cyanosis of the lips, ears, cheeks, hands, and forearms. The forearms and legs were cold and white and the fingers and toes cyanotic. Pulsation of the brachial and radial arteries was palpable, but in both dorsalis pedis and the popliteal and femoral arteries it was feeble. The pulse rate was rapid—approximately 130—but regular. The blood pressure was

300/170. The pupils were much dilated; the temperature was in the region of 100° F. (37.8° C.) and the urine contained albumin and sugar. *Neck*: The thyroid was not enlarged; there was no lymphadenitis; the trachea was central; there was no venous pulsation or engorgement. *Chest*: Signs of disease were absent save for a few scattered rales and rhonchi at both bases. *Heart*: This was slightly enlarged to the left. The cardiac impulse was forcible and diffuse and the sounds were normal. *Abdomen*: There was some fullness and marked tenderness below the right costal margin, but no rigidity in the right hypochondrium. Dullness to percussion was present over an area of about 2½ in. (6.3 cm.) in this region. The liver and spleen were not palpable. *C.N.S.*: Some oedema was observed round the eyes, which watered profusely. The pupils were dilated, but reacted promptly to light and accommodation. The retinae showed bilateral papilloedema, greater on the left side than on the right, and multiple haemorrhages and discrete exudates. The reflexes were brisk. There were marked dermographism and tremor of the fingers, tongue, and upper lids. There were no other abnormalities.

The urine passed during an attack was copious, clear, and almost watery in colour, with a specific gravity varying between 1004 and 1010. It contained a slight trace of albumin and sugar (0.1 and 0.2%). The average duration of an attack was about half an hour.

After the attack the patient, though exhausted, was calm and placid, talking slowly and normally. The cyanosis and pallor soon disappeared, the skin became dry and the pulse slow (approximately 72), and the blood pressure fell below normal, varying between 90 and 100 systolic and 70 and 80 diastolic, only to rise slowly to about 200/120, which was her usual blood pressure between attacks. It remained fairly constant at that level until a further attack occurred, when it abruptly rose again to 300/170.

From the history and from physical examination during and after attacks a preliminary diagnosis of medullary suprarenal chromaffinoma was made. That it was on the right side was suggested by the fullness present below the right costal margin and by the fact that the attacks were precipitated by turning on to that side. There were no symptoms or signs indicating involvement of the cortex in the way either of hyperfunction resulting in "adreno-cortical syndrome" or of hypofunction resulting in Addison's syndrome.

Ancillary investigations were carried out with the following results.

A catheter specimen of urine after an attack showed: specific gravity, 1008; albumin, slight trace; sugar, 0.2%; deposit, occasional leucocytes, epithelial cells, a fair number of erythrocytes, hyaline and cellular casts; culture, sterile. Blood urea was 54 mg. per 100 ml. From a urea-clearance test the renal function appeared to be 34% of the average normal maximum clearance and 24% of the average standard clearance. The glucose-tolerance test showed diminished tolerance, as the fasting blood sugar was 150 mg. per 100 ml.; 3/4 hr. after 50 g. of glucose, 270 mg.; and in another 3/4 hour, 260 mg. Lumbar puncture revealed cerebrospinal fluid under normal pressure. This was clear and colourless and contained 1 lymphocyte per c.mm.; protein, 30 mg. per 100 ml.; sugar, 130 mg. per 100 ml.; and chlorides, 740 mg. per 100 ml. The Queckenstedt test was positive.

Radiological examination of the skull showed no evidence of abnormality. The pituitary fossa was rather small, but the clinoid processes appeared normal. Radiographs of the chest revealed some fibrosis in both lungs probably due to old healed tuberculous infiltration. Bronchial thickening was present at both bases.

X-ray examination showed that the heart was globular in shape, suggestive of concentric hypertrophy of heart muscle without dilatation. An electrocardiogram revealed depression of S-T intervals in all leads, but otherwise it was within normal limits. Intravenous pyelograms showed that the kidneys were normal in size and position. Both excreted well. There was no evidence of any organic lesion in the urinary tract. No suprarenal calcification was found, but there was a depression in one calix of the right kidney, probably due to external pressure and suggesting that the tumour was on the right side.

The erythrocyte sedimentation rate was 59 mm. in 1 hour (Westergren). The white cells numbered 12,700, with a normal differential count. The Wassermann and Kahn reactions were negative.

As these clinical investigations strengthened the diagnosis of a right suprarenal chromaffinoma, it was decided to perform an exploratory laparotomy. Before this could be done, however, the patient had another severe attack, which proved fatal.

Post-mortem examination.—This was carried out at Charing Cross Hospital on Dec. 5, 1944, and the report by Dr. Vines was as follows. "*Brain.* No abnormality was found. The pituitary appeared normal. *Thorax: Lungs:* Both lungs showed some emphysema and chronic bronchitis; they were closely bound to the thoracic wall by chronic fibrous adhesions. In both lungs there was extensive patchy fibrosis at the apices, but no active tuberculosis was seen. *Heart:* There was gross hypertrophy of the left ventricular wall. The tone of the muscle was good. The aorta was rather small and some early atheroma was present. *Abdomen:* The only pronounced changes were seen in the kidneys and the right adrenal. In the latter there was a spherical tumour about 1 in. (2.5 cm.) in diameter projecting forwards. It felt soft and almost cystic, and was not incised. It did not appear to infiltrate, and no metastatic deposits were found. In both kidneys the cortical band was reduced in width and the capsules were adherent, leaving a finely granular surface. In the lower two-thirds of the right kidney there were numerous areas of cortical necrosis suggestive of malignant hypertensive changes; the total changes in the kidneys suggested uraemia as the ultimate cause of death."

Histological examination of the adrenal tumour revealed a fairly typical medullary chromaffinoma. In accordance with the clinical evidence and the presence of an actively secreting tumour, the kidneys histologically showed well-marked hypertensive changes with arteriosclerosis and necrosis. Although the adrenal tumour should have been easily removable, in view of the extensive renal damage it is unlikely that the patient would have survived operation.

There are, of course, reasonable grounds for belief that the accessory chromaphil bodies also secrete adrenaline. It is not surprising, therefore, that similar tumour masses should be found associated with one or other of these bodies anywhere in the sympathetic autonomous nervous system, causing similar paroxysmal attacks of hypertension. For instance, cases of paroxysmal hypertension associated with tumours of the carotid sinus and carotid body and also of the aortic bodies have been reported. The structure and function of both are probably identical. They consist of masses of glandular-looking cells arranged in characteristic clumps resembling those of chromaphil cells. Such a tumour of the carotid sinus or carotid bodies has often been mistaken for an enlarged cervical gland.

In essential hypertension cerebral hypertensive crises may simulate paroxysmal hypertensive attacks such as I have described. It is important to remember these occurrences in order that errors of diagnosis, and thus of treatment, may be avoided.

Summary and Conclusions

A case of medullary chromaffinoma of the right adrenal medulla in a woman aged 38, diagnosed during life, is reported and the syndrome discussed. The patient, unfortunately, died from secondary malignant hypertension and uraemia before operation could be performed. The history of the case and the results of various examinations conducted during and after attacks are described.

The symptom-complex was caused by a tumour of the right adrenal medulla which consisted of adrenaline-secreting chromaffin cells discharging into the blood paroxysmally, though possibly also constantly, small quantities of adrenaline. This caused hypertension with periodic exacerbations to very high levels—sometimes to over 300 mm. Hg—glycosuria, and, when the kidneys became ischaemic, albuminuria and uraemia. The vasomotor crises were also due to a discharge of adrenaline into the blood stream.

The treatment is removal of the tumour, but this is likely to be successful only if it is recognized early and before permanent changes in the kidneys have occurred. The tumour, as in the present case, is seldom malignant and is usually unilateral. It can therefore be concluded that a certain proportion of all cases of paroxysmal hypertension are due to a hyperfunctioning medullary tumour. In this case there was evidently gross hyperfunction, but cases also occur in which the tumour is less active and the symptoms correspondingly less pronounced.

Within the past few years a number of tumours of chromaphil tissue have been reported, some authenticated at necropsy, others actually removed surgically with beneficial results.

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A CASE OF PHAECHROMOCYTOMA

BY

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The surgical removal of a chromaffin tumour or phaeochromocytoma of the adrenal medulla is rare enough to warrant the report of a recent case.

Case History

The patient, a 24-year-old secretary, was admitted on June 4, 1945, under the care of Prof. Ellis, complaining of blurring of vision and of attacks of upper abdominal pain and vomiting with palpitations and headache. There was nothing relevant in her past history. The attacks began two years before admission and at first recurred about once every four weeks. They became gradually more frequent, and on admission she was having one or more daily. Immediately before an attack she had a premonition that it was about to occur. She then felt a burning pain in the epigastrium, followed by severe headache, nausea, and often vomiting. She had severe palpitations, "as though her heart would burst through her ribs," her hands and face became pale, and she felt cramp-like pains in the limbs. The attacks occurred at any time of the day or night and usually lasted 10 to 20 minutes, but might last an hour. On three occasions she had lost consciousness. After an attack she felt weak, her arms and face were flushed, and she had a sensation of "pins and needles" in her arms. Five months before admission she was examined elsewhere and found to have a raised blood pressure. The fundi and urine were normal, but the cerebrospinal fluid contained 80 mg. of protein per 100 ml. No record of the pressure of the cerebrospinal fluid at this time was available. She was noted as unduly excitable and her condition was dismissed as functional, a