1/1,000 adrenaline hydrochloride by intravenous injection and his blood pressure immediately rose to 125/65. However, within an hour it had dropped to 75/40; he was given an intramuscular injection of ephedrine hydrochloride, ¼ gr. (50 mg.), and it rose to 105/60. During the next day he was maintained on ephedrine hydrochloride 1 gr. (65 mg.) by mouth, and his condition was satisfactory, his blood pressure remaining about 115/75, although on sitting up it dropped to 70/60. From an absence of urine on the previous day his output rose to 66 oz. (1,870 ml.). From then on he continued to improve, although his pupils remained fixed and dilated for a further four days, and he remained on ephedrine by mouth for another week.

During his stay in hospital, routine investigation showed that his urine contained 6 parts per 1,000 of albumin but no casts or red cells. His blood urea was 142 mg. per 100 ml. and his blood bromide 30 mg. per 100 ml. It was thought at first that these changes might have been due to continued low blood pressure, but three weeks later his urine still contained albumin, his blood urea was 87 mg. per 100 ml., and a urea-clearance test showed a standard clearance of only 15% of normal. It is therefore more likely that his kidneys had already been damaged by his attack of Bright's disease. An electrocardiogram was normal. A barium meal revealed a considerable degree of pyloric stenosis, which was secondary to chronic duodenal ulceration as it did not show the characteristic picture produced by this drug.

COMMENT

It seems probable that this case represents true intolerance to hexamethonium bromide. A comparatively small oral dose produced a very marked effect which was prolonged over several days.

It is possible that the pre-existing renal damage might have interfered with the elimination of the drug, though without exact knowledge of its fate in the human body this would merely be a guess. Furthermore, it would seem unlikely, as hexamethonium bromide has been used in other cases with just as severe interference with renal function without producing such an alarming result.

This case does, however, suggest that small doses, probably no more than 120 mg. by mouth, should be used when beginning treatment with hexamethonium bromide, that the patient should be within easy reach of a bed, and that a careful watch should be kept on his blood pressure.

Our thanks are due to Dr. A. H. Douthwaite for his permission to publish this case and for his help and encouragement.

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Phaeochromocytoma Presenting as a Cardiac and Abdominal Catastrophe

In view of a report of a phaeochromocytoma presenting as an abdominal emergency (Gilliland and Daniel, 1951), it is of interest to record this very similar case.

CASE REPORT

A housewife aged 47 was admitted to hospital as a case of coronary thrombosis. She had a 12-hours history of sudden severe substernal and epigastric pain lasting half an hour, nausea, vomiting, sweating, and collapse. The attack had begun at 11 p.m. while strolling by the seashore. A local doctor recorded a blood pressure of 220/140 and a pulse of 160, gave her morphine, and asked for her to be admitted. The patient gave a past history of diabetes mellitus for over three years, stabilized at a Birmingham hospital at 20 units P.Z.I. daily, urine tests being done twice a year. She had had her insulin the day of the attack, but not on the morning of her admission. She had also had a similar but less severe attack of pain of a few minutes' duration three months previously.

On admission she was in an obviously shocked condition; pale and grey about the lips, cold, nauseated, and sweating profusely. Her temperature was 101.8° F. (38.8° C.), pulse 160, respirations 26, and blood pressure 60/?. The heart sounds were distant, with a soft apical systolic murmur. There was no abdominal tenderness or palpable tumour. The lungs and nervous system appeared normal. The urine was brick-red to Benedict's test and showed quantities of acetone, 1+ of albumin, and 8 g. of chloride per litre. Her haemoglobin was 113% and W.B.C. 12,500. She was put on anticoagulants and soluble insulin twice a day, with glucose feeds.

By the second day her blood pressure had risen to 140/80. She complained of encircling abdominal pains and appeared as shocked as on admission. Her abdomen was soft but tender in the left hypochondrium. The morning specimen of urine showed green to Benedict's test and no acetone.

On the third day she was found to have generalized extreme abdominal tenderness, some distension with gas, absence of bowel sounds, and a mobile mass the size of an orange to the left of the umbilicus. She said she had passed only a little flatus and no faeces since admission. A loud apical systolic and softer diastolic murmur had developed. Electrocardiograms showed no definite evidence of a coronary thrombosis but diffuse myocardial damage. *X*-ray films of the chest and abdomen were negative. Her haemoglobin was now 88% and W.B.C. 6.500. Her blood pressure was still 140/80, her pulse settled to about 120, and she remained slightly febrile.

A surgical opinion was obtained, but in view of her critical state and the likelihood of a coronary thrombosis no interference was recommended.

For the next ten days her general condition remained practically unchanged; the abdominal pain passed off but the distension remained; occasionally bowel sounds were audible and a little flatus was passed. Enemata and flatus tubes had little effect. The mass moved to the right of the umbilicus and became more palpable. The diastolic murmur vanished and the character of the systolic murmur changed. Meanwhile difficulty was experienced in stabilizing both her diabetes and her blood prothrombin time. She could take only small quantities of fluids by mouth, even through Ryle's tube, without vomiting, and finally an intravenous drip was set up.

Her blood pressure was 125/80 on the ninth day, rose to 200/110 on the tenth day, and fell to 180/80 by the twelfth day. She sweated a great deal during this phase. On the twelfth day she passed three motions and the distension disappeared, but next day she complained of severe pain in the left chest, had an epistaxis, became dyspnoeic, and died after a short period of Cheyne–Stokes respiration.

Pathology.—At necropsy (Dr. J. A. V. Morgan) all organs were found to be normal except the lungs, which were congested and oedematous, and the left suprarenal, which "was replaced by a soft encapsulated haemorrhagic tumour 8.5 cm. in diameter with a cystic centre. There had been a recent haemorrhage into the capsule of the tumour and the compressed tissues surrounding it. The left kidney was compressed and grossly distorted by the growth." Histology showed mainly necrotic matter with areas of cells compatible with those of a phaeochromocytoma.

COMMENT

As with the case of Gilliland and Daniel, there was much conjecture over the diagnosis of this case. The substernal pain and fall in blood pressure made a coronary thrombosis

seem most likely, with a mesenteric infarct to account for the second-day ileus. Phaeochromocytoma was considered because of the glycosuria and hypertension, but, after perusal of the papers by Crowther (1951) and Swan (1951), was rejected as unlike any previous case so far reported at that time. Unfortunately, the paper of Gilliland and Daniel was not noted until the day of the necropsy, when the similarity in presentation and signs was remarked upon.

Presumably haemorrhage into the cystic, friable, necrotic tumour produced the severely shocked state which, with a curious lividity of the skin, persisted throughout the whole illness. The use of anticoagulants must have aided this considerably, as must the bout of hypertension. A troublesome feature of the case was the diabetes, more and more insulin being required to keep the urine clear of sugar as her blood pressure rose after the original fall.

I would like to express my thanks to Dr. J. C. Prestwich for permission to publish this case, which was under his care.

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Phaeochromocytoma Simulating Thyrotoxicosis

In spite of a growing literature of case reports and methods of investigation, the diagnosis of phaeochromocytoma presents many difficulties, and it is probable that only a minority of cases are recognized during life. This state of affairs may be attributed to the apparent rarity of the tumour and the wide range of symptom-complexes it may produce. The tumour itself is often too small to demonstrate by physical methods, and other investigations may yield ambiguous results. The following case illustrates the similarity which may exist between thyrotoxicosis and the syndrome of hyperadrenalism.

CASE REPORT

A married woman aged 58 was referred to hospital on June 19, 1950, by Dr. G. C. Campbell, who suspected thyrotoxicosis. For 12 months she had suffered from paroxysmal vertigo associated with tinnitus and deafness on the right. More recently she had lost weight and complained of weakness and apprehensiveness. Her appetite remained excellent. There had been amenorrhoea since the normal birth of her second child 28 years before. Her previous health had been good. There was no family history of goitre and none of deafness.

Of slight build, the patient was evidently emaciated. Her weight was 5 st. 10 lb. (36.3 kg.). She wore a rather strained expression and was pale, but not obviously anaemic. Her hair was fine; the eyebrows and axillary and pubic hair were scanty. There was an impression of restlessness about her movements, which were rapid and bird-like. Other features were a resting pulse rate of 120, a blood pressure of 180/100, an apex beat in the sixth left intercostal space in the mid-clavicular line, and a loud systolic murmur heard over all areas and maximal at the apex. No exophthalmos was evident. There was a fine tremor of the outstretched fingers. The thyroid gland could not be felt. Examination of the patient's abdomen revealed no abnormalities. She was mildly breathless at rest, but no physical signs were found over the lung fields. The nervous system appeared normal apart from some nerve deafness on the right. The tympanic membranes were healthy and intact. The urine contained neither albumin nor sugar.

A threefold diagnosis of thyrotoxicosis, old rheumatic heart disease, and possibly otosclerosis was made. Mr. Arnold Gourevitch kindly saw the patient and agreed that thyrotoxicois seemed likely. At his suggestion an *x*-ray film of the thoracic outlet was taken. This showed no evidence of a retrosternal goitre.

On July 13 the patient was admitted to hospital, and on the 17th she was given methyl thiouracil, 600 mg. daily, and iodine. By July 23 she was able to say she felt much less agitated, but the pulse rate seldom fell below 100. Iodine was discontinued on July 27. By August 4 her condition was clearly not responding in the manner anticipated. Tachycardia and tremor persisted. On August 11 the pulse rose to a sustained rate of 150 and breathlessness increased. Venous engorgement appeared in the neck. The pulse rate was 170 on August 14. The pupils were constricted and the skin was moist. The blood pressure had fallen to 116/96. Methyl thiouracil was discontinued. On August 17 the patient was semi-conscious, pulse 120, and B.P. 100/70. On the 21st consciousness returned for a few hours, pulse 110. Death occurred on the 22nd.

Dr. A. G. Marshall conducted a necropsy, and I am indebted to him for the report, the positive findings of which were: "Thyroid 34 g. Smooth encapsulated surface. Cut surface showed excess colloid with no adenomata. One or two very small haemorrhages present. Lungs: gross emphysema with severe engorgement. Pericardial effusion (about 3 ml. clear yellow). Heart: no dilatation, no hypertrophy. Mitral valve moderately distorted, with thickened chordae tendineae, and clusters of small fibrinous vegetations on it. Other valves normal. Myocardium normal. Coronary arteries: double orifice of left. No atherosclerosis. Aorta: slight sclerosis. Stomach: engorged, containing altered blood. Liver: severely engorged. Adrenals: right, 6 g. normal; left, 20 g. Sections from the left adrenal show it to be composed of cords and columns of polyhedral cells with much cytoplasm and small nuclei. It is highly vascular, but there are no features to suggest malignancy. The appearance is highly reminiscent of the adrenal medulla. Unfortunately, as the tumour was unsuspected, no fixative agent containing chrome salts was available, and specific staining has failed."

DISCUSSION

In one of two cases of phaeochromocytoma recorded by McCullagh and Engel (1942) a similar error of diagnosis was made and death took place shortly after thyroidectomy. In this case the diagnosis of thyrotoxicosis seemed to be further supported by the presence of a thyroid adenoma. Those authors observed that the B.M.R. may be significantly raised in hyperadrenalism.

Espersen and Dahl-Iversen (1946) describe another case in which a diagnosis of thyrotoxicosis led to treatment with methyl thiouracil. This was followed by improvement, but was discontinued because of toxic manifestations. Subsequent thyroidectomy led to a recession of symptoms for five months, when further attacks of hyperadrenalism suggested the correct diagnosis of phaeochromocytoma, which was successfully treated by adrenalectomy.

In view of the similarity of the two syndromes it is surprising that this diagnostic pitfall has not been more widely published. Clinically the two conditions share many features. Of the symptoms [*sic*] of phaeochromocytoma Hartman and Brownell (1949) include "palpitations, hypertension, headache, nausea, pallor, perspiration, agitation, anxiety, tremulousness, dizziness, dyspnoea, pain (precordial or epigastric, or of the extremities), vomiting, glycosuria, and sometimes prostration." In the case here reported there was considerable weight loss but no loss of appetite.

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