would seem—as the Table would suggest—that this syndrome is becoming more frequent.

We thank Dr. I. S. Buchanan and Dr. S. C. Melnick for their permission to publish and for their criticism; also Dr. D. D. Gibbs for his help in writing the report. Mr. W. B. Yeoman, biochemist, performed the serum lipase estimation.

J. C. ROBERTSON,* M.B., B.S., D.C.H.,
Senior House Officer in Medicine.

G. H. EELES, M.B., B.S., M.R.C.PATH.,
Senior Registrar in Pathology.

Dudley Road Hospital,
Birmingham 18.

* Now Registrar in Medicine, Good Hope Hospital, Sutton Coldfield, Warwickshire.

REFERENCES

Auger, C. (1947). *Archives of Pathology*, **43**, 400.
Bell, E. T. (1957). *American Journal of Pathology*, **33**, 499.
Belsky, H., and Cornell, N. W. (1955). *Annals of Surgery*, **141**, 556.
Berner, P. (1908). *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin*, **193**, 510.
Bockus, H. L. (1965). *Gastroenterology*, vol. 3, 2nd ed. Philadelphia, Saunders.
Gullick, H. D. (1959). *Medicine*, **38**, 47.
Hegler, C., and Wohwill, F. (1930). *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin*, **274**, 784.
Jackson, S. H., Savidge, R. S., Stein, L., and Varley, H. (1952). *Lancet*, **2**, 962.
MacMahon, H. E., Brown, P. A., and Shen, E. M. (1965). *Gastroenterology*, **49**, 555.
Osborne, R. R. (1950). *Archives of Internal Medicine*, **85**, 933.
Schmid, M. (1957). *Zeitschrift für klinische Medizin*, **154**, 439.
Titone, M. (1936). *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin*, **297**, 416.

Houssay Phenomenon in a Diabetic

British Medical Journal, 1970, 2, 709

We believe the following case to be an example of the Houssay phenomenon caused by a pituitary neoplasm which underwent resorption, leaving the sella turcica empty.

CASE REPORT

A 49-year-old man had been diabetic for 10 years before his first admission to hospital in 1966. Initially the diabetes had been controlled with 20 units of insulin daily, but since 1960 he had had many episodes of hypoglycaemia in spite of reduction of dosage to 12 units. On admission to Limerick Regional Hospital with a history of vomiting, drowsiness, and disorientation, he was confused and dehydrated. The systolic blood pressure was 70 mm.Hg and the axillary temperature 94° F. (34·4° C.). A clinical diagnosis of hypopituitary coma was made on the basis of findings such as smooth depigmented skin, scanty pubic and axillary hair, and testicular atrophy. Optic disc pallor on the left suggested a space-occupying lesion. Serum sodium and chlorides were low. Blood sugar was 70 mg./100 ml. Intravenous fluids with hydrocortisone produced improvement. *X*-ray examination showed an enlarged sella turcica and visual field examination a left temporal scotoma. In view of the acute onset, replacement therapy was begun with cortisone and thyroxine. The diabetes was controlled with 32 units of insulin daily.

On his referral to the Mater Misericordiae Hospital the clinical findings were confirmed. Diabetes mellitus had developed at the age of 39. Four years later hypoglycaemic symptoms were observed, beard growth had decreased, and there was lessening of libido and reduction of nocturnal emissions. There had been no headaches, intolerance to cold, lethargy, or enlargement of the extremities. Fundoscopy showed a normal disc on the right with pigmentary changes at the macula. There was grade 2 pallor of the left disc, with choroidal vascular sclerosis and slight macular degeneration. Perimetry showed loss of the left temporal field.

Treatment was withdrawn and investigations (see Table) confirmed the diagnosis of hypopituitarism. Urinary follicle-stimulating hormone was less than 0·48 mg./24 hours. Skull *x*-ray film showed an enlarged pituitary fossa, with erosion of the floor and dorsum sellae. Biochemical studies showed nothing abnormal. There was a mild normochromic anaemia. The E.C.G. revealed low-voltage complexes and T-wave changes consistent with hypothyroidism. Diabetes insipidus was excluded. Therapy was resumed and the patient was assessed for exploratory craniotomy. Angiographic and air encephalographic studies were inconclusive. At operation the right optic nerve was visualized running horizontally to a prefixed chiasma. The left optic nerve was not raised. There was no suprasellar extension of a tumour. The diaphragma sellae was incised and the pituitary fossa was found to be empty. A biopsy specimen of the floor of the fossa was taken. Histological examination showed areas of haemorrhage, with a small zone of pituitary cells arranged haphazardly and not conforming to a tumour pattern. The patient was discharged on substitution therapy and 24 units of insulin daily.

Results of Investigations

	Basal Value	After 4 Days' Tetracosactrin 50 U. Intramuscularly	After 2 Days' Metyrapone 4·5 g./Day By Mouth	After 3 Days' Thyroid Stimulating Hormone
Thyroid function:				
Protein-bound ^{131}I ..	1·9 µg.			3·3 µg.
24-hour uptake of ^{131}I	19%			29%
Adrenal function:				
Plasma cortisol at:				
08.00 hours	1 µg.	10·6 µg.	1·4 µg.	
24.00 hours	1 µg.	4·0 µg.	1·0 µg.	

When seen three months later he felt better. Physical examination gave the same results, though visual field estimation suggested improvement in the left temporal field defect and this was confirmed by perimetry. The E.C.G. had reverted to normal and the anaemia had resolved spontaneously. There was slight improvement in the visual field defects from this time, possibly due to the improvement in general well-being. When last seen (June 1969) there was no further increase in the size of the sella.

COMMENT

Pituitary necrosis of unknown aetiology occurs more often in diabetics than in the normal population (Williams, 1952) and is probably of vascular origin (Frey, 1959). It is the commonest cause of spontaneous Houssay phenomenon, whereas destruction of the pituitary gland by a tumour with amelioration of the diabetic state is rare. In this case the long history of hypoglycaemic episodes in conjunction with concomitant failure of secondary sexual characteristics suggests that hypopituitarism developed gradually owing to compression of the gland by tumour. Repeated minor episodes of infarction of this tumour may have occurred before the final acute episode.

We wish to thank Dr. J. Noel and Dr. M. S. Honan, of Limerick Regional Hospital, who referred the patient; Mr. P. Carey, who performed the exploratory craniotomy; Mr. Alan Mooney, who carried out the fundoscopic and perimetric studies; and Dr. P. J. Bofin, who reported on the histology.

M. I. DRURY, M.D., F.R.C.P.I.
S. O'LOUGHLIN,* M.B., M.R.C.P.I.
E. SWEENEY, M.B.

Mater Misericordiae Hospital, Dublin, Eire.

* Now at the Mayo Clinic, Rochester, Minnesota, U.S.A.

REFERENCES

Frey, H. M. (1959). *Journal of Clinical Endocrinology and Metabolism*, **19**, 1642.
Williams, F. W. (1952). *Diabetes*, **1**, 37.