South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 3 August 1957
Weekly 2s. 6d.
Vol. 31 No. 31
Kaapstad, 3 Augustus 1957
Weekliks 2s. 6d.

GENERAL PRACTICE SERIES

THE PITUITARY GLAND IN DISEASE*

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It is not yet certain how many hormones are secreted by the pituitary gland. Among the more important secretions—from a clinical point-of-view—are:

1. *Adrenocorticotrophic Hormone (Corticotrophin, ACTH)*

This stimulates the adrenal cortex, but *not* the medulla.

2. *Thyrotrophic Hormone (Thyroid-stimulating Hormone, TSH)*

This promotes the formation of thyroid hormone from iodine by the thyroid gland, and its secretion into the circulation.

3. *Gonadotrophins*

These consist of: (i) Follicle-stimulating hormone (FSH) (ii) Luteinizing hormone (LH), and (iii) Prolactin (lactogenic hormone, luteotrophin, mammotrophin).

FSH stimulates ovarian follicular growth mainly; LH is chiefly responsible for oestrogen and progesterone secretion by the ovary; prolactin has an action auxiliary to that of LH in promoting function of the corpus luteum and is the chief hormone concerned with lactation.

These hormones have analagous effects in the male. Thus FSH stimulates spermatogenesis by the testicular tubules, whereas LH—known as interstitial-cell-stimulating hormone (ICSH) in males—promotes androgen secretion.

4. *Somatotrophin (Growth Hormone)*

This is important in the regulation of the rate of body growth and its ultimate size. Its effects are manifested throughout the body. It plays an important role in the metabolism of protein, fat, carbohydrate, water and minerals.

5. *Vasopressin (Antidiuretic Hormone, ADH)*

This hormone of the posterior pituitary gland is concerned primarily with water metabolism.

DISORDERS OF THE PITUITARY GLAND

Disorders of the pituitary gland may arise from:

1. *Non-functioning Tumours*

These cause 'neighbourhood' pressure effects particularly involving the optic chiasma or tracts, classically producing bitemporal hemianopsia.

2. *Hyperfunction*

The effects vary according to the type of hormone produced in excess:

(i) *ACTH* acting through the adrenal glands may produce Cushing's syndrome in some patients.

(ii) *TSH*—possibly responsible for some cases of thyrotoxicosis by stimulating the thyroid excessively.

(iii) *Growth hormone*—responsible for gigantism (if a growing individual is affected) or acromegaly (in a fully-grown adult). This is the only clear-cut example of pituitary-gland hyperfunction, neither of the other states being accompanied by enlargement of the pituitary fossa.

3. *Hypofunction*

This usually affects all the 'trophic' hormones. Pituitary deficiency states include:

(i) *Panhypopituitarism* (Simmond's disease, Sheehan's syndrome).

(ii) *Pituitary dwarfism*—resulting from the rare state of hypopituitarism in a growing child.

* This is the 3rd article in a series of 5 on endocrine disturbances by the same authors which are appearing weekly.

(iii) *Diabetes insipidus*—where the posterior pituitary gland is inactive.

SOME COMMON CLINICAL ERRORS

1. *Cushing's Syndrome*

Previously called 'pituitary basophilism', this condition was once uniformly attributed to a basophil adenoma of the pituitary gland. Present concepts incriminate the adrenal glands, although cases due to adrenal hyperplasia possibly are mediated by excessive ACTH production at some time. Therapy is directed, therefore, toward the adrenal glands in most cases. This condition is not common and will be considered in a subsequent article on the adrenal glands.

2. *Obesity*

Obesity always results from a calorie intake excessive for the metabolic needs of the patient. This is *not* the result of of 'glandular' dysfunction. In particular, recent thought has tended to cast discredit on the concept of 'Fröhlich's syndrome'. This designation should be reserved for patients with obesity and true hypopituitarism due to a recognizable hypothalamic lesion—a very rare state of affairs indeed. Obese children of the 'Fröhlich's' type need calorie restriction, *not* hormonal therapy.

3. *Pituitary Dwarfism*

This is another overworked diagnosis. When general metabolic disorders and bone dysplasias have been excluded, a small core of endocrine diseases remains as a cause of dwarfism. The majority of these appear to be primarily hypogonadal. Cases of dwarfism require specialized investigation.

4. *Panhypopituitarism*

Most of the confusion about this condition has arisen from the equation of Simmond's disease with 'pituitary cachexia'. It is realized today that the great majority of these patients do not present a cachectic picture. Cases of anorexia nervosa are often mistakenly diagnosed as having pituitary hypofunction. Extensive investigations may be required to distinguish these two conditions, as secondary endocrine-gland suppression frequently accompanies anorexia nervosa.

Clinical points which distinguish anorexia nervosa from the unusual thin patient with pituitary failure are:

(i) Despite considerable emaciation, patients with anorexia nervosa exhibit restlessness and often cheerfulness; hypopituitary cases are generally inert and apathetic.

(ii) Retention of sex hair is characteristic of anorexia nervosa; in addition these patients often show fine downy hair-growth over the face and back (as seen in other states of inanition). In hypopituitarism loss of sex hair is a cardinal feature.

In hypopituitarism, as a result of diminished TSH stimulation, the production of thyroid hormone is deficient. The majority of patients with hypopituitarism thus present a hypothyroid picture—so-called 'pituitary' or 'secondary myxoedema'. These patients are as a rule well-nourished or slightly overweight.

The commonest etiology is necrosis of the pituitary following postpartum haemorrhage or severe shock. These patients almost invariably do not lactate. Amenorrhoea, loss of libido and sex hair, and hypothyroid features, complete the diagnosis.

It is important to exclude a pituitary cause in cases of myxoedema, as treatment with thyroid alone is unlikely to succeed and may be hazardous. Hypopituitarism often presents a picture of insiduous decline in health with loss of libido and physical and mental torpor as its only features. Occasionally it is the cause of an obscure 'refractory' anaemia.

5. *Diabetes Insipidus*

This is a rare cause of polyuria and polydipsia. The basic defect is an inability to concentrate the urine, and polyuria is obligatory in order to rid the body of its excretory products. As a secondary effect polydipsia occurs. Other conditions which may simulate diabetes insipidus are:

(i) Psychogenic polydipsia—where the patients drink excessive amounts of fluid from habit or because they feel that it is good for them.

(ii) Renal disease.

(iii) Diabetes mellitus.

The second and third conditions are not difficult to distinguish. A simple test to exclude psychogenic polydipsia is a water-deprivation test. Patients with true diabetes insipidus will seldom tolerate this test and will not produce a concentrated urine. Psychogenic polyuria manifests itself by concentration of the urine—usually within 6-12 hours of water deprivation. An early-morning urinary specific gravity will often suffice to distinguish the two conditions. Specialized tests are available in difficult cases.