Abnormalities of form and function of the human breast. By G. L. Foss. *The Endocrine Clinics, United Bristol Hospitals*

Gynaecomastia and galactorrhoea were discussed. A simple clinical test was described for differentiating gynaecomastia from the pseudo-condition.

The incidence of gynaecomastia in American wartime hospitals was seven to sixteen cases per 100,000 admissions, and it was unilateral in 87% and bilateral in 13%. It may be associated with pigmentation. After prolonged administration of synthetic oestrogens for carcinoma of prostate, there is considerable pigmentation and keratinization of nipples and areolae which in the course of treatment disappears. With the co-existent action of prolactin and somatotrophin, oestrogens cause growth of nipple and areola and proliferation of the ducts of the male mammary glands.

The cause of spontaneous gynaecomastia is reasonably explained by a sensitive end-organ response of one or both breasts to small amounts of endogenous oestrogens. Following administration of androgens to eunuchoids, it may be caused by breakdown in the body to oestrogens.

Chorionic gonadotrophins probably stimulate production of oestrogens from Leydig cells and possibly the adrenal cortex.

Gynaecomastia is seen clinically after administration of oestrogens to males for various conditions such as carcinoma of prostate and acne.

It may occur spontaneously in newborn infants and in 90% of adolescent males. Hormone assays are inconclusive and inconsistent.

Gynaecomastia is seen in a wide range of endocrine conditions. It is particularly associated with testicular disorders—tumours, surgical removal or disease and gonadal syndromes such as Klinefelter's or those named after McCullagh, Beck & Schaffenburg [1953] and Sohval & Soffer [1953]. There may be a familial incidence.

Gynaecomastia also occurs in some cases of adrenocortical hyperplasia, in male pseudo-hermaphrodites, associated with pituitary tumours and thyrotoxicosis and diabetes. Another group is associated with hepatic disease owing to deficient inactivation of oestrogens by the liver, which seems to be dependent on an adequate amount of protein and vitamin B complex.

Gynaecomastia was seen after re-feeding starved prisoners of war.

In addition, male breast development has been reported in a wide range of chronic diseases affecting most of the systems of the body, either during the course of the disease or later during convalescence, and it may be a premonitory sign.

Although this condition is usually physiological before the age of 25 years, after this age—provided that hormone administration can be excluded—it is an important clinical finding which demands a full and searching investigation.

Galactorrhoea has been known from the earliest times [see Foss & Short, 1951], and lactation has been prolonged for many years by repeated suckling. It is recognized now that galactorrhoea is associated with varying degrees of oestrogen deficiency and genital and ovarian atrophy.

Since Ahumada & del Castillo [1932] found absent gonadotrophins in such a case, there have been numerous reports of cases of galactorrhoea and amenorrhoea in which FSH has been low or absent.

Forbes, Henneman, Griswold & Albright [1954] described fifteen non-acromegalic cases of this syndrome, in eight of whom there was evidence of a pituitary tumour.

Nine cases seen in Bristol in 9 years are briefly described and include a virgin, a nulliparous married woman, two examples of Chiari-Frommel syndrome, a woman who has lactated for 20 years through five pregnancies, a woman who developed signs of cerebral tumour 4 years after galactorrhoea started, a unilateral case starting 3 years after a breast abscess, an acromegalic and a patient with evidence of Simmond's syndrome.

Of these nine cases, four have a lesion in the brain or hypophysio-hypothalamic region.

In the reported cases there is a strong incidence of psychotic disturbances.

Amenorrhoea may be primary or secondary according to time of onset, and may or may not precede the onset of galactorrhoea. Varying degrees of atrophy of the genital tract are found. The quantity of milk secreted is very variable and is not influenced by hormone therapy. Menstruation may be induced by cyclical oestrogen treatment or FSH therapy.

The important point is the frequency of pathological lesions in the hypophysio-hypothalamic region or in the brain. Various theories are discussed and further support to a hypothalamic origin is suggested by the report of galactorrhoea occurring in a number of psychotic women after chlorpromazine hydrochloride.

The finding of galactorrhoea requires a full investigation to exclude a pathological lesion in the brain or pituitary area.

Based on the theories of lactogenesis and stimulated by the success of Lyons, Li, Johnson & Cole [1955], who succeeded in producing lactation in male rats, an attempt was made to initiate lactogenesis in a male transvestist.

Six years ago this patient had been given oestrogens. Both testes and penis were then removed and an artificial vagina was constructed by plastic surgery.

The patient was implanted with 500 mg oestradiol in September 1954, and 600 mg in July 1955. The breasts were then developed more intensively with daily injections of oestradiol dipropionate and progesterone for 6 weeks. Immediately following withdrawal of this treatment, prolactin 22·9 mg was injected daily for 3 days without effect.

After a second month on oestradiol and progesterone daily, combined injections of prolactin and somatotrophin were given for 4 days and suction was applied by a breast pump—four times daily. On the 4th and 5th days a few drops of colostrum were expressed from the right nipple.

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