Technique

Naturally the technique of preparation and administration affects results and therefore bare details are given below :

(*a*) The solution and all apparatus must be pyrogen-free and administration must be by a closed method, *i.e.* not by tube and funnel.

(*b*) The intravenous needle is inserted into a vein without incision.

(*c*) Veins in the forearm are used, not in the cubital fossa, and the forearm is supported in a vertical position against the stand by the bedside from which the saline flask hangs.

Summary

Physiological and clinical facts regarding water and salt metabolism are described. ½ N 5 per cent glucose saline is considered to be a desirable solution for almost all kinds of cases requiring intravenous administration of these substances.

The details regarding fluid and salt balance in 36 cases on intravenous therapy with ½ N 5 per cent glucose saline have been studied and illustrative cases are recorded. From a study of these records it is revealed that provided ½ N 5 per cent glucose saline is used estimations of plasma or urine chloride are unnecessary. Only the urine volume, which depends on alimentary intake and output, intravenous intake and other subsidiary factors, need be recorded and studied with a view to estimation of intravenous requirements for the ensuing 24 hours.

A simple table for the estimation of the amount of ½ N 5 per cent glucose saline required when the urinary volume is at various levels is appended.

Intravenous administration of salt is contra-indicated in œdematous, thirsty patients and is undesirable in cases of sulphonamide hæmaturia or anuria.

In cholera it is not certain whether ½ N 5 per cent glucose saline is a satisfactory solution. In all other cases it is recommended that this solution should be administered as a routine.

I have pleasure in thanking Dr. W. B. Roantree, Chief Medical Officer, Kolar Gold Field, for his encouragement and advice, Dr. J. M. Daly for permission to include some of his cases, and Messrs. John Taylor and Sons (India), Ltd., the Managers, for permission for reporting.

REFERENCES

EDWARDS, H. C. (1948). *Recent Advances in Surgery.* J. and A. Churchill Ltd., London.

MARRIOTT, H. L. (1947). *Brit. Med. J., i,* 245, 285, 328.

MARTINDALE (1941) .. *The Extra Pharmacopœia.* The Pharmaceutical Press, London.

WRIGHT, S. (1938) .. *Applied Physiology.* Oxford University Press, London.

SQUAMOUS METAPLASIA AND KERATINIZATION IN CYSTIC HYPERPLASIA OF THE HUMAN BREAST

By C. MOHAN RANGAM, M.D.

From the Department of Pathology, St. James's Hospital, Leeds

THE histological appearances in cystic hyperplasia of the breast, the condition still commonly referred to as 'chronic mastitis', have been widely discussed. This case is reported to record a rather unusual degree of intraduct squamous metaplasia and keratinization.

Case history.—An unmarried woman of 40 years had noticed, ten months prior to admission into hospital, a small 'gathering' on the areola of the right breast discharging pus. After a course of diathermy, there was a blood-stained discharge. Clinical examination revealed bilateral 'chronic mastitis' with a small mobile sub-areolar swelling in the right breast. Small soft lymph nodes were palpable in both axillæ. A simple mastectomy was performed, and the patient made a good recovery.

The specimen received in the laboratory was a rather fat breast, showing slight retraction of the nipple and a sub-areolar, firm, yellowish, spherical nodule, an inch in diameter. Pieces taken for histology included the nipple, the nodule and the underlying breast tissue which looked normal.

Histology.—All the sections studied showed features of cystic hyperplasia (figures 1, 2 and 3, plate I). Some of the larger and many of the subsidiary ducts showed considerable dilatation, often frankly cystic, the desquamation, partial or complete, to hyperplasia. One of the most striking features was the complete replacement of the normal epithelium in some of the dilated ducts by stratified squamous epithelium, several cells in thickness, with keratinization at the surface. Laminated pink-staining material (H. & E.), the outer laminæ consisting of keratin with some cellular débris, was seen in the cysts. Where the lining epithelium was desquamated, giant cells of foreign body type, with as many as 50 nuclei, were found along the inner surface of the cyst wall and in juxtaposition with the laminated material. The periductal connective tissue showed collections of lymphocytes and histiocytes with occasional polymorphs, though in some places plasma cells were prominent. In parts, the cells had infiltrated the entire thickness of the duct wall, reaching the epithelial lining. Similar cell collections were noticed in the interstitial tissue of the breast and in relation to normal ducts. The deeper parts of the sections presented features of intra-canalicular fibro-adenoma. Sections stained Gram, and treated with acid alcohol, showed varying amounts of keratin in the laminated material.

The condition was diagnosed as cystic hyperplasia with unusual features, and since an amputation had already been done, no further treatment was considered necessary.

Discussion

Epithelial metaplasia occurs in neoplastic as well as in non-neoplastic conditions. No specific causative factor is recognizable in the former, but in the latter, chronic inflammation, nutritional impairment and alteration in the function of the organ concerned are amongst the causes. It is also observed in conditions of disordered hormonal control. Squamous metaplasia in the glands is a frequent finding in endometrial cystic hyperplasia, the result of persistent œstrogenic over-stimulation, and in benign glandular hyperplasia of the prostate, particularly in stilboestrol-treated cases. Hyperplasia and metaplasia have been reproduced in experimental animals, in the latter organ, by œstrin and its synthetic substitutes (Burrows, 1935*a*).

In cystic hyperplasia of the breast, a condition generally accepted as resulting from disordered hormonal control, in particular an œstrogen excess, squamous metaplasia seems uncommon. Willis (1948) in his 'Pathology of Tumours' mentions that it is very rare in the breast, save in carcinomatous tumours, and observes that even here, it is rarer than might be expected, in view of the development of the organ from the ectoderm. Though changes in the human breast associated with cystic hyperplasia have been experimentally induced in animals by œstrogen, squamous metaplasia has not been observed frequently. Bonser (1945) saw it only on rare occasions in œstrogen-treated mice, but Kirschbaum *et al.* (1946) observed it constantly in mice of certain genetic types painted with methyl-cholanthrene.

The rarity of squamous metaplasia, in the relatively common cystic hyperplasia of the human breast, renders it necessary to seek an explanation, when it does occur, in factors other than hormonal. Retention of secretions with an alteration in their character would be a possible cause, but this would not be consistent with the rarity of metaplasia. Two factors, (*a*) infection and (*b*) deficiency of vitamin A, deserve consideration.

Squamous metaplasia of epithelial linings following suppuration is well known, the change in infected bronchiectatic cavities being a typical example. In the present case, the clinical history of a 'gathering' discharging pus would point to a suppurative process. Unfortunately, the discharge was not cultured for organisms. On microscopic examination, there was no active inflammation. The presence of lymphocytes and histiocytes, described in the histological report, is a common finding in cystic hyperplasia and does not necessarily denote inflammation at any stage, nor can the cellular infiltration in the walls of the ducts, sometimes reaching the epithelial lining, be interpreted as due to pre-metaplastic inflammation. On the other hand, leucocytic infiltration seems to be an accompaniment of squamous metaplasia and keratinization (Burrows, 1935*b*).

Clinical and experimental evidence have made it clear that the fat-soluble vitamin A is necessary for the preservation of epithelial integrity. Widespread epithelial atrophy, followed by squamous metaplasia and keratinization, have been observed in animals maintained on vitamin A deficient diets (Wolbach and Howe, 1926), and in children suffering from similar deficiency (Blackfan and Wolbach, 1933). The findings in mice receiving œstrogen are even more significant. Removal of vitamin A from their diet hastened squamous metaplasia and keratinization in the coagulating gland and increased the degree of keratinization to a remarkable extent (Bonser, 1935). It would appear that the two factors act synergistically, and it is very likely that deficiency of vitamin A can induce squamous metaplasia in the human breast already under the influence of œstrogen excess.

Summary

A case of cystic hyperplasia with an unusual degree of intraduct squamous metaplasia is reported.

The known causes of squamous metaplasia are discussed.

It is suggested that deficiency of vitamin A can induce squamous metaplasia in the human breast already under the influence of œstrogen excess.

I am grateful to Dr. W. Goldie and Dr. G. M. Bonser for their advice. My thanks are due to Mr. J. Hainsworth for the photomicrographs.

REFERENCES

BLACKFAN, K. D., and WOLBACH, S. B. (1933). *J. Pediat.*, **3**, 679.
BONSER, G. M. (1935) .. *J. Path. and Bact.*, **41**, 217.
Idem (1945) .. *Ibid.*, **57**, 413.
BURROWS, H. (1935*a*) .. *Ibid.*, **41**, 43.
Idem (1935*b*) .. *Ibid.*, **41**, 423.
KIRSCHBAUM, A., WILLIAMS, W. L., and BITTNER, J. J. (1946). *Cancer Res.*, **6**, 354.
WILLIS, R. A. (1948) .. *Pathology of Tumours.* J. and A. Churchill Ltd., London.
WOLBACH, S. B., and HOWE, P. R. (1926). *Arch. Path.*, **1**, 1.

PULMONARY MONILIASIS

By KHUSHDEVA SINGH, M.B., B.S., F.C.C.P., T.D.D.
MAJOR
Medical Superintendent, Hardinge Sanatorium, Dharampore (Simla Hills)

FOR many years mycotic diseases of the lungs have been regarded as rare infections which were of interest to the specialists of high order

PLATE I

SQUAMOUS METAPLASIA AND KERATINIZATION IN CYSTIC HYPERPLASIA OF THE HUMAN BREAST : C. MOHAN RANGAM. (O. A.) PAGE 9

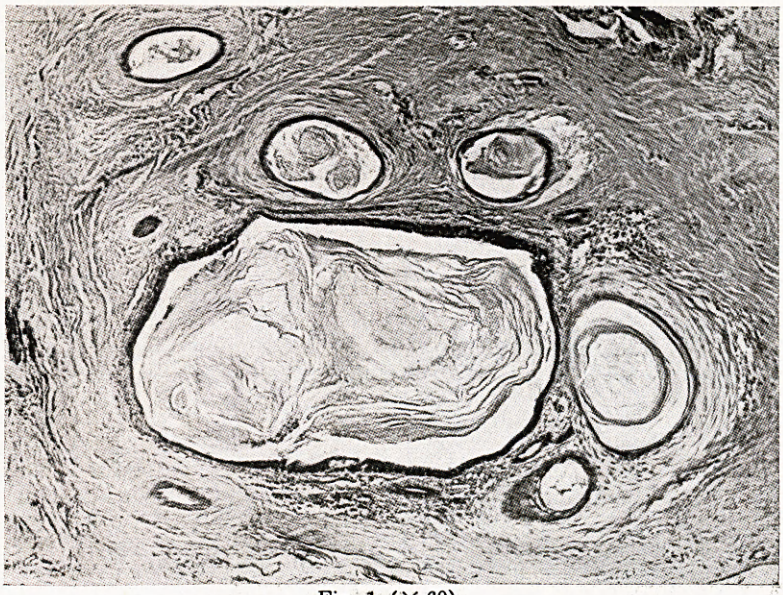

Fig. 1 (× 60).

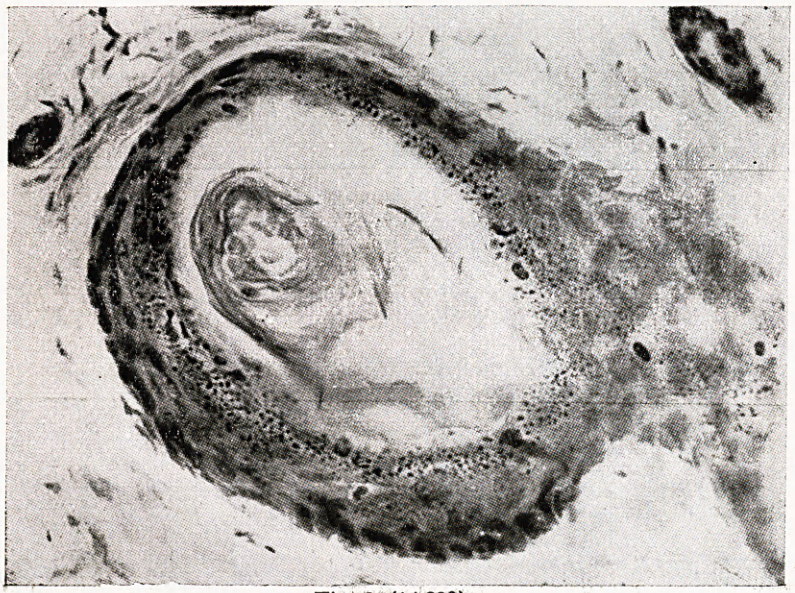

Fig. 2 (× 320).

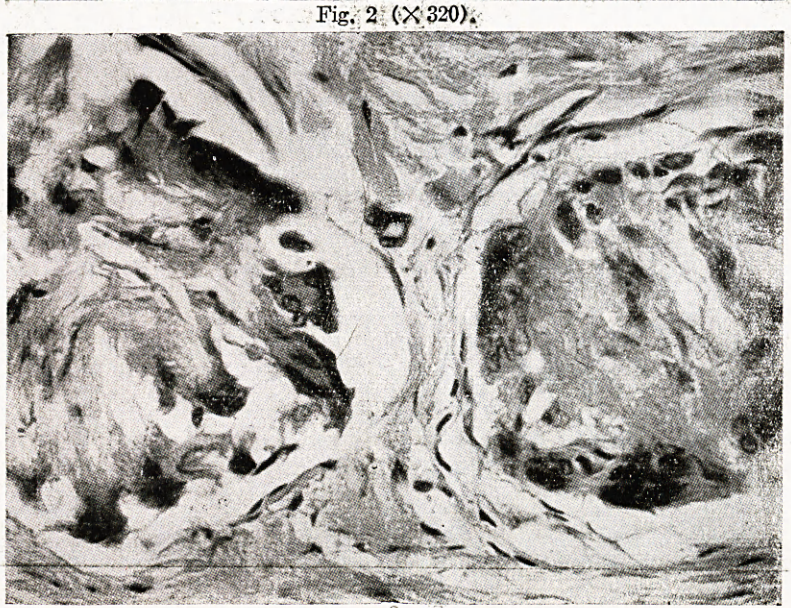

Fig. 3 (× 320).