condition of the patient, the deepest part of the cyst wall cannot be removed, then drainage through a lumbar incision presents no difficulty.

BIBLIOGRAPHY.

- Doran: *Proc. Roy. Soc. Med.*, 1907-8, i, Surg., p. 201.
Hartwell: *Annals of Surgery*, 1909, xlix, p. 125.
Hedinger: *Ztschr. f. Path.*, Frankfurt, 1911, vii, p. 112.
Pacinatti: *Gazz. d. Osped.*, Milano, 1908, xxix, p. 847.
Potherat et Chambard: *Bull. et Mém. Soc. de Chir. de Paris*, 1909, p. 1187.
Rolleston: *Trans. Path. Soc. Lond.*, 1898-99, i, p. 214.
Pearse: *Trans. West. Surg. Assoc., Minneapolis*, 1916, xxvi, p. 329.
Major and Black: *Amer. Journ. Med. Sci.*, 1918, clvi, p. 459.
Ausenda: *Osp. Maggiore, Milano*, 1919, vii, p. 191.

EARLY AND CURABLE DISEASE OF THE BREAST.

BY

SIR G. LENTHAL CHEATLE, K.C.B., C.V.O., F.R.C.S.,
SURGEON AND LECTURER IN SURGERY, KING'S COLLEGE HOSPITAL.
(With Special Plate.)

SINCE the publication of my article on the proemial breast in the *BRITISH MEDICAL JOURNAL* of June 3rd, 1922,* I have gained valuable information on the interpretation placed upon it by many of my readers. They have asked whether the breasts of patients whom they have sent to me were in what I termed the proemial state. From them I have been able to select instances of diseased breasts in stages which, under proper treatment, can be saved from becoming examples of the established proemial condition.

The most typical conditions of an early and curable disease are exemplified in one of my own patients. She was 26 years old, single, by occupation a saleswoman. At the time of the consultation her tongue, teeth, tonsils, bowels, and urine were normal. The breast had not been injured by a blow, and her stays were not high enough to touch it in any position which she assumed. The left breast was normal. She had complained of pain in the upper and outer segment of the right breast for three and a half months; the pain was increased at the time of her periods.

Palpation revealed an irregularly swollen duct which could be traced from the ampulla in the areola region to the part towards the periphery, where the gland was more tender than elsewhere. On observation of the nipple three minute raised yellowish dry spots could be seen, situated at the upper and outer margin of the nipple, and I concluded that probably one of them was the exit of the duct which belonged to the affected segment of the breast. I believed them to be points of exit of ducts which were plugged with dry epithelium and excretions, either from the breast or from the sebaceous glands which open into the outlet of the duct (Special Plate, Fig. 1). Acting on this belief I advised the woman to carry her arm in a sling and to put hot fomentations on the breast; but the most important part of my treatment was to make sure that she constantly soaked the nipple with hot sterilized water applied with cotton-wool. This treatment was continued for fourteen days until the plug in the outlet of each duct was macerated enough to enable me to remove it by wiping away the film that covered it, and by gentle pressure to squeeze it out. In two months the breast gradually became normal. I then advised her to keep the nipples clean always, by means of the same process, and to avoid all kinds of soap, powder, methylated spirit, hydrogen peroxide, and so on, for fear of irritating or plugging afresh the outlets. If soap be not properly washed away it dries and cakes and fills the outlet. In addition to this it must be realized that soaps are irritating and some brands more so than others.

My interpretation of the state of affairs in this woman's breast was as follows. The presence of the plug was detrimental in many possible respects—for instance, as an irritant it might have encouraged the local action of non-virulent micro-organisms upon the epithelial lining of the duct outlet, and in consequence the whole duct might have suffered a mild infection, or the products of a mild infection might have been disseminated in the same directions. The observations of Mr. A. J. Walton in 1914 and 1915¹ and those of Dr. A. H. Drew² suggest another possible explanation—that the products of epithelial irritation created by the plug in

the duct outlet caused changes in the epithelium throughout the distribution of the duct and acini. My belief is that the change was probably a desquamative hyperplasia of epithelium which distended this duct and caused the woman pain. I further believe that similar treatment applied to the breast in this early stage of disease will prevent it from becoming an established proemial breast. Thus the prevention of disease in the care of the breast can be taken a step further back than the proemial state.

Many authorities seem able to connect changes in a sebaceous gland with plugging at the outlet of its duct, but do not seem able to connect changes in the breast with a plugged outlet of a mammary duct. I do not understand why there should be this difference, and it is all the more incomprehensible when it is realized that many sebaceous ducts open into the outlets of a mammary duct (Fig. 2), so that any detrimental discharge from these sebaceous glands would open direct into the mammary duct at this point. In addition to this anatomical feature, there is this fact: in a previous article on cysts and primary cancer in cysts of the breast,³ I pointed out the consummate ease with which foreign material is distributed throughout the ducts of the breast when once entrance be gained. Further, to show what a definite pathway for entrance there is, I draw attention to the anatomical section in Fig. 2, which shows a complete duct in the nipple and the outlet on the surface. The duct lumen is seen distinctly throughout its course. The duct widens at its outlet, into which enter the openings of many sebaceous glands. To all intents and purposes each duct system of the breast may be regarded as a separate gland. This notion may create a doubt in the minds of those authorities to whom I have alluded.

It is essential that women should keep these duct outlets free from plugs. I say this for another reason: I have in my possession whole sections of a breast affected with the earliest Paget's disease of the nipple I have seen. Only half the top of the nipple is affected by the disease. There is no carcinoma in the deeper parts of the breast. The main focus of the disease is in a plugged duct outlet, from which the process is spreading into the duct and on to the surface of the nipple. Epithelial cells are invading deeper tissues and have entered lymphatic vessels. It is a typical case of Paget's disease of the nipple, and this section, together with many other reasons, convince me that Paget's disease of the nipple is carcinoma from the beginning. This is another investigation, on the writing of which I am at present engaged. I mention it here to emphasize the importance of keeping duct outlets clean and free from irritating agents.

REFERENCES.

- ¹ Variations in the Growth of Adult Mammalian Tissue in Autogenous and Homogenous Plasma, *Proc. Roy. Soc., B*, vol. 87, 1914. On the Variation in the Growth of Mammalian Tissue *in vitro* according to the Age of the Animal, *Proc. Roy. Soc., B*, vol. 88, 1915. The Artificial Production in Mammalian Plasma of Substances inhibitory to the Growth of Cells, *Journ. Exper. Med.*, August 1st, 1915, vol. xxii. The Technique of Cultivating Adult Animal Tissues *in vitro* and the Characteristics of such Cultivations, *Journ. of Path. and Bact.*, vol. xviii, 1914. On the Survival and Transplantability of Adult Mammalian Tissue in Simple Plasma, *Journ. Exper. Med.*, vol. xix, No. 2, 1914. The Effect of Various Tissue Extracts upon the Growth of Adult Mammalian Cells *in vitro*, *Journ. Exper. Med.*, vol. xx, No. 6, 1914.

² Three Lectures by Dr. A. H. Drew on the Cultivation of Tissues and Tumours *in vitro*, Lectures II and III, *Lancet*, April 28th, 1923.

³ *British Journal of Surgery*, vol. viii, No. 30, 1920, Figure 113.

POLYTHELIA.

BY

R. DOUGLAS HOWAT, L.R.C.P., L.R.C.S. Edn.,
L.R.F.P.S. Glas.

CASES of polymastia (supernumerary breasts) are not uncommon. Goldberger¹ makes mention of some 262 such cases. Williams (Baltimore)² says: "Probably one in every few hundred women has one or more accessory breasts," and points out that the condition is usually regarded as an atavistic reversion, though it is not associated with any increased tendency towards multiple pregnancy. Ahlfeld,³ on the other hand, holds that the distribution of the mammary tissue is to be attributed to the transference at an early period of development, by means of the amnion, of some of the cells which ordinarily go to form the breasts to other

* See also the *British Journal of Surgery*, April, 1923, p. 436.

portions of the body. When polymastia occurs the supernumerary nipples are usually functionless. In numerous instances an hereditary influence has been traced.

Polythelia is a very much rarer abnormality. In fact, with the exception of occasional allusions to the condition, I have been unable to find any report of an authentic case in medical literature. In November, 1921, I reported in this JOURNAL a case of polythelia, in which the accessory nipple functioned; it occurred in my private practice. Since then I have been fortunate in securing the accompanying plate illustrating the case.

The woman, a primipara, aged 23, first recollected noticing the accessory nipple in early childhood and regarded it as a fleshy mole. It is about the size of a sixpenny piece, and is situated close to the left mammillary line in the inferior half of, and towards the periphery of, the left breast. It resembles the normal nipple of the same breast in everything but size, possessing lactiferous ducts and erectile tissue.

Even Montgomery's secondary areola is faithfully reproduced. There is no macroscopical evidence of a supernumerary breast. The right and left breasts are symmetrical except for the small accessory nipple on the latter, and no demarcation of the gland tissue could be distinguished by inspection or by palpation. The condition has caused the woman no discomfort other than that, during the period of lactation subsequent to her pregnancy three years ago, there was a very free secretion of milk from the secondary nipple as well as from the normal nipples, the medical practitioner then attending her having dissuaded her from nursing the child.

There is no evidence of any hereditary influence. The woman, who is one of a family of eleven children, is the only one exhibiting this abnormality.

REFERENCES.

- ¹Goldberger: Ein seltener Fall von Polymastia, *Arch. f. Gyn.*, 1895.
²Whitridge Williams: *Obstetrics*, 1920, p. 987. ³Ahlfeld: Spaltung der Anlage der Brustdrüse, Polymastia, *Die Missbildungen des Menschen*, Leipzig, 1880, 110-113.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

ACUTE OEDEMA OF THE LUNGS.

ACUTE oedema of the lungs is one of the most striking and terrifying emergencies with which the practitioner may be called upon to deal. The most tragically celebrated case on record is that of Charcot himself. During a journey he was seized with a sudden attack of suffocation, expelled a quantity of frothy albuminous fluid, and died in half an hour. Often the sufferer is dead before the doctor gets to him, and is found lying with an abundant frothy pinkish exudation filling the mouth and nose.

On February 23rd, 1923, on the R.M.S.P. *Desna*, I was called hurriedly about midnight to a gentleman taken suddenly ill. During the evening he had been well, dining in the saloon with the rest of us. I did not know then that he had not been in good health, but learnt subsequently that he was suffering from arteriosclerosis, or what I would prefer to call cardio-vascular breakdown.

On reaching the cabin I found him standing up struggling for breath, and bringing up an enormous quantity of frothy soapsudgy expectoration, coughing it up, and brushing it away from his nose and mouth with his fingers. There was no time to make exhaustive inquiries; but his wife said he was 56, and that it was his first attack. The pulse was small and rapid, and owing to the râles in the chest the heart sounds could not be heard. Even during this rapid examination he became very much worse, and it was clear that unless something was done quickly the first attack would also be the last. I ran to the surgery for a knife and a bandage, picking up a steward on the way to help me. On again reaching the cabin the patient had fallen back exhausted but was still conscious. We put the bandage round his right upper arm, and cut across the first vein easy to get at. This was probably the anterior ulnar; the branches of the median are best avoided when in a hurry. He bled freely, the blood being at first very dark, although his face was pallid. I bled him well over a pint, and it was most gratifying to watch the change. The vivid colour and dyspnoea disappeared, the expectoration gradually ceased, and his dreadful anxiety passed off. At the end of the bleeding breathing, colour, voice, mental attitude, and pulse were all reassuring. It was surprising how the pulse improved; it was not necessary to inject camphor. Incidentally, why is camphor in oil so much used, when a saturated solution of camphor in alcohol is so much more convenient? I have used the latter for years and never found any disadvantage, and a much larger dose can in this way be given.

The patient did well afterwards; he was kept rigidly in bed on a spare milk diet until Liverpool was reached.

Neither the diagnosis nor treatment was difficult. As to the former, asthma, cardiac dyspnoea, angina pectoris, and pulmonary infarct might occur to one's mind, but the extraordinarily abundant frothy expectoration, coming on quite suddenly, was decisive. The acute oedema is evidently due to a condition of vascular engorgement in the pulmonary area, and partially draining the systemic veins gives the right ventricle time to deal with it.

VINCENT MOXEY, M.R.C.S., L.R.C.P.,

Southampton.

Surgeon, Royal Mail Steam Packet Company.

SINUS THROMBOSIS FOLLOWING PNEUMONIA
IN AN ADULT.

THE following case appears to be of interest on account of the difficulty presented in the diagnosis, and because the condition is unusual in the adult.

A woman, aged 35, was admitted under Dr. A. Stanley Barnes on April 11th, 1923. A letter from her doctor explained that on March 31st she was taken ill with symptoms referred to the respiratory system, and that when he saw her for the first time, on April 2nd, she had a dry cough and pleuritic pain in the left side. Next day it was evident that she had lobar pneumonia affecting the left base. The crisis occurred on April 7th; on April 9th the temperature, pulse, and respirations were normal and the patient felt so well that she asked to be allowed to get up. During the following night, however, she complained of severe headache and vomited several times; early next morning, while sitting up in bed, she had an epileptiform convulsion which left her with left hemiplegia.

On admission, some twelve hours later, she was in a semi-comatose condition, but could be roused sufficiently to speak a few words. The temperature was 97° F., the pulse 48, and the respirations 20. There was left hemiplegia with an extensor plantar reflex on that side, while the other plantar reflex was flexor and both knee-jerks were somewhat brisk. There was complete incontinence of urine. There were no retinal changes suggesting vascular disease, and except for the bradycardia no abnormality in the circulatory system was detected. The lungs also appeared normal.

A diagnosis of cerebral embolism was made, although it was realized that this would not account for the headache and vomiting prior to the convulsion.

On April 12th, about twenty-four hours after admission, she suddenly became worse. The face was flushed, the breathing stertorous, the coma more profound, and some weakness with an extensor reflex appeared on the right side. Lumbar puncture was performed, and 50 c.cm. of fluid under considerable pressure and deeply tinged with blood were withdrawn. On April 13th the temperature had risen to 100.5°, and lumbar puncture again yielded a blood-stained fluid. It was concluded that haemorrhage had taken place at the site of embolism.

The condition of the patient rapidly grew worse, the temperature fell below normal, the pulse rate rose to 136, and death occurred the same night.

At necropsy the heart (300 grams) was found soft and flabby, and considerable fatty infiltration was present in the muscle of the right ventricle. There was a slight degree of mitral stenosis, but no thrombi were present in the auricles and no vegetations on the valves. The aorta was normal. The lungs were intensely congested and oedematous. There was extensive *ante-mortem* thrombosis in all the dural sinuses and in many of the cortical veins, especially near the vertex. The surface of the brain was intensely congested and a considerable amount of free blood was seen in the meshes of the pia arachnoid. On section large areas of brain substance showed the condition of red softening. The largest, on the right side, involved the optic thalamus, the genu of the internal capsule, the lenticular nucleus, and the caudate nucleus, so extending into the lateral ventricle. A second area, 1 inch in diameter, was situated in the right frontal lobe, while yet a third affected