the C, Pm_2 , and M_2 teeth in the upper jaw; and the I_1 tooth followed by the M_1 tooth, and the C, Pm_1 , M_2 , and Pm_2 teeth in that order in the lower jaw.

The analysis indicates that the times of eruption of teeth with similar mean eruption times on the same side are independent of each other, but that the eruption times of corresponding teeth on the right and left sides are correlated.

We gratefully acknowledge the receipt of a grant from the Medical Research Council, and much assistance and advice from Professor S. Zuckerman and Mr. M. J. R. Healy.

REFERENCES

- Clements, E. M. B., Davies-Thomas, E., and Pickett, K. G. (1953). *British Medical Journal*, 1, 1421.
 Leslie, G. H. (1951). *A Biometrical Study of the Eruption of the Permanent Dentition of New Zealand Children*. Government Printing Office, Wellington.
 Livy, J. (1885). *British Medical Journal*, 2, 241.

MAMMARY ABSCESS

DISADVANTAGES OF ANTIBIOTICS IN ITS MANAGEMENT

BY

C. P. MILLS, F.R.C.S.

(From the Department of Surgery, Postgraduate Medical School of London)

In days gone by the cardinal signs of inflammation, heat, pain, redness, and swelling were all too familiar. Just as the classical picture of lobar pneumonia with its typical crisis is seen no more, so the clinical features of acute pyogenic infections have been greatly altered since the general introduction of antibiotics. No one will deny their immense value in combating what were earlier most severe infections, but in less severe cases they can be a mixed blessing if not used with judgment and restraint. The need for surgery is as great as ever, although its extent may be much restricted. Since antibiotics can mask so effectively many of the arresting features of acute infections, the timing of an operation is becoming more and more important.

A good deal of suffering can, in my opinion, be avoided by a general appreciation of the limitations of antibiotics in the treatment of acute infections, and this point I want to underline by citing the records of four cases of acute mammary abscess. Cases of this sort are no doubt the everyday experience of most active general surgeons, but it is their very frequency which has prompted me to record the following cases.

Case 1

The patient was aged 26. Immediately after a normal delivery her left nipple became sore and cracked, and led to weaning. Two weeks later, 24 days after her confinement, she developed a painful swelling in her left breast. On the twenty-ninth day she was seen by her own doctor, who noticed a painful, red, indurated area in the lower half of the left breast. "Distaquaine penicillin," 400,000 units, was given daily, together with local applications of heat for four days. She was admitted to hospital on the thirty-sixth post-partum day, and 12 days after the onset of symptoms.

The left breast was fuller and more pendulous than the right, which seemed quite normal. There was an obvious swelling in the lower and outer quadrant. Peau d'orange was not seen. The nipple was not retracted, but the lower half of the areola was tethered to a hard, slightly tender non-fluctuant mass, measuring 8 by 8 cm., and easily felt

by the flat of the hand. The skin over the mass was not discoloured and was no hotter than the surrounding breast tissue. There was some fixity of the mass to the underlying pectoral fascia. Glands were not palpable in the axilla.

When she was first seen in hospital the absence of the signs of inflammation and the size, hardness, and fixation of the mass in the breast strongly suggested the diagnosis of a lactation cancer.

On the following day a radial incision was made over the most prominent part of the mass. A very thick-walled multiloculated abscess cavity was entered, which was involving most of the lower half of the breast. Free drainage was established. The pus yielded a profuse growth of a coagulase-positive *Staphylococcus pyogenes*, resistant to penicillin but sensitive to aureomycin, streptomycin, and chloramphenicol. Streptomycin, 0.5 g., was given twice daily for four days. Two weeks later the wound was completely healed, and there was no more than a diminishing thickening. Her total period of disability was 26 days.

Case 2

The patient, aged 32, noticed pain, tenderness, and swelling in her right breast three days after a normal confinement. For the next ten days she was given daily injections of distaquaine penicillin, 300,000 units, and local applications of kaolin poultices. The swelling became less painful but no smaller. On the twenty-fourth post-partum day the baby had to be weaned, when the patient developed a generalized rash. On the thirtieth day her own doctor again prescribed injections of penicillin of the same dosage as previously because the breast and upper arm had become stiff and swollen.

On admission to hospital on the thirty-fifth day—that is, 32 days after the onset of symptoms—her temperature was 98.4° F. (36.9° C.), pulse 80, respirations 20, and white blood cell count 11,000. She was looking in the very best of health. The left breast was normal. The right breast was enlarged and deformed by a large, easily visible swelling in the right lower quadrant. There was no peau d'orange, but a little reddish discoloration and oedema over the centre of the swelling. The upper part of the areola was tethered to the tumour. The mass was hard, smooth, and slightly tender, non-fluctuant, and a little hotter than the surrounding breast tissue. It was fixed to the underlying pectoral fascia. There were three firm, tender glands in the right axilla. There were even fewer signs and symptoms of an acute inflammatory condition in the breast than in Case 1. The patient was a very healthy-looking woman with no evidence of fever or toxæmia, complaining only of a large, slightly tender mass in her right breast.

The fixity of the mass to the skin, areola, and underlying fascia; the oedema of the breast and the upper arm; and the presence of enlarged glands in the axilla were once again very suggestive of a lactation carcinoma. The next day a radial incision was made over the most prominent part of the mass. A great depth of dense inflammatory tissue had to be penetrated to find one small loculus of pus in its centre. Bleeding was profuse and difficult to control. Culture of the pus yielded a profuse growth of *Staph. pyogenes* resistant to penicillin but sensitive to streptomycin, aureomycin, and chloramphenicol.

A section from the abscess wall showed an inflammatory reaction with infiltration by foamy macrophages, plasma cells, and polymorphonuclear cells. There was an area of fat necrosis.

Streptomycin, 1 g. twice daily, was given for six days. She was discharged on the sixteenth post-operative day with a clean granulating wound but still some residual thickening in the breast.

Case 3

This patient, aged 28, was confined five weeks before coming to hospital, and complained of pain in the left breast for two days immediately prior to admission.

The left breast was diffusely swollen and tender. There was no discoloration or oedema. After eight days of distaquaine penicillin, 300,000 units daily, the breast was indurated in the left upper and outer quadrant, but less tender and still non-fluctuant.

Chloramphenicol, 0.5 g. four times daily, was given for seven days, by which time fluctuation had developed. Exploration revealed a multiloculated breast abscess with a thick and densely hard wall. Brisk bleeding was encountered, and this could be controlled only by packing. Six daily doses of 300,000 units of distaquaine penicillin were then given, the wound being dressed every third day. The patient was discharged on the thirty-fifth day after the onset of symptoms. She returned, however, one month later with a recurrence of symptoms. Distaquaine penicillin, 600,000 units daily, was given for seven days, and then streptomycin, 1 g. a day for a further seven days. The old incision was then reopened, and pus encountered which grew a penicillin-resistant coagulase-positive *Staph. pyogenes*. The patient was discharged with the wound well healed 54 days after the recurrence of symptoms. In all, she had been attending hospital 116 days, with a brief respite in the middle, after first being seen with a simple acute breast abscess.

Case 4

This patient, aged 32, complained of pain and swelling in the right breast on the thirty-first day after her confinement, and came to hospital four days later.

The left breast appeared normal. The right breast was red and swollen, with a very tender, smooth lump measuring 5 by 5 cm. just below the areola.

Stilboestrol, 10 mg. thrice daily, was given for five days, and 5 mg. for a further ten days. At the same time a dose of 600,000 units of distaquaine penicillin was given daily until the tenth day after the onset of symptoms. The lump was then still the same size but distinctly less tender.

On the thirteenth day a large breast abscess was drained. Daily injections of 600,000 units of distaquaine penicillin were continued until the nineteenth day, when the wound was still discharging and a new area of induration and tenderness had appeared above the areola. Two days later a second thick-walled abscess was drained of approximately 5 ml. of pus, which again yielded a penicillin-resistant *Staph. pyogenes*. After a further 17 days the wound was well healed. The patient took 34 days to recover from an acute breast abscess.

Discussion

Cases 1 and 2 serve to illustrate how the symptoms and signs of an acute breast abscess, by prolonged penicillin therapy, can be masked to such an extent that the condition may be difficult to differentiate from a lactation carcinoma.

Cases 3 and 4 illustrate well how an abscess can be so attenuated by antibiotics that the need for early incision is not appreciated and an undue amount of disability results. Perhaps it might be said that all that these last two case histories make evident is a lamentable lack of judgment in timing a surgical excision. This I freely admit, but claim in extenuation that the giving of antibiotics so relieved symptoms, and so modified the local tissue reaction, that the necessity for incision was less evident. Further, I claim to be not alone in making these mistakes.

Lake (1952) makes it quite clear that there is a growing awareness of the dangers and drawbacks of the indiscriminate use of the antibiotics. My experience in the casualty department of a large general hospital has certainly made me acutely aware of their obvious limitations. Used early and vigorously, they undoubtedly abort completely what were previously the most severe and disabling infections; one can confidently expect prompt resolution with complete

restoration of function. When, however, their administration is not begun until the infection is firmly established, complete resolution unaided is most unlikely. Gratifying as may be the alleviation of symptoms and the abatement of the toxæmia, timely surgical incision to drain what may be only a small collection of sterile pus is no less necessary than it was in earlier days. Antibiotics in many instances render surgical intervention no less necessary, though they may greatly modify its extent. The relief of pain which their use affords makes it only too easy to defer a necessary incision until long after the proper time. Meantime there develops a dense local inflammatory reaction which, when operation is ultimately undertaken, bleeds alarmingly, and which interferes considerably with the establishment of free drainage.

Perseverance with the administration of antibiotics long after they have ceased to have any value is a malpractice which is in no way confined to the management of acute mammary infections. The same mistakes are made in infections at other sites, but more evidently in situations like the finger pulp or the parotid, where the clinical sign of fluctuation is in any event long delayed.

My experience suggests that their abuse presents, in the case of breast infections, a problem peculiar to this situation—namely, the development of a hard, painless, fixed mass which in its clinical features mimics very closely a lactation carcinoma. This problem in diagnosis is especially seen when the patient first presents for treatment at hospital, having been previously under the care of her own practitioner. This point has recently been briefly alluded to by Stammers (1953).

It may well be that the unsatisfactory treatment of some of these infections can be attributed to the presence of resistant organisms. This is undoubtedly all too often the case in mammary abscess, especially if the patient is confined in hospital, where by a process of selection a permanent reservoir of resistant strains seems to be maintained. This was true of one of the above cases.

Heavy dosage with, say, penicillin will in the case of a light infection lead to clinical improvement so long as the local production of penicillinase does not build up to an effective level.

Stammers has suggested that in the presence of antibiotics the character of the local inflammatory reaction is greatly modified. There may, however, be a simple explanation. On the one hand, we have an infection with prompt antibiotic treatment which clears up quickly and completely. At the other end of the scale we have the old-fashioned abscess of the pre-antibiotic days, a large collection, representing a considerable loss of tissue. Somewhere in between, when the forces of invasion and those of repair are closely matched, it seems reasonable to assume that there may develop a hard inflammatory mass surrounding a small collection of pus such as was encountered in the breast cases described above.

Summary

The management of four cases of acute mammary abscess is reported. Treatment was primarily with penicillin, one case receiving in addition a seven-day course of chloramphenicol.

It was observed that the classical signs and symptoms of acute inflammation were considerably altered by the use of antibiotics.

A dense hard mass of fibrous and granulation tissue surrounding a loculus of pus was a feature of all the cases. The vascularity of the mass presented a real danger when it was explored surgically, in the form of torrential haemorrhage (as illustrated by Case 2). The undue delay of operative drainage because of the masking of symptoms and signs led in turn to a long and tedious recovery.

The lessons that might be learnt from these observations are : (1) Antibiotics, though of immense value in the treatment of acute infection, especially the localization of the disease in the breast, have their limitations. (2) The treatment of a localized collection of pus is still by surgical drainage, the timing of which is now as important as ever.

My thanks are due to Mr. Maurice R. Ewing for encouragement and help in the preparation of this paper.

REFERENCES

- Lake, N. C. (1952). *British Medical Journal*, 2, 1307.
Stammers, F. A. R. (1953). *Ibid.*, 1, 272.

PROSTHETIC MUCOSAL HYPERTROPHY IN THE MOUTH, SIMULATING MALIGNANCY

BY

J. WALTER, B.M., M.R.C.P., F.F.R., D.M.R.E.

Consultant Radiotherapist, Sheffield National Centre for Radiotherapy ; Clinical Teacher in Radiotherapy, University of Sheffield

Now that a realization of the importance of early diagnosis and treatment of cancer is spreading, not only in the medical profession but among the lay public, it is more than ever necessary to separate those benign lesions that mimic malignancy. This paper seeks to focus attention on one type that is common enough to merit detailed discussion—the mucosal hypertrophy due to chronic irritation by dental prostheses. Experience both at a large centre dealing primarily with cancer and at a general hospital dental department shows that this lesion is a regular cause of suspicion and alarm among patients, their general practitioners, general surgeons, and dental surgeons. Descriptions of the lesion are hard to find outside the dental literature. The only account that I can trace in recent general medical literature is by Simmons (1941). More recently, recognition seems to be increasing among dental surgeons. With the relative increase of the older age groups, and the increased number of dentures supplied under the National Health Service, many more of these lesions are to be expected in the future.

Clinical Picture

There are two principal sites : (1) the alveolo-buccal sulcus, immediately adjacent to the alveolus, more commonly lower than upper ; and (2) the hard palate, more or less centrally grouped round the midline.

First Type (Alveolo-buccal).—The typical lesion is a projecting elongated fleshy mass or tag on the mucosal surface adjoining the outer side of the edentulous alveolar margin (Figs. 1 and 2). Its length, parallel with the alveolus, ranges from a few millimetres to several centimetres, and its width up to about 1.5 cm. Its surface is ridged, with one or more deep furrows, and if the patient inserts his denture the edge can be seen to lie accurately in, or slip easily into, a furrow. The alveolus is atrophic and the denture fits very loosely ; usually it will be found to be an old one, commonly dating back ten years or more. The mass is fairly soft in the early stages, becoming harder with age and fibrotic changes, but there is not the spreading induration at the base that characterizes

malignancy. There may be ulcerations of the furrow with a little bleeding ; inflammation may cause tenderness and increased induration at the base, which can arouse suspicion of malignancy. The patient may complain of a little lump which has inconvenienced or alarmed him, or of intermittent pain due to sepsis. In some cases the lesions are not noticed by the patient at all, but are discovered by the dental surgeon when the patient attends for new dentures. Histologically there is considerable acanthosis and thickening of the surface epithelium but no evidence of invasion ; there may be increased fibrous tissue secondary to inflammatory changes.

Second Type (Palatal).—Here the presenting lesion is on the hard palate, in the form of one or more flat or pedunculated papillomatous patches, typically a few millimetres across. There may be a complete ring of separate or fused patches, corresponding with the relief chamber or suction ring on the upper denture. The surface of the denture may be roughened and irregular. Ulceration and bleeding are uncommon. The patches may also appear at the posterior end of the hard palate, on a transverse line due to the "food line" at the back of the denture.

Case Material

The clinical material over the last eight years at the Sheffield National Centre for Radiotherapy has been reviewed. Twenty cases were found ; 10 of these were of the first type, 8 of the second, and 2 were in atypical sites, and not definitely of similar nature. (These numbers are small in comparison with the material in a dental department ; for example, at the Sheffield Royal Hospital 24 cases were seen in the dental department in a single year (1951) and approximately the same number were seen in the facio-maxillary surgical unit.)

Alveolo-buccal Cases.—Of the 10 cases in this group nine were in females. The lower jaw was the site in all but two. This preponderance in females and the lower jaw was also found by Simmons (1941). Ages ranged from 37 to 78, the mean being 51 years. The length of history varied between three weeks and three years, usually two to four months. Most lesions were 1–3 cm. long ; one unusual case showed hypertrophy extending symmetrically along nearly the whole length of the lower alveolus on both sides. The age of the denture was not routinely recorded in all cases, but was typically at least several years, the oldest set being 16 years.

Palatal Cases.—In the eight cases the sex distribution was equal. Ages ranged from 39 to 75, the mean being 63 years. The length of history ranged from a few weeks to a few months. None of the individual lesions was larger than 1 cm.

Atypical Cases.—The first case was a proliferative papilloma, 2 cm. across, on the floor of the mouth just behind the alveolus. The other was a hypertrophic patch at the posterior end of the lateral border of the tongue, apparently an enlarged papilla.

Treatment

The only danger these lesions are likely to cause arises from a mistaken diagnosis of cancer leading to radical

FIG. 1

FIG. 2

FIG. 1.—A typical hypertrophic mucosal lesion on the upper alveolus. FIG. 2.—The lesion in Fig. 1 in relation to the denture.