intestinal secretions is known to be synthesised locally by subepithelial plasma cells.¹⁰ Many plasma cells are seen in the lactating human breast,³ and this has led to the assumption that the IgA in human milk is also locally synthesised. Local synthesis of immunoglobulin by mammary tissue has been shown in several animal species,^{11,12} but there is only one brief report of such a study in the human breast.¹³

Our observation that the non-lactating breast produces IgA rather than other immunoglobulins suggests that this synthesis is not a non-specific inflammatory response but a process associated with secretion. This interpretation is supported by the localisation of IgA in plasma cells associated with the lobules. Deposits of IgA are also concentrated in the lobules and particularly in the lumina of ductules, whereas IgG-positive cells are randomly distributed throughout the lobules and stroma.

Many of our biopsy specimens were taken from women who had breast nodules and were therefore not truly "normal." Mammoplasty specimens gave similar results, however, and we therefore conclude that IgA synthesis is not a reaction to benign breast disease but a property of normal breast tissue.

The secretion of IgA into the ductules may represent a form of defence against the entry of infection, but it seems more likely that the IgA secretion is simply a basal level of activity in an organ whose primary function is full lactation. In our primitive ancestors the breast was probably lactating almost continuously during the reproductive years,¹⁴ and it is unlikely that mechanisms adapted specifically for the non-lactating state have had time to evolve.

IgA synthesis occurs in both parous and nulliparous patients, but the observation that cyclical changes in IgA synthesis occur only in parous women implies some functional difference in the breast after the first pregnancy. The increase in IgA synthesis in the luteal phase of the cycle among parous women suggests that after the first pregnancy the breast is more sensitive to progesterone. A study of DNA synthesis by human breast epithelium *in vitro*¹⁵ has also shown cyclical variation in tissue from parous women and no cyclical variation among nulliparae.

The nulliparous breast is more susceptible to cancer, and the risk of subsequent breast cancer increases with increasing time elapsed between menarche and first pregnancy.^{16,17} The suggestion in our results that the nulliparous breast is less responsive to progesterone than the parous breast may simply be a reflection of the "immaturity" of the nulliparous breast, but the difference in hormonal sensitivity of breast tissue may have a direct bearing on its susceptibility to malignant change. It has been suggested that "unopposed" oestrogen stimulation

can lead to neoplastic changes¹⁸: if the nulliparous breast is relatively insensitive to progesterone this might mean that oestrogen stimulation during the menstrual cycle is inadequately "opposed" by progesterone.

We have no evidence that oral contraceptives produce abnormal stimulation of breast tissue. Our results suggest that the nulliparous breast will be relatively insensitive to the progestogen component of the combined oral contraceptive; the effect of the oestrogen component remains a matter for conjecture.

We are grateful to Professor A P M Forrest and his staff at Edinburgh Royal Infirmary, to the staff of the surgical units at the Chalmers, Bruntsfield, Deaconess, and Longmore Hospitals, Edinburgh, and to the staff of the plastic surgery unit, Bangour General Hospital, Broxburn, for collecting the specimens. We thank Mrs Pamela Chambers, Mrs Sandra Maciver, Miss Eileen McDonald, and Miss Jacqueline Scarisbrick for assaying steroid levels. Mr R Sharpe gave statistical advice, and Mr R Hogg and Mr R R Samson gave expert technical help. We thank Professor R V Short and Professor A R Currie for advice, and we are most grateful to the patients who volunteered to take part in the study.

References

- 1 Petrakis, N L, *et al*, *Journal of the National Cancer Institute*, 1975, **54**, 829.
- 2 Milligan, D, Drife, J O, and Short, R V, *British Medical Journal*, 1975, **4**, 494.
- 3 Beer, A E, Billingham, R E, and Head, J, *Journal of Investigative Dermatology*, 1974, **63**, 65.
- 4 Van Furth, R, Schuit, H R E, and Hijma, W, *Immunology*, 1966, **11**, 1.
- 5 Lai A Fat, R F M, *et al*, *Clinical and Experimental Immunology*, 1976, **23**, 9.
- 6 Brandtzaeg, P, *Immunology*, 1974, **26**, 1101.
- 7 McClelland, D B L, Shearman, D J C, and Van Furth, R, *Clinical and Experimental Immunology*, 1976, **25**, 103.
- 8 Neal, P, *et al*, *Journal of Endocrinology*, 1975, **65**, 19.
- 9 Cameron, E H D, and Jones, D A, *Steroids*, 1972, **20**, 737.
- 10 Tomasi, T B, and Bienenstock, J, *Advances in Immunology*, 1968, **9**, 1.
- 11 Watson, D L, and Lascelles, A K, *Australian Journal of Experimental Biology and Medical Science*, 1973, **51**, 247.
- 12 Lascelles, A K, and McDowell, G H, *Transplantation Reviews*, 1974, **19**, 170.
- 13 Hochwald, G M, Jacobson, E B, and Thorbecke, G J, *Federation Proceedings*, 1964, **23**, 557.
- 14 Short, R V, in *Physiology and Genetics of Reproduction*, Part A, ed E M Coutinho and F Fuchs. New York, Plenum, 1974.
- 15 Masters, J R W, Drife, J O, and Scarisbrick, J J, submitted for publication.
- 16 MacMahon, B, Cole, P, and Brown, J, *Journal of the National Cancer Institute*, 1973, **50**, 21.
- 17 Shapiro, S, *et al*, in *Host Environment Interactions in the Etiology of Cancer in Man*, ed R Doll and I Vodopija, p 169. Lyon, International Agency for Research in Cancer, 1973.
- 18 Siiteri, P K, Schwarz, B E, and MacDonald, P C, *Gynecologic Oncology*, 1974, **2**, 228.

SHORT REPORTS

Simplified ECG monitoring with an electrode mat

After myocardial infarction continuous ECG monitoring allows early recognition of arrhythmias, and impending ventricular fibrillation and asystole.¹ In sick neonates progressive slowing of cardiac rate gives early warning of hypoxia and potential cardiac arrest.² Conventional ECG electrodes take time to apply correctly, tend to become disconnected, and in neonates are prone to cause maceration of the skin. A newly available electrode mat* incorporates three electrodes of thin metal foil on to a single card (34 × 23 cm) and in some circumstances has definite advantages over conventional electrodes. The electrode mats are often used during surgery but have other applications: in casualty, after cardiac arrest, for intensive care, or during transit by ambulance. We report the successful monitoring of ECG and cardiac rate in 26 neonates.

The electrode mat

The mats are disposable, x-ray translucent, require no conductive gel, and can be positioned in seconds with minimum patient disturbance; infants can be laid across the whole electrode. For small neonates (< 1.75 kg) the mat should be trimmed to one-third of its original length and inserted halfway under the infant (see figure), to give direct skin contact with each of the three foil strips. ECG tracings are comparable with those from conventional electrodes and heart rate can be metered audibly. In neonates any material interferes with signal clarity and, in particular, "silver-swaddlers" obliterate meaningful recording. The electrode is connected to the monitor by a three contact clamp and single lead, and any mains-powered monitor can be used.

Infants studied

Twenty-six neonates weighing from 770 to 4700 g were monitored, two while in transport incubators. A pneumatic apnoea alarm³ may be used simultaneously if the electrode is placed directly between the infant and the apnoea mattress. With infants weighing < 1.75 kg the apnoea mattress may

An infant of 1200 g with the leading edge of a shortened electrode mat inserted under her right side and lying on an apnoea mattress.

give rise to a false alarm unless the electrode is reduced in size and inserted so that it makes direct contact only with one side of the infant, leaving his other side lying directly on the apnoea mattress (as in the figure).

When monitoring during transport, the electrode mat was connected to a battery powered ECG monitor† which performed well and without interference during ambulance travel. Recent reviews from two centres indicate the great value of continuous heart rate monitoring during transfer.^{4,5} Adequate observation during transit is difficult for many reasons: background noise renders heart and breath sounds inaudible and vibration obscures respiratory movement and accurate palpation of pulses. Bradycardia is an extremely important sign in any sick newborn infant, and particularly during transport, when a high proportion of babies (49%) have endotracheal tubes in situ.⁵ Here, early recognition of bradycardia provides immediate warning of accidental extubation or similar impending problems.

Because the mat warmed quickly in the incubator infants were placed naked on the electrode and then wrapped round with warm blankets. Continuous monitoring ensures better protection from heat loss as no part of the infant need be left exposed for observation of respiratory movement or skin colour. In both babies transported a small rise in rectal temperature occurred during the journey.

Conclusion

This electrode mat is not intended to replace conventional electrodes. Nevertheless, it has great advantages of speed and ease of insertion, lack of trauma and minimal disturbance, together with the benefits of simplified monitoring during transport. No important problems were encountered provided the mat was reduced in size for small infants and excessive movement did not occur. We suggest that wider use of this electrode mat could simplify ECG and heart rate monitoring.

We are grateful to Dr G Katz and Dr K Norton for allowing us to study their patients and for the interest and care given to these infants by the medical and nursing staff of the units concerned.

*Intek Cardiomat ECG Electrode, Intek (UK) Limited, London.
†Visicard 8 portable cardioscope, Linton Instrumentation, Harlow.

¹ Julian, D G, *Medicine*, 1973, 17, 1089.

² Klaus, M H, and Faranoff, A A, *Care of the High Risk Neonate*. Philadelphia, Saunders, 1973.

³ Blake, A M, et al, *Lancet*, 1970, 2, 183.

⁴ Storrs, C N, and Taylor, M R H, *British Medical Journal*, 1970, 3, 328.

⁵ Blake, A M, et al, *British Medical Journal*, 1975, 4, 13.

Intensive Therapy Unit, Barnet General Hospital and the Special Care Baby Units of Barnet and Edgware Hospitals

ANDREW J MACNAB, MB, BS, acting senior paediatric registrar (present address: Department of Paediatrics, University of British Columbia, Vancouver, BC)

ROSEMARY POPE, SRN, nursing officer, intensive therapy unit

Seasonal allergic symptoms due to fungal spores

The commonest cause of seasonal hay fever is a type 1 IgE-mediated allergic reaction to grass pollen, which is sometimes accompanied by asthma. In England several grasses release their pollen at about the same time; pollens from different grasses are antigenically similar and all can cause seasonal conjunctivitis, rhinitis, and asthma in susceptible patients. The diagnosis is suggested by the seasonal nature of the symptoms, which begin in the south of England towards the end of May, reach a peak towards the end of June, and diminish gradually during July. A simple prick test with a commercially available extract of grass pollen will produce an immediate weal and flare response, which correlates well with the results of direct intranasal challenge.¹ Some patients, however, complain of seasonal respiratory symptoms that do not coincide with the release of grass pollen. At any given time the atmosphere contains several other pollens² and a large variety of fungal spores,³ some of which may cause seasonal respiratory symptoms. With the use of a Hirst spore trap⁴ the air may be sampled continuously throughout the 24-hour period, both qualitatively and quantitatively, and the "count" of any particular pollen or spore expressed as the number of particles per cubic metre of air per 24 hours. The typical seasonal pattern of the common pollens and spores in central London is shown in the figure.

Patients, methods, and results

Patients who were referred to the allergy clinic with a diagnosis of seasonal rhinitis or asthma were asked to define closely the weeks of the year when their

Seasonal pattern of common pollens and spores in central London.

symptoms were present and at which period they were at their worst. They were then given prick tests with all the common airborne pollens and fungal spores. Allergy to a given pollen or spore was considered established if the symptoms occurred when the allergen was at maximal aerial concentration and if the patient showed a weal and flare response to the allergen greater than 5 mm diameter when prick tested and the results of prick tests with other allergens were negative.

Out of 2916 patients seen during 1973 (1425) and 1974 (1491), 2637 (90%) were allergic to grass pollen and 279 to other airborne allergens. Of these 279 patients, 86 (30.8%) were allergic to pollen of either the silver birch (*Betula*) or plane tree (*Platanus*) and had their symptoms between the end of March and the middle of May, and 156 (55.9%) were allergic to spores of *Alternaria tenuis* and had experienced their symptoms between the second half of July and the beginning of September. The remaining 37 patients were allergic to