(it is significant in this respect that "tank-seat" does not figure in the British soldier's vocabulary). Ladies should dispense with the bra and pico-panties on the journey at least. The attractive "tank-suit" worn by the elegant Italian or a loose-fitting Saharien are ideal for the long, hot summer drive and will preserve both the comfort and appearance of the wearer so that she can enjoy the whole of her holiday without cutaneous mishap.

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Shoulder-cuff lesions

SIR,—With reference to your leading article (20 March, p 672), for many years now I have been using steroid injections into shoulder lesions, but it was not until I attended a course so ably presented by Dr J H Cyriax that the success of my treatments increased considerably. Using his method to ascertain an accurate diagnosis, I find it is possible to treat most tendinous lesions with great success, and capsulitis can be equally treated if approached at an early stage in the condition.

I think it would be true to say that my consultant colleagues see shoulder lesions only when they are fairly well advanced and established. The success I have obtained over the past three years prompts me to promote the contention that injection therapy should be performed by a general practitioner, who sees the patient at an early stage.

B M G CLARKE

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Tattooing for port-wine stains

SIR,—In dealing with this subject, which was raised in "Any Questions" some time ago,¹ much was written about surgery but extremely little about tattooing, the latter being considered "generally unsatisfactory." Similarly, in two subsequent reviews on "Aspects of Plastic Surgery"^{2,3} only cursory references to this method of treatment were made.

Two years ago I heard that a cousin in Toronto, now aged 76, had been treated by tattooing with a very satisfactory result. His port-wine lesion had been unusually extensive, involving practically the whole right side of the face. After as much plastic surgery as was considered feasible and expedient had been performed there still remained a large area (two-thirds of the original) of deep-purple-coloured skin. On visiting him a few months ago I was astonished at the remarkable change. This had been obtained by an advanced technique developed in Canada.⁴ For the extremely important factor of pigment colour-matching a method has been devised producing clinical results which demonstrate the advantage of electronic equipment in determining the proper pigment mixture for each patient's skin colour. The new mechanical device used in the operation is a high-speed turbine to which is attached a carrier head holding the tattooing needles, the turbine being driven by compressed air.

In the answer to the original question it was suggested that "the best results are often produced, with the least trauma, by a kindly acceptance . . . of some inevitable and permanent disfigurement." The patient referred to above maintains that one *never* learns to live

with it. The least acceptable aspect of this lesion is its intense livid discoloration and, to the observer, it is this feature which makes the patient the object of pity and curiosity, even of aversion and rejection. The life-long experience of this patient bears ample evidence of these social inflections. Now, virtually free from the discoloration, he feels presentable; to quote him: "I enjoy my new looks every day of the week." This emphasises the complete change in outlook that tattooing has brought about in this case.

In view of the possible attainment of such satisfactory results from surgical tattooing it is difficult to accept that this technique should not be advocated and made available for these lesions.

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Son,
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¹ *British Medical Journal*, 1974, **1**, 450.

² Cobbett, J R, *British Medical Journal*, 1974, **2**, 718.

³ Morgan, B D G, *British Medical Journal*, 1974, **3**, 35.

⁴ Thomson, H G, and Wright, A M, *Plastic and Reconstructive Surgery*, 1971, **48**, 113.

New look at malaria

SIR,—Your leading article on this subject (1 May, p 1029) reiterates the simplistic conclusion that "malaria must be defeated if the standards of health and living in the endemic areas are to be raised permanently."

This is the traditional attitude to a killing disease. The question arises whether it can square up to the facts of the new situation resulting from burgeoning population increase and limited food supplies. Most African countries where malaria is endemic have population increasing at well over 2.5% resulting, if unchecked, in a doubling of population in under 30 years. It is really humane to eradicate malaria and leave the population to die of starvation? It would seem to be better for the WHO to concentrate first on intensive aid in population control in these areas. The elimination of malaria and other natural checks on population increase will then be a blessing rather than a bane.

It is all too easy to espouse comfortable and well-established nostrums in medical policies without realising that interference with natural balances has to be fully justified in a world threatened more by man's short-sighted application of his apparent power to do good than by any other single factor.

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NHS cost of domiciliary oxygen

SIR,—Your leading article on domiciliary oxygen in chronic bronchitis (28 February, p 484) quotes the annual cost of 14 1.35-m³ cylinders per week as £1372. In my view this figure is too low since it takes into account only the cost of the gas.

Under the terms of the Drug Tariff professional fees and transport costs for cylinders would amount to a minimum of £770 and a maximum of £1499—depending on the time and urgency of the deliveries—if the patient lives within three miles of a pharmacy handling oxygen. The costs for delivery to patients living 10 miles or more from the pharmacy will vary between £1737 and £2466. When you add the hire charges for regulator, flowmeter

stand, etc at £10 per year you will see that the total cost to the NHS of supplying 14 "F" size cylinders of oxygen each week to a patient at home will be at least £2152 per annum and may be as high as £3848 per annum. An average cost of £3000 per patient is therefore more realistic.

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Management of threatened abortion

SIR,—Permit me to comment on your leading article (1 May, p 1034). I doubt the existence of "threatened abortion" as an entity. However, fetal wastage is quite a problem. One of the preventable causes is indeed hypothyroidism.

I encountered not long ago the case of a 22-year-old primigravid woman who presented with vaginal bleeding and abdominal pain at 26 weeks' pregnancy. Signs of premature labour were noted. The patient was rather lethargic and showed signs of hypothyroidism. She gave a history of congenital hypothyroidism being diagnosed at the age of 18 months. Thyroid extract had been prescribed continuously since that time but she admitted to not taking the tablets regularly. Thyroid function tests showed hypofunction, and replacement therapy was started. The condition improved and premature labour was averted. Normal "thyroid test profile" for pregnancy was maintained on 0.3 mg of L-thyroxine daily. Eventually this patient was delivered of a healthy infant near term.

Despite the well-documented link between hypothyroidism or hypothyroxinaemia and fetal wastage^{1,2} it is still unclear whether thyroid replacement would prevent fetal loss or not. The case mentioned indicates that it might do so. The suggestion of Winikoff and Malinek³ of "thyroid test profile" is quite a valid one. Since thyroid replacement does not inflict real harm on the unborn infant it is wiser to start the supplement therapy despite the doubts regarding its therapeutic value.

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¹ Jones, W S, and Man, E B, *American Journal of Obstetrics and Gynecology*, 1969, **104**, 909.

² Winikoff, D, Dickinson, R D, and Wade, G, *Journal of Obstetrics and Gynaecology of the British Empire*, 1960, **67**, 56.

³ Winikoff, D, and Malinek, M, *British Journal of Obstetrics and Gynaecology*, 1975, **82**, 760.

Myringitis bullosa

SIR,—In otitis media surely the general practitioner comes into his own, seeing as he does the entire spectrum of cases, not the red end only which reaches the consultant. In his otherwise comprehensive article (21 February, p 443) and in his letter (3 April, p 836) Mr J F Birrell makes some statements which are true for a consultant but not for a GP. This has been pointed out by Dr V W M Drury (13 March, p 648), Dr E O Evans (13 March, p 653), and Dr S J Carne (24 April, p 1018), but further amplification seems needed on the subject of that common (in general practice) complaint, myringitis bullosa.

Among the many cases of children with ear-ache for which the GP's help is sought, it not infrequently happens that, having cried for some hours, the child went peacefully to sleep shortly before being seen. Usually, of course, the cause has been a blister on the drum, bursting or regression resulting in relief from the pain. Only a small proportion of such blisters are haemorrhagic and of the dark colour commonly described, most being like those elsewhere. Surely it was the bursting of the former kind which accounted for the blood in discharge mentioned by Dr Carne, if it was not from suppurative otitis media. They vary much in size from some quite small to others almost covering the drum. One wonders how often a large bulla has been mistaken for a true "bulging drum." Any degree of reddening may be present, as in non-vesicular inflammations, or there may occasionally be none. After bursting or regression the site presents a very characteristic, rather crinkly appearance, presumably the "grey mottling" mentioned by Mr Birrell. Once this stage is reached there is rarely any further trouble. The condition is usually classified as part of catarrhal otitis media, but the pathology is debatable. Clearly of virus origin (incidentally often the first intimation that yet another of the never-ending succession of such epidemics is prevalent), there is much to suggest that the vesicles are herpetic—in lay language, "cold sores on the drum." Pain is apt to be severe, but fortunately usually lasts only a matter of a few hours. In view of the virus origin it is doubtful whether antibiotics can achieve much, but as a few cases go on to more definite otitis media they would appear to be indicated while pain or other evidence of inflammation is present. When pain has gone and the drum clearly shows regression it is at least questionable whether treatment need be begun or continued.

May it be said that more than half the battle in seeing the drums of young children is won by having the head firmly held. In the absence of a nurse, many mothers soon get good at this. A convenient position is with the child on one side, then on the other, across the mother's lap, head to the observer's left. Rapid assessment is usually essential.

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Once-daily atenolol for hypertension

SIR,—I read with interest the paper by Drs A P Douglas-Jones and J M Cruickshank (24 April, p 990) in which atenolol is claimed to be effective in once-daily dosage in reducing the resting pulse rate and blood pressure after 24 hours in patients with mild or moderate hypertension. If such patients had been exercised, which the authors agree is necessary to assess beta-blockade, their findings might well have been different.

Our department has investigated the effect of metoprolol, a cardioselective beta-adrenergic blocking agent, given in a single daily dose to previously untreated hypertensive patients.¹ Although apparently controlled at rest by 100-200 mg daily, the post-24 hour exercise blood pressure and pulse rate in some patients tended to exceed normal limits but was subsequently well controlled by dosage increases to levels of 300-400 mg. These

results will form the basis of a future publication.

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¹ Ablad, B, Carlsson, E, and Ek, L, *Life Sciences*, 1973, 12, 107.

Does anticonvulsant hypomagnesaemia exist?

SIR,—In two recent letters on this subject the authors—contrary to our findings in 1974¹—report that they were unable to demonstrate hypomagnesaemia in epileptic patients on anticonvulsant therapy. Dr S H Katz and his colleagues (7 February, p 341) believe that this difference may be due to our use of "an unmatched normal outpatient control population," while Dr M J Stewart (13 March, p 649) thinks this explanation unlikely. We tend to agree with the latter statement since serum magnesium values are independent of sex and age differences.²

Experts in the field of magnesium metabolism agree that the biological variation of serum magnesium concentration is quite small^{3,4}; furthermore, about 25% of serum magnesium is protein-bound. The latter fact infers that dehydration and/or venous stasis (which influences the serum level of protein) simultaneously may influence the serum magnesium level. In our study on 226 epileptic outpatients and 95 controls¹ these possible sources of variation were dealt with in the following way: all blood samples were drawn without stasis; the magnesium concentrations were corrected to a constant protein level; the blood samples were run in series of 60 duplicates, each batch containing samples from patients and controls; and the technicians were unaware of the clinical data. In the conflicting reports of Dr Katz and his colleagues and Dr Stewart (which contain data from a mixture of inpatients and outpatients) no information is given as to the blood sampling technique and apparently no correction for serum protein was made. This alone might explain the different findings, but it appears that this is not the sole cause.

In the accompanying table we have summarized data of interest from your correspondents' reports and from our own study. For the control subjects it is interesting to see that all three reports agree as to the mean values; only our biological variation is much smaller than those found in the other studies. The main reason for this is probably a larger inter-assay variation of the methods used by our opponents. The same remarkable difference in biological variation is found between our patients and those of Dr Katz and his colleagues and of Dr Stewart. But whereas we,

with our apparently superior laboratory method in a large population, can demonstrate a small but significant lowering of serum magnesium in epileptic outpatients, our opponents, with their methods in smaller series of inpatients (or mixed in- and outpatients) apparently cannot.

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¹ Christiansen, C, Nielsen, S P, and Rødbro, P, *British Medical Journal*, 1974, 1, 198.

² Petersen, B, et al. To be published.

³ Hunt, B J, *Clinical Chemistry*, 1969, 15, 979.

⁴ Wacker, W E C, et al, *New England Journal of Medicine*, 1968, 278, 658.

Benign proliferative lesions of the breast

SIR,—Confusion still exists over the conditions included under the above heading and the alternative terms you mention—cystic hyperplasia and mammary dysplasia—in your leading article (8 May, p 1106). May I suggest that this is largely due to a failure to differentiate between three separate and distinct entities?

The first is a purely histological condition, of importance as a field of study of the potential relation of various types of epithelial change to carcinoma but with no diagnostic clinical features.

The second is an objective clinical condition—the development of macroscopic cysts. These usually present as an obvious solitary lump in one breast, hard in consistency and thus simulating carcinoma. Further solitary lumps in either breast commonly develop over the years. Because of the suddenness of their appearance the proximate cause must be some temporary acute upset of intraductal hydrodynamics. The condition occurs in women from the late thirties to the menopause and is completely innocent, the one-time belief that it might be precancerous being due to a failure to separate the rare cysts developing secondarily to an intraduct papilloma or papillary carcinoma.

The third condition, which I have termed "the pain syndrome," is a purely functional one in both the physiological and psychological senses of the term. It arises from a hyper-awareness of the physiological and para-physiological cycle of changes in the female breast. As far as I can gather, it is not seen in societies in which women are unaware of the

Authors	Epileptic patients			Control subjects			Serum magnesium determination	
	No	Serum magnesium		No	Serum magnesium		Intra-assay variation (%)	Inter-assay variation (%)
		Mean (mmol/l)	Coefficient of variation (%)		Mean (mmol/l)	Coefficient of variation (%)		
Christiansen <i>et al</i> ¹ ..	226*	0.78	6.4	95	0.81	4.9	0.9	1.6
Katz <i>et al</i> ..	100†	0.82	11.0	100	0.82	8.5	0.6	Not stated
Stewart ..	80§	0.84	10.3	71	0.82	10.2	Not stated	4.1

*Outpatients. †Inpatients. §In- and out-patients.