menstrual period to see if the string is now visible. If the films suggest that the IUCD is no longer within the uterus the matter should be fully discussed with the patient and a laparoscopy or laparotomy performed to remove the IUCD. Fitting a further IUCD following such an abdominal procedure would seem unwise.

The apparent increase in the missing tails problem may in part be due to cutting the string too short after insertion (at least 5 cm (2 in) should be left). Investigations should not be started if there is any possibility of pregnancy (itself a cause of missing tail).

If the patient is pregnant—but that is another problem.

JOHN FRAMPTON

Warneford Hospital,
Leamington Spa, Warwick

Disopyramide in ventricular fibrillation

SIR,—In the case referred to by Dr D P Atukorale (25 December, p 1564) the ventricular arrhythmia appears to have been "torsade de pointes."¹ This may result from overdosage with cardioselective drugs such as lignocaine, procaine, quinidine sulphate, alprenolol, or pindolol, all used by him. The arrhythmia responded to omission of these drugs. In our patient, however (2 October, 1976, p 795), the lignocaine was tailed off only when the patient responded to disopyramide; hence the cessation of ventricular fibrillation in this case could not have been due to the omission of lignocaine. Comparison of disopyramide with cardioactive agents such as lignocaine is currently under test in an open ward trial.²

T D I M S DE LANEROLLE

General Hospital,
Columbo, Sri Lanka

¹ Krikler, D M, and Curry, V L, *British Heart Journal*, 1976, **38**, 117.

² Jennings, A, et al, *Lancet*, 1976, **1**, 51.

Tetracycline resistance of group A streptococci from different sites

SIR,—The nationwide survey of tetracycline resistance in pneumococci and Group A streptococci (15 January, p 131) very wisely points out the necessity for making local surveys before deciding which antibiotic to use. This prompted us to review the tetracycline resistance of strains of Group A streptococci isolated in this laboratory over the past 15 months. We found that in the 305 strains isolated tetracycline resistance was almost the same in the two centres from which we receive specimens (Alton 29 out of 107 (27.1%); Basingstoke 51 out of 198 (25.8%)). The incidence of tetracycline resistance in 187 respiratory strains was 29.4%, and in 118 non-respiratory strains 21.2%. This difference is not significant ($0.2 > P > 0.1$). This latter finding does not support the nationwide survey conclusions.

However, more interesting was the analysis of the tetracycline resistance of strains causing infection of skin and soft tissues. These were divided into three groups: "impetigo/infected eczema" (all from general practice); "superficial lesions" (consisting predominantly of infections of the hands and feet, often clinically referred to as paronychia, from general practitioners and the accident and emergency

department); and "deep lesions" (consisting of abscesses from general practice and the accident and emergency department and three inpatient wound infections). The results are shown in the table.

Group	Total No of isolates	No (%) resistant to tetracycline
Impetigo/eczema ..	41	4 (9.8)
Superficial ..	38	8 (21.1)
Deep ..	19	8 (42.1)

These figures suggest that, while the locality of the patient may be important in deciding initial treatment, the locality of the lesion may be equally important.

P A BOSWELL
G E HILLIER

Department of Microbiology,
Basingstoke District Hospital,
Basingstoke, Hants

Possible immunological effect of oxprenolol

SIR,—The report of Dr L F W McMahon on a possible immunological effect of oxprenolol (4 December, p 1388) is rather in contrast with previous observations on this subject reported in the literature. Other beta-blockers in fact, like butoxamine and propranolol, are known to enhance in-vivo antibody synthesis to heterologous protein in rats,¹ although propranolol added in culture does not modify PHA-induced transformation of human lymphocytes.² On the contrary, a beta-agonist like isoprenaline inhibits lymphocyte transformation³⁻⁴ and IgE synthesis in vitro.⁵

Since alpha-adrenergic stimulation by norepinephrine at physiological concentrations enhances lymphocyte transformation² while alpha-blockade by phenoxybenzamine depresses antibody synthesis in animals,⁶⁻⁹ it might be postulated that immunodepression is related to a beta-adrenergic effect and that it can be modified according to the usual pattern of adrenergic stimulation and blockade. It could be very interesting, then, if further studies would confirm this hypothesis or, so far as Dr McMahon's letter is concerned, would provide conclusive evidence against it.

RENATO NESSI

Corporate Medical Department,
Gruppo Lepetit SPA,
Milan, Italy

¹ Benner, M H, et al, *Journal of Allergy*, 1968, **41**, 110.

² Hadden, W J, Hadden, E M, and Middleton, E, *Cellular Immunology*, 1970, **1**, 583.

³ Smith, J W, et al, *Federation Proceedings*, 1969, **28**, 566.

⁴ Szentivanyi, A, *Journal of Allergy*, 1968, **42**, 203.

⁵ Patterson, R, et al, *Journal of Immunology*, 1976, **117**, 97.

⁶ Rosenblatt, E, and Johnson, A G, *Proceedings of the Society of Experimental Biology and Medicine*, 1963, **113**, 156.

⁷ Pieroni, R E, and Levine, L, *Nature*, 1967, **213**, 1015.

⁸ Pieroni, R E, *New England Journal of Medicine*, 1971, **284**, 793.

⁹ Flier, J S, *New England Journal of Medicine*, 1971, **284**, 1159.

Antibiotic myths

SIR,—I have recently discovered that two old beliefs about the use of antibiotics are still so widely held and so misleading that they deserve publicity.

The first is that if you have started to give a patient an antibiotic and shortly discover that antibiotic to be inappropriate you must

nevertheless complete the course. Five days is the usual time mentioned. Prevention of the emergence of resistance in some unspecified bacterial population is the usual reason cited.

The second misconception is that if you have decided on clinical grounds that a patient needs antibiotic treatment and have also decided to send appropriate material to the laboratory for culture you should wait for the results of the laboratory investigation before starting treatment. If a patient needs treatment with an antibiotic, he needs treatment *now*—at the time the indication for treatment is recognised. Of course, if material for culture is to be taken it should be collected before the antibiotic is given, but only just before. The antibiotic most likely to be effective should then be given. If later that agent proves inappropriate it should be stopped immediately and a more appropriate one substituted.

MALCOLM LEWIS

Public Health Laboratory,
City and Sherwood Hospitals,
Nottingham

Access to oral contraceptives

SIR,—The signatories to the letter under this heading (22 January, p 228) omit any reference to consideration of British women's own individual needs and wishes in respect of their health, wellbeing, and future fertility in using oral contraceptives. We are doctors, not vets. Many women know what the "pill" is doing—and say so.

Whatever happens in the Third World does not, surely, invalidate our medical responsibility here. When we as doctors know that the oral contraceptives' long-term effects on individual women's physiology are unknown and women themselves (for example, Sonia Behr in the *Sunday Times* of 23 January) are questioning the suppression of facts—such as the increasing frequency of post-pill amenorrhoea—are we prepared to lie to our patients about the need for continuing medical responsibility and prescription?

Is this the "exemplar to the developed world" that Judith Bury, Malcolm Potts, John Lorraine, and others want?

BETTY ELLIOTT

West Walton, Norfolk

Local gold toxicity

SIR,—Dr D C Dick (1 January, p 51) has suggested that the patient I described (27 November, p 1294) had a recurrence of a nickel dermatitis and in support of his contention he cites the observations of Stoddart.¹

The needles used in our clinic contain 8–12% nickel and my patient was exposed to such needles on two occasions before sodium aurothiomalate was given and on several occasions since, for the purpose of venepuncture, without any untoward effect. The duration of exposure for both venepuncture and intramuscular injection is similar and short. The two cases of recurrent nickel dermatitis described by Stoddart¹ occurred in patients in whom nickel-steel-shafted infusion sets were inserted into a forearm vein during elective surgery. The duration of exposure was hours rather than seconds and the reaction manifestations were generalised in both and localised in one. In his conclusions the author

was careful to point out that an allergic reaction to the infused blood or plasma could not be excluded.

Formal patch-testing conducted on the patient in question failed to evoke a response to nickel, dichromate, or cobalt. The results of patch-testing to gold leaf and gold chloride (1% aqueous solution) were also negative. The lack of response to the application of gold leaf or ring scrapings was also noted by Comaish,² and in a review of the literature of contact hypersensitivity to metallic gold Young³ found that patch-testing was often positive only to gold salts rather than metallic gold and often positive with one salt and not another. I regarded the administration of sodium aurothiomalate as a form of exposure to gold salt in a woman one might have regarded as being allergic to gold jewellery.

NORRIS RENNIE

Centre for Rheumatic Diseases,
Glasgow

¹ Stoddart, J C, *Lancet*, 1960, 2, 741.

² Comaish, S, *Archives of Dermatology*, 1969, 99, 720.

³ Young, E, *Dermatologica*, 1974, 148, 294.

Lymph-node puncture

SIR,—I have used the ordinary disposable Gillette venepuncture needle size 19G × 2 for doing lymph-node punctures and have found it more useful than the conventional lymph-node puncture needle with the stylet.

The advantages are: (1) it is a disposable needle and easy to hold when attached to a syringe; (2) a local anaesthetic is not required; (3) even a small lymph node can be successfully punctured; (4) a sufficient amount of material is easily obtained by a few forward and backward movements of the needle inside the lymph-node substance; and (5) it does not distort the morphology of the lymph node, so that the same node can be removed for a proper histological examination if required.

P C SRIVASTAVA

Burton District Hospital Centre,
Burton-on-Trent, Staffs

Chemoprophylaxis of malaria

SIR,—In his letter (1 January, p 49) commenting on your recent leading article on this subject (20 November, p 1215) Professor W Peters asks what recommendations this company is now making for Fansidar, particularly with regard to the dosage in non-immune subjects.

Our current prescribing information for the drug does include recommendations for its prophylactic use in high-risk subjects in areas where resistance to other antimalarial agents is known to occur or in cases of chloroquine intolerance. For semi-immune subjects the recommended dosage interval is once every four weeks—a regimen for which there is ample evidence of efficacy, as indicated in your reply to Professor Peters. For non-immunes the evidence we have is that the once-weekly dose of one tablet and the once-fortnightly dose of two tablets are equally effective; the choice between these regimens rests on patient preference or convenience.

The work in non-immunes is still largely unpublished except for the extensive study by Ebisawa *et al*¹ among Japanese and native dam construction workers in Laos, in an area

where chloroquine resistance was known to occur. The study incidentally underlines the importance of regular dosage by non-immunes; the few instances of breakthrough infection recorded in this group on the weekly prophylactic regimen over an 18-month period were all attributed to missed doses.

The occasional breakthrough infections observed during Fansidar prophylaxis in south-east Asia have nearly always been due to *Plasmodium vivax* and are effectively suppressed by the subsequent dose of the drug.^{2 3}

I hope this will serve to put our position on record.

VICTOR BERGSON
Medical Adviser,
Roche Products Ltd

London W1

¹ Ebisawa, I, *et al*, *Japanese Journal of Experimental Medicine*, 1971, 41, 209.

² O'Holohan, D R, and Hugoe-Matthews, J, *South-east Asian Journal of Tropical Medicine and Hygiene*, 1973, 2, 164.

³ Lewis, A N, and Ponnampalam, J T, *Annals of Tropical Medicine and Parasitology*, 1975, 69, 1.

Subclinical hypothyroidism

SIR,—Professor R Hoffenberg writes an excellent review of Dr D C Evered's monograph on diseases of the thyroid (15 January, p 176). I was interested in Professor Hoffenberg's "personal protest against the continued use of the term 'subclinical hypothyroidism.'" He expresses fear that "its acceptance will only give rise to misunderstanding and unnecessary thyroxine therapy." Some other thyroid experts share his view, but unnecessary thyroxine therapy based on a misunderstanding can scarcely be an adequate reason for rejecting the validity of a concept.

Nilsson *et al*¹ do not question the validity of subclinical hypothyroidism (or premyxoedema) but are rightly more concerned with the practical significance of the condition. Their study offered no support for my contention that hypercholesterolaemia can be a premonitory sign of hypothyroidism.² Other leading investigators such as Hall's group in Newcastle³ or Bastenie in Brussels⁴ have failed to relate an increased incidence of coronary artery disease in autoimmune thyroiditis to hypercholesterolaemia. I continue to see patients suffering from premyxoedema with hypercholesterolaemia and degenerative arterial disease.⁵⁻⁷ Recently Professor Baschieri⁸ and his colleagues in Pisa and Padua have found that 31% of unselected patients with hypercholesterolaemia have a raised basal serum thyroid-stimulating hormone (TSH) concentration or an exaggerated response of TSH to the thyrotrophin-releasing hormone. Those who know Professor Baschieri's work will protest against any summary dismissal of a concept that may play a key role in the prevention of degenerative arterial disease.

P B S FOWLER

Charing Cross Hospital,
London W6

¹ Nilsson, G, Nordlander, S, and Levin, K, *Acta Medica Scandinavica*, 1976, 200, 63.

² Fowler, P B S, and Swale, J, *Lancet*, 1967, 1, 1077.

³ Tunbridge, W M G, *et al*, VII International Thyroid Conference, Boston, USA, June 1975.

⁴ Bastenie, P A, *et al*, *Lancet*, 1971, 1, 203.

⁵ Fowler, P B S, Swale, J, and Andrews, H, *Lancet*, 1970, 2, 488.

⁶ Ikram, H, Banim, S, and Fowler, P B S, *Lancet*, 1973, 2, 1405.

⁷ Greenwood, T W, and Fowler, P B S, *Proceedings of the Royal Society of Medicine*, 1976, 69, 225.

⁸ Baschieri, L, *et al*, *Revue Française d'Endocrinologie Clinique*, 1975, 16, 211.

Too many doctors and in the wrong places?

SIR,—The debate about RAWP¹ has died down and the special pleading by the London regions that shocked and astounded the rest of the country has been in large part successful. Mr David Ennals, like Sir Keith Joseph four years before him, believes it will take 10 years to redistribute the resources of the NHS. Perhaps equality in this as in so many things is a mirage.

One outcome of the discussion is the realisation that we may be producing too many doctors and that the geographical distribution of those we do produce is obviously faulty. We are all familiar with the argument that the need for medical treatment is never-ending and that there will always be work for doctors; the question is (as with school teaching), can our society afford to pay for such a bottomless demand at the salaries that professionals themselves feel represent their value and standing in the community? I doubt it.

Many of us in Manchester, struggling to teach a yearly clinical intake of 270 students, hardly dare to complain or question the need for such a vast output (and big is certainly not beautiful or easy in this context) when there is an obvious shortage of doctors in the north-west of England.

Greater London, historically the site of most English medical education, has a rapidly shrinking population. It might not be very important that London medical schools should continue to produce most of our profession if it were not that for very many of them outer darkness seems to start north of Potters Bar. A few graduates escape to the northern regions of their childhood but most are conditioned by a London education to stay in or near that city. Moreover, as has often been stated, a "centre of excellence" raises the standards of the whole of medicine in its vicinity.

Similar arguments apply to other cities and regions, which is why at Manchester we have reluctantly accepted 270 students in each clinical year. But why couldn't the redistribution have been brought about by opening two or three more clinical schools in the north at such places as Preston and Stoke and closing or transferring half the London schools? It takes about 10 years to create a "centre of excellence" in medicine and it takes almost as long to divert the output of new doctors from one region to another. Why not St Thomas's for Preston and Barts for Bolton? After all, the oil sheikhs could always fly into Ringway Airport.

PETER O YATES

Department of Pathology,
University of Manchester

¹ Department of Health and Social Security, *Sharing Resources for Health in England*. London, HMSO, 1976.

DHSS maternity leave

SIR,—I find it laudable that the Department of Health and Social Security, together with the profession, are currently endeavouring to encourage a return to breast-feeding as opposed to "the bottle" for our new generation of babies. The DHSS even go so far as to suggest that a minimum period of three to four months of mother's milk is best.

I was therefore disgusted to find that the DHSS maternity leave scheme demands that