kept unequivocally biochemically euthyroid. It is possible (and I feel worthy of serious consideration) that the addition of a small maintenance dose of L-thyroxine might enable the required dose of steroids (which of course also have a valuable role to play) to be reduced to a lower level than 10 mg of prednisone a day with an accompanying lessening of the risk of long-term steroid-induced complications.

J F FALCONER SMITH

Nuffield Department of Clinical Biochemistry,
Radcliffe Infirmary,
Oxford

Possible environmental hazards of gas cooking

SIR,—Recent letters in your columns from Mr E A K Patrick and Dr M C S Kennedy (19 August, p 567) discussed possible hazards of indoor air pollution from gas stoves. In community studies of respiratory disease in Connecticut and South Carolina[1 2] in which we obtained data on respiratory symptoms and lung function from about 7000 residents aged 7 years and over we compared residents in homes with gas stoves with those who used electric power for cooking. We took age, sex, race, height, weight, and smoking habits into account in these comparisons and found that respiratory symptoms were equally prevalent and lung function similar among men, women, and children living in homes with gas stoves and in homes with electric stoves. Using portable sampling equipment in selected homes[3] we confirmed that nitrogen dioxide (NO_2) concentrations are higher in homes equipped with gas stoves than in those where electricity is used for cooking. Twenty-four-hour average NO_2 levels in 11 homes with gas stoves ranged up to 500 $\mu g/m^3$, and short-term (2 h) peaks up to 3000 $\mu g/m^3$ were observed in one such home. However, we have found no evidence that the use of gas for cooking is associated with increased respiratory illness among adults or among children aged 7 years and older. We did not investigate younger children, but any effect of indoor air pollution from gas stoves on their lungs would appear to be temporary, since no changes were detected after the age of 7.

H ROLAND HOSEIN

Department of Preventive Medicine and Biostatistics,
University of Toronto,
Toronto, Ontario

AREND BOUHUYS

Department of Internal Medicine,
Yale University School of Medicine,
New Haven, Connecticut

[1] Mitchell, C A, Schilling, R S F, and Bouhuys, A, *American Journal of Epidemiology*, 1976, **103**, 212.
[2] Bouhuys, A, Beck, G J, and Schoenberg, J B, *Nature*, 1978, **276**, 466.
[3] Binder, R E, *et al*, *Archives of Environmental Health*, 1976, **31**, 277.

Ankylosing spondylitis in HLA-B27-positive individuals: use in diagnosis

SIR,—We wish to take issue with several of the major points in your leading article (2 September, p 650) "HLA-B27 and risk of ankylosing spondylitis." You recognise the obvious conflict between the early and the more recent population studies of the prevalence of ankylosing spondylitis (AS) but, for reasons not given, prefer to believe the more recent studies. In order to resolve this conflict we have recently conducted a study of the population of Busselton, Western Australia, and have found a prevalence of AS similar to that predicted by the early studies rather than the much higher prevalence suggested by Calin and Fries.[1] We HLA-typed some 80% of the adult population of this relatively isolated, stable, small country town. Of 2745 people, 168 (6·1%) were B27-positive. Careful questionnaire and clinical assessment was undertaken on 139 of these together with an age- and sex-matched control group from the same population. Radiographs, which were available from 22% of the B27-positive individuals, were reviewed. Not one case of AS was detected and there were no differences in the frequency and character of the back pain between the two groups. We used a questionnaire similar to that of Calin and Fries and obtained sacroiliac radiographs in a higher percentage of the study group. These data suggest a maximum prevalence of AS of 0·04% in the population as a whole and 0·7% in B27-positive individuals and are therefore consistent with the earlier "classical" surveys rather than some surveys[1] which may be explained either on the basis of the population studied or over-interpretation of symptoms, signs, and *x*-rays.

Your article states that in a suspicious clinical setting (for example, persistent backache) the *presence* of B27 will help in making a diagnosis. We contend that it is the *absence* of B27 which should be used to exclude AS, while the finding of B27 in a patient is of little help. Such a contention is based on the relative rarity of AS and the relative frequency of B27. Even if one accepts the figures from the study of Calin and Fries four out of five people with B27 will not have AS. Furthermore, recent studies[2] indicate that the incidence of B27-negative AS may be as low as 1-2%. Therefore it is clear that HLA-typing for B27 should be used as an *excluding* test rather than a confirming test.

Because the prevalence of AS in B27-positive individuals is nearer 1% than 100% genetic counselling will cause needless anxiety. In fact, with modern treatment, as outlined in your article, AS is nowadays an infrequent cause of significant morbidity.

F CHRISTIANSEN
PAUL ZILKO
R L DAWKINS

Department of Clinical Immunology,
Royal Perth Hospital,
Perth, Western Australia

[1] Calin, A, and Fries, J F, *New England Journal of Medicine*, 1975, **293**, 835.
[2] *Joint Report on HLA and Disease*, Seventh International Histocompatibility Workshop Conference, Oxford, 1977. In press.

**These new data from Dr Christiansen and his colleagues are of considerable importance and it will be of great interest to read a full report, when the size and age structure of the population and its ethnic origins will be clearer. The great variations which are known to exist in the frequency with which AS is found in different populations must be taken into account and it may be that a small, relatively isolated country town in Western Australia is not typical of the generality of Caucasians. Certainly the observations made by Calin and Fries are similar to those of Kidd *et al*[1] in suggesting a much higher frequency of clinical and subclinical AS in B27-positive individuals than Dr Christiansen has found. No doubt more studies will be completed and the mystery resolved in time. We agree with Dr Christiansen that genetic counselling for ankylosing spondylitis should be approached with caution, and this was stated in the leading article. Also consistent with the leader is Dr Christiansen's point that the absence of B27 might be helpful in diagnosis.—ED, *BMJ*.

[1] Kidd, K K, *et al*, in *HLA and Disease*, ed J Dausset and A Svejgaard, p 72, 1977.

Another hazard of pierced ears

SIR,—I should like to draw readers' attention to another hazard of the present fashion for pierced ears. Three children from the same family attended our casualty department 24 h after having their ears pierced at a reputable Sheffield jewellers. They had all received ethyl chloride spray to anaesthetise their ear lobes before piercing.

However, instead of the usual 20-30 seconds' worth of spray, each received several minutes of spraying to each lobe (the mother thought about seven minutes in one case). All the children had deep purple areas of chemical "frost bite" from their ears in a thick area 2 in (5 cm) wide across their necks running to the midline of their throats. These burns have subsequently completely healed with conservative treatment without leaving a scar.

D A NOBLE

Accident and Emergency Department,
Children's Hospital,
Sheffield

Polymyalgia rheumatica and primary biliary cirrhosis

SIR,—In reply to the comments of Drs A L Ogilvie and P J Toghill (25 November, p 1501) on our case reports (21 October, p 1128), we are aware that hepatic biochemical abnormalities occur in polymyalgia rheumatica (PMR) and, indeed, not uncommonly in the investigated population. However, the abnormalities we found were gross, consistently so, and were indicative of hepatic disease. The antimitochondrial antibody titres were high and remained so in our unbiopsied patients. Our patients did not have serositis, fever, or peripheral arthritis or arthralgia. Therefore we cannot agree that the systemic lupus erythematosus-like syndrome they mention should enter in the differential diagnosis.

Liver biopsy was not performed on two patients. One was a recluse and it reflects credit on her daughters and her general practitioner, Dr Nigel Shield, that we were able to investigate her as far as we did. A biopsy would not have altered management and neither would it have done so in our first case, in which it would probably not have contributed to diagnosis either. We believe that careful and repeated biochemical examination is sufficient to establish a probable diagnosis of chronic hepatitis in appropriate clinical circumstances. Walker *et al*[1] noted that in all of their biopsied cases the results confirmed the biochemical findings of significant hepatic abnormality. We also note that one of their patients, a doctor, refused liver biopsy because there were no hepatic symptoms.

With regard to Drs Ogilvie and Toghill's comments concerning our mention of a first case report of PMR and chronic hepatitis, we are surprised at their use of the word "categorically." Our reading of the text was that