erythromelalgia-like conditions under secondary erythralgia. As yet, none the less, we have not observed this association.

The suggestion that the relation between connective tissue disorders and secondary erythralgia is purely coincidental is highly speculative and certainly not based on scientific evidence. Dr Carmichael and colleagues state that "All is not red or white in the classification of erythromelalgia"; true enough in every clinical classification. Our objective was to clarify the existing information. We agree that the arrangement in primary and secondary erythralgia is made merely on clinical grounds in the absence of clear cut laboratory indicators. In general, however, patients with burning pain and red swollen extremities can be assigned with our classification.

Finally, the threat of therapeutic nihilism is limited because using our classification does not interfere with common and sound therapeutic practice.

J P H DRENTH

Dutch Medical Centre,
Afcnt Brunsum,
The Netherlands

J J MICHIELS

University Hospital,
Erasmus University,
Rotterdam,
The Netherlands

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Tanning with ultraviolet A sunbeds

SIR, — Dr B L Diffey and others argue against using ultraviolet A sunbeds.¹ This is quite logical as the evidence suggests that exposure to ultraviolet light is harmful. The public, however, are unlikely to heed such advice unless the risks are perceived as overwhelming, and current behaviour indicates that sunbathing is viewed more like alcohol than smoking: enjoyable in moderation and dangerous only in excess.

Two questions therefore need to be asked. Firstly, Is it possible to "cheat" by obtaining a tan with ultraviolet A and not run the risk of skin cancer? And, secondly, Can using a sunbed before an annual holiday in the Mediterranean "protect" against the risk of melanoma? The answer to the first question is probably no. In humans the action spectrum for tanning exactly parallels that for erythema,² and in hairless albino mice the action spectrum for erythema is a good approximation of the spectrum for carcinogenesis induced by ultraviolet.³ Experimental data also show that the induction of squamous cell carcinoma is a function of total dose.⁴ In terms of non-melanoma skin cancer the idea of a safe tan is therefore a delusion.

The answer to the second point is more difficult. The incidence of cutaneous malignant melanoma is increasing, and this has been attributed to changing recreational habits.⁵ Epidemiological data indicate that intensity of exposure to ultraviolet is more important than cumulative dose.⁶ Skin type is a risk factor, but so is a history of sunburn in childhood.⁷ Apart from lentigo maligna melanoma, cutaneous malignant melanoma is the only skin cancer induced by ultraviolet for which indoor workers are more at risk than outdoor workers. The relative risk of cutaneous malignant melanoma is lower in those who are exposed all year round than in those who are exposed for

short periods, even though total doses may be equivalent.⁸ These data suggest that tanning is protective and that people planning holidays in areas of high ultraviolet intensity might reduce their melanoma risk by promoting a tan before departure. An ultraviolet A sunbed is arguably a better method of tan promotion than sunlight as ultraviolet dose can be predicted more accurately and burning can be avoided. Unfortunately, ultraviolet A tanning offers little protection against erythema induced by ultraviolet B.⁹ Tolerance to ultraviolet radiation is not dependent just on melanogenesis. Increased thickness of the epidermis and stratum corneum is also important, and this seems to be mainly caused by ultraviolet B.¹⁰

In summary, the use of sunbeds cannot be justified on either count. Gentle tan promotion may be protective for those contemplating holidays abroad, but those with skin types I or II would be better advised to avoid exposure to ultraviolet altogether.

ROBIN RUSSELL JONES

St John's Dermatology Centre,
St Thomas's Hospital,
London SE1

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The new new general practice

SIR, — Those thousands of us who came home from the war in the late 1940s to approve, join, and establish the NHS must defend ourselves from the patronising inaccuracies of the article by Drs Anna Livingstone and David Widgery in the anniversary issue.¹

Country general practice in Somerset, with our long established cottage hospitals, added a range of consultant outpatient clinics and "cold" surgery in our operating theatre within months in 1948 and, by 1953, our own maternity unit with resident staff, and soon a geriatric unit as well, sufficient for our needs. The domiciliary consultant service was prompt and rewarding to patient and practitioner alike. Certainly we practised from our homes, and our lives were an integral and vital part of the service we gave. We knew patients by their names in a relaxed atmosphere and in complete confidentiality. Our local medical societies held lectures by well known experts speaking on their own original work, and of course vocational training began 20 years before 1980, with away courses all over the country. And the trainee system was well established.

The 1966 charter arose from general practitioners' pressure, and two thirds of us placed our resignation papers in the hands of the BMA to strengthen its position. The minister responded

within hours and many reforms came in, which we wanted. We were not hampered by suspension of the Rent and Rates Scheme if we attempted to resign then, for we owed them nothing.

To suggest that we had no antibiotics available before 1948 is ridiculous. The growing range of sulphonamides was available to us from 1939 onwards, and families of "panel patients" who were not covered for the cost of them were often supplied from the practice if they could not otherwise afford drugs. Penicillin was available in 1949.

Our mentally ill and mentally handicapped patients were cared for in hospital when necessary, and were not on the streets or in prison or under the underground trains. And waiting lists were a fraction of what they are now because Nye Bevan called them "a blasphemy."

The first 25 years of the NHS were years of achievement of which we were proud. And the constant and justified complaint by the public, which is now sadly so common, had not come to pass—ask the older patients.

MICHAEL BOURKE

Shepton Mallet,
Somerset BA4 5LW

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Sodium-lithium countertransport activity in red blood cells

SIR, — We would like to comment on the paper by Dr James D Walker and colleagues.¹ The authors found no significant difference in mean blood pressure between the two groups of parents. Previous reports from the authors' own² and other³ laboratories have shown significantly higher blood pressure in parents of patients with diabetic nephropathy. This family history of hypertension has been held responsible for the raised sodium-lithium countertransport observed in diabetes. This latest study disagrees with this hypothesis and its genetic implications, but this is not commented on in the discussion.

The authors calculate a mid-parental value for sodium-lithium countertransport and use this in postulating modes of inheritance. But they give no justification for this calculation and no precedent in other biological measurements. They state that an increased maximum velocity was observed. We and others have shown that the standard assay for countertransport is performed at a non-saturating external sodium concentration so maximum velocity is not measured.^{4,5}

Pearson correlation coefficients are dubious with such outliers as in their first figure. In addition, our statistical tables indicate that an *r* value of 0.37 (*n*=40) has borderline significance, with a *p* value around 0.05 not <0.001.

It is known that hyperlipidaemia can lead to raised sodium-lithium countertransport, yet this seemed to be excluded by history alone. The authors argue that previous studies have shown that 80-90% of variance in sodium-lithium countertransport in the general population is explained by genetic transmission.^{6,7} One study⁶ showed 80% variability in pedigrees selected because of cardiovascular disease and is unlikely to be applicable to the general population, and the study in the general population found that only 50% of variability was attributable to genetic factors.⁷

Finally, they state that raised sodium-lithium countertransport in hypertensive patients is associated with increased risk of renal and cardiovascular complications. The studies quoted in fact showed the incidence of raised countertransport in

patients with renal disease and hypertension, not the incidence of renal disease in subjects with raised countertransport (which is not known).^{8,9} It cannot be concluded, therefore, that patients with essential hypertension and raised countertransport are at increased risk of nephropathy.

P A RUTHERFORD
T H THOMAS
R WILKINSON

Freeman Hospital,
Newcastle upon Tyne NE7 7DN

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AUTHORS' REPLY.—We appreciate the comments made by Dr P A Rutherford and colleagues concerning our study. In the penultimate paragraph of our discussion we commented on the question of arterial blood pressure. Although actual blood pressure was similar in the two groups of parents, more parents of patients with proteinuria were being treated for hypertension. We believe that this does not contradict earlier reports but we recognise that the relation between blood pressure and sodium-lithium countertransport is complex.

The overactivity of sodium-lithium countertransport is believed to represent the expression of a susceptibility rather than a disease gene¹; thus arterial hypertension must result from the interaction of different factors, both environmental and genetic, with abnormalities of countertransport. Our original finding of higher blood pressure in the parents of diabetic patients with proteinuria was obtained in a population studied in the early 1950s, when treatment of hypertension was not readily available. The introduction of pharmacological or other interventions in recent years (for example, diet, exercise, increased awareness of risk, etc) may also have contributed to blur this relation.

We showed a difference in sodium-lithium countertransport activity not only when mid-parental values were used but also when all individual parental values were analysed. Moreover, we used all values to calculate proportions below and above the median value and to postulate modes of inheritance. Maximum velocity in our paper relates to velocity measured at physiological external sodium concentration, which, we are aware, is not saturating.

The p value for the correlation of countertransport between parents and patients was 0.033 by Pearson correlation and 0.006 by Spearman's. We apologise for the typing error. This correlation remained significant even when the outlier value was removed (Pearson's $r=0.36$, $p<0.024$; Spearman's $r=0.40$, $p<0.012$). The

association between hyperlipidaemia and countertransport does not imply a cause and effect relation, and we thought that excluding hyperlipidaemia by history was adequate in this study.

We agree that in the general population roughly half of sodium-lithium countertransport is attributable to genetic factors and simply stated that in certain subgroups this could be as high as 80-90%. We accept that the higher prevalence of raised countertransport in patients with renal disease and hypertension does not imply that hypertensive patients with high countertransport are at greater risk of renal and cardiac complications. Strong evidence for this is emerging, however, from several recent studies that show an increased renal and cardiovascular risk in hypertensive patients with high countertransport.^{2,3}

G C VIBERTI
R TREVISAN
T TARIQ
J D WALKER

Guy's Hospital,
London SE1 9RT

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Early HIV infection

SIR.—The peripheral blood CD4+ cell counts quoted in the editorial by Dr Ann Marie Swart and colleagues as risk markers of progressive HIV disease are wrong by a factor of 10.¹ The critical CD4+ cell counts quoted by Volberding *et al* are 200/mm³ and 500/mm³,² which convert to $0.2 \times 10^9/l$ and $0.5 \times 10^9/l$ respectively (not $2 \times 10^9/l$ and $5 \times 10^9/l$ as stated in the editorial).

This confusion arises presumably because the authors, as do most doctors, use counts per cubic millimetre in everyday medical language, whereas the SI convention adopted by the *BMJ* and many automated cell counters refers to cells in thousands of millions per litre. The attempt to standardise cell counts in this cumbersome way has been sustained for long enough. Perhaps " $\times 10^9/l$ " should be given the same treatment as the SI unit for blood pressure, the kilopascal.

MARK WALKER

Royal Preston Hospital,
Preston PR2 4HG

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*Sorry—Ed, *BMJ*.

Drug Points

Colitis associated with ibuprofen

Drs D CLEMENTS and G T WILLIAMS, and Professor J RHODES (University Hospital of Wales, Cardiff CF4 4XW) write: Proctitis due to indomethacin suppositories is well recognised.¹ Oral non-steroidal anti-inflammatory drugs have been implicated in exacerbations of colitis, but they have been thought to be the cause in surprisingly

few cases.^{2,7} The fenamates, mefenamic acid and flufenamic acid, are well recognised as causing diarrhoea, and colitis associated with their usage has been reported.^{3,6} Patients who develop colitis when taking mefenamic acid may tolerate other nonsteroidal anti-inflammatory drugs without a recurrence of diarrhoea or colitis. Two cases of proctitis associated with ibuprofen and naproxen have been reported.³ We report on a patient who developed colitis after taking ibuprofen.

A 63 year old man was admitted as an emergency with diarrhoea, urgency, and rectal bleeding. He had had no gastrointestinal symptoms until eight weeks before admission, when he had started taking ibuprofen (400 mg three times a day) for back pain. Shortly after starting this his diarrhoea began and was associated with urgency. He was passing watery stools with blood 15-20 times a day. He had some left iliac fossa pain before opening his bowels. He had no family history of inflammatory bowel disease and had had no contact with anyone else with diarrhoea. He had given up smoking seven years previously, his general health was otherwise good, and he had not lost weight.

On examination he was well with no fever or tachycardia. Abdominal examination gave normal results. There was no perianal disease, and sigmoidoscopy confirmed a symmetrical proctitis. A rectal biopsy specimen showed diffuse moderate acute-on-chronic inflammation of the large bowel mucosa. Neutrophil polymorphs invaded the crypt epithelium, and there were occasional crypt abscesses. No granulomas, lymphoid aggregates, or parasites were present, and there was no appreciable architectural disturbance. A full blood count and biochemical profile gave normal results, and the plain abdominal film showed faecal loading of the right and proximal transverse colon, while the descending colon appeared oedematous. Three stool cultures were obtained but no pathogens were isolated.

Ibuprofen was withdrawn, and because of his severe symptoms he was started on both oral and topical steroids, with mesalazine 400 mg three times a day. Within two days his motions were normal and he was discharged home. Three weeks after discharge he had maintained his improvement and steroids were withdrawn. Three months after presentation sigmoidoscopy and rectal biopsy gave normal results. He remained well and asymptomatic after 12 months without receiving all treatment. Barium enema examination, sigmoidoscopy, and rectal biopsy gave normal results. He has not been rechallenged with ibuprofen.

This case shows that non-steroidal anti-inflammatory drugs other than the fenamates can cause colitis. Ibuprofen is now available without prescription, and patients may not volunteer its usage; the association could therefore pass unrecognised. In the only other report of colitis associated with ibuprofen histological examination was unavailable; in our case there was complete histological resolution. There were no clinical or histological features suggestive of Crohn's disease or specific infection. Non-steroidal anti-inflammatory drugs should be considered in all patients presenting with colitis.⁸

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