for skilled advice on occupational health matters, and in consequence many previously neglected environmental problems have come to light which call for medical help. It is the duty of those who undertake to advise them to make it plain that these are the important tasks, and that occupational medicine does not consist of routine examinations of new employees on the pretence that the doctor can forecast future sickness absence experience or survival.

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¹ Taylor, P J, *British Journal of Industrial Medicine*, 1967, 24, 93 and 169.

² Taylor, P J, *British Journal of Industrial Medicine*, 1968, 25, 106.

³ Taylor, P J, *Transactions of the Society of Occupational Medicine*, 1968, 18, 96.

Uniform style for biomedical journals

SIR,—The decision of the International Steering Committee of Medical Editors (20 May, p 1334) to recommend the adoption of the style of abbreviations of names of journals as used in *Index Medicus* will be welcomed by most medical librarians, as this decision follows the recommendations of the Library Association Medical Section.¹ May I, however, point out a current trend which is rather disturbing?

The redesign of journal covers, in line with modern design practice, often gives prominence to the initial letters of the title. The example known to most of us is the *Journal of the American Medical Association* (known almost everywhere as *JAMA*). Thus the current list of journals indexed in *Index Medicus*² includes, as well as *JAMA*, the following: *AJR*, *JAOA*, *JCU*, *JNM*, *JOM*, and so on, but the most striking example is that two journals are listed as *ZFA* (Dresden) and *ZFA* (Stuttgart), where the A is not even the abbreviation for the same word.

The adoption of an international standard for the abbreviation of titles of periodicals is highly desirable, and was pioneered by the *British Medical Journal* with its compilation of *World Medical Periodicals*,³ now sadly out of date. The scheme used by the National Library of Medicine for *Index Medicus* follows very closely ISO-4-1972,⁴ with the exceptions noted above, but I wonder how long it will be before it is reduced to a set of incomprehensible initials.

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¹ Library Association Medical Section, *Reference Citation Recommendations*. London, Library Association, 1972.

² United States National Library of Medicine, *List of Journals Indexed in Index Medicus*. DHEW Publication No (NIH) 78-267. Washington, US Govt Printing Office, 1978.

³ Fleurent, C H A, (editor), *World Medical Periodicals*, 3rd edn. New York, World Medical Association, 1961.

⁴ International Standards Organisation, *International Standard ISO-4-1972 (E). Documentation—International Code for the Abbreviation of Titles of Periodicals*. Geneva, ISO, 1972.

Axillary hyperhidrosis

SIR,—Dr David Bird's helpful letter (29 July, p 357) adds a further five patients to the lengthening list of cases of axillary hyperhidrosis whose distressing symptoms have been eliminated by the use of a modification

of the regimen originally reported by Hurley and Shelley.¹

I write only to amplify a sentence of Dr Bird which I think might perhaps mislead. He suggested: "It is too soon to abandon surgery completely since the long-term results of this topical treatment are not known." He clearly refers to the possibility of loss of effect of the aluminium chloride hexahydrate preparation with the passage of time. Although we considered it too early to include it in our paper, the average time between treatments in our patients has steadily lengthened, so that an increasing number now have to make applications only once every two to three weeks.

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¹ Hurley, H J, and Shelley, W B, *Journal of the American Medical Association*, 1963, 186, 109.

Dangers of glass doors

SIR,—I have just come across yet another incident of an extremely serious injury involving a glass door. One wonders whether the aesthetic value of such doors justifies the injuries that occasionally occur. It seems to me that it ought to be illegal to use full length glass doors, screens, or windows without a horizontal dividing or preventive bar, strong enough to take the weight of a fairly heavy person falling against it. An alternative would be to use armoured glass.

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Atenolol self-poisoning

SIR,—The recent reports provide several examples of self-poisoning with β -adrenoceptor blocking agents. The outcome of these cases has varied from being relatively uncomplicated¹ to fatal.² The opinion that these agents, even in massive overdosage, represent little hazard to the normal healthy subject whose myocardial performance is not dependent on sympathetic drive has already been expressed.³ We therefore consider it of interest to report what we believe is the first recorded case of atenolol self-poisoning in a patient with normal cardiac function.

The patient, a 24-year-old woman with a psychiatric history, was being treated for hypertension secondary to chronic pyelonephritis. In recent months her blood pressure had been well controlled on a combination of clonidine and atenolol. A recent assessment of her renal and cardiac function (including serum urea, electrolyte, and creatinine estimations and electrocardiography) showed nothing abnormal. In attempting suicide she took about 1200 mg of atenolol, the number of tablets being reliably confirmed by a relative who was familiar with her treatment and her current supply.

On admission to hospital, two to three hours later, her general condition was very good. Her

pulse rate was 80 beats/minute, with a recumbent blood pressure of 150/110 mm Hg. There were no signs of cardiac decompensation. None of the tablets were recovered from the gastric contents on lavage. She was closely monitored over the ensuing days both clinically and electrocardiographically, during which time her pulse rate varied between 66 and 80 beats/minute in sinus rhythm. Renal function remained normal. With the temporary withdrawal of all medication, however, her blood pressure rose gradually, reaching 190/120 mm Hg five days later. Control was then re-established with a combination of atenolol and prazosin. Her further clinical course was uncomplicated.

In addition to normal myocardial function, other features are worthy of mention. Although tolerance to most beta-blockers is very variable, atenolol has a much narrower effective dose range.^{4,5} Doses over the average 100-200 mg/day regimen, although further affecting pulse rate, do not confer any additional benefit in hypertension. Large doses of atenolol have been well tolerated by volunteers,⁴ after gradual titration, and perhaps the existing beta-blockade in our patient conferred a similar protective titrating effect.

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¹ Wermut, W, and Wojcicki, M, *British Medical Journal*, 1973, 3, 591.

² Khan, A, and Muscat-Baron, J M, *British Medical Journal*, 1977, 1, 552.

³ Boakes, A J, and Boeree, B H, *British Medical Journal*, 1973, 4, 675.

⁴ Zacharias, F J, and Cowen, K J, *Postgraduate Medical Journal*, 1977, 53, suppl No 3, p 111.

⁵ Zacharias, F J, Hayes, P J, and Cruickshank, J M, *Postgraduate Medical Journal*, 1977, 53, suppl No 3, p 114.

Efficacy of rubella vaccination

SIR,—The timely letter from Dr W J C Roberts on the efficacy of rubella vaccination (5 August, p 433) reminds us that not all women shown by laboratory tests to be susceptible to rubella develop rubella haemagglutination-inhibiting (HI) antibodies after vaccination. Unfortunately he does not name the vaccine he used or the dilution of the patients' serum without antibody which indicated susceptibility. If facilities are available we would support his suggestion of testing a postvaccination serum sample and revaccination if the patient has not developed antibodies.

In some maternity units in this area post-natal rubella vaccination with Cendevax is offered to patients if previous laboratory tests have shown that they are susceptible to rubella. A serum sample is collected approximately eight weeks after vaccination (unfortunately this period varies with circumstances), and the pre- and post-vaccination serum samples are tested in parallel for rubella antibodies. The table shows that during the period February 1977 to March 1978 4.9% of women with a titre of <1/8 failed to produce HI antibodies after vaccination. Of the four postvaccination serum

Post-vaccination rubella HI antibodies in serum from 82 women with titres <1/8 who were vaccinated with Cendevax

No tested	Weeks after vaccination when serum was collected	Post-vaccination rubella HI antibody titres							
		<1/8	1/8	1/16	1/32	1/64	1/128	1/256	1/512
62	5-9 (mean 7)	4	1	4	16	25	10	2	—
20	≥10 (mean 22)	—	2	3	5	6	1	2	1

samples from patients who failed to develop antibody, three were collected at 7½–8 weeks after vaccination and one at 6 weeks. It is possible that if these samples had been collected later antibody may have developed, so in the future it may be advisable to collect the postvaccination serum after a longer interval.

The results on 82 patients indicate that successful vaccination was higher in our group than in that described by Dr Roberts, but it would still appear to be worth while to examine samples collected at least 10 weeks after vaccination to confirm the presence of rubella antibodies, or the possible need for revaccination.

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Infection with Epstein-Barr virus

SIR,—There are two puzzling features in the report by Drs Hillas Smith and A M Denman on a new manifestation of infection with Epstein-Barr (EB) virus (22 July, p 248).

Firstly, they say that the antibody titre to EB virus capsid antigen (indirect immunofluorescence) was 1/5 on 4 March and rose to 1/20 on 26 July and 1/40 on 13 September. This was a remarkable delay in an antibody which normally rises so early as to be of little help in the diagnosis of infectious mononucleosis. Two sentences later they state that "EB virus IgG was 1/512 on 9 March." The most commonly measured "EB virus IgG" is antibody to the virus capsid antigen, said to be 1/5 five days earlier and 1/20 three months later. Can the authors offer some clarification of these conflicting statements?

Secondly, they report that four weeks after admission the titre of complement-fixing antibody to cytomegalovirus was 1/1024 and specific IgM antibody was 1/32. These titres, which would normally be considered good evidence of recent infection, are dismissed as "an anamnestic response." How vigorously was cytomegalovirus sought by repeated culture of urine, throat, and cervical swabs?

Their evidence for infection by EB virus is (a) a Paul-Bunnell-Davidson titre which they describe as "weakly positive," (b) the presence of anti-i antibody, titre unstated, and (c) their conflicting statements about EB antibodies.

Should not cytomegalovirus either alone, or with EB virus infection, also be considered in discussing the aetiology of this illness?

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Intravascular coagulation in falciparum malaria

SIR,—Professor J Vreeken and Mr Th M Cremer-Goote (19 August, p 533) studied nine patients with falciparum malaria and reported a fall in plasma fibrinogen together with thrombocytopenia. They concluded that diffuse/disseminated intravascular coagulation (DIC) was not present, and postulated the

adsorption of fibrinogen to cell membranes or leakage from the microcirculation as alternative mechanisms for the hypofibrinogenaemia.

Inflammatory conditions, including malaria, give rise to an acute-phase rise in the concentration of plasma fibrinogen and other proteins. The activity of coagulation factors in falciparum malaria should therefore be compared not only with normal values obtained from healthy controls but also with reference values obtained from other patients undergoing a similar inflammatory response.

We recently completed a serial coagulation study of 37 patients admitted with bacterial, viral, or protozoal infection.¹ Patients with vivax malaria, compared with healthy uninfected controls, showed statistically significant thrombocytopenia, hyperfibrinogenaemia, and raised factor VIII antigenic (VIII:Ag) and coagulant (VIII:C) activity. Patients with falciparum malaria, when compared with vivax malaria, showed a statistically significant reduction in fibrinogen and platelets, increase in the ratio of factor VIII:Ag to factor VIII:C, and a positive serial-dilution protamine sulphate test for fibrin monomer. There was also a reduction in antithrombin III and increase in fibrin degradation products.

We would recommend the above approach in future coagulation studies in malaria to distinguish the acute-phase protein effect. Patients with falciparum malaria show evidence of coagulation activation; in some patients this may be interpreted as DIC, although the term is now used so loosely as to lack precise biological meaning.

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¹ Richardson, S G N, et al, *British Journal of Haematology*. In press.

Treatment of narcotic poisoning

SIR,—The most recent articles on this subject still recommend that patients severely poisoned by certain narcotic drugs should be treated by eliminative procedures. In the popular *Treatment of Common Poisonings*,¹ which is widely used as a reference book by casualty officers, the authors repeatedly emphasise that most patients recover without using methods to eliminate the poison. They nevertheless state categorically that when the plasma salicylate concentration exceeds 500 mg/l intensive forced diuretic treatment *must* (my italics) be started, and they also advocate eliminative measures for severe lithium, meprobamate, chloral, primidone, barbitone, and pheno-barbitone poisoning. Lawson² again insists that most poisoned patients should not be given anything beyond supportive therapy, but he also states: "There is no doubt that there are instances when these measures of elimination are extremely valuable and may be life-saving." Vale,³ dealing with paediatric poisoning, likewise says that eliminative measures have a small place, but lays down that if the plasma salicylate concentration exceeds 500 mg/l forced alkaline diuresis should be instituted.

The only reason why eliminative measures are recommended is that some of the poison is thereby removed from the body. But this does not prove that the patient's life will be saved. In assessing the value of treatment for a condition which varies infinitely in severity and from which the vast majority of patients

recover without treatment—and poisoning is a typical example of such a condition—the only means of reaching the truth is by a controlled trial on a very large scale. Indeed, this has become part of the conventional wisdom of medicine when drugs and operations are being assessed, but for reasons which are not clear to me a less critical approach has been taken towards eliminative treatment for poisoning. We would do well to remember that analeptic drugs were widely recommended by the experts 25 years ago in treating the poisoned, and a *Lancet* leading article⁴ commented: "A succession of case-reports illustrated how bemegride could aid the recovery of many seriously poisoned patients; and experienced clinicians believed that many of these patients would have died if bemegride had not been used." I pointed all this out in an article in the *Lancet* in 1973,⁵ and no one disputed my views in the correspondence columns. I even wrote to one of the most eminent advocates of eliminative measures and urged him to reply to me in the *Lancet*, but he declined on the main ground that any comments would occupy several pages.

As eliminative treatment is without doubt hazardous and as there is no worthwhile evidence that it is beneficial, I urge that it should no longer be used (except perhaps by those taking part in a controlled trial). Failing this, I urge that those who advocate eliminative measures should at least point out that they are doing so solely for theoretical reasons.

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¹ Matthew, H, and Lawson, A A H, *Treatment of Common Acute Poisonings*, 3rd edn. Edinburgh, Churchill Livingstone, 1975.

² Lawson, A A H, *British Journal of Hospital Medicine*, 1976, 16, 333.

³ Vale, J A, *Prescribers' Journal*, 1978, 18, 67.

⁴ *Lancet*, 1956, 2, 980.

⁵ Todd, J W, *Lancet*, 1973, 2, 1067.

Secret list of MCQs

SIR,—The value of employing repeatedly a secret list of multiple choice questions in various final medical examinations including the MRCP is now suspect.

Either a single copy of these questions may be smuggled out of the examination room despite the strict rules regarding such papers or students are asked to memorise two or three of the questions and give them to their teachers for future reference. By this means the secrecy is destroyed. The comments of students suggest that many of the questions are controversial and cover rare conditions. Surely there is no reason why the profession should not be allowed to see these questions, and if they are so limited in number the object of the examination is lost. Again, regarding the marking of papers, the mark-down for an error or guess seems irrationally severe.

Although marking the papers has been made easier, it is regrettably essential to reconsider the advisability of this type of examination. Although there are many books being produced displaying multiple choice questions and answers, the tendency does seem to invite abuse as well as rewarding some students who have a particular aptitude for this type of question?

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