distances or who have a compulsive attraction to the set may be provided with a pair of spectacles with one lens polarised with an axis orthogonal to that of a sheet of polariser placed over the television screen.[6] The occlusion is cosmetic, is specific to the television, and has been shown to prevent the occurrence of seizures and to reduce considerably the incidence of paroxysmal activity. The protection is restricted to televisions that have been "treated," and acceptability varies considerably from one patient to another, many patients complaining of discomfort—sometimes such that the glasses are not tolerated. Any discomfort that does occur may be partially overcome by providing two pairs of spectacles, one occluding the left eye and the other the right, the patient alternating between pairs as desired. Alternatively, the axes of polarisation can be altered to admit binocular viewing at low luminance levels.

In the extensive series of photosensitive patients collected by Jeavons and Harding[1] half were sensitive to intermittent photic stimulation at 50 Hz, whereas only 15% were sensitive at 60 Hz. At 25 Hz and 30 Hz the proportion was over 70%. These findings suggest that System M televisions are less epileptogenic, but only at normal viewing distances. The clinical picture of television epilepsy in the United States may, therefore, differ from that in Europe.

[1] Jeavons, P M, and Harding, G F A, *Photosensitive Epilepsy*. London, Heinemann, 1975.
[2] Charlton, M H, and Hoefer, P F, *Archives of Neurology*, 1964, **11**, 239.
[3] Stefansson, S B, *et al*, *British Medical Journal*, 1977, **2**, 88.
[4] Darby, C E, *et al*, *Electroencephalography and Clinical Neurophysiology*, 1977, **43**, 577.
[5] Wilkins, A J, Darby, C E, and Binnie, C D, *Electroencephalography and Clinical Neurophysiology*, 1978, in press.
[6] Wilkins, A J, *Epilepsy News*, 1978, **6**, 6.
[7] Darby, C E, and Hindley, A T, *Proceedings and Journal of the Electrophysiological Technologists' Association*, 1974, **21**, 4.
[8] Pantelakis, S N, Bower, B D, and Douglas Jones, H, *British Medical Journal*, 1962, **2**, 633.

Declaration of Vancouver

Authors have long complained about the lack of uniformity in the house styles of biomedical journals—for so long, indeed, that editors have probably shrugged off their grouses as yet another component of the redactorial scene, along with complaints about delays in deciding on publication and about anonymous editorials. Although there have been other irritants, almost certainly the major complaint has been about the different styles of references. At last November's *BMJ Else** conference one speaker claimed that a random survey of 52 journals had shown no fewer than 33 different styles,[1] but this was an underestimate: there are said to be 2632 possibilities.[2] Secretaries and subeditors waste much valuable time in adapting references to a particular journal's style, and not surprisingly ten years ago a medical secretary finally wrote to six American clinical journals to ask why every journal should have "its own little pet form of references." This time, however, the complaint led to action: a group of editors of 30 American clinical journals met and agreed to use the style of *Index Medicus*,[3] and three years later the editors of a group of biochemical journals took a similar step.[4]

These American agreements were based on adopting the numbering system of references in the text, and since then several organisations have tried to see whether this style could not be extended to other journals in other countries. Experience has shown that in practice neither of the major objections always raised to the numbering system are valid. Thus in multi-author papers using the Harvard system (where the names of the authors and the date are cited in the text and the reference system is in alphabetical order) the name of the head of the unit is often so well down the list that in the shortening imposed during subediting to Brown, Jones, Smith *et al* it does not appear—and hence the seal of quality given by a well-known name is no longer present. In practice, also, authors are found not to want to add another reference to the middle of a long list at the proof stage, and if this is essential, the editor can usually persuade the author to delete another less-essential reference near the added one.

Two other factors have emphasised the advantages of considering switching to the numbering system, Firstly, this makes the reader's task in finding what he wants quicker and easier,[5] and, secondly, by 1977 over two-thirds of American biomedical journals were using this style and the proportion was growing. On the other hand, there remains the problem that many scientists submit articles to journals in several different disciplines where the principal reference style is still the Harvard system. Recognising this, the Else Ciba Foundation workshop (which was attended by editors and others representing the earth and life sciences, chemistry, physics, and engineering) made an ingenious proposal that would avoid the necessity to have the complete text retyped if after rejection by one journal it had to be sent to another with a different style of references. Essentially this envisages a master text in which names and dates are typed initially but may then be deleted in favour of the numbering system, and the reference lists are styled accordingly but in a standard way.[6]

Clearly to bring the 2632 possibilities for reference styles down to two at a stroke would be no mean feat. Nevertheless, a group of editors of major clinical journals from the USA, Canada, and Britain believed that they could go one better for their own publications. Meeting in Vancouver last January they produced a set of uniform requirements for manuscripts, which we print in draft on p 1334. These cover more than just reference styles, dealing with standard requirements for the various sections of the article, abbreviations, preparation of the manuscript, illustrations, and so on. After comments have been received on this draft, a final version will be produced, printed as a booklet, and made available to authors in both Europe and the USA. The international steering group hopes that any changes will be introduced on or before 1 January 1980.

These uniform requirements are not intended as a directive to editors about their house styles—merely that they should be prepared to accept manuscripts which follow the requirements and to insert their own conventions (such as italics or full points) during subediting. Moreover, they are only a beginning of an approach to greater uniformity among journals, which could include in future, for instance, an agreement on when these should start using SI as well as traditional units. But, given that the proposals are widely adopted, editors might consider being harsher in what they require from authors.

Handling original articles is expensive: each of the 3500 or so papers the *BMJ* receives every year costs upwards of £15 in office and postal charges alone—a fact recognised by some journals by the introduction of handling charges (recently, for example, *Blood* has begun to charge its contributors $30 per article). Once accepted, articles are even more expensive to subedit,[7] yet many of the changes have to be made merely because their writers have taken no pains to follow our instruc-

*European Life Sciences Editors.

tions to authors (printed in full in the first issue of every year and latterly, abbreviated, in the News pages every week). We do not want to impose handling charges for every article and page changes for the accepted paper, because all these would do is deprive medicine of much needed funds for research. Nevertheless, eventually we and many other journals may be forced by economic considerations to decline to consider for publication any article that does not use the standard conventions.

[1] *British Medical Journal*, 1977, **2**, 1428.
[2] Williams, P C, *Units, Symbols, and Abbreviations*, ed G Ellis, p 35. London Royal Society of Medicine, 1972.
[3] *New England Journal of Medicine*, 1970, **282**, 48.
[4] IUB Commission of Editors of Biochemical Journals, *Biochemical Journal*, 1973, **135**, 1.
[5] Garfield, E, *New Scientist*, 1968, **39**, 565.
[6] O'Connor, M, *British Medical Journal*, 1978, **1**, 31.
[7] Smith, J, *British Medical Journal*, 1978, **1**, 222.

Ebstein's anomaly

Ebstein's anomaly of the tricuspid valve[1] is one of the rarer congenital cardiac malformations. Attention has been called to it in two recent articles: one showed that the prognosis was generally good with conservative management,[2] while the other showed the feasibility of surgical treatment.[3]

The basic anomaly is a downward displacement of the attachment of the base of the tricuspid valve from the true valve ring into the cavity of the right ventricle. The medial and inferior leaflets are those most frequently affected, but sometimes the whole valve is displaced.[4] As a result of the displacement the right ventricle is divided by the tricuspid valve into a distal, effective ventricle and a proximal "atrialised" portion which is thin-walled and poorly contractile. While the thickness of the distal ventricle is usually normal in some instances it, too, is attenuated. The relative sizes of the cavities of the effective right ventricle and the atrialised portion depend on the degree of displacement of the valve, and this is extremely variable.[4] The tricuspid valve usually shows additional abnormalities. The leaflets may be fused at their edges, sometimes leading to tricuspid stenosis, or they may be perforated and sometimes portions are entirely absent.[6]

Even in its simplest form Ebstein's anomaly is, then, of highly variable severity, depending on the degree of displacement and dysplasia of the valve and the size and function of the effective right ventricle. Even this is not the whole picture, since half the affected patients have additional cardiac abnormalities. The most common is atrial septal defect or patent foramen ovale. About 20% have more serious abnormalities including ventricular septal defect, pulmonary stenosis or atresia, the tetralogy of Fallot, mitral stenosis, and corrected transposition of the great arteries. Finally there is a high incidence of cardiac arrhythmias, sometimes associated with Wolff-Parkinson-White type B pre-excitation.

The rarity of Ebstein's anomaly (less than 100 children per year in Britain) and the diversity of the lesions have made the assessment of its clinical course and natural history difficult. The presentation may be with an asymptomatic cardiac murmur, with an arrhythmia, with cyanosis due to a right to left shunt through an atrial septal defect or foramen ovale, or with congestive cardiac failure. The course of the disease depends on the severity of the basic tricuspid valve lesion and the presence or absence of associated cardiac abnormalities.[7 8] Certain facts are evident. A poor prognosis is associated with presentation in the first year of life, almost half of such patients dying early.[7 8] Once through this danger period the prospects for survival into adult life are good.[2] Nevertheless, at any age the appearance of cyanosis indicates a poor prognosis, while the onset of congestive cardiac failure is a particularly grave sign, few patients surviving for more than a few years.

Fortunately, in older children and adults surgery is feasible.[3] The procedure consists of tricuspid valve replacement, obliteration of the atrialised portion of the right ventricle, and closure of the patency of the atrial septum, if present.[3 9] Surgical treatment is generally reserved for those patients with cyanosis or congestive cardiac failure, while asymptomatic patients are treated conservatively. This approach should maximise the chances of survival for adults and older children with Ebstein's anomaly; but the management of the highest-risk cases, the infants, remains an unsolved problem.

[1] Ebstein, W, *Archiv für Anatomie und Physiologie*, 1866, **33**, 238.
[2] Fischer-Hansen, J, *et al*, *Acta Medica Scandinavica*, 1977, **201**, 331.
[3] Jugdutt, B I, *et al*, *Journal of Thoracic and Cardiovascular Surgery*, 1977, **73**, 20.
[4] Lev, M, *et al*, *Archives of Pathology*, 1970, **90**, 334.
[5] Genton, E, and Blount, S G, *American Heart Journal*, 1967, **73**, 395.
[6] Becker, A E, Becker, M J, and Edwards, J E, *Archives of Pathology*, 1971, **91**, 167.
[7] Kumar, E A, *et al*, *American Journal of Cardiology*, 1971, **28**, 84.
[8] Watson, H, *British Heart Journal*, 1974, **36**, 417.
[9] Hardy, K L, *et al*, *Journal of Thoracic and Cardiovascular Surgery*, 1964, **48**, 927.

Treatment of osteoporosis

As long ago as 1941 Albright and his colleagues suggested that postmenopausal osteoporosis in women is caused by oestrogen deficiency.[1] Other possible contributory factors are a small skeleton at maturity,[2] the calcium malabsorption of old age,[3] and a high-protein diet.[4 5] Bone mass may not be the only factor which determines bone strength. Old bone is more fully calcified than young, and this may explain why less energy is required to fracture bones in adults than in children.[6] Racial differences may also be important: among women aged over 70 in Johannesburg those who are white fracture their necks of femur at least 10 times as frequently as those who are black —in spite of their having no less bone mass as estimated by metacarpal radiogrammetry.[7]

Osteoporotic patients have about 20% less bone than their normal peers,[8] whereas in both groups the annual rate of renewal of old bone is only about 5%.[9-11] This means that even if bone breakdown could be arrested completely with formation continuing unabated at least four years' treatment would be needed for the average patient with osteoporosis to regain a bone mass normal for her age. Even this is unattainable because a substantial fraction of new bone formation occurs in the Haversian systems of cortical bone and is necessarily preceded by bone breakdown. Furthermore, many treatments proposed for osteoporosis, such as oestrogens, calcium supplements, and thiazide diuretics, reduce turnover and would thereby delay recovery further.[12-14]

The two fundamentally different approaches to treatment are prevention and restoration. Prevention aims at reducing bone resorption so that formation can keep pace. Hormone replacement, calcium supplements, and vitamin D (together