Among the many cases of children with ear-ache for which the GP’s help is sought, it is not invariably that, having cried for some hours, the child went peacefully to sleep shortly before being seen. Usually, of course, the cause has been a blister on the drum, bursting or regression resulting in relief from the pain. Only a small proportion of such blisters are haemorrhagic and of the dark colour commonly described, most being like those elsewhere. Surely it was the bursting of the former kind which accounted for the blood in discharge mentioned by Dr Carne, if it was not from supplicative otitis media. They vary much in size from some quite small to others almost covering the drum. One wonders how often a large bulla has been mistaken for a true “bulging drum.” Any degree of reddening may be present, as in non-vesicular inflammations, or there may occasionally be none. After bursting or regression the site presents a very characteristic, rather crinkly appearance, presumably the “grey matter” mentioned by Mr Birett. Once this stage is reached there is rarely any further trouble. The condition is usually classified as part of catarrhal otitis media, but the pathology is debatable. Clearly of virus origin (incidentally often the first indication that yet another of the never-ending succession of such epidemics is prevalent), there is much to suggest that the vesicles are herpetic—in lay language, “cold sores on the drum.” Pain is apt to be severe, but fortunately usually lasts only a matter of a few hours. In view of the virus origin it is doubtful whether antibiotics can achieve much, but as a few cases go on to more definite otitis media they would appear to indicate while pain or other evidence of inflammation is present. When pain has gone and the drum clearly shows regression it is at least questionable whether treatment need be begun or continued.

May it be said that more than half the battle in seeing the drums of young children is won by having the head firmly held. In the absence of a nurse, many mothers soon get good at this. A convenient position is with the child on one side, then on the other, across the mother’s lap, head to the observer’s left. Rapid assessment is usually essential.

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Once-daily atenolol for hypertension

Sir,—I read with interest the paper by Drs A P Douglas-Jones and J M Cruickshank (24 April, p 990) in which atenolol is claimed to be effective in once-daily dosing in reducing the resting pulse rate and blood pressure after 24 hours in patients with mild or moderate hypertension. If such patients had been exercised, which the authors agree is necessary to assess beta-blockade, their findings might well have been different.

Our department has investigated the effect of metoprolol, a cardioselective beta-adrenergic blocking agent, given in a single daily dose to previously untreated hypertensive patients. Although apparently controlled at rest by 100-200 mg daily, the post-24 hour exercise blood pressure and pulse rate in some patients tended to exceed normal limits but was subsequently well controlled by dosage increases to levels of 300-400 mg. These results will form the basis of a future publication.

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Does anticonvulsant hypomagnesaemia exist?

Sir,—In two recent letters on this subject the authors—contrary to our findings in 1974—report that they were unable to demonstrate hypomagnesaemia in epileptic patients on anticonvulsant therapy. Dr S H Katz and his colleagues (7 February, p 341) believe that this difference may be due to our use of an unmatched normal outpatient control population, while Dr J Stewart (13 March, p 649) thinks this explanation unlikely. We tend to agree with the latter statement since serum magnesium values are independent of age and sex differences.

Experts in the field of magnesium metabolism agree that the biological variation of serum magnesium concentration is quite small 1; furthermore, about 25% of serum magnesium is protein-bound. The latter fact infers that dehydration and/or venous stasis (which influences the serum level of protein) simultaneously may influence the serum magnesium level. In our study on 226 epileptic outpatients and 95 controls 2 these possible sources of variation were dealt with in the following way: all blood samples were drawn without stasis; the magnesium concentrations were corrected to a constant protein level; the blood samples were run in series of 60 duplicates, each batch containing samples from patients and controls; and the technicians were unaware of the clinical data. In the conflicting reports of Dr Katz and his colleagues and Dr Stewart (which contain data from a mixture of in-patients and out-patients) no information is given as to the blood sampling technique and apparently no correction for serum protein was made. This alone might explain the different findings, but it appears that this is not the sole cause.

In the accompanying table we have summarized data of interest from your correspondents’ reports and from our own study. For the control subjects it is interesting to see that all three reports agree as to the mean values; only our biological variation is much smaller than those found in the other studies. The main reason for this is probably a larger inter-assay variation of the methods used by our opponents. The same remarkable difference in biological variation is found between our patients and those of Dr Katz and his colleagues and of Dr Stewart. But whereas we, with our apparently superior laboratory method in a large population, can demonstrate a small but significant lowering of serum magnesium in epileptic outpatients, our opponents, with their methods in smaller series of in-patients (or mixed in- and out-patients) apparently cannot.

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Benign proliferative lesions of the breast

Sir,—Confusion still exists over the conditions included under the above heading and the alternative terms you mention—cystic hyperplasia and mammatory dysplasia—in your leading article (8 May, p 1106). May I suggest that this is largely due to a failure to differentiate between three separate and distinct entities?

The first is a purely histological condition, of importance as a field of study of the potential relation of various types of epithelial change to carcinoma but with no diagnostic clinical features.

The second is an objective clinical condition—the development of macroscopic cysts. These usually present as an obvious solitary lump in one breast, hard in consistency and thus simulating carcinoma. Further solitary lumps in either breast commonly develop over the years. Because of the suddenness of their appearance the proximate cause must be some temporary acute upset of intraductal hydrodynamics. The condition occurs in women from the late thirties to the menopause and is completely innocent, the one-time belief that it might be precancerous being due to a failure to separate the rare cysts developing secondarily to an intraduct papilloma or papillary carcinoma.

The third condition, which I have termed ‘the pain syndrome,’ is a purely functional one in both the physiological and psychological senses of the term. It arises from a hyper-awareness of the physiological and pathological cycle of changes in the female breast. As far as I can gather, it is not seen in societies in which women are unaware of the...