larger professional contribution in posts appropriate to their capabilities, and have a chance to influence departmental and hospital policies. At a time when specialist training is becoming more prolonged we see the increase in posts of senior house officer grade as giving us registrars by another name, and pushing the bottle-neck further down the career ladder.

If both foreign and British graduates are to be doing the same rotations, and if the posts are to be of equal quality, then the contracts must all be held at regional headquarters so that acceptable rotations can be organised. As a majority of registrars will eventually obtain consultant appointments in district general hospitals, we believe that it should be obligatory for all registrars to have at least two years’ experience in such hospitals. Opportunities for overseas graduates to work in teaching hospitals should also be available.

Despite much time and thought having been given to the problem of hospital staffing in 1980–81, little progress had been made by 1985. What has happened since then that could not have been foreseen? We now have imposed on us an ill thought out, hasty, and short sighted solution in a struggling hospital service, which remains unsympathetic to the requirements of flexibility that are now needed for 50% of medical school graduates, so that their full potential may be realised.

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Concern over safety of SAGM blood

Sir,

We read with interest the letter from Drs Robertson and Chiswick. We recently carried out an exchange transfusion in a neonate using 500 ml of partially packed SAGM blood. She was born at 38 weeks’ gestation, weighed 3000 g at birth, and required an exchange transfusion for ABO group incompatibility when 19 hours old.

Before the transfusion the infant’s total serum protein concentration was 58 g/l, and the serum albumin concentration was 36 g/l. These fell to 43 g/l and 28 g/l, respectively, immediately after the transfusion. By the following day the patient’s total serum protein concentration was 52 g/l with a serum albumin concentration of 32 g/l. A coagulation screen immediately after the transfusion showed a prothrombin ratio of 1:3, a kaolin cephalin coagulation time of 39 seconds [control 38 seconds], and a prothrombin time of 22 seconds [control 19 seconds].

This infant tolerated a SAGM exchange transfusion with a temporary fall in plasma protein concentration and no disturbance of coagulation. Partially packed SAGM contains protein concentrations that are one third to one half of those of normal plasma. Though SAGM donor blood may be safe for use in exchange transfusions in the well term infant, further assessment is required in the ill low birthweight baby.

Peptic ulceration: long term outcome

Sir,

Murphy et al reviewed 110 children with duodenal ulcers.¹ We are carrying out a long term follow up study of 36 children with peptic ulcers. The clinical features are similar but the long term outcome is different. The aim of our study, begun in 1982, was to determine endoscopically the healing rate after eight weeks of treatment with ranitidine, and the rate of relapse after stopping the drug. Endoscopy was repeated every six months or when symptoms recurred. For ethical reasons no placebo group was included.

The average number diagnosed annually was seven, the mean age at diagnosis was 9 years 7 months (range 18 months to 19 years), the male:female ratio was 1.8:1 and the mean duration of symptoms before diagnosis two years four months. There was a family history of peptic ulcer in 47%. The commonest symptoms were abdominal pain (91%), vomiting (47%), and bleeding (11%). The maximal acid output after pentagastrin stimulation was two standard deviations above normal in 66%. Of six patients with antral and prepyloric ulcers the maximal acid output was raised in five and normal in one, suggesting an identical pathogenetic factor.²

The table shows the sites of the ulcers, the rate of healing in 34 patients, and the rate of relapse in the 30 who were followed for more than a year. Though our series is smaller than that reported by Murphy et al, some of these preliminary data can be compared with his.

The main difference is in the rate of relapse (36% compared with 70%) and the length of time before relapse

<table>
<thead>
<tr>
<th>Site</th>
<th>No healed after eight weeks</th>
<th>No relapsed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duodenum</td>
<td>22/23</td>
<td>9/21</td>
</tr>
<tr>
<td>Antrum or prepyloric zone</td>
<td>10/11</td>
<td>2/9</td>
</tr>
</tbody>
</table>

Reference

2. Murphy et al.
Enuresis in children

Sir,

I was interested to read your correspondent’s personal view of her enuresis and her observations on its management. One can’t but help sympathise with her. In my experience most parents of enuretic children are bothered not so much by the nuisance that it causes them but by whether it could be a symptom or sign of disease in soma or psyche; if they are reassured on that score they can usually cope until the child demands to be ‘cured’. As we all know, wetting can be a manifestation of many diseases: of the urinary tract, of the upper respiratory tract (obstruction at night), of the endocrine system (diabetes mellitus, diabetes insipidus, and Addison’s Disease), of the gut (coeliac disease with intestinal water retention), of the cardiovascular system (improving renal perfusion at rest), of the hypothalamus (failure of development of diurnal rhythms), of the central nervous system (nocturnal or early morning fits), or of the psyche; these should be carefully excluded before the symptom is ‘treated’ on its own merits. For this reason, and because it represents a stigma, attendance at so called enuretic clinics may be harmful, as is the provision of a star chart—implying that the wetting is under voluntary control—or the imposition of an alarm (although this can be very helpful when managed by the child himself). Drugs like imipramine do seem to help but they bring ‘magic’ into the management, are dangerous in overdose, and do not have a convincing rationale in relation to bladder innervation, if indeed they do act by increasing capacity. In this context it is interesting that nearly all nocturnal enuretics are wet before their parents go to bed.

Perhaps as members of a profession whose practice is supposed to be science based we should confine ourselves to sorting out and acting on what we do know of the pathogenesis and leave the rest to growing up, practical sympathy, education for all concerned—particularly teachers—and common sense measures to mitigate nuisance like the provision of a washing machine, using a covered mattress and easy to wash sheets (supplemented by newspapers used as blotting paper), and a potty under the bed.

Reference


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Hearing loss due to mumps

Sir,

We share the opinion of Hall and Richards that mumps is a major cause of severe sensorineural hearing loss. During an epidemic of mumps in Israel in 1984, 85 children with mumps were admitted to the paediatric department of the Beilinson Medical Center, which serves an area of roughly 68 000 children (age 0–14 years). Seventy-nine of the patients had symptoms or signs of meningoencephalitis. Three children (3.5%) developed unilateral profound sensorineural deafness in association with mumps.

The first patient was a 3 year old boy who had bilateral parotitis and meningeal irritation. Cerebrospinal fluid examination showed 1020 cells/mm³ (98% mononuclear cells), and mumps virus was subsequently isolated from this fluid. During his stay in hospital his parents noticed that he did not respond to calls, and audiometry showed right severe sensorineural hearing loss.

The second patient was a 10 year old girl with bilateral parotitis, severe headache, and vomiting. On the sixth day in hospital she complained of inability to hear a telephone conversation through her right ear, and severe sensorineural hearing loss was documented by audiometry.

The third patient was a 8 year old girl who was admitted for observation with fever of 39°C. In the ward she complained of inability to hear with the right ear, and this was confirmed by audiometry. As we were aware of the association of hearing loss and mumps this possibility was tested, and her complement fixation antibody for mumps rose from 1/20 to 1/240 within three weeks, which confirmed recent infection. Routine hearing screening tests done on these three patients before the disease gave normal results. All the other children with mumps who were in hospital were tested and no significant hearing abnormalities were found.

A postal inquiry, albeit with an incomplete response, showed three additional cases of deafness after mumps in 1984, one of them with severe bilateral hearing loss.

References


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