Correspondence

Child health services after re-organisation

Sir,
The annotation by Dr Wilson\(^1\) was interesting but I think that her message of gloom is too strong.

Child health area specialists were an essential part of the last reorganisation; they acted as catalysts in setting up child health services on a firm paediatric (as opposed to public health) basis, but these having been created can now move on to new fields and fresh challenges.

Dr Wilson only gives one model for the organisation of the child health services from 1 April 1982; the British Medical Association sees a different model and I suggest that every district should press for the second option. I quote (Dr Wilson’s model is first):

First, the model which exists in many areas at present where SCMOs and CMOs are managerial accountability to the authority through a community physician.

Secondly, accountability to the authority through one SCMO (often titled Principal Medical Officer) with a community physician monitoring and co-ordinating their work, and responsible for evaluation . . .\(^2\)

In Scotland this person is called the ‘Clinical co-ordinator of child health services’ and such a doctor works closely with consultant paediatricians to ensure that appropriate training programmes are carried out (until such time as national programmes are implemented) and suitable doctors appointed. The community physician will expect to be consulted and to have his approval sought for such decisions, but surely he will be fully occupied elsewhere if he has a competent SCMO. However, one very real concern is the community child health representation at district level and where this will come from.

The managerial change of accountability for work is not in itself important. I was pleased that Dr Wilson stressed that CMOs and SCMOs are clinically autonomous; this fact has not been recognised by many people, which has not enhanced the image of the child health doctor.

Four years ago the community child health services were at serious risk of becoming part of a generic third force but with the valiant help of consultant paediatricians (many of whom confessed that they had never before given the community service a thought), and with the help of GP colleagues, we fought off that threat. Some of those paediatricians have since built up departments of child health (closely integrated with community services) which no reorganisation can destroy. In particular I would mention the supreme efforts made by Professor Forfar on behalf of the training of CMOs and SCMOs, and the clear recommendations which his committees have made towards a unified service. In the last four years we have come a very long way, which is why I feel optimistic about the future.

Although Scotland always seems to be one step ahead, there are exciting developments in child health services in inner cities, Nottingham and Newcastle to mention only two. These changes have been initiated by paediatricians who can see that paediatrics without prevention is like treating an overdose while the patient is still swallowing the tablets. Their vision is the same as that of the hard-done-by, hard working Court committee who said it all many years ago,\(^3\) but unfortunately that report was far ahead of its time. However, slowly but surely we are all beginning to recognise that the Court report was wise and that it recommended practical solutions to our problems, many of which may still be relevant.

If we have the foresight and the courage needed to set up integrated child health services in our districts, and if we play our cards right, no management structure can destroy what we build, but if we hesitate and fail to grasp the opportunity then gloom may well be justified.

References


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Congenital villous atrophy

Sir,

Dossetor\(^4\) described a patient with presumed congenital villous atrophy not secondary to intraluminal events. However, barium follow-through showed a dilated small-bowel and very slow passage of barium. We suggest for this child the diagnosis of intestinal pseudo-obstruction, a term that is used for the syndrome in which there are symptoms and signs of intestinal obstruction without any evidence for an actual lesion obstructing the intestinal lumen.\(^5\)

Chronic intestinal pseudo-obstruction exists in two forms, primary and secondary. The latter can be the result of many diseases.\(^6\) \(^4\) Primary chronic intestinal pseudo-obstruction is a primary disorder of gastrointestinal motility. Manometric and myoelectric studies...
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of the gastrointestinal tract are often abnormal.\(^8\) The pathological findings vary: smooth muscle atrophy and degeneration can be seen on a full thickness biopsy; degeneration of the plexus myentericus is described in other patients; however, normal small bowel histology under light and electron microscopical examination is possible.\(^8\)

Severe villous atrophy is well known in the syndrome. It is probably secondary to intraluminal factors—such as bacterial overgrowth and toxic bacterial metabolites.\(^5\)

We therefore suggest that Dossetor's patient had a primary abnormality of motility with secondary bacterial overgrowth and villous atrophy.

References


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Fifth day fits

Sir,

The paper by Pryor et al.\(^1\) was interesting because it described similar observations to those we published in 1977;\(^2\) since then the reality of this syndrome has become more convincing. In our neonatal intensive care unit we see this type of convulsion, with identical features, between 5 and 15 times a year (47 cases between 1974 and 1980), and authors in France\(^3,4\) and Australia (J I Manson, 1977, personal communication) have identified it.

We should like to make the following comments on the work of Pryor et al.:

(1) EEG recordings at the time of the seizures show true electroclinical status epilepticus lasting 12 to 36 hours, with a special inter-ictal tracing that we call 'sharp alternant theta'.\(^5\) This special tracing is useful for the diagnosis of the syndrome since it is found in 80% of the cases even though it is not specific.

(2) Anticonvulsive therapy seems to be ineffective in stopping the fits and it prolongs post-ictal alterations of the baby's tone and consciousness. Currently we use this kind of treatment only occasionally and then limit the prescription to one or two intravenous injections of diazepam, or to one dose of phenobarbitone.

(3) We have never prescribed maintenance anticonvulsive therapy. Long-term prognosis seems good. Among 37 infants followed up for longer than one year (six aged between 1 and 2 years, seven between 2 and 3, six between 3 and 4, four between 4 and 5, six between 5 and 6, and eight between 6 and 7) one had febrile convulsions at age 17 months (he is well at 6 years) and one had fits without fever at age 3 years (he is now 5\(\frac{1}{2}\) years old and doing well on phenobarbitone treatment).

We think that it is easy to differentiate between fifth day fits and other types of neonatal convulsions using the following criteria: term newborn, no perinatal asphyxia, no pathological event in the first 4 to 5 days, electroclinical seizures with inter-ictal tracing, 'sharp alternant theta' lasting about 24 hours, spontaneous favourable outcome, negative aetiological investigations. The long-term prognosis of such a syndrome appears to be favourable but needs confirmation. The main question is still the aetiological background: in common with Pryor et al. our metabolic, toxicological, and virological investigations remain negative. It would be helpful if other teams with new concepts and different techniques could tackle the problem.

References


Dr Dossetor comments:

Whether one labels the primary abnormality of motility with secondary bacterial overgrowth as 'stagnant loop syndrome' or 'intestinal pseudo-obstruction' is to some extent semantic, but since my patient presented with steatorrhoea and failure to thrive and did not have any 'nausea vomiting and cramping abdominal pain'\(^4\) I preferred the former term. The suggestion that the villous atrophy was secondary to intraluminal factors might be right in that although the illness started in early infancy, the first biopsy showing severe villous atrophy was obtained at 29 months, and the recent biopsy from the same level as the previous abnormal one has shown substantial improvement. However, the atrophic mucosa showed no increased cellularity which is usually a feature of villous atrophy caused by luminal toxins, and appears to be a feature of the villous atrophy of the intestinal pseudo-obstruction syndrome.\(^5\) For this reason I thought it more likely that the villous atrophy was congenital, together with the motility disorder.