More work on the spontaneous respiratory activity of ventilated babies is needed.

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References


Drs Field, Milner, and Hopkin comment:

We thank Drs Greenough and Morley and Drs South and Morley for their comments. Both letters raise similar questions, and in response we would just like to make three points.

(1) Our aim was to investigate the role of ventilator manipulation in controlling active expiration against the ventilator. We felt that we have shown this to be possible in a proportion of ventilator dependent infants.

(2) To elucidate the situation further will require a prospective study of ventilator dependent infants in whom pressure and volume monitoring is continuous from the time of beginning ventilation. Currently, such a system is not available.

(3) A discussion and clarification of what constitutes ‘active expiration against the ventilator’ would be a useful step in aiding further research in this area.

A service for problem families

Sir,

Dr Polnay’s encouraging account of the Radford Family Centre gives welcome attention to an area central to child health today—namely, how best to help families handicapped by disadvantage. There are analogies with the management of similar problems in developing countries that are worth exploring, in this case with Nutrition Rehabilitation Centres.

Nutrition Rehabilitation Centres were set up in many countries in Africa and elsewhere in the 1960s and 70s with the aim of preventing malnutrition by teaching mothers how to feed their children better. The concept is now being reassessed as a result of evaluations that cast doubt over their effectiveness. Yet the aims of the centres were broadly similar to those formulated for the Nottingham families by Dr Polnay. What went wrong?

Firstly, the concept that malnutrition is due mainly to ignorance has been falsified: usually, adverse socio-economic circumstances are the prevailing aetiological factor. Secondly, though the centres were successful in rehabilitating children with malnutrition in the short term, their impact on long term problems was limited: eight months after admission the children’s nutritional state and family’s dietary practice were little different from a control group. It was considered that the families had ‘practical difficulties in implementation, chiefly related to low income’. These findings were confirmed in Lukmanji’s study.

As a result of such evaluation, nutrition policies in developing countries are changing to emphasise community based programmes that train community workers who will help the local people (not just the parents of malnourished children, who have low social status) to confront the root causes of malnutrition. Though the analogy with family centres should not be pursued too closely, the problems the families face are similar: poverty, lack of community support, and a feeling of ‘powerlessness’. As with malnutrition, the children admitted represent the tip of an iceberg—there are many more less severely affected outside.

In finding remedies, ultimately, the professionals must stand aside and assist in the development of community based health initiatives, involving local people as activators, to combat the root causes of ‘problem families’.

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Dr Polnay comments:

I would accept that there are three problems, namely, poverty, ignorance, and neglect, rather than the two, ignorance and neglect. Whether these factors lead to problems in the child depends on the balance that exists between the individual vulnerability of the family, the quality of the professional services that are available, and the hostility that they meet from their environment. This is illustrated in the attached diagram. In the Family Centre we have worked very much in the areas of personal vulnerability. However, other social, medical, and educational initiatives in the area have also addressed themselves to the other two issues, namely the stresses that the environment produces and the services available outside to support the families.

Long term follow up studies of early intervention programmes are few and far between. However, Guteillus et al and Schweinhart et al have produced promising long term results. Viewed in its wider context, our record for dealing with disadvantaged communities may not be quite as impressive. References to the Medical Officer of Health for the Report...
Plasma fibronectin concentrations in mucocutaneous lymph node syndrome

Sir.

Recently, we have reported that patients suffering from mucocutaneous lymph node syndrome (MCLS, Kawasaki disease) show a distinct trend in plasma fibronectin concentrations during the course of the disease.\(^1\) In that paper we discussed the possibility that decreased plasma fibronectin concentrations in the early stage of the disease are due to endothelial damage, as the endothelium is the major site of plasma fibronectin synthesis. The underlying pathology of Henoch-Schönlein purpura is known to be vasculitis as well as MCLS. Hence we have attempted to compare these two common vasculitis diseases in a paediatric population with regard to the concentrations of plasma fibronectin.

The study population consisted of 10 children aged between 2 and 8 years suffering from Henoch-Schönlein purpura. Altogether, 49 samples were drawn in days 1–210 of the disease. The method used has been described previously.\(^1\)

Plasma fibronectin concentrations in patients suffering from Henoch-Schönlein purpura showed normal to slightly increased values, and there was no apparent trend of changes throughout the course of the disease, unlike the situation in MCLS. Symptoms such as purpura, abdominal pain, or arthralgia were not significantly related to any differences in plasma fibronectin concentrations. Similarly, whether the patient showed proteinuria or haematuria at the time of sampling had no apparent influence on the concentrations of plasma fibronectin.

One supposed reason for the difference between MCLS and Henoch-Schönlein purpura is the difference in intensity of the inflammatory response as well as the endothelial damage between these two diseases. There is also a possibility that the hypothesis that the decreased plasma fibronectin concentrations are at least in part due to the endothelial damage itself is incorrect and that the vascular damage has no influence upon the plasma fibronectin concentrations.

Reference


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