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Diagnosis and treatment of *Pneumocystis carinii* pneumonia

Sir,

Lipson *et al.* (1977) report success in all 7 of their patients with *Pneumocystis carinii* pneumonia treated with cotrimoxazole alone. We would like to make some cautionary observations based on a successfully treated child.

A 4-year-old girl with acute lymphoblastic leukaemia in remission for 15 months was seen at a routine follow-up clinic. Although symptom free, she appeared slightly cyanosed and tachypnoeic and a chest *x*-ray showed diffuse changes. A diagnostic needle aspiration of the lung was performed and *Pn. carinii* was identified (by Dr John Lever). Cultures of the remainder of the aspirate for viruses, fungi, and bacteria were sterile. Despite early treatment with high-dose cotrimoxazole, the child's condition deteriorated steadily over 3 days. Pentamidine isoethionate was added and her condition thereafter improved; there were no complications.

We would like to make 3 points: (1) The child was symptom free on the day of diagnosis. (2) Needle aspiration was a safe, rapid means of diagnosis. Of the cases reported by Lipson *et al.* (1977), 2 out of 14 had serious complications from open lung biopsy. This contrasts with one mild episode of transient haemoptysis complicating 8 needle aspirations in children with pneumonia at Bristol Children's Hospital (personal observations). Open biopsy may of course be indicated after a negative needle aspiration when *Pn. carinii* infection is strongly suspected (Hughes, 1977). (3) Cotrimoxazole appeared to be ineffective in the first 3 days of treatment, in contrast to the observation by Lipson *et al.* that the drug 'seemed to have a rapid onset of action'. Hughes (1977) reported failure of cotrimoxazole therapy in 3 out of 14 children with *Pn. carinii* pneumonia.

We conclude that needle aspirate is the investigation of choice when *Pn. carinii* pneumonia is suspected, and that cotrimoxazole is not necessarily effective in all cases.

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Sir,

The report by Lipson *et al.* (1977) prompts us to report our recent experience with 2 cases of *Pneumocystis carinii* pneumonia, one aged 9 years and one 3 months, who were under treatment for systemic lupus erythematosus and acute lymphatic leukaemia respectively. Clinical diagnosis of *Pn. carinii* pneumonia was made on the basis of fever, increased respiration rate, and chest *x*-rays. The agent was shown in tracheal washings (2 ml saline solution under anaesthesia using N_2O-O_2-halothane), but was absent from sputa and gastric juice (Chan *et al.*, 1977). The patients' general conditions did not permit lung biopsy or lung aspiration (Cohen and Weiss, 1971). Both were treated first with pentamidine isoethionate, further

clinical improvement was obtained after this was combined with trimethoprim-sulphamethoxazole (Hughes *et al.*, 1975). This is the first report from Japan of the treatment of the disease by trimethoprim-sulphamethoxazole. We favour the use of tracheal washings to demonstrate the agent, since the procedure is safe and the chance of finding the agent seems to be better than in sputum or gastric juice.

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Identifying children 'at risk' from unexpected death in infancy

Sir,

The lay press has recently acclaimed the system which is in use in Sheffield for identifying children 'at risk' from unexpected death in infancy. Carpenter *et al.* (1977) point out, however, that this system may not apply in other communities. One of the purposes of the DHSS Multicentre Postneonatal Study is to discover whether a system based on Sheffield data might be equally applicable in other communities.

The obstetric and perinatal records of 234 cases of unexpected death in infancy have been studied in retrospect in 11 centres: Manchester, Liverpool, Leeds, Edinburgh, Oxford, Gateshead, Newcastle upon Tyne, Barnsley, Rotherham, Doncaster, and Birmingham. For each case a living control was chosen at random from those born on the same day and in the same centre as the index. Each case and control was 'scored' numerically according to the 'at birth' data analysis used in Sheffield (Carpenter *et al.*, 1977).

In all, only 49% of index cases scored 'at risk', together with as many as 27% of controls. This represents a low sensitivity, despite having over one-quarter of the population labelled 'at risk'. The poor discrimination between cases and controls would confer a low degree of efficiency on a prospective prevention programme. Although numbers in some centres were small, in only one centre was there case/control discrimination of the degree which is still shown in Sheffield.

I present these preliminary findings in advance of a more complete communication for several reasons; (1) Primary care teams could be led into a false sense of security concerning infants who did *not* score 'at risk', if prospective programmes are initiated prematurely using a system of low sensitivity. (2) The 'scoring' system used in Sheffield should be validated in other areas before use in those areas. It appears that the system may need revising before it can be of use in other communities. (3) Objective environmental data are not included in the present system. The subjective observation of the home environment which is used would be difficult to apply accurately elsewhere using multiple observers. (4) *If* an 'at risk' system is necessary to reduce the numbers of unexpected infant deaths in this country, a system should be derived with a high sensitivity, and which could be applied to many communities.

Finally, I would like to emphasise that the 'at risk' factors used in Sheffield do not have an aetiological basis, and that the aetiology of 'cot death' remains unexplained.

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Rickets in preterm infants

Sir,

We were interested to read Glasgow and Thomas's (1977) description of respiratory distress owing to rickets in 4 very small preterm infants. Over the past year we have diagnosed rickets (rachitic changes at the wrist metaphyses associated with raised levels of serum alkaline phosphatase) in 3 preterm infants but in none was rickets associated with respiratory problems. The Table gives salient clinical and biochemical details and an example of the severity of osteodystrophy is shown in the Fig. At the time of diagnosis the infants were receiving 400 IU supplementary vitamin D, which had been started 2 weeks after birth. Rickets was diagnosed in Cases 1 and 2 on finding craniotabes at a routine follow-up examination and these positive findings prompted investigation of Case 3 who showed no clinical abnormality. None of the infants was in any way unwell when rickets was diagnosed. They were treated by increasing daily vitamin D intake. Cases 1 and 3 were given 1000 IU and Case 2 was given 2000 IU for one month with gradual reduction of the dose to 400 IU over the next 2 months. Within 3 months of diagnosis there were well defined radiological signs of healing in the 3 infants.

To explain their syndrome of subacute respiratory distress and severe metabolic bone disease, Glasgow and