research. This will require a multicentre study to provide sufficiently large numbers of births. Priority of the Asian mother may lie in preconceptual clinics and genetic counselling. A long term solution may lie in the establishment of Pakistani Moslem women genetic counsellors as is being done in Bradford for families with thalassaemia.

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Neonatal tuberculosis

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SUMMARY Tuberculosis rarely presents in the neonatal period. Though treatable, it may be fatal despite modern treatment. The diagnosis of congenital tuberculosis should be considered in any neonate with pneumonia that fails to respond to conventional treatment, particularly in a child from an ethnic or socioeconomic environment where tuberculosis is prevalent.

Congenital tuberculosis is rarely reported[1] and is unlikely to be considered in areas where the prevalence of adult tuberculosis is low. A case of congenital tuberculosis is described.

Case report

A boy weighing 1380 g was born vaginally at 28 weeks' gestation to a 21 year old primigravida. Both parents were Indian, but neither had been to the Indian subcontinent for over two years. No maternal illness had been recorded during pregnancy, although a history of cough and night sweats was subsequently elicited. Immediately after delivery the infant was transferred to the special care baby unit and nursed in an incubator. He remained well until 22 days of age, when he had a number of short apnoeic episodes, became tachypnoeic, and exhibited signs of a patent ductus arteriosus. A chest *x* ray film showed cardiomegaly with bronchopneumonia, and a full infection screen proved noncontributory. Treatment with parenteral penicillin and gentamicin was started and the infant transferred to a regional paediatric unit, where minimal hepatomegaly was noted and echocardiographic confirmation of a patent ductus arteriosus made.

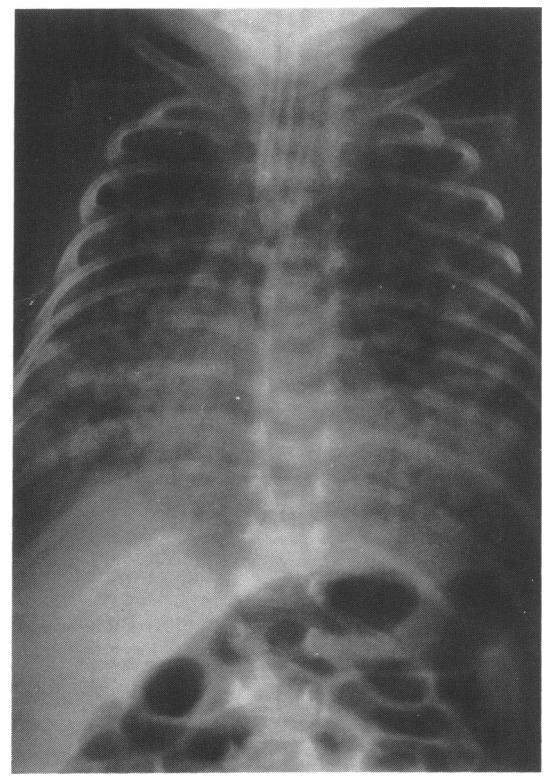

Fig. 1 *Chest* x *ray film showing widespread bronchopneumonia.*

Over the next three days the baby's respiratory state worsened (Fig. 1) and ventilatory support was begun.

On the 28th day of life a tracheal aspirate was noticed to contain particles, which showed up poorly on Gram staining (Fig. 2). There was no clinical suspicion of tuberculosis infection, but Ziehl-Neelsen staining of this aspirate showed acid/alcohol fast bacteria which were subsequently identified on culture as *Mycobacterium tuberculosis*.

Antituberculous treatment with streptomycin, rifampicin, and isoniazid was begun. The heart murmur varied intermittently and was not thought to contribute appreciably to the infant's clinical state. The infant's clinical course fluctuated for three weeks against a background of deteriorating pulmonary function, and he died aged 7 weeks after a period of hepatic failure.

Postmortem examination confirmed extensive tuberculous bronchopneumonia and accompanying cytomegalovirus pneumonia. Neither hepatic cytomegalovirus involvement nor tuberculous granuloma was identified. The placenta was not available for examination. Screening of the infant's family and attendants showed radiographic changes of non-caseating pulmonary tuberculosis in the mother alone, which responded to appropriate antituberculous treatment. *M. tuberculosis* was neither identified nor grown from maternal sputum, gastric washings, or cervical smear.

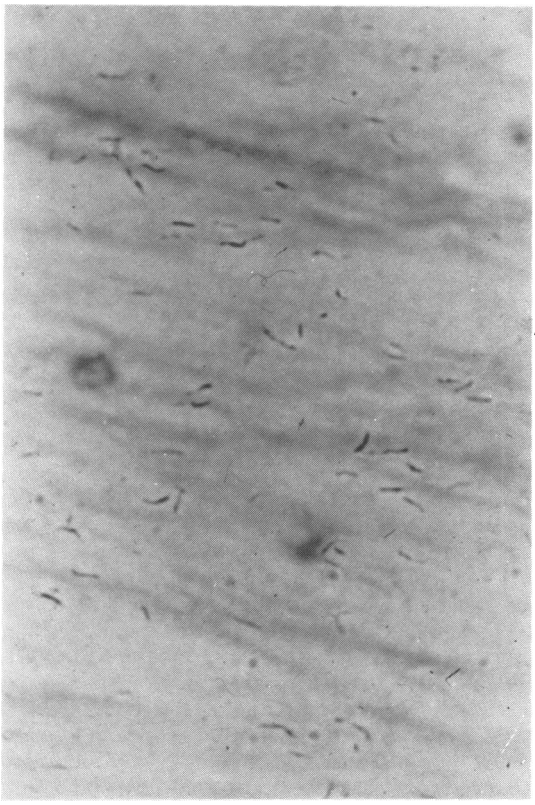

Fig. 2 *Tracheal aspirate: particles showing up on Gram staining.*

Discussion

Congenital tuberculosis is uncommonly found in the infants of women known themselves to have pulmonary or placental tuberculosis.[2 3] In 1935 Beitzke laid down diagnostic criteria: *M. tuberculosis* should be grown from the infants tissues.[4] In addition, a primary complex should be shown in the liver indicating carriage of bacilli through the umbilical vein, or tuberculous lesions be discovered within a few days of life where the possibility of extrauterine infection could be excluded. The advent of specific chemotherapy has reduced the prevalence of tuberculosis and made histological confirmation in the newborn rarely necessary so that these precise criteria can only infrequently be met. Furthermore, it is recognised that in addition to its hematogenous spread to the liver, the best oxygenated fetal organ, congenital tuberculosis may occur from aspiration of infected liquor in utero or during delivery.[5] Our patient's only tuberculous contact was with his mother, who did not have open disease, and it is presumed that the blood borne congenital infection lay dormant for the first three weeks of postnatal life.

The clinical presentation of congenital tuberculosis has a peak onset three to four weeks after delivery but has been reported during the first week of life.[6] Affected children are often born prematurely and may present with respiratory disease, hepatosplenomegaly, and non-specific fever, failure to thrive,[6] or sudden death.[5] When positive bacteriological cultures are not available diagnoses may be made by open lung biopsy examination, though this may be precluded by the severity of the infant's condition.[7] Histology of affected tissues shows large numbers of organisms with a poor inflammatory response and only rarely tuberculous meningitis. Although almost 50% of the placentas of tuberculous mothers contain acid fast bacilli, infants born to mothers with active tuberculosis rarely have congenital infection.[2] How the fetus is protected is unclear. Since the introduction of antituberculous chemotherapy, infants have been successfully treated,[8] and the seven day delay in diagnosis in this case may have influenced the outcome.

Postmortem examination unexpectedly showed concurrent cytomegalovirus pneumonia. This is not uncommonly seen in immune suppressed patients. Cytomegalovirus was not grown from the maternal cervix, and although the possibility that the infection was acquired from one of several top up transfusions was considered, this could not be confirmed.

In any neonate with non-responsive neonatal pneumonia, congenital tuberculosis should be considered.

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Specific malabsorption of vitamin B_{12} in Down's syndrome

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SUMMARY A 3 year old girl with Down's syndrome became lethargic and withdrawn, and investigations showed a specific malabsorption of vitamin B_{12} without proteinuria.

Megaloblastic anaemia due to specific malabsorption of vitamin B_{12} is usually associated with proteinuria. Urban *et al* reviewed the published reports in 1981 and found only six cases without proteinuria,[1] and Conway *et al* reported a further case with neuropathy but without proteinuria.[2] There is often delay in diagnosing a new problem in a child with Down's syndrome, and we report this association to emphasise the need to take seriously the development of new symptoms, rather than attributing them to the syndrome.

Case report

A 3 years and 4 months old girl of unrelated parents who was under regular review because she had Down's syndrome presented with new symptoms developing over one month. She suffered one episode of unproven haematuria followed by two weeks of watery diarrhoea. She then developed diurnal enuresis, cold cyanosed peripheries, lethargy, irritability, anorexia, and a scaly erythematous non-itching rash over an elbow and became withdrawn. She had lost 350 g in six months, her weight being 9650 g (2000 g below the third centile). Her height was just below the third centile with a normal height velocity. On examination she had typical features of Down's syndrome and, in addition, was pale and miserable and had an area of discoid eczema over her left elbow.

The following investigations yielded normal results: plasma urea, creatinine, electrolytes, calcium, phosphate, albumin, glucose, bilirubin, and thyroxine concentrations, alkaline phosphatase, alanine transferase, and γ-glutamyltransferase activities, urine analysis and culture, throat and cough swabs, faecal microscopy and culture, and chest *x* ray films.

The peripheral blood film showed a pancytopenia with macrocytic normochromic red cells (haemoglobin=5·9%, mean corpuscular volume 107·6 fl, mean corpuscular haemoglobin 35·9 pg, white cell count $3{\cdot}41\times10^9$/l, neutrophils $0{\cdot}550\times10^9$/l, platelets 47×10^9/l). Erythrocyte sedimentation rate was 9 mm in the first hour. The bone marrow was megaloblastic. Serum total cobalamins were 80 ng/l (normal range 300–1100 ng/l), red cell folate 240 mcg/l (normal range 130–600 mcg/l), and serum folate 15·4 mcg/l (normal range 2·6–14 mcg/l). Intrinsic factor antibody, parietal cell antibody, and