whereas the peak expiratory flow meter exercises the muscles of the abdomen and other accessory muscles, which are less important. In their study of training Martin *et al* claimed to show improvement of muscle endurance,[2] but the clinical value of this is uncertain. The vital capacity is possibly the best index of lung function as it has been repeatedly shown to predict both survival, and the ability to withstand spinal surgery without respiratory complications.

In this study we have attempted to eliminate bias in lung function testing by a double blind placebo design. When learning effects are allowed for, this study does not convincingly show any improvement in lung function from short term respiratory muscle training. It is important to note that lung function is significantly worse in the supine position—a factor likely to be relevant in postoperative care.

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# Congenital tuberculosis localised to the ear

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SUMMARY We report two infants who had localised congenital tuberculous otitis. In both cases the infants presented with an ear discharge and both mothers had been diagnosed as having miliary tuberculosis. Infection is thought to have occurred in utero or during birth.

Congenital tuberculosis is seldom reported, and when it occurs it tends to be a multisystem disorder with a poor perinatal outcome.[1–3] In older children isolated tuberculous otitis is well described[4 5]; isolated congenital tuberculosis of the ear, however, has not been previously reported.

## Case reports

CASE 1

A boy, weighing 670 g, was born vaginally at 27 weeks' gestation. The mother, a 27 year old primigravida, had booked at 14 weeks. At 23 weeks she was admitted for minor antepartum haemorrhage and unexplained fever. There were no respiratory symptoms. Several blood and urine cultures were negative. A fluctuating temperature persisted despite treatment with paracetamol, indomethacin, and co-trimoxazole. Eight days before delivery, on radiological evidence, a diagnosis of miliary tuberculosis was made and treatment was started with isoniazid, ethambutol, rifampicin, and pyrazinamide. Despite indomethacin and hexoprenaline suppression, spontaneous premature labour and delivery ensued.

Histological examination of the placenta showed small areas of infarcted villi with associated haemorrhage and fibrin deposits on maternal surfaces. Areas of caseous necrosis were noted (figure). Numerous acid-alcohol fast bacilli were seen on Ziehl-Nielsen staining. This information was, however, only discovered after discharge of the infant.

Immediately after delivery the infant was taken to the neonatal intensive care unit. He had clinical and radiological features of hyaline membrane disease, but required headbox oxygen only. Apnoeic spells occurred from days 15 to 45. Investigations for sepsis and metabolic abnormalities were negative,

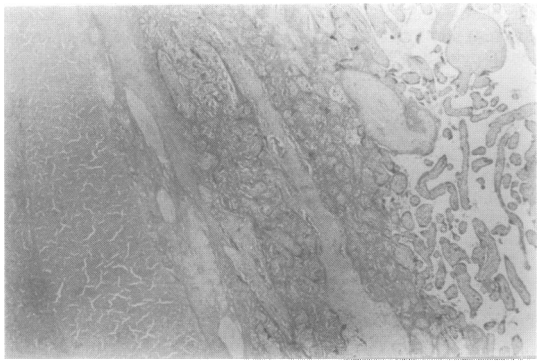

Figure *Photomicrograph of placental tissue showing normal villi on the right and caseous necrosis of placenta on left. (Haematoxylin and eosin stain×5000.)*

and chest radiographs were clear. The infant thrived. BCG was not given at birth, in accordance with routine nursery practice not to administer BCG at discharge to premature infants.

A whitish seropurulent discharge of the infant's right ear was noted on day 68 (when he weighed 1650 g); a pus swab of the discharge grew *Proteus mirabilis*. An otorhinolaryngologist prescribed dry mopping, amoxacillin orally, and framycetic sulphate eardrops locally. Twelve days later (day 88) progressive right preauricular and upper cervical lymphadenopathy became apparent. The aural discharge persisted. In view of the duration of the otitis and the perinatal history, the diagnosis of tuberculous otitis was considered; and at repeat otoscopy a granuloma was removed from the auditory canal.

Histology showed acutely inflamed granulation tissue, with epitheloid granuloma; acid-alcohol fast bacilli were shown. Multiple drug treatment was commenced (isoniazid, rifampicin, ethambutol, and pyrazinamide). Systemic examinations throughout his hospital stay and after discharge, on days 95 and 125, were normal as were the chest radiographs. By day 150 the aural discharge had cleared and lymphadenopathy was appreciably reduced. The infant was thriving and had normal developmental milestones.

CASE 2

A boy, weighing 1560 g, was born vaginally at an estimated 36 weeks' gestation. After four uneventful weeks in the nursery the infant was discharged well and weighing 2100 g. BCG immunisation was given before discharge.

One week later the mother presented with cough, chest pain, and night sweats, of two weeks' duration. Miliary tuberculosis was diagnosed on the basis of a typical chest radiograph, though her sputum was negative for acid-fast bacilli.

On admission, the mother stated that her baby had had a discharging left ear for four days, but had been otherwise well. Examination of the baby showed firm, non-tender, left sided periauricular lymphadenopathy. Otoscopy showed friable granulation tissue and thin pus filling the auditory canal. Microscopy showed moderate acid-alcohol fast bacilli, but culture for acid-fast bacilli was negative. No other pathogens were cultured. A chest radiograph was normal, and Mantoux test negative. Based on maternal history, clinical features, and pus microscopy, a diagnosis of tuberculosis was made. Quadruple chemotherapy (isoniazid, rifampicin, ethambutol, and pyrazinamide) was commenced.

At 3 months, the infant was thriving, with reduction in lymphadenopathy and aural discharge. Systemic examinations were normal throughout. Investigations for tuberculosis at other sites were negative.

## Discussion

Congenital tuberculosis, although rare, still occurs sporadically and is preventable.[1-3] The fetus and newborn infant are susceptible to several potential routes of infection: directly via placental circulation by in utero aspiration of infected amniotic fluid, or in the case of maternal genital tuberculosis, at birth during descent through the birth canal.[3]

The clinical presentation and diagnosis of congenital tuberculosis has recently been well summarised by Bate *et al.*[6] In recent English language literature, congenital tuberculosis with otitis has been reported, but all these patients had systemic involvement.[1 2] Aural discharge as a presenting sign of tuberculosis in early infancy has also been reported,[4 5] but it is not clear whether infection was acquired congenitally or postnatally.

Our patients were unusual in that they showed no signs of systemic infection either before or up to five months (case 1) and three months (case 2) after diagnosis and initiation of treatment. Investigations for tuberculosis at other sites were consistently negative. Both infants have thrived, in contrast with the usual perinatal outome of congenital tuberculosis.

The mother of our first patient had no respiratory symptoms, had been on antituberculous treatment from eight days before delivery, and was considered to be non-infectious at the time of handling her baby. Although she visited regularly, her infant was in an incubator for many weeks and she only

cuddled him later on. The infant is therefore presumed to have been infected in utero.

Although the second infant was home for seven days before the mother's diagnosis, it is unlikely that this infant was infected postnatally, as this period was considerably less than the six to eight weeks required by the tubercle bacilli to multiply.

The ear would be a most unusual site for a primary infection. It is speculated that transmission occurred by infected amniotic fluid being lodged in the fetal auditory canal, either in utero or during birth. After birth, the auditory canal provides a moist, relatively well oxygenated environment suitable for growth of tubercle bacilli. The delay in clinical presentation accords well with the time required by the organism to multiply. The absence of systemic manifestations make infection from transplacental spread, or by aspiration of infected liquor, unlikely.

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# Munchausen's syndrome in a 4 year old

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SUMMARY From the age of 4 a boy repeatedly feigned epileptic fits. He later admitted that his mother had taught him that he was epileptic and had trained him to behave like that.

Munchausen's syndrome by proxy is the fabrication by a mother of false symptoms and signs of illness in her child. The commonest form of this syndrome is fictitious epilepsy. We describe a case where a mother not only fabricated a false history of epilepsy in her 4 year old son, but also trained him to feign epileptic fits.

## Case report

A 2 year old boy was referred to the outpatient department by his family doctor because his mother had reported that he had episodes of cyanosis. No abnormality was found. He was again referred at the age of 3 because of episodes of shaking and unconsciousness. The electroencephalogram was normal, and no firm diagnosis was made. His mother continued to report that he had fits and when he was 4 years old a diagnosis of epilepsy was made and he was treated with sodium valproate. Three months later he was admitted to hospital because of fits, and on several occasions was observed lying apparently unconscious and trembling. He was, however, easily roused by the mention of his favourite food, and it was obvious that he was feigning the fits. The diagnosis of epilepsy was withdrawn and treatment was stopped.

His mother continued to claim that he was having fits, and so they were both referred for psychiatric help. Despite this she repeatedly brought him to hospital claiming he had been having fits. On one occasion he was brought to the clinic in a pushchair, wearing nappies and a crash helmet, and being fed from a baby's bottle. When he was 5 he was admitted to hospital during an apparent fit, and was given intravenous diazepam by a house officer who did not know him. During this admission he feigned further fits, and pretended to have a paralysed arm for two days. He also stated that he was epileptic. Two weeks later he was again admitted to hospital during an apparent fit but the next day was able to give a clear account of all that had occurred at that time.

At a case conference the family doctor told us that the boy had been brought to the surgery 130 times.