Surface swabs were all negative but *L. monocytogenes* was grown from blood cultures and CSF. She made a complete recovery after treatment with ampicillin, gentamicin, and chloramphenicol. Her follow up assessments did not show any sequelae.

The strains of *L. monocytogenes* from the two infants were both of serovar 4, and on phage typing were indistinguishable giving the same octal code* at 000 520 000 suggesting either a common source outbreak or cross infection. The fact that the two babies were born by caesarean section in the same obstetric theatre within a 12 hour period, and that they were attended by the same paediatrician on the same resuscitaire makes, in our view, the latter the more likely probability.

This report strongly supports the plea that all future *L. monocytogenes* isolates from cluster or outbreaks of listeriosis should be phage typed and detailed epidemiological investigations made in an effort to elucidate the mechanisms involved.¹ The survival of both babies supports the view that the high mortality rate must no longer hold for today's infants receiving swift adequate treatment and supportive management.¹

### References


### Correspondence

**Cleft palate and oesophageal atresia**

Sir,

In a most useful study, paediatric surgeons in Glasgow noted that 6 of 114 (5.3%) patients with oesophageal atresia also had cleft palate.² In fact this is a pretty high incidence. The authors quoted an American series that reported the association in 3.1% cases. However, in a series of 345 cases in the South West of England, only 4 had a cleft palate (with cleft lip in one), an incidence of 1.2%.³ The difference between the Scottish and English figures is statistically significant at the 2.5% level.

It seems likely that oesophageal atresia is aetologically heterogeneous, and a rather non-specific consequence of several teratological processes.³ The rather high incidence of cleft palate in the Glasgow series suggests the local operation of some different aetiological factors. Oesophageal atresia is a common defect (incidence 0.34/1000 births),³ yet has attracted remarkably little epidemiological research—surprising in view of Knox's albeit unconfirmed report⁴ of clustering of cases. Similar studies from other paediatric surgeons could be most productive.

**References**


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**Caseating regional lymphadenitis complicating BCG vaccination**

Sir,

We strongly disagree with Tam *et al.*¹ in their management of axillary adenitis after BCG immunisations. They describe 6 cases treated by surgical dissection and excision of the axillary nodes followed by multiple antituberculous chemotherapy for 16 months.

We believe that such treatment is unjustified and will eventually adversely affect the acceptability of their immunisation policy in infants. We have seen some 16 cases in Dominica. Initially we began treatment with isoniazid alone, but lately we have given no treatment. The adenitis has slowly resolved over many weeks in all cases. One patient required surgical incision because of abscess formation and in two the abscesses drained spontaneously. This policy of no treatment is supported

**References**