

Correspondence

Congenital oesophageal stenosis and herpes simplex infection

Sir,

In the May issue of the *Archives* (1977, 52, 414), Valerio and co-workers report congenital oesophageal stenosis in a 7-week-old baby with swallowing difficulties. Oesophagoscopy showed a stenotic area and dilatations were successful. They mention that recurrent regurgitation had occurred during the third and fourth week, and that serological evidence of herpes simplex infection was reported in both mother and child. We believe that the latter might well be the cause of the lesion of the oesophagus. In 1973, we reported 2 cases of neonatal herpes simplex infection proved by culture and serological reactions, resulting after 5 weeks in oesophageal stenosis which was cured by dilatations (Laboureau *et al.*, 1973). In a third observation (unpublished) a newborn had herpes vesicles in several areas of the skin (positive virus culture) and developed severe oesophagitis with a number of small ulcers and purpuric spots over the whole extent of the mucous membrane, seen by fibroscopy. X-ray showed a 'congenital stenosis', but earlier the barium swallow had shown many small defects of the wall of the oesophagus, interpreted as ulcers. Topical treatment and levamisole were given and no stenosis occurred.

The oesophageal localization of neonatal herpes simplex infection seems to be a frequent occurrence (Felder *et al.*, 1960; Langvad and Voigt, 1963; Becker *et al.*, 1968; Miller *et al.*, 1970), but so far most of such lesions have been found at necropsy. We emphasize that they should be routinely looked for by fibroscopy in any baby having herpes simplex infection, and our findings support the hypothesis that they may not infrequently result in oesophageal stenosis.

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Mr P. F. Jones comments:

There is no doubt that herpes simplex can affect the oesophagus and this is confirmed in some of the references which Prof. Rossier quotes. The main lesion appears to be mucosal ulceration (which can be quite widespread). This does not entirely explain why the lesion in our case was localized and, in particular, was notably elastic and resilient—a feature which seems to be typical of described cases of congenital stenosis. One would expect ulceration to be followed by fibrosis.

Whatever the aetiology of the stenosis in our patient, our main object as surgeons was to draw attention to a method of treatment which proved to be safe, simple, and effective when standard intermittent dilatation through an oesophagoscope had failed to relieve almost complete oesophageal obstruction.

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Perinatal cytomegalovirus infection

Sir,

The studies of Granström *et al.* (*Archives*, 1977, 52, 354) support the view that most perinatal cytomegalovirus (CMV) infections of infants are acquired from their mothers, but it is not possible to draw any conclusion from their data about the risk of infection from exchange transfusion in other European countries. Their high perinatal infant infection rate (39%) is attributed to a high frequency of reactivation of the mothers' virus during pregnancy—a frequency similar to that found in the East (18-28%) rather than that in most European and North American countries (3-4%) (Stern, 1977). The Finnish rate of perinatal infection in normal infants could very well mask infection transmitted by exchange transfusion.

In England Tobin *et al.* (1975) found that 29% of 51 babies excreted CMV after exchange transfusion, whereas none of 42 nontransfused babies did so. In Oxford we found a similar increase in CMV infection of 4 out of 16 infants after exchange transfusion with blood not screened for CMV antibodies, an increase which did not occur when CMV antibody-free donations were used (unpublished results).

The hazards of CMV transmitted to infants by blood