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. Oesophagography 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In a third observation (unpublished) a newborn had herpes vesicles in several areas of the skin (positive virus culture) and developed severe oesophagitis with a number of small ulcers and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 fibreoscopy. 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 fibreoscopy 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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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