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Effect of ascorbic acid on urinary hydroxyproline in children receiving corticosteroids

Sir,

Saphyakhajon *et al.* (1977), studying children receiving adrenal steroids for the treatment of juvenile rheumatoid arthritis (JRA), criticize our observations (Liakakos *et al.*, 1974a), stating that they did not find an increase in the amount of urinary hydroxyproline (HOP) with the simultaneous administration of large doses of ascorbic acid. Our study material, however, did not include children with JRA. It consisted of 6 children with rheumatic fever and 2 with the nephrotic syndrome. The observed discrepancy between their results and ours may well be attributed to differences in the study material, as in JRA the amount of HOP in the urine is found to be diminished (Smith *et al.*, 1968).

The same authors do not state the amount of steroids administered to their children. Our children received prednisolone 2 mg/kg body weight per day. If, by any chance, the dose of corticosteroids administered was too small it could not cause a further reduction in the amount of already low values of HOP found in JRA. The authors also do not mention in their letter whether their children were receiving salicylates simultaneously with steroids on a standard therapeutic basis. As we have previously shown (Liakakos *et al.*, 1974b), administration of therapeutic doses of aspirin to children causes a decreased urinary HOP which cannot be restored with administration of large doses of ascorbic acid.

Our recent observations in guinea pigs receiving corticosteroids have shown that prednisolone causes a narrowing in the epiphyseal plate of the tibia bones of these animals, whereas this effect is entirely inhibited with the simultaneous administration of ascorbic acid (unpublished data).

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Congenital abnormalities associated with extrahepatic portal hypertension

Sir,

In the May issue of the *Archives* (1977, **52**, 383), Odièvre and co-workers draw attention to the association of congenital abnormalities in children with extrahepatic portal hypertension of unknown cause. None of the children described by them nor those recorded in published reports had affected sibs.

We reviewed 8 patients with extrahepatic portal hypertension seen by us in the last 7 years. Of these, 1 had had omphalitis and 7 had extrahepatic portal hypertension of unknown cause. Of those 7 children, 3 had associated congenital abnormalities and 2 of these were in sibs. 1 patient had pyloric stenosis and congenital stricture of the sphincter of Oddi. The other 2 cases with extrahepatic portal hypertension were in brothers, 1 of whom had pulmonary stenosis and atrial septal defect, and the other pulmonary stenosis and pyloric stenosis. Atrial septal defect, pulmonary stenosis, and pyloric stenosis have all been described previously in association with extrahepatic portal hypertension.

This is the first report to our knowledge of the association of extrahepatic portal hypertension and congenital stricture of the sphincter of Oddi, and of the occurrence of extrahepatic portal hypertension and congenital abnormalities in sibs. These findings suggest that some cases of extrahepatic portal hypertension are not only congenital in origin, but might be genetically determined.

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