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Drs Durbin and Winterborn comment:

We are pleased that Dr Rylance agrees with the aim of our report which was to stress the need for regular monitoring of blood levels when chloramphenicol is used in combination with other drugs, particularly enzyme-inducing anticonvulsants. We did not measure serial phenobarbitone levels but neither patient showed evidence of toxicity in the form of unduly prolonged or increasing drowsiness. Any possible advantage of sodium valproate remains to be established by direct observation, but the evidence cited by Dr Rylance suggests that by inhibiting degradation, sodium valproate may actually increase the antibacterial activity of a given dose of chloramphenicol.

We thank Dr Rylance for drawing our attention to the report by Windorfer and Pringsheim (1977) which stated that addition of phenobarbitone to the *combination* (our italics) of chloramphenicol and penicillin reduced the serum chloramphenicol concentration in the neonate but not in infants or older children. We do not agree that their report invalidates our claim to the first specific report of the interaction between chloramphenicol and phenobarbitone in man. Windorfer and Pringsheim used a photometric assay which fails to distinguish between active chloramphenicol and its breakdown products. Their finding of increased chloramphenicol levels in neonates and infants treated with penicillin and chloramphenicol can be attributed at least in part to penicillin-induced renal retention of the breakdown products (Windorfer, 1972). It is therefore difficult to interpret their finding in neonates and infants that serum chloramphenicol concentration did not differ significantly between patients given chloramphenicol alone and those given the combination of chloramphenicol + penicillin + phenobarbitone. In the only group they were able to assess, they found no significant difference in the serum chloramphenicol levels of infants given chloramphenicol alone (n = 45) and of those given chloramphenicol + phenobarbitone (n = 40). The contradiction between their findings and ours may be explained by the relatively insensitive method of their study. They compared single measurements of serum (chloramphenicol + breakdown products) to the weight-related dose of chloramphenicol in children of different ages given different combinations of drugs, without allowing for the earlier duration of treatment. We suggest that Windorfer and Pringsheim would have demonstrated a significant effect of phenobarbitone on the serum level of biologically active chloramphenicol if, as we did, they had used a bioassay to measure the serum levels serially. In the event they did not.

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Henoch-Schönlein syndrome after chickenpox

Sir,

On the basis that one swallow does not make a summer, readers may not have paid too much attention to the letter from Halle (*Archives*, 1979, **54**, 166) suggesting that it was the first account of Henoch-Schönlein syndrome after chickenpox. However, the association has been reported before. In a series of 88 children with Henoch-Schönlein nephritis (Meadow *et al.*, 1972) we found that 5 children had had a specific infectious fever in the 6 weeks preceding onset of purpura. These illnesses were chickenpox in 2 children, and measles, rubella, and scarlatina in the other 3. The onset of chickenpox had been 5 weeks in one child and 10 days in the other before the onset of purpura.

Furthermore Pedersen and Petersen (1975) reported a 2-year-old boy who developed Henoch-Schönlein syndrome 16 days after the onset of chickenpox. Their paper illustrates the difficulty in defining Henoch-Schönlein syndrome. Chickenpox can be followed by a nonthrombocytopenic purpura: it can also be followed by a nephritis. If the two were to happen together and the child also had a tummy ache or an aching joint the diagnostic label would be Henoch-Schönlein syndrome.