responsiveness as severe as that of the asthmatics. Moreover, with very few positive airway responders in a small study group it would be difficult to show a significant difference statistically, even if such a difference had existed. Additionally, without a reference population, they cannot say whether their findings were normal or abnormal.

In our own study of a cohort of low birthweight children we found that the distribution of a positive airway response to histamine formed a continuum.³ At very low doses of histamine very few subjects in either the low birthweight cohort or a reference group showed a positive airway response. On the other hand, a significantly greater proportion of low birthweight children had a positive airway response, at moderate histamine doses, suggesting a shift in the frequency distribution of airway responsiveness. Children with bronchopulmonary dysplasia were slightly more responsive than other low birthweight children.

We agree with the findings of Duiverman and colleagues that very few children with bronchopulmonary dysplasia have a degree of airway responsiveness to histamine similar to recognised asthmatic subjects. Many do, however, show a mild to moderate increase in airway responsiveness when compared with an appropriate reference population. We are therefore unable to agree with their conclusion that 'bronchial responsiveness to histamine in these subjects (bronchopulmonary dysplasia survivors) was normal'.

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Maternal narcotic abuse and neonatal thrombocytosis

Sir,

We read with interest the recent report by AlRoomi *et al* on maternal narcotic abuse and its effects on newborn infants.¹ We wish to draw attention to an additional complication, not mentioned in this report, but first regarded by Burstein *et al* in 1979.² These authors noted significant thrombocytosis in a prospective study of 33 infants of mothers using narcotic drugs. They also specu-

lated that platelet microaggregates in these babies may give rise to cerebral infarcts. We have recently seen this complication in two preterm infants admitted to the neonatal intensive care unit. Both mothers were registered heroin addicts on a methadone maintenance programme during pregnancy, and at least one was still using heroin regularly.

Case reports

CASE 1

A boy, weighing 2320 g, gestational age 31 weeks, had a normal platelet count until day nine when it began to rise reaching an initial peak of $838 \times 10^9/l$ in the third week of life and a second peak of $919 \times 10^9/l$ in the sixth week of life. Other than the mother's substance abuse, there had been no particular antenatal problems and the baby's condition at birth was excellent. On day 4 cranial ultrasound examination showed a periventricular flare, which surprisingly persisted. Cystic degeneration then occurred and at 6 months of age the child had spastic quadriplegia and developmental delay.

CASE 2

A boy, weighing 2280 g, gestational age 32 weeks, had a platelet count which rose from $721 \times 10^9/l$ at the end of the second week to a peak of $969 \times 10^9/l$ on the fourth week and at the time of discharge, at 8 weeks, was still $818 \times 10^9/l$. This baby showed normal growth and appropriate development at follow up to 1 year of age.

In neither baby were there other abnormal haematological indices. Neither baby had perinatal asphyxia but both showed clinical features of narcotic withdrawal in the early neonatal period. Neither infant had received any antiplatelet drugs. Review of cumulative haematology reports of all other babies admitted to the intensive care unit at this time showed that thrombocytosis of this degree was not present in any infants of non drug abusing mothers during the same period of time.

The periventricular leukomalacia in case 1 was unexpected and it must be considered that the thrombocytosis contributed to the ultimate cerebral atrophy. The true incidence of thrombocytosis in babies of narcotic addicted mothers is unknown as not all babies require admission to a neonatal intensive care unit and only in those who have serial full blood examinations over several weeks is this essentially asymptomatic abnormality revealed.

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