Correspondence

Spina bifida and maternal Rh blood type

Sir,

Baker and Sherry¹ reported that of 28 mothers of children with spina bifida they found twice as many with Rh-negative blood group than would normally be expected. They stated that they had found no previous reports of such an association. In fact, Wiener² strongly propounded the existence of such an association, and McKeown and Record³ reported a significant excess of spina bifida infants delivered to Rh-negative mothers with antibodies. Other population data (Table 1) are consistent with the hypothesis that a woman who is Rh-negative is at a slightly increased risk of bearing such a child.

Information taken from the 1958 British Perinatal Mortality Survey⁴ is shown in Table 2. These data only include stillbirths and neonatal deaths with spina bifida. They show that the excess risk of spina bifida to women who were Rh-negative was apparent only when the woman was of parity 2 or more. This would again be compatible with the theory of an association between spina bifida aperta and maternal rhesus isoimmunisation.

Table 1 Percentage of mothers to be Rh-negative

Place	Spina bifida infant (%)	Control infant (%)	Reference
Rhode Island, USA	15.5	15.0	MacMahon <i>et al.</i> ⁵
Melbourne, Australia	18.8	17.0	Collmann and Stoller ⁶
Budapest, Hungary	25.0	14.8	Czeizel and Révész ⁷
South Wales, UK	17.8	16.0	Carter <i>et al.</i> ⁸
Birmingham, UK	18.5*	17.0*	McKeown and Record ³
Glasgow, UK	22.6	17.2	Wilson ⁹
1958 British Perinatal Mortality Survey†	18.9	17.5	Golding ¹⁰

*Estimated from graph, †stillbirths and neonatal deaths only.

Table 2 Percentage of Rh-negative mothers by parity

Parity	Mothers of spina bifida		Control mothers	
	(No.)	%	(No.)	%
0	(141)	16.3	(5974)	17.8
1	(50)	16.0	(4942)	17.0
2	(36)	25.0	(2459)	18.3
3	(17)	23.5	(1183)	17.7
4+	(21)	28.6	(1326)	15.5
Total with known Rh (265)		18.9	(15 884)	17.5

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An arteriovenous malformation of the liver giving rise to persistent transitional (fetal) circulation

Sir,

We report the case of an infant with an hepatic arteriovenous malformation who presented with cyanosis and heart failure in whom cardiac catheterisation suggested a persistent transitional (fetal) circulation.

A girl was delivered by forceps at 36 weeks' gestation after a normal pregnancy; birthweight was 2.950 kg. Apgar score at one minute was 6 with a heart rate of 120/min. She failed to establish regular respiration and required ventilation via an endotracheal tube for 12 minutes. She was externally a normal infant without skin blemishes. The placenta was oedematous and weighed 1250 g.

A systolic murmur was noted during resuscitation. She remained cyanosed in 40% O₂ after extubation. At 2 hours she was noted to have a 4 cm enlarged liver with a prominent left lobe. She was in heart failure with a sinus tachycardia of 180/min, a grade 4/6 pansystolic murmur at the lower left sternal border, and prominent peripheral pulses. BP was 75/45 mmHg. A N₂-washout with 100% O₂ raised the Pao₂ (descending aorta) to 6.3 kPa (47 mmHg). Chest x-ray showed a large heart with apparently oligemic lung fields, and an ECG showed biventricular enlargement. An echocardiogram showed all chambers and valves to be normally related.