tumours, we will not find it easy to confirm histologically what we at present regard as true negative results. We agree with Tullo *et al.* that investigation of other antibodies is warranted. In vitro we found monoclonal antibody (MoAb) 763.24T to have greater affinity for melanoma cells than MoAb 225 28S.[5]

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Circulatory collapse and ROP

SIR, Ng *et al.* (*BJO* 1989; **73:** 111-4) report an association between circulatory collapse, the development of severe retinopathy of prematurity (ROP) and periventricular leucomalacia (PVL). They suggest that postnatal hypoxic/ischaemic brain and retinal injury may form a common pathway in the pathophysiology of severe ROP and PVL in predisposed infants, though no data on the cause, severity, frequency, or duration of the circulatory collapse are given.

This theory is indirectly supported by metabolic data published from our unit. The frequency and duration of episodes of arterial pH falling below 7·2 due to metabolic, respiratory, or mixed acidosis, and also the duration of episodes of both hypoxia (PaO_2 <5·5 kPa) and hyperoxia (paO_2 >12 kPa) were shown to be significant variables in the development of ROP.[1]

Tissue hypoxia resulting from vascular insufficiency is a potent cause of metabolic acidosis, with consequent loss of cerebral and choroidal autoregulation and further loss of perfusion pressure, so it can be suggested that descriptions of circulatory collapse and descriptions of acidosis may refer to the same underlying processes. Quantified cotside measurements of cerebral blood flow using near infrared spectroscopy,[2] and continuous blood pressure monitoring are two techniques which may help to elucidate this important association further.

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SIR, We thank Messrs Acheson and Schulenburg for their interest in our article and also for their support for the idea that severe ROP and PVL may both be the consequence of cerebral circulatory hypoperfusion. We fully recognise the inadequacy of our data regarding the details of the circulatory collapse, but this is probably inevitable, as during this clinical catastrophe monitoring must necessarily take a back seat.

We are also looking at the metabolic risk factors for ROP. While on univariate analysis we obtained similar results,[1] using linear regression analyses for both the incidence and severity of ROP the variables PaO_2, pH, and PCO_2 failed to remain independently significant (in preparation). The relationship of metabolic acidosis to further loss of perfusion pressure remains speculative, and we are unable to comment further on this issue. The idea of prospectively continuously monitoring the cerebral blood flow and blood pressure to investigate this subject further is attractive but the logistics are probably overwhelming, as in our study this association developed in only 6 of the 299 infants at risk.

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Book review

Clinical Ophthalmology: A Text and Colour Atlas. By JAMES L KENNERLEY BANKES. Pp. 130. £14·95. Churchill Livingstone: Edinburgh. 1987.

This is a very helpful book for the beginner in ophthalmology and hence should be of quite wide interest. Medical students are probably its principal target, but opticians might also find the book of interest as well as some general practitioners. It is probably of somewhat limited value to anyone specialising in ophthalmology, but even for this group a read through during the first few days of the first residency would certainly not come amiss. Definitely a good book to have in the library. REDMOND J H SMITH