Correspondence

Renin and aldosterone response in human newborns to acute blood volume change

Sir,
We read with interest the report of Dillon and associates (Archives, 1978, 53, 461) on this subject. In a paper to be submitted to Rivista Italiana Pediatria we studied plasma renin activity (PRA) in 13 newborn infants undergoing exchange transfusion with ACD stored blood for hyperbilirubinaemia of various aetiology during the first 6 days of life. Exchange transfusions were such that the infants were kept normovolaemic. Weights ranged from 2400 to 3810 g, and gestational ages from 37 to 41 weeks. PRA and haematocrit were determined on umbilical vein samples before, midway, and at the end of the procedure, and from the transfused blood. The amount of blood administered ranged from 63.7 to 155.8 ml/kg, and the exchange rate from 0.964 to 5.288 ml/kg per min.

Statistical analysis of data was performed by a step-wise multiple regression program (UNIVAC 90/30), taking into account as dependent variable the average rate of renin production during the period of exchange transfusion and as independent variables the weight, postnatal age, basal PRA of the newborn, donor's PRA, and exchange rate. Allowing for the dilution caused by the donor's blood, hyperproduction of renin occurred in all cases. All these variables influenced, in various directions, the rate of renin release but the rate of exchange appears to be the most important factor in the activation of the renin-angiotensin system (multiple correlation coefficient = 0.774; F-value for analysis of variance = 16.404; standard error of estimate = 0.078; t = 4.050; P <0.01).

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Peripheral pulmonary artery stenosis

Sir,
The observation by Salisbury and Keeling (Archives, 1978, 53, 428) of polypoid lesions in the peripheral pulmonary arteries of an infant, recalls a controversy that was resolved over 20 years ago. Originally described in systemic arteries by Bucciante in 1945 and later by many others, these structures became known as 'pedunculated nodules' or 'polypoid cushions' and were interpreted as flow regulation mechanisms. Later studies

Familial neurodegenerative disorder associated with raised urinary VMA

Sir,
The belief held by Young and Hosking (Archives, 1978, 53, 682) that 'disturbances of catecholamine excretion have not been known to occur in children with a progressive neurological disorder or other neurological insult' is not true. Excessive urinary excretion of 4-hydroxy-3-methoxymandelic acid (HMMA) has been demonstrated in hydrocephalic infants with spina bifida (Barson and Hodge, 1971). It was felt from this study that a raised urinary HMMA reflected a persistently raised intracranial pressure, since it was not a feature of spina bifida alone or in cases where hydrocephalus was well controlled by a valve. It is likely that increased excretion of catecholamines is a nonspecific consequence of degenerating nerve tissue, whether this is in turn due to increased intracranial pressure or to an intrinsic neurological disorder.

Reference

GWILYM HOSKING
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Dr Hosking comments:
We are grateful to Dr Barson for drawing our attention to his important paper. It was remiss of us not to be aware of the data in that publication.

The raised levels of urinary HMMA seen in Drs Barson and Hodge's papers are most likely nonspecific as Dr Barson suggests. Similar increases are seen from time to time in a number of 'stress' situations. However, in our patient there was not only biochemical evidence of impaired catecholamine metabolism or excretion, but also clinical features apparently related directly to this abnormality. It is for this reason we suspect our patient's disordered metabolism was rather more specific.
produced morphological evidence that they were artefacts and that they resulted from invaginations of the arterial wall when tissues are stretched and then cut. This may happen during surgery as well as at necropsy. Moffat (1956) produced these changes, which are identical with those described by Salisbury and Keeling, experimentally.

As for the pulmonary vasculature, in our experience they are found particularly often in the thick-walled pulmonary arteries of newborn infants and of patients with pulmonary hypertension of whatever cause (Wagenvoort et al., 1964), while in normal adult individuals they are usually limited to the elastic pulmonary arteries and do not occur in the thin-walled muscular arteries in which the tendency to invaginate is apparently much less.

References


C. A. WAGENVOORT

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Weaning very low birthweight infants from mechanical ventilation using intermittent mandatory ventilation and theophylline

Sir,

We would like to support the observations of Dr Barr (Archives, 1978, 53, 598) who found that theophylline therapy facilitated the weaning of infants from mechanical ventilation when apnoea and bradycardia occurred at low IMV rates. We have used this combination therapy in 2 infants with hyaline membrane disease (1620 g, 32 weeks' gestation; 2000 g, 34 weeks' gestation). Theophylline serum concentrations were closely monitored, and all were in the therapeutic range (6 to 11 mg/l). Both infants were severely ill. Each had bilateral pneumothoraces requiring chest tube drainage. Both developed significant apnoea and bradycardia at IMV rates less than 5, necessitating increased ventilatory support primarily in the form of repeated bagging during apnoeic episodes. The infants had dramatic responses to treatment with theophylline, and we were able to extubate both infants within 48 hours.

We too believe that theophylline can aid in weaning infants from ventilators when apnoea and bradycardia occur at low IMV rates. Theophylline can be beneficial not only for low birthweight infants but also for those in whom rapid extubation or a reduction in mean intrathoracic pressure is desirable. Our infants with pneumothoraces are good examples, and others (such as those with pneumopericardium) come readily to mind.

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Dr Keeling comments:

We are aware that vascular 'abnormalities' may be produced by the casual treatment of tissues. The lungs in this case were fixed before blocks were taken for histological examination and we note that Moffat (1956) was unable to produce vascular 'lesions' when tissues were treated in this way; nor were we, on re-examination of semiserial sections of the lungs, able to find evidence of local vessel damage, thus excluding reported causes of artefact.

Reference


J. W. KEELING

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Detection of phenylketonuria

Sir,

Guthrie testing is performed at this hospital on blood samples received from the whole of Scotland, from children aged 6 to 14 days (Stevenson and Kennedy, 1974). Samples are assayed for phenylalanine, tyrosine, methionine, leucine, and galactose, blood samples being collected by hospitals or health visitors as appropriate, and a check on samples received compared with the number of registered births by the district medical officer.

Theoretically, therefore, all children should be included in this national scheme; although in practice the percentage of children sampled varies from 93–99% (Clayton, 1976).

In a recent search for the PKU cards of children born in Glasgow of known birth dates for another purpose, a surprising number of samples had either never been