LETTERS TO THE EDITOR

Clotting profiles and coagulation factors

EDITOR,—Neonates who have clinical signs of a bleeding diathesis warrant a coagulation screen to detect an underlying coagulation disorder. If the specimen collected for a clotting profile clots then the test cannot be done and a repeat specimen is required. Often samples that are clotted are not repeated due to the commonly held misconception that for a specimen to clot it must have normal coagulation factor concentrations.

We aimed to test the hypothesis that if a coagulation profile specimen clots then the patient's clotting profile must be normal. A retrospective analysis was performed, examining all neonatal clotting profile specimens that had clotted, from August 1 1996 to December 26 1998 at the Royal Women's Hospital, Melbourne, Australia. Clotted specimens were identified and data were collected from the clotting profile of specimens repeated later the same day. The clotting profile included the prothrombin time (PT) (seconds), activated partial thromboplastin time (APTT) (seconds), fibrinogen concentration (g/l) and D-dimer (mg/l). The reference ranges for each parameter were corrected for postnatal and gestational age.

Eighteen clotted specimens were taken from 11 neonates. Only nine were repeated on the same day, two of which clotted again and blood was not re-collected. Of the remaining seven specimens, five had abnormal results. Of those five abnormal results, all had increased PT, APTT, and D-dimer, and two had an accompanying low fibrinogen concentration. In summary, half of the clotted specimens were not repeated, and 71% of repeated specimens were abnormal.

Only 20–30% of most coagulation factors are required for clot formation in vivo,[1] and this may also be true for blood collected for analysis. These data support the principle that it is wise not to assume that clotted specimens come from patients with normal clotting profiles. We conclude that if a clotting profile is clinically indicated and the specimen clots, this does not imply normal coagulation, and another specimen should be collected for analysis.

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Clomipramine withdrawal in newborns

EDITOR,—Maternal clomipramine use during pregnancy may cause epilepsy in newborns.[1–3] We present the case of a neonate who developed a thus far unrecognised manifestation of clomipramine withdrawal.

A 38 year old woman had been treated for depression throughout pregnancy with clomipramine (100 mg/day). No other medication had been taken. A boy weighing 3370 g was born at term; he had excellent Apgar scores. Two days later, the child was jittery following startling impulses. On the fifth day, simultaneous and symmetrical jerks of arms and legs developed, but the child remained alert. The jerks could be provoked by touch or auditory stimuli, and stopped by briefly stabilising the limb. Neither the face nor the eyes showed abnormal movements. Clomipramine serum concentration was <10 ng/ml. Other laboratory tests, cerebrospinal fluid analysis, and ultrasound brain scans were unremarkable. Suspecting a status epilepticus, the child was treated intravenously with clonazepam and phenobarbital, without success. Subsequently, a 10-channel electroencephalogram, recorded while limb jerks were present, showed no epileptic activity and a normal background pattern.

We diagnosed a generalised, stimulus sensitive, status myoclonus. A single dose of 0.5 mg clomipramine suppressed the myoclonus immediately, but caused somnolence. Over the next four days, the patient had only sporadic myoclonic jerks, while consciousness improved. Examination three weeks later was normal, except for mild jitteriness in response to touch.

This report shows that the clinical spectrum of clomipramine withdrawal in newborns includes myoclonus. Limb jerks appeared several days after birth and were suppressed by clomipramine, which suggests a withdrawal effect[1–4] Myoclonus has hitherto not been recognised, but epileptic seizures are commonly reported.[1–3] However, EEGs are never performed, except for one neonate with generalised clinical manifestations and a focal epileptogenic focus.[3] Hence, epilepsy was not unambiguously demonstrated and myoclonus may have been present in some patients.

In this case, several observations argued against epilepsy. Antiepileptic drugs were ineffective, limb jerks were stimulus sensitive, and the child remained responsive during generalised jerks. Importantly, the EEG showed no abnormalities during the generalised jerks, which proved they were not epileptic.

Recognition of myoclonus has therapeutic consequences because status epilepticus requires aggressive treatment, with phenobarbital as the first choice. However, phenobarbital is often ineffective for myoclonus[1 3] and may theoretically aggravate withdrawal signs due to liver enzyme induction which facilitates clomipramine clearance.[4] Low dose clomipramine, which allows for graduate tapering of clomipramine concentrations, seems more effective for myoclonus.[1 3]

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Neonatal diabetes and severe intracerebral haemorrhage

EDITOR,—A 20 day old baby boy was referred to our hospital with a history of right sided facial palsy, nystagmus, and seizures. He had been born by normal vaginal delivery, at 36 weeks of gestation, with good Apgar score (8 and 9 at five minutes, respectively). He had jaundice, for which he required phototherapy for four days at the referring hospital, but was otherwise normal.

Ten days after discharge he presented again with a history of poor feeding and lethargy for one day. On examination, he looked pale with a full and tense anterior fontanelle. He was febrile and tachypnoeic. A presumptive diagnosis of sepsis/meningitis was made and he was given cefotaxime and ampicillin. Spinal tap was deferred as the fontanelle was tense. He developed intractable seizure, requiring diazepam, phenobarbitone, and phenytoin.

In view of his deteriorating condition, he was transferred to the university hospital. On arrival, he was noted to be febrile and lethargic. After stabilisation an emergency computed tomography scan of the brain was performed, which revealed cerebral oedema and a large, left sided, intracerebral bleed, with extension into the subarachnoid spaces and ventricle. Investigations on admission revealed severely deranged electrolytes. His serum glucose was 51.2 mmol/l, sodium 170 mmol/l, potassium 7.5 mmol/l, urea 39 mmo/l and creatinine 246 µmol/l. The white cell count was 17×10^9/l, with a haemoglobin of 156 g/l. The platelet count was obtained from the referring hospital because our specimen had a small clot (125×10^9/l). The coagulation profile was normal (PT 15 seconds, APTT 45 seconds, D-Dimer > 0.5 < 1 µg/ml, fibrinogen 3.1g/l). The infant was treated symptomatically with supportive care, but died on the second day of admission.

The common causes for bleeding were ruled out. There was no history of birth asphyxia or trauma. The platelet counts and coagulation profile were normal. Although the chance of arterio-venous malformation as a cause of spontaneous bleed cannot be ruled out completely, the computed tomography scan gave no indication of this. The diagnosis of neonatal diabetes was supported by concomitant hyperglycaemia (51.2 mmol/l), glycosuria, metabolic acidosis (pH 7.29, baseline excess –16.3), dehydration and weight loss (weight at admission was 2050 g compared with birthweight of 2580 g).

Neonatal diabetes is rare. The incidence rates reported from the UK and Germany are 1 in 400 000 and 1 in 500 000, respectively.[1] As far as we are aware, no reports have been published on the association between neonatal diabetes and intracerebral haemorrhage. However, previous reports have associated hyperglycaemia with haemorrhagic transformation of the cerebral infarct.[2 3] In a recent study, Scott *et al* showed an increased incidence of intracerebral haemorrhage with

admission hyperglycaemia in adults.[4] In an animal model study, de Courten-Myers *et al* reported 25-fold more extensive haemorrhage in cats with hyperglycaemia compared with normoglycaemic cats.[2] Similarly, Broderick *et al* associated hyperglycaemia with cerebral bleed in two human adults.[3] The exact mechanism by which hyperglycaemia induces cerebral bleeding is not clear. It enhances lactic acidosis in brain tissue and the combination of hyperglycaemia and acidosis enhances endothelial damage with subsequent extravasation of red blood cells through the leaky vessels.[3] There have been studies on the effect of glucose and acid base changes on brain ischaemia.[5 6]

A causal link between hyperglycaemia and intracerebral haemorrhage in neonates may be difficult to prove, but we look forward to other prospective studies on the risks and mechanisms of brain haemorrhage in neonates with severe hyperglycaemia.

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Neonatal paracetamol poisoning

EDITOR,—Neonatal paracetamol poisoning is rare. To date, there have been no definitive therapeutic guidelines for its management. We describe the successful use of N-acetylcysteine in the management of a term infant who had transplacentally acquired paracetamol poisoning.

A 2.88 kg term infant was born at home to an 18 year old single mother. Five hours after delivery the mother admitted to taking about 40 tablets (20 g) of paracetamol three hours before delivery. Investigations performed on both mother and baby nine hours after ingestion confirmed the presence of significantly high serum concentratios of paracetamol (table 1). The mother's liver enzymes and coagulation profile were both normal. The infant, however, had evidence of hepatotoxicity with a prolonged International Normalised Prothrombin Ratio (INR) of 3, increased liver enzymes, and hypoglycaemia (blood glucose 2 mmol/l) (table 1). At admission to our unit, N-acetylcysteine was given according to the following protocol:150 mg/kg in 5% dextrose over 30 minutes, then 50 mg/kg in 5% dextrose over 4 hours, then 100 mg/kg in 5% dextrose/24 hours until the INR returned to normal.

The infant was mildly jaundiced (day 1 of life), alert, without enlarged liver and spleen or evidence of a bleeding diathesis. Serial investigations showed a gradual fall in the serum paracetamol concentration. N-acetylcysteine was continued until the INR returned to normal. Urine output was good and the infant remained active. The synthetic function of liver showed gradual improvement with normalisation of INR at 48 hours. The infant was discharged well on day 7 of life.

Even though no guidelines on safe limits for paracetamol overdose in neonates have been established, it would be safe to use those in use for older children, while erring on the side of caution. The beneficial effects of N-acetylcysteine for paracetamol overdose have been documented in infants and children,[1–3] but there is no published evidence of its use in the management of neonatal paracetamol overdose. Exchange transfusions have been used,[4 5] but increase the potential for a rebound increase in blood concentrations, necessitating repeat exchange transfusions, because of tissue sequestration of paracetamol.

N-acetylcysteine is a relatively safe drug, an effective antidote for paracetamol, and because of its antioxidant effect, has also been used in the management of non-paracetamol fulminant liver failure.[6] We suggest that N-acetylcysteine should continue to be used in neonatal paracetamol poisoning until the INR returns to normal.

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Table 1 Biochemical profile of mothers and infants

	Neonates							*Mothers*	
Time from ingestion (h)	9	18	27	32	48	72	7 days	9	32
Paracetamol (mg/l)	133	67	25	<0.1	<0.1			147	<0.1
PT (sec)	44							14	
INR	3		1.68	1.14	0.94				1.01
Bilirubin (mmol/l)				90	99	85	25	11	
AST (U/l)				86	48	55	32	28	
Creatinine (mmol/l)	89	81		89	61	53	50	52	

"Dewatering" of the lungs

EDITOR,—The letter from Hills and Masters[1] in response to my commentary of their paper[2] raises some interesting points which require further comment.

I stand corrected over my description of their model of the alveolus as "dry." This was a short hand term on my part to distinguish Hills' theory on which he has published widely from the more traditional theories of how pulmonary surfactant works.

Their letter, however, does not challenge the basic points I made in reluctantly refuting their hypothesis. These were, firstly, that fetal lung liquid can be absorbed before birth—that is, before the establishment of an air–liquid interface and thus before surface forces can act—and secondly, that even in neonates who have intact pulmonary surfactant function, there is total failure of lung liquid removal when sodium ion transport is abolished.[3] The latter finding, in particular, must make us question whether surfactant has any role in lung liquid removal at birth.

The authors make the novel suggestion in their letter that the oligolamellar structures of surfactant could be the barrier to diffusion at intercellular junctions—an action which they claim makes the lung epithelium relatively tight and thus enhances its ability to secrete or absorb liquid through generating osmotic forces by ion movement. Before birth the pulmonary epithelium is already "tight" and restricts the passage of molecules the size of sucrose and larger.[4] The low permeability is constant during gestation[5] and does not decrease (at least in the liquid filled lung) when surfactant appears near term, a finding which argues against this newly proposed function for surfactant.

Measuring permeability in the air filled postnatal lung is difficult, but the evidence suggests that there is a temporary increase in permeability over the first 12 hours after birth (probably as the result of stretch) which then reverts to near fetal permeability levels.[6 7] The reason that it takes days to clear fluid from the lungs in respiratory distress syndrome (RDS) even after administration of exogenous surfactant, is because the epithelial barrier has been breached in the early stages of the disease and cannot maintain an osmotic gradient, however hard the Na^+ "pump" works. Indeed, damage to the pulmonary epithelium in RDS has been shown to occur within two minutes of birth.[8] It is the slow healing of the epithelium, hindered by persistent barotrauma and high oxygen tensions, which causes the prolonged recovery from severe surfactant deficient RDS.

The picture included in the author's letter seems to provide evidence for the traditional theory—that surfactant rests on a very thin alveolar liquid layer and not vice versa. The thickness of this layer measured in vivo by physiological means in the air filled lung, rather than by microscopy, is indeed very small. The mean thickness being calculated at only 0.1– 0.2 microns, indicating that in some areas it will be even thinner.[9 10]

I accept that these physiological measurements are no more able to settle the arguments about how precisely surfactant is distributed in the alveolus than can microscopy. However, for ion transport and water movement to work, there must be ready access to the

apically placed epithelial ion channels by ions in the alveolar liquid—the alveolar liquid would be better placed below any surfactant forms rather than above them. A more detailed discussion of the possible interactions between surfactant and lung liquid movement has been given elsewhere.[11]

The authors are to be complimented on their thought provoking suggestions, but, ultimately, all hypotheses must be supported by experimental evidence.

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Detection of heart disease in infancy

EDITOR,—Gregory *et al*[1] state that after neonatal examination, the 6–8 week examination provides the only opportunity for detection of heart disease in infancy, other than the opportunistic recognition of a murmur or the development of symptoms. This is supported by the most recent report of the joint working party on Child Health surveillance,[2] which states: "there is no justification for carrying out a screening check for heart murmurs after 8 weeks of age."

In the Bath clinical area routine auscultation of the heart was, until recently, carried out at the 8 month check, and indeed in the Wiltshire catchment area this is still current practice. A retrospective study was performed, looking at the records of around 10 000 children seen over 27 months. Hospital records, community child health records, and General Practitioner records were reviewed, as appropriate, to determine the outcome of children referred to general paediatric outpatient clinics with a murmur. Forty seven children were seen and notes were available for 45.

When examined in outpatients, six children (13.3%) had no audible murmur and 30 children (66.7%) were felt to have an innocent murmur. Nine children (20%) were thought to have a cardiac lesion. These children were all asymptomatic and all had had normal six week checks. Of the nine children, two were thought to have ventricular septal defects and seven were referred to a paediatric cardiologist. Subsequently, three were found to have structurally normal hearts, two pulmonary valve stenosis, and one a ventricular septal defect. There was no further information on one child.

Therefore, as a result of the 8/12 check, three children with ventricular septal defects and two with pulmonary valve stenosis were identified.

Our study highlights the importance of vigilent opportunistic screening if the routine 8/12 check is dropped. Ways to promote opportunistic screening must be sought.

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BOOK REVIEW

Brain damage in the newborn and its neurologic sequels. Towbin A. [Pp 764, hardback $302]. PRM Publishing Ltd, 1998. ISBN 0-9660551-0-1.

Clinicians working with children with cerebral palsy see the end result of damage which occurred many years before. To understand the causes and timing of this damage, you need to look at its origins in the fetal and newborn periods, because it is now abundantly clear that most cerebral palsy arises in prenatal life; in only a small proportion of children is there evidence of injury at birth.

The causes are by no means understood. Hypoxia and ischaemia have long been regarded as the most important, but pathological and epidemiological studies show that very similar forms of damage also result from maternal and placental infections, possibly mediated by cytokines crossing the placenta into the fetal brain, by metabolic disease, iodine deficiency, etc.

The contribution of very early intrauterine damage to birth asphyxia was first noted by Sigmund Freud over 100 years ago; not all birth asphyxia is what it seems. Many of these infants were set up months beforehand for a difficult birth.

Dr Abraham Towbin recognised the value of studying the fetal and neonatal brain. He worked with the most eminent of neuropathologists, Paul Yakovlev, and from him learned the method of celloidin embedding. Using this technique, these tiny, fragile, and precious specimens can be embedded whole in plastic and entire brain sections cut and examined under the microscope, with unparalleled preservation of the pathologic anatomy. You won't persuade a technician to undertake this technique today; it is far too costly, both in time and resources for modern laboratories.

This book represents a celebration of Dr Towbin's 40 plus years in paediatric neuropathology. It is a meticulously documented collection of over 200 case studies accompanied by detailed obstetric and clinical histories. It is beautifully produced and the illustrations are superb. They include gross and microscopic pathology of the brain, placenta, and body organs as well as many radiographs. Most are full colour, every one is clean and crisply focused, and the legends are economical.

The weakest part of the book is Dr Towbin's interpretation of the pathogenesis of developmental brain damage. It is idiosyncratic and he paints with a very broad brush. The clinical chapters are scarcely more informative.

But the real value of this book is that it allows us to share in a rare wealth of carefully archived material; an invaluable resource for pathologists, obstetricians, neonatologists, neurologists and developmental psychiatrists.

This book has a timely message in an age of ever-increasing litigation. Only a small proportion of developmental brain damage occurs at birth; in over 80% we still have to seek causes and establish prevention in the prenatal period.

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CORRECTIONS

In last month's edition (Arch Dis Child Fetal Neonatal Ed) 1999;80, the following were inadvertently published incorrectly:

In the Letters to the Editor section, several names were omitted from **Vascular ring: an important cause of upper airways obstruction (F252–3)**. In addition to A Sharples, I N Keengwe, I Ahmad, and O Dearlove should have been included.

The figure legends in **Serum malondialdehyde concentration in babies with hyperbilirubinaemia (Yigit, et al, F235–7)** should have been:

Figure 1 Correlation between malondialdehyde and bilirubin concentrations in group 1.
Figure 2 Correlation between malondialdehyde and bilirubin concentrations in group 2.

The first author of **Body composition of preterm infants during infancy (F188–91)** should have been R J Cooke and not D J Rawlings, as published.