PostScript

LETTERS

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Neonatal sepsis in Peshawar: Author's reply

I am responding to the letter of Ali *et al*.[1]

(1) The total number of babies (1598) and number of culture positive babies (1003) in our article[2] represent the number of cases after removal of patients meeting the exclusion criteria. We did find isolated cases of *Streptococcus* sp, *Salmonella*, and *Enterococci*, but they happened to fall in the excluded group.

These organisms accounted for < 0.5% of the spectrum as a whole. This is similar to the findings of Maryam *et al*[3] in a public sector institution with a population of similar socioeconomic, cultural, religious, and climatic background. Their study was carried out in the same time period as ours but completely independently and blinded from ours. In their series of 284 cases, they grew *Escherichia coli* (130 cases; 45.7%), *Klebsiella* (49 cases; 17.2%), *Pseudomonas* (46 cases; 16.2%), *Staphylococcus aureus* (39 cases; 13.7%), *Staphylococcus epidermis* (18 cases; 6.34%), *Strepococcus* sp (1 case; 0.3%), and *Salmonella* (1 case; 0.3%).

The numbers are slightly different among the studies from south of Pakistan[4]. This is not surprising as neonatal sepsis is known for the temporal and regional variation of the spectrum of its organisms even in different hospitals within the same city.

(2) The basic message from the majority of studies from Pakistan is the same: Gram negative organisms are the main cause of neonatal sepsis in Pakistan, followed by *S aureus*. This group of organisms is responsible for > 99% of the spectrum, and unfortunately the grave situation of multidrug resistance is emerging among these organisms. That is where research needs to be concentrated, instead of on the organisms responsible for < 0.5% of the spectrum (*Salmonella*, *Streptococcus* sp, etc), which do not carry any significance for overall neonatal mortality and morbidity.

(3) Out of 296 cases of *S aureus* in our series, ampicillin was tested on 285 cases, with 171 (60%) sensitive to it, and 279 were tested with augmentin, with 75 (26.9%) sensitive to it. I agree with Ali *et al* that this pattern of sensitivity looks unusual as far as *S aureus* is concerned, although this phenomenon is known to occur with β lactamase-producing *E coli*. It may be due to the various strengths of augmentin discs available, the known biochemical instability of clavulonic acid, or the difficulty of interpretation when a combination of two antibiotics is used in one disc using the disc diffusion technique. However, I would be interested to hear more expert opinion on this. Our series did not exclude hospital acquired infections.

(4) The longitudinal analysis of our data shows an increasing sensitivity to penicillin and decreasing sensitivity to cephalosporins, particularly cefotaxime, over the last half decade. This is consistent with the change in antibiotic use in Pakistan since the early 1990s when penicillin/gentamicin was replaced by cephalosporins/amikacin as the first line antibiotic treatment. Most of the Gram negative organisms in Pakistan maintain a very high degree of sensitivity to amikacin[3 4] but not to gentamicin. I feel that penicillin/amikacin may be a very good choice as the first line antibiotic in neonatal units in Pakistan. It is high time that we reviewed our antibiotic policies and at the same time approach the government to rationalise antibiotic marketing in this country.

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Serum S-100 protein does not correlate with cerebral ultrasound scans in preterm infants

Whitelaw *et al*[1] recently reported that S-100 protein concentration was 20–200 times higher than control levels in the cerebrospinal fluid of infants with posthaemorrhagic ventricular dilatation. S-100 protein is produced only within the brain by astrocytes, but it can be detected in the serum after cerebral damage in adults with stroke, where it is a marker of infarction volume,[2] and it has also been found to be useful in head injury.[3] A study of term infants found that serum S-100 protein levels were detectable after uncomplicated delivery.[4] Cerebrospinal fluid is not often taken from preterm infants, and an easily obtainable serum marker for brain cell damage would be of value as an indicator of the degree of cerebral insult in such infants.

The aims of our study were to determine firstly whether S-100 protein could be measured in the serum of preterm infants during the first week after birth, and secondly whether there was a significant difference in these levels between infants who had and had not suffered parenchymal damage as diagnosed by cerebral ultrasound scans taken during the first week after birth.

Thirty preterm infants of 25–35 weeks gestation were recruited after signed parental consent, and 0.5 ml blood samples for S-100 determination were taken at the same time as routine phlebotomy on days 1, 2, 3, 5, and 7 after birth. Routine cerebral ultrasound scans were undertaken during the stay on the unit. The study was approved by the Manchester research ethics committee (central).

S-100 protein was detectable in the serum of preterm infants and concentrations ranged from 0.85 to 22.0 μg/l (table 1).

Seven of the 30 infants had features of parenchymal damage on cranial ultrasound scan, with a median S-100 protein level of 3.39 μg/l. The remaining 23 infants had normal scans and had a median S100 protein level of 3.18 μg/l (table 2). The data were analysed using a Mann-Whitney U test, and no significant difference in levels of S100 protein was found between the two groups (p = 0.774).

Table 2 Serum S-100 protein levels in preterm infants with and without parenchymal damage as shown on cerebral ultrasound scanning

	Parenchymal damage	No damage
Number	7	23
Median S-100 (μg/l)	3.39	3.18
Range	0.76–22.0	0.59–13.42

Table 1 Serum S-100 protein levels in preterm infants of 25–35 weeks gestation by day after birth

	Day				
	1	2	3	5	7
Median (μg/l)	2.90	3.11	2.65	3.11	2.56
Minimum (μg/l)	0.92	0.91	0.91	0.85	0.59
Maximum (μg/l)	13.3	22.0	10.0	11.9	14.1

This study confirmed the presence of S-100 protein in the serum of preterm infants, but, in view of these findings, its measurement would not be useful as a marker of cerebral damage.

Acknowledgements: Cambridge Life Sciences performed the S-100 protein analyses.

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Neonatal shaken baby syndrome—historical inexactitudes

I read with interest the article on neonatal shaken baby syndrome.[1] Although a fascinating account of the sequence of events in this saga, it is factually incorrect in several respects. As the perinatal pathologist involved in the Birmingham series, I raised the possibility that the brain damage was due to the effects of physiotherapy prior to the publication of our report. My co-authors felt that the suggestion was too speculative to be included. It is, however, of note that the physiotherapy regime was changed at this time as a precaution and as I remember I encountered only one further case until my retirement in 2000. At the time I presented the pathological data at several scientific meetings both in the UK and abroad, suggesting physiotherapy was relevant and also that the method used in Birmingham appeared to be unique in allowing free movement of the baby's head during treatment of the chest.

Some years later I received a telephone call from Dr David Becroft, the perinatal pathologist concerned with the New Zealand cases that pathologically appeared very similar to our own. They had no explanation for their cases at this time and I indicated that I had always been of the opinion that physiotherapy was responsible. As I understand it this resulted in changes in the physiotherapy regime in New Zealand and the disappearance of the lesion.

In retrospect, I should have insisted that my hypothesis, however speculative, was included in our original paper or expressed the view in the correspondence columns at the time because it might have prevented or at least reduced the number of affected cases in New Zealand. Certainly today I would not have been so reticent. In the event it is gratifying that detailed clinical analysis of the cases confirmed my original opinion.

As to the pathology of the condition, detailed unpublished studies of the affected brains in our series suggest the lesion is more akin to that of hydranencephaly than infantile shaken baby syndrome and leads me to raise the possibility that hydranencephaly may be the result of intra-uterine brain trauma.

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Neonatal shaken baby syndrome—lessons to be learned

Williams and Sunderland,[1] and the accompanying commentary from Rosenbloom and Ryan,[2] discuss a severe cystic brain lesion associated with chest physiotherapy in very preterm infants. Rosenbloom is correct that the topic lacks topicality, but mainly because neonatal chest physiotherapy is now used very little if at all. I disagree that there is an abundant literature detailing appropriate treatment and the absence of brain damage associated with neonatal chest physiotherapy. Older data suggested benefit,[3–5] but more recent publications demonstrate none.[6–8] The reported benefits were transient improvements in oxygenation and slight increased removal of secretions. The older studies are all too small to adequately address safety. Chest physiotherapy, by whatever method, has little or no place in neonatal intensive care.

There are several lessons to be learned from the experience of the units who found these brain lesions. Firstly, a treatment generally recognised as being beneficial may not be so, especially with other changes in care over the passage of time. Continued reassessment of the usefulness of treatment is needed. Secondly, side effects can appear, even when a treatment has supposedly passed the test of time. Ongoing audit is needed. Thirdly, there is a dilemma that clinicians face in reporting complications. The first hospital to find this lesion did not further investigate the cause, report its suspicions, or inform the parents of the affected babies.[9] The second hospital did all of these.[10] That hospital has been subject to a long official public inquiry and law suites, and had 20 medical, nursing, and physiotherapy staff investigated by registration authorities, lasting 8 years. All this happened in the supposedly non-litigious medicolegal environment of New Zealand. There needs to be the ability to be open about complications and side effects and have an atmosphere of learning from, rather than blame for, them.

I would like to correct one statement by Williams and Sunderland. In our nursery there was no change in the vigour of chest physiotherapy from the introduction of the technique in 1985 until we stopped all chest physiotherapy at the end of 1994. The cerebral lesions appeared 1992–94. From 1985, the same physiotherapist was teaching and supervising the technique. During those 3 years, babies who developed the brain lesion had more chest physiotherapy than matched concurrent controls, but considerably less than many infants in previous years. Why the brain lesion began to appear remains a mystery.

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Authors' reply

We are grateful to colleagues for their comments on our annotation.[1] We would stress that we merely abstracted the views of others, so any criticisms (apart from our brevity) will be of the lawyers, doctors, nurses, physiotherapists, and parents who contributed to the Royal Commission Report. We found it to be systematic, rational, and objective.

We strongly refute any suggestion that any of the New Zealand professionals should be criticised let alone made scapegoats (witness our final paragraph). We are puzzled that Drs Rosenbloom and Ryan discount the quoted witness statements of the parents and involved clinicians.[1] The lawyers and doctors are clear that the physiotherapy and nursing practices did occur and that the levels of head shaking were not monitored.

We are concerned with infant brain injuries not lung disease and consider this to be topical. We share colleagues' concern at the need to base opinions on speculative presumption extrapolated from animal or accident research and are aware of the limited evidence that identifies the minimal forces needed to cause shaken brain damage in neonates or older infants. We found the reported experiences to be a helpful insight.

We are delighted that Dr Rushton has taken this opportunity to state he thought vigorous chest physiotherapy without supporting the head was responsible for the porencephalic lesions and to inform of his pivotal involvement in advising New Zealand colleagues.[2] We understand there were earlier concerns that publishing the speculation about physiotherapy would open liability to litigation. Lawyers might consider the inference that

fear of litigation led to suppression of information that might have prevented the New Zealand deaths and the dilemma facing clinicians who reported the cerebral implications of vigorous physiotherapy. Dr Knight reports their unit has been 'subject to a long official public inquiry, law suits and had 20 medical, nursing, and physiotherapy staff investigated by registration authorities, lasting 8 years.'[3]

We do not accept criticisms of inaccurate references. The Cochrane review we both cited was last updated in 1997. There has been an updated review this year (dealing with lung not brain disease), which was unavailable to the editors or us at the time of submission. Dr Knight states[3] there was no change in the vigour of chest physiotherapy from 1985 until the end of 1994 but he co-authored the paper[4] we cited that states that there was no policy to support the head during chest physiotherapy and no data on the extent the head moved during physiotherapy, whether given by nurses or physiotherapists. The Royal Commission Report found no record of the vigour of chest percussion and understood there was considerable variation with no standardisation of training.

We recommend interested colleagues to read this report and the publications of Knight *et al*[4] before dismissing the possibility that vigorous chest physiotherapy without supporting the head may cause brain injuries in certain circumstances.

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SIDS, smoking, and arousal thresholds: conclusions not supported by data

The investigation of the effect of maternal tobacco smoking on arousal in healthy infants[1] concluded that maternal tobacco smoking increases arousal thresholds (i.e. impairing arousal) in infants of 2–3 months of age, during quiet sleep in the supine position. It is suggested that this may provide an explanation for the association between smoking and sudden infant death syndrome (SIDS).

This conclusion is not supported by the data because the study also found that maternal tobacco smoking reduces arousal thresholds in 2–3 month old infants, in active sleep in the prone position—the very position in which victims of SIDS are still most commonly found.[2 3] Could passive smoking then be protective of death from SIDS?

Smoking is undoubtedly associated with SIDS. However, these contradictory findings do not support the hypothesis that an alteration of infants' arousal thresholds by passive smoking is explanatory.

Is it not time that SIDS research concentrated less on smoking and more on alternative mechanisms?

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Author's Reply

In response to the letter from Tom Blyth and Sheila McKenzie,[1] I wish to clarify the following points. Our study is the first to examine the effects of both sleeping position and maternal smoking, both factors that are associated with an increased risk for SIDS, on arousal from sleep. We had hypothesised that the effects of these two risk factors might be additive. Our findings, however, showed that sleeping position had no effect on arousal threshold in the smoking group, but arousal was impaired in the non-smoking group when they slept prone. The arousal responses to both stimulus induced and spontaneous arousal were, however, impaired in the smoking group in the supine position. The significant findings that Blyth and McKenzie highlight as being supportive of the idea that passive smoking is protective of SIDS may be explained by this finding that prone sleeping elevated arousal thresholds only in the non-smoking group.

We strongly disagree with the suggestion that passive smoking might be protective of SIDS when infants sleep prone. Our finding of depressed arousal responses in infants of smoking mothers is also supported by those of other workers.[2 3] As yet the mechanism(s) that causes some infants to die suddenly and unexpectedly is unknown, it is thus of great importance that research should focus on how the known risk factors for SIDS might act. At present, a failure to arouse from sleep in the face of a life-threatening event is a leading hypothesis for SIDS. In support of this, prone sleeping, maternal smoking, recent infection, head covering, overheating, and prematurity—all risk factors for SIDS—have all been demonstrated to decrease arousability in otherwise healthy infants. Conversely, the use of pacifiers, which decrease the risk of SIDS,[4] has been shown to increase arousability.[3]

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Swallowing induced supraventricular tachycardia in a neonate

We would like to report on an infant with swallowing induced supraventricular tachycardia (SVT). This has rarely been described in adults,[1] but, to our knowledge, has not been previously reported in neonates or children.

A male infant weighing 4500 g was born by vaginal delivery at term after a normal pregnancy. He was admitted to the neonatal unit for phototherapy and was diagnosed with SVT. There was no haemodynamic instability and he was cardioverted with adenosine. He had recurrent SVT and was started on antiarrhythmic treatment. He subsequently had recurrent SVT which was noted to be precipitated by feeding. SVT did not appear to be triggered by sucking a pacifier but occurred within seconds of feeding. Termination of the SVT usually occurred spontaneously about 30 minutes after the end of a feed. On a few occasions, longer periods of SVT required adenosine for cardioversion. In an attempt to control his SVT, oral feeds were discontinued and he was started on intravenous fluids. He remained in sinus rhythm throughout 18 hours of fasting. When feeding was resumed, he again developed SVT. Treatment with digoxin or propranolol at therapeutic levels did not control the SVT.

There was no evidence of Wolff-Parkinson-White syndrome on the electrocardiogram, which showed a normal cardiac anatomy with good ventricular function. A barium swallow was performed, which revealed a normal oesophageal anatomy and swallowing mechanism. An abdominal ultrasound was normal and showed good diaphragmatic movement with respiration.

Treatment with increasing doses of flecainide resulted in a reduction in the frequency and duration of the SVT. Before discharge on flecainide 3.5 mg/kg/day, a 24 hour continuous ECG recording did not produce any evidence of SVT. This patient subsequently had recurrent SVT unassociated with swallowing and remains on antiarrhythmic treatment.

Swallowing induced SVT has been recognised in adults without evidence of gastro-oesophageal disease[2] and in association with pharyngoplastic manipulations.[3] In one intractable case, intrapleural repositioning of the oesophagus to physically separate it from the right atrium successfully achieved control.[4] Swallowing increases vagal tone, and thus termination of SVT is often associated with swallowing and other vagal

manoeuvres. Changes in vasosympathetic tone or perhaps mechanical stimulation are possible causes for this unusual phenomenon.

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Management of mothers of neonates with vertically transmitted sexually transmitted infections

Vertical transmission of sexually transmitted infections such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae* can cause ophthalmia neonatorum and rarely pulmonary infection.[1]

To optimise the management of mothers, and their partners, of neonates diagnosed with these infections, we set up a system of direct fax referrals between our Microbiology Department and the local genital-urinary medicine (GUM) clinic. This is in line with the national strategy for sexual health,[2] which stresses greater collaboration between agencies. A similar direct referral system is already in place for gynaecology patients diagnosed with sexually transmitted infections.[3]

We undertook a retrospective case notes review of all neonatal referrals between January 1998 and December 2000. Of 25 neonates referred, 24 had chlamydial and one had gonococcal eye infections.

Five mothers made contact with clinic health advisors and opted for management by their general practitioner. Eleven mothers attended the GUM clinic. No infection was found in three cases that had already received treatment. Genital chlamydia were confirmed in the remaining eight cases. One had co-infection with *N gonorrhoeae,* which was not suspected in the baby, and another had *Trichomonas vaginalis*. Contact tracing yielded only eight partners (0.73 per index case). Of these, six attended the same GUM clinic (55% of index cases) and one attended his general practitioner. Four had confirmed chlamydia infection, but no other co-infection was discovered in these men. The remaining nine mothers did not contact the GUM clinic. We ensured that their general practitioners would agree to take over follow up.

There were no complaints from the mothers or their partners about being contacted by the GUM clinic.

The two year review of this initiative shows it to be an effective way of treating and contact tracing mothers of infected neonates. Joint protocols and good communication between all the healthcare professionals are paramount to the success of such a scheme.

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Decreasing oxygen saturation in very early preterm newborn infants after transfusion

Very early preterm infants are born at a time when more than 90% of their red blood cells contain fetal haemoglobin (HbF)[1] and therefore their blood has a high affinity for oxygen.[2] Because of blood sampling, early preterm newborns often receive transfusions for blood volume replacement. These transfusions are carried out with adult red blood cells containing adult haemoglobin (HbA) and decrease the HbO_2 affinity.

The change in P_{50} after transfusion has not previously been published in human preterm infants. A study was therefore planned to be carried out during the first week of life in very early preterm infants to determine the decrease in HbO_2 affinity (Po_2 required to achieve a saturation of 50% at pH 7.4 and 37°C) after a single transfusion as well as the relation of P_{50}.

Four very early preterm infants of about 26 weeks gestation, free of any congenital anomalies and transfused during the first week of life, were included in the study. The oxygen dissociation curve and the P_{50} were determined by Hemox-Analyser (TCS Scientific Corp, New Hope, Pennsylvania, USA) as previously described.[3] A 50 µl volume of whole blood was added to 4 ml buffer (135 mM NaCl, 30 mM TES, 5 mM KCl, and NaOH adjusted to pH 7.4 ± 0.02; TCS buffer; TCS Scientific Corp), 10 µl antifoam solution, and 20 µl 20% bovine serum albumin. Samples were analysed immediately on collection from the patient. Nitrogen (100%) was bubbled through the sample at a constant rate that resulted in complete deoxygenation within 20 minutes, followed by reoxygenation with air for 15 minutes. The analyser measured the oxygen tension with a standard Clark oxygen electrode (model 5331 Oxygen Probe; Yellow Springs Instrument Co, Yellow Springs, Ohio, USA).

The mean (SD) gestational age was 25.3 (1) weeks and the mean (SD) birth weight was 755 (185) g. The mean (SD) volume of red blood cells transfused (packed cell volume 0.55%) was 21.5 (6) ml (28.47 ml/kg). Haemoglobin levels before and after transfusion were 98 (9) and 134 (10) g/l respectively. The percentage of HbF was 92.9 (1.2) before and 43.3 (5.8), 2.8 (1) days after a transfusion. The value for P_{50} increased from 18.1 (1.0) to 21.0 (1.1) mm Hg ($p = 0.007$). The P_{50}, if known after a transfusion, could be useful to predict the range of adequate saturation.

This work was supported by Canadian Institutes of Health Research grant no MOP 49464.

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Risks of treating infected neonatal lines

Hodge and Puntis[1] suggest that "up to 80% of coagulase negative staphylococcus infection . . . in young children can be eradicated with antibiotics". The study referenced, Raad et al,[2] was carried out in adults (mean age 43 years) with underlying malignancy, most of whom had non-tunnelled subclavian lines. It may not be appropriate to apply Raad et al's results to children with long term parental nutrition, in view of the differences in age, illness, and catheter type.

The authors endorse the treatment of infected central venous catheters in situ without an adequate appraisal of the risks. In the neonatal population retention of catheters has a lower success rate than suggested, with only 50% of catheters being successfully treated.[3]

Importantly, treating catheters rather than immediate removal significantly prolonged the bacteremia. The risk of bacterial end organ damage increases with each day that there are positive cultures.[4]

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BOOK REVIEW

The Lazarus case, life and death issues in neonatal intensive care

John D Lantos. The Johns Hopkins University Press, 2001, pp 178. ISBN 0–8018–6762–2

When things go badly wrong in the perinatal period there has developed a culture in many "advanced societies" that demands a search for someone to blame. This search for guilt, accountability, punishment, and recompense often results in litigation.

In this thought provoking book John D Lantos describes such lawsuits as "our public morality plays" and uses his experience as a neonatologist, expert witness, and ethicist to create, debate, and crystallise relevant issues of ethics related to the neonatal intensive care of a fictional preterm infant who should have died but did not—*The Lazarus Case*.

A fictitious neonatologist, Dr Miller, decides to stop resuscitation of a very preterm infant who seems past reasonable care. The baby who might have died survived with severe neurological problems and the parents sue Dr Miller, alleging that stopping treatment was negligent. John Lantos places himself in the role of expert witness and uses questions put by the plaintiff's lawyers to explore the moral, ethical, legal, and social factors and to illustrate the ambiguities, misunderstandings, responsibilities, and evasions highlighted by the perinatal care of a 25 week gestation infant.

A key question put to Dr Lantos by one lawyer was "Can studying philosophy tell you whether what a doctor does in a particular case is right or wrong?" Probably not is the final conclusion reached by Dr Lantos, but it was just as unlikely that definitive guidance would come from sociology, religious doctrine, strict medical protocols, or any other single source.

There have been many attempts over the past half century to face and explain the moral dilemmas associated with our attempts to save the lives, prevent damage, and encourage optimal development of critically ill preterm infants. *The Lazarus Case* reviews in a most effective, compelling, erudite, and compassionate way the enormous complexity of these issues. It is highly recommended to all who are concerned with the care of preterm infants and their families and is essential reading for those required to provide medicolegal advice on life and death issues in neonatal intensive care.

Forrester Cockburn

CD REVIEW

Echocardiography for the neonatologist. Part 2. Structural and transitional haemodynamic problems in the newborn. Practical

N Evans, G Malcolm. Royal Albert Hospital, Sydney.

This second CD, covering structural and transitional haemodynamic problems in the newborn, is the companion volume to part 1, which dealt with normal 2D imaging and Doppler. In this latest volume, the authors deal comprehensively with the use of echocardiography in the diagnosis and assessment of preterm patent ductus arteriosus, pulmonary hypertension, and low output states. They also provide an introduction to recognition of structural cardiovascular malformations.

The CD is divided into six sections that deal, respectively, with the ductus arteriosus, atrial shunting, measurement of flow and ventricular output, assessment of ventricular function and hypertrophy, measurement of pulmonary artery pressure, and an introductory description of common types of cardiovascular malformation. Images are mainly provided as a mixture of text and video clips.

The first section on patent ductus arteriosus is excellent, providing many examples of the diagnosis and assessment of functional importance. The section on measurement of pulmonary artery pressure provides a valuable insight into one of the more important applications of cardiac ultrasound in neonatology. The sections on atrial shunting, flow measurement, and assessment of ventricular function cover applications of cardiac ultrasound that are likely to be less familiar to most neonatologists and will require more assimilation. The final section, on structural congenital heart disease, is really only an introduction to what is obviously a very large subject. The authors stress that suspicion or confirmation of a cardiovascular malformation should lead directly to a cardiological referral and they highlight the difficulty in exclusion or confirmation of some diagnoses, particularly total anomalous pulmonary venous connection and coarctation of the aorta. Some of the examples of cardiovascular malformations are of rather disappointing quality and some of the more difficult problems, such as coarctation of the aorta, are dealt with only very briefly. There is also possible confusion between coarctation of the aorta and interruption, which cardiologists regard as different abnormalities. The self assessment section offers the opportunity of testing your skills in recognition of various examples but there is no leeway in terminology. For example, TAPVC (connection) is counted as a wrong answer for TAPVD (total anomalous pulmonary venous "drainage").

Overall this is a valuable guide to the way in which neonatologists should use echocardiography and what they should expect to be able to achieve. I think it is likely to be of more use than a textbook as echocardiography is mainly about interpretation of moving images. Details of both CDs are available at www.cs.nsw.au/rpa/neonatal/default/htm.

C Wren

PostScript

LETTERS

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Amiodarone and breast feeding

An infant was born at 33+2 weeks gestation by caesarean section after an in utero diagnosis of fetal ascites and tachycardia. The mother had received treatment during pregnancy with flecainide, amiodarone, and propranolol. The amiodarone was prescribed initially at 200 mg three times a day and was reduced to twice a day after 11 days.

The mother was keen to breast feed the baby. In previous reports of amiodarone and breast feeding, amiodarone treatment was for a maternal indication and hence continued post partum.[1 2] In this case, the amiodarone treatment stopped at delivery. However, because of the long terminal half life of amiodarone (about 50 days[3]), it could take several months for the level to fall. As one of the adverse effects of amiodarone is thyroid toxicity, the baby's thyroid function was assessed and found to be normal. A decision was made to allow the mother to breast feed, and the baby was closely monitored.

Breast milk was sent for analysis to determine the amiodarone level on days 5, 11, 18, and 25. It had increased on day 11 (2.1 mg/l) compared with day 5 (0.6 mg/l). This may be due to changes in composition of the milk. We do not know at what time of day the milk was expressed or whether the sample was taken at the beginning or the end of the feed. The fat content of the milk was likely to be greater after 11 days than after 5 days, which may affect the distribution of amiodarone. McKenna *et al*[4] described changes in amiodarone concentration in breast milk throughout the day. By 25 days, amiodarone was undetectable. Throughout this period the baby remained well and thyroid function was normal.

Although we would not recommend that breast feeding is necessarily safe for all babies exposed to amiodarone, this case illustrates that, in some circumstances, with close monitoring, breast feeding can be initiated.

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Reducing antibiotic use on the neonatal unit by improving communication of blood culture results: a completed audit cycle

It is common clinical practice to discontinue antibiotic treatment of asymptomatic babies if the blood cultures are negative at 48 hours.[1–3] However, if blood culture results are only available during the normal working day, then antibiotic treatment of some babies may continue into the next working day. In our neonatal unit, blood culture results were routinely received from the microbiology laboratory via fax as a list every morning. Extra positive results would be telephoned through, if they became available, during the normal working day. Results could also be checked by the clinical staff telephoning the laboratory during "office hours". This gave the potential for inadvertent prolongation of antibiotic courses for up to a day. In a previous study, McDonald *et al*[4] found this to be a common occurrence. It is of concern because unnecessary antibiotic use may contribute to antibiotic pressure within the neonatal unit and may encourage the selection of drug resistant organisms.

We performed two audits into this problem within our neonatal unit. Our audit standard on each occasion was that antibiotics should be stopped at 48 hours, if blood cultures were negative, unless a decision to continue was clearly documented in the case notes. Babies with negative blood cultures were identified from the microbiology database. Each episode was classified into one of four groups: (*a*) antibiotics not started; (*b*) antibiotics stopped within 48 hours; (*c*) antibiotics given for more than 48 hours deliberately; (*d*) antibiotics given for more than 48 hours unintentionally. The results are summarised in table 1.

The first audit was conducted on 451 babies with negative blood cultures between January 1997 and December 1998. We were able to collect complete data from case notes and drug charts for 376 (83.4%) of these blood cultures. We found that the audit standard was not met in 144/376 (38.3%). The median (range) duration of antibiotic treatment for each baby was 60 (16.9–332) hours.

The blood culture analyser in use in our laboratory (BacT/Alert Microbial Detection System; Organon Teknika Corporation, Durham, North Carolina, USA) tests for bacterial growth every 10 minutes and communicates the blood culture status (positive or negative) to a computer. After our initial audit, we established a computer link between the blood culture analyser and the neonatal unit. This allows the clinical staff to check the status of any blood culture in the analyser in real time, 24 hours a day.

The second audit was performed on babies with negative blood cultures between May 2000 and August 2000. Two hundred negative blood cultures were identified. Complete data were available for 179/200 (89.5%). The audit standard was not met in only 20/179 (11.2%); $p<0.001$ compared with the first audit. The median (range) duration of treatment was reduced to 48 (1–182) hours ($p<0.0001$). There was an overall reduction of two doses of antibiotic per baby (from a mean of 8.8 to 6.8 doses per baby).

Overall, we estimated that we gave 21 684 doses of antibiotics on the neonatal unit between January 1997 and December 1998. If the computer system had been in operation during this period, we estimate that we could have reduced this by 16.2% to 18 169. We think that this magnitude of reduction in antibiotic pressure on the neonatal unit is worth achieving.

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Table 1 Reduction in unintentional antibiotic use over 48 hours after introduction of real time availability of blood culture status by a computer link between the blood culture machine and our neonatal unit

Results	No antibiotics started	Antibiotics stopped after <48 h	Antibiotics continued for >48 h deliberately	Antibiotics continued >48 h unintentionally
First audit	25 (6.6%)	132 (35.1%)	75 (19.9%)	144 (38.3%)
Second audit	15 (8.4%)	117 (65.4%)	27 (15.1%)	20 (11.2%)

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Swaddling and heat loss

The letter of Hawkes *et al*[1] raises the important issues of swaddling and temperature on admission to the neonatal unit. Besch *et al*[2] carried out a limited comparison of different swaddling materials and found a transparent plastic bag together with radiant heat to be effective in preventing heat loss in infants over 2 kg. Following a report in the literature,[3] we have begun wrapping all preterm infants < 1000 g in a thin plastic wrap. The wrap is preheated on a radiant warmer and the infant is immediately placed (undried) on the plastic sheet, which is folded over to completely (but loosely) enclose the torso and extremities from the neck down. The infant is left in the wrap until transported to the neonatal unit and the temperature has stabilised in a humidified environment. The median temperature of the 19 < 1000 g infants admitted since wrapping was commenced was 36.7°C on arrival to the nursery compared with 35.5°C for the previous 86 unwrapped infants (p = 0.002; using Mann-Whitney U test). There were no significant differences in birth weight, gestational age or Apgar scores between the groups.

Although our experience is in smaller preterm infants (who are more prone to hypothermia), our results are in keeping with those of Vohra *et al*, who studied infants < 32 weeks.[3] We now plan to wrap all preterm infants < 1500 g.

The plastic wrap is likely to be more effective than towels because of reduction in evaporative heat loss and because it allows observation of the infant. However, the plastic wrap is unlikely to significantly reduce radiant heat loss, so an additional heat source is essential for preterm infants. Some form of head swaddling is also important and needs further study. Aluminum foil may reduce evaporative, convective, and radiant heat loss but does not allow observation or radiant warming.

It appears there are many aspects of swaddling that require further investigation.

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Preventing hypothermia at birth in preterm babies: at a cost of overheating some?

In the Epicure study, the odds ratio of death before discharge for babies whose temperature on admission to the neonatal unit was > 35°C was 0.58 (95% confidence interval (CI) 0.39 to 0.85) compared with those with lower temperatures.[1] In 2001, we therefore introduced a policy of wrapping neonates < 30 weeks gestation in polythene bags at birth without first drying them. Temperatures on admission to the neonatal unit after the introduction of this policy were compared with those of historical controls of < 30 weeks gestation admitted unwrapped between 1996 and 2000. The admission temperatures were analysed by stepwise multiple regression against being "bagged" or not, time to admission to the unit, birth weight, gestation, mode of delivery, month of delivery, and maternal temperature. Significant coefficients of variation existed between admission temperature and:

- being bagged +0.35°C (0.09 to 0.62) (coefficient, 95% CI);
- time to admission –0.02°C (–0.01 to –0.03) per minute;
- birth weight +0.07°C (0.02 to 0.1) per 100 g;
- gestation +0.0007°C (0.0002 to 0.001) °C per week.

Thus "bagging" increased admission temperatures by 0.35°C, which is rather less than the rise of 1.9°C in babies < 28 weeks gestation reported in a previous study.[2]

Table 1 shows that, in the comparable groups, this rise of 0.35°C resulted in a significant reduction in incidence of hypothermia (< 35.5°C) in "bagged" babies. However, significantly more of them (12%) were hyperthermic (> 37°C), a phenomenon previously reported but not discussed.[2] The risks of hyperthermia are less well defined than those of hypothermia, but it may increase the risk of neurological damage, particularly after ischaemia.[3] The technique of wrapping babies in polythene bags would seem to benefit very preterm babies, although we may yet have to learn to use it appropriately.

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Technique for insertion of percutaneous central venous catheters in the newborn period

The use of percutaneous central venous catheters is of proven value for the provision of parenteral nutrition and intravenous drug treatment in neonates. They have become an integral part of the management of very low birthweight infants in most intensive care units.

At the Royal Children's Hospital in Melbourne we used a silastic catheter, which has an external diameter of 0.6 mm and comes in a variety of different lengths (Epicutaneo-cava catheter manufactured by Vygon; lengths 15, 30, and 50 cm; ref nos 2184.015, 2184.00, and 2184.005; cost AU$59.10). It is packaged with a metal 19 GA butterfly needle for use in insertion of the line.

This technique has some drawbacks.

(1) The 19 GA needle is difficult to put directly into neonatal veins because of its large size.

(2) It can be difficult to appreciate "flash back" of blood into the metal needle.

(3) It is not possible to "flush" the needle to ensure correct positioning of the line as well as patency of the vessel.

(4) It is not feasible to place femoral venous lines using this method.

We therefore use a method whereby the vein, using the Seldinger technique, is ultimately cannulated with a 20 GA catheter through which the silastic line can be inserted.

(1) The procedure should be carried out under optimal conditions using an aseptic technique. If the infant is already ventilated, we advocate the use of a muscle relaxant as well as adequate sedation. This is especially advisable for insertion of femoral venous lines.

(2) The vein is initially cannulated with a 24 GA (external diameter 0.7 mm) cannula. The sites most often used are the great saphenous vein at the ankle or knee joint, the femoral vein, the basilic or cephalic veins in the antecubital fossa, or, occasionally, the superficial temporal vein. A transilluminator or "cold light" inserted into the finger of a sterile glove can be of use in locating deep veins as well as protecting the sterile field.

(3) A guidewire is then inserted through the cannula into the vein. We use a "duoflex spring wire guide": diameter 0.45 mm, length 25 cm (duoflex spring wire guide manufactured by Arrow; product no AW-04018; cost

Table 1 Incidence of hypothermia and hyperthermia in control babies and babies wrapped in polythene bags (study group)

	Control group	Study group	Difference (95% CI)
Number	230	48	–
Gestation (weeks)	27.5 (23–29)	28 (23–29)	–
Weight (g)	1020 (400–1900)	1027 (500–1700)	–
Number <35.5°C	96 (42)	12 (25)	–17 (–2 to –29)
Number >37.0°C	1 (0.4)	6 (12.5)	12 (5 to 24)

Values are either median (range) or number (%).
CI, Confidence interval.

AU$13.00; Insyte intravenous catheter manufactured by Becton Dickinson catheter; GA 24, 22, and 20; cost AU$2.00). This has the advantage of having a soft tip at both ends of the wire and being a snug fit to the smallest catheter. Care must be taken not to advance the wire if any resistance is met.

(4) A small nick is made in the skin at the site of wire to facilitate the insertion of the larger intravenous cannulae.

(5) A 20 GA (external diameter 1.1 mm) cannula is then threaded over the wire into the vein (a 22 GA (external diameter 0.8 mm) can be used to dilate the vein before the larger cannula is inserted). This can be flushed with saline to ensure patency of the vein.

(6) The silastic catheter can then be fed up the vein through the 20 GA cannula with a pair of toothless forceps. Occasionally the silastic line coils up in the hub of the cannula. This can be overcome by cutting the cannula flush to the hub and reinserting the silastic line.

(7) The silastic catheter is placed to the required length and the other cannula is withdrawn.

(8) The silastic catheter should be placed outside the cardiac outline in accordance with new guidelines.[1–3] The position is always confirmed radiologically either by plain radiograph or, if necessary, by injection of radiopaque dye. We have seen neonates with pericardial tamponade associated with malpositioned catheters, which has been well documented in the literature.[1–3]

We have found this method to be extremely reliable in the insertion of percutaneous venous catheters.

The use of the guidewire incurs additional costs (see above). In our experience these are partially offset by an improved success rate using the above method. We do not open the silastic catheter until the 20 GA is in place within the vein. This means that a line is not wasted if the vein cannot be cannulated.

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Umbilical granulomas: a randomised controlled trial

The Archimedes section has previously contained a brief section on the treatment of umbilical granulomas.[1] We have now conducted a randomised controlled trial of the management of umbilical granulomas. The trial compared silver nitrate cauterisation with the use of alcoholic wipes at each nappy change (conservative management). The impetus for this work was a series of three burns to the anterior abdominal wall after silver nitrate cauterisation, seen in a single London hospital over a two year period.

The trial aimed to show equivalence between the two treatment modalities. On the basis of equal efficacy, we intended to change practice to conservative management. More than 40 infants were referred, but a large number of parents chose conservative management rather than randomisation. Difficulty in recruitment meant there were inadequate numbers to show statistical significance within the limited time span available.

The salient results were that two of three granulomas resolved over a three week period without cauterisation. Those infants whose granulomas did not resolve went on to treatment with cauterisation following a protocol that involved drying the area both before and after silver nitrate application, surrounding the umbilicus with white soft paraffin, and leaving the area exposed for 10 minutes after application. This resulted in resolution in all remaining cases without harm due to delay in treatment.

On the basis of this work, we suggest a change in current practice to initial conservative management followed by cauterisation only when conservative treatment fails.

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Progressive ventricular dilatation (PVD) over the past 22 years

We read with interest the article of Murphy *et al*,[1] and it prompted us to review our own experience with progressive ventricular dilatation (PVD) over the past 22 years at the Maine Medical Center (MMC). Since 1980, we have used a single approach to management of PVD. As noted in previous publications, we have considered the need for intervention to be rapid head growth defined as an increase in occipitofrontal circumference of 2 cm a week or more rather than relying on imaging.[2 3] As this degree of head growth suggests increased intracranial pressure,[4] we have intervened by directly draining ventricular fluid through a 21 gauge angiocath placed through the right coronal suture into the right lateral ventricle. This catheter is connected to a ventriculostomy drainage system, and drainage is continued for seven days if possible. The catheter is then removed and the decrease in head circumference and ventricular size recorded. The infant is watched for return of rapid head growth, and an angiocath is reinserted as needed. This procedure is repeated until the infant reaches about 2 kg in weight, and if rapid head growth continues, a permanent ventriculoperitoneal shunt is placed.[3] We do not use pharmacological treatment or repeat lumbar puncture to treat PVD.

As pointed out by Murphy *et al*, PVD sufficient to require intervention occurs almost exclusively in infants with grade 3 or 4 intraventricular haemorrhage (IVH). As expected, the very low birthweight infants with high grade IVH have a high mortality. Table 1 shows a comparison between the outcomes for grade 3–4 IVH at MMC during the 1980s and over the past five years (1997–2001 inclusive), and the data of Murphy *et al* grouped in the same way. As noted, there is little difference over time or between studies. Overall mortality for grade 3–4 IVH was 33% (26/79) for Murphy *et al*, 33% (31/94) for MMC in the 1980s, and 31% (9/29) for MMC in 1997–2001. Until grade 3–4 IVH can be eliminated, posthaemorraghic hydrocephalus will continue to occur with high morbidity and mortality.

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Table 1 Comparison between the outcomes for grade 3–4 intraventricular haemorrhage (IVH) in the three studies

	Murphy *et al*[1]	MMC 1980s[3]	MMC 1997–2001
Grade 3–4 IVH (% of all <1500 g)	79 (7%)	94 (6%)	29 (6%)
Death <14 days	18/79 (23%)	29/94 (30%)**	8/29 (28%)**
PVD requiring treatment	34/61 (56%)	24/65 (37%)	11/21 (52%)
VP shunt/late death (% of PVD treatment)	18/8 (26/34=76%)	12/3 (15/24=63%)	6/1 (7/11=63%)

*Rate for all infants <35 weeks.
**Rate for all deaths <30 days.
MMC, Maine Medical Center; PVD, progressive ventricular dilatation; VP, ventriculoperitoneal.

Do we need to assess the thyroid function in the infants of mothers with Hashimoto's thyroiditis?

We read with interest the recent comprehensive review of neonatal thyroid disorders, which gave evidence-based answers to many important questions. The author recommended that all babies born to mothers with Hashimoto's thyroiditis should be reviewed at 10 days to 2 weeks and a thyroid function test taken because infants may develop transient hypothyroidism or, very rarely, hyperthyroidism.[1]

As paediatricians, in a hospital with a paediatric endocrine caseload similar to some tertiary centres and a subregional neonatal intensive care unit with local deliveries of 6000 per annum, we think that the potential benefits of this practice are difficult to justify. We do understand that such practice will help in identifying babies who may develop transient congenital hypothyroidism caused by maternal thyrotropin receptor blocking antibodies. However, the incidence of this form of hypothyroidism has been estimated to be 1 in 180 000 normal infants (~2 % of congenital hypothyroidism) and the majority of them will have raised thyroid stimulation hormone levels that can be detected by the current neonatal screening.[2] Based on a simple calculation, in a unit of our size only one baby will be detected every 30 years. We feel that there would be major disadvantages if we are to adopt the author's recommendation. Firstly, an extra hospital visit for babies and parents; secondly the need to bleed many healthy infants; and finally the potential for confusion and unnecessary anxiety. Until objective evidence emerges about the significance of subtle thyroid dysfunction in early life we feel that the current screening programme should not be extended.

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CORRECTIONS

In the CD Review (*Arch Dis Child Fetal Neonatal Ed* 2003;**88**:F164) reviewed by C Wren, please note that the affiliation of the authors is published incorrectly. This should have read Royal Prince Alfred Hospital, Sydney. Also, the web address in the final paragraph is incomplete. The correct address is: http://www.cs.nsw.gov.au/rpa/neonatal/default.htm. The errors are much regretted.

The authors would like to acknowledge and apologise for an error in our article Socioeconomic status and preterm birth: New Zealand trends, 1980 to 1999. ED Craig, JMD Thompson, EA Mitchell (*Arch Dis Child Fetal Neonatal Ed* 2002;**86**:F142–6).

Paragraph four in the Results section should read "Figure 2 summarises changes in preterm birth rates by Deprivation Index decile between 1980 and 1999. During this period rates rose from 5.2% to 5.9% among those living in the most deprived areas (a 13.5% increase), from 4.0 to 5.5% amongst those living in average areas (a 37.5% increase) and from 3.1% to 5.4% amongst those living in the least deprived areas (a 74.2% increase). Thus while in 1980 a marked social gradient in preterm birth existed, by 1999 this had diminished markedly." Table 2 and table 3 are amended. These errors do not significantly change the reported trends in preterm birth or the interpretation of the findings previously published.

Table 2 Multivariate odds ratios for preterm birth by gestational age category and Deprivation Index decile; New Zealand singleton live births 1980, 1990, and 1999

		Gestational age category			
Year	NZDep Index Decile	All preterm (n=51 711) OR*	20–27 weeks (n=2697) OR**	28–33 weeks (n=12 703) OR*	34–36 weeks (n=36 311) OR*
1980	1	1.00	1.00	1.00	1.00
1980	5	1.15	1.08	1.16	1.15
1980	10	1.36	1.18	1.39	1.36
1990	1	1.30	1.31	1.21	1.33
1990	5	1.44	1.45	1.34	1.47
1990	10	1.63	1.66	1.52	1.67
1999	1	1.64	1.67	1.44	1.72
1999	5	1.76	1.91	1.53	1.84
1999	10	1.93	2.25	1.64	2.02

Multivariate analysis adjusted for gender, maternal age, parity, birth year, decile and birth year*decile, year*age, year*parity, decile*age, decile*parity.
*Odds ratios (OR) with reference category Deprivation Index decile 1, 1980.
**Odds ratios for the 20–27 week category did not reach statistical significance.

Table 3 The "social gradient in preterm birth"; risk of preterm birth amongst decile 10 women compared to decile 1 women (same year), New Zealand singleton live births 1980, 1990, and 1999

		Gestational age category		
Year	All preterm (n=51 711) OR*	20–27 weeks (n=2697) OR**	28–33 weeks (n=12 703) OR*	34–36 weeks (n=36 311) OR*
1980	1.36	1.18	1.39	1.36
1990	1.26	1.27	1.25	1.26
1999	1.17	1.35	1.14	1.17

Multivariate analysis adjusted for gender, maternal age, parity, birth year, decile and birth year*decile, year*age, year*parity, decile*age, decile*parity.
*Odds ratios (OR) for preterm birth amongst decile 10 women compared to those in decile 1 for each particular year reflects the social gradient for that year.
**Odds ratios for the 20–27 week category did not reach statistical significance.