abated on the 4th day in hospital and her CSF returned to normal on the 12th day. However, her haemoglobin concentration, which had been 117 g/l on the 4th day fell to 90 g/l by day 36; the haemoglobin concentration and the reticulocyte count improved gradually and returned to normal on day 73. There were no increases in serum antibody titres for measles, herpes simplex, mumps, rubella, cytomegalovirus, enterovirus 70/71, mycoplasma, toxoplasma, or chlamydia. She was discharged on the 21st hospital day without sequelae.

Unfortunately, we could not make a definitive diagnosis of B19 infection by polymerase chain reaction (PCR), hybridisation, etc. at that time, but we suppose that the haematological manifestations of fever, meningitis, and anaemia are more likely to have been related to the B19 infection because B19 IgM was detected in serum, and because the mother simultaneously developed adult type B19 infection. Epidemiologically, the source of infection was thought to be the brother, considering that the incubation period for B19 infection is 17 to 18 days.1

Postnatal B19 infection could cause severe complications such as a hydrops fetalis, but the outcome of primary B19 infection in newborns is still unknown.

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Outcome of triplet pregnancies

EDITOR.—The incidence of triplet and higher order pregnancies has more than doubled since 1980 as a result of new techniques for the treatment of infertility. Because of the high incidence of prematurity, triplets are at a high risk of neonatal complications and death. The poor reproductive histories of some mothers treated for infertility has led to the suggestion that such triplets carry a poorer prognosis than those naturally occurring, possibly as the result of earlier delivery.

Since 1980, 41 sets of liveborn triplets have been admitted to the Mersey Regional Neonatal Unit, Liverpool. Twenty eight sets were natural, and 13 the result of fertility treatments (six ovulation induction, six IVF, and one GIFT). The mean gestational age at delivery was 30-2 weeks in the natural triplets and 30-3 weeks in the others. There were five deaths in both groups; seven infants in the natural group and two in the infertility group have survived with major disabilities (cerebral palsy and/or blindness). Survival without major disability was not significantly different between the two groups (86% natural v 82%). However, only 75% and 61% triplet pregnancies admitted, respectively, resulted in three live children without subsequent disabilities.

It remains important to counsel couples undertaking infertility treatments concerning the morbidity and mortality associated with higher order pregnancies, but such risks are probably not greater as the result of mode of conception, but, rather, relate mainly to prematurity.

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Acute blood pressure response to surfactant administration

EDITOR.—Saliba et al reported a significant increase in cerebral blood flow velocity (CBFV) and transcutaneous carbon dioxide tension (TcPCO₂) following rapid instillation of surfactant.1 The rise in CBFV was related to, but not solely explained by, the increase in TcPCO₂. They found no alteration in one minute averages of mean arterial blood pressure (MABP). It is reasonable to expect that swings in systemic blood pressure could be caused by rapid intratracheal administration of surfactant and we would like to offer an explanation as to why the current method of measuring short term blood pressure change may obscure potentially important information.

All infants in our neonatal intensive care unit have continuous physiological variables transferred from a multiparameter monitor to a bedside computer,2 and displayed in real time. Using this system to display second by second data during surfactant administration, we often detect significant blood pressure surges which can be biphasic in character and could therefore be obscured by looking at the mean change in pressure over a selected time period.

The figure shows the blood pressure trace from an infant receiving Exosurf as an infusion over five minutes. Each data point is a one second value and a total of 21 minutes is displayed. The trace is analysed as three equal time periods: before, during, and after surfactant administration. If analysis were confined to mean values for each period of five minutes, the finding would be a 1 mm Hg fall in MABP during, and a rise of 4 mm Hg after, administration. These findings would not be impressive. However, detailed analysis within the time periods of administration shows a drop in MABP of 10 mm Hg below the pre-treatment baseline followed by a rise in MABP of 12 mm Hg above the baseline. An overall swing in MABP of 22 mm Hg against a background MABP of 33 mm Hg would certainly be considered important.

It is our frequent observation that administration of surfactant can provoke significant fluctuations for blood pressure which may be overlooked when examining mean changes alone.

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Dr Grantly Dick-Read
EDITOR.—Grantly Dick-Read’s contribution to obstetrics was not limited to the labour ward.1 His books shifted the emphasis in antenatal education from mothercraft to preparation for childbirth, with a new emphasis on physiology and informed choice. These, together with the antenatal classes developed by his second wife, Jessica Bennett, are the basis of most antenatal preparation today.

In 1956 a group of mothers influenced by Dick-Read’s work formed a charity to...
promulgate his aims. Now Britain’s foremost childbirth charity, The National Childbirth Trust, offers information and support in pregnancy, childbirth, and early parenthood. It aims to enable every parent to make informed choices. The first president of the Trust, Dr Grantly Dick-Read, said he had welcomed the way his work has become today’s good practice, even if recognition of his contribution is belated.

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Formula mistake

EDITOR,—We would like to point out an erroneous formula. The correct formula is:

Galbladder volume \( (\text{mm}^3) = \frac{1}{6} \times \text{maximum length (mm)} \times \text{maximum width (mm)} \times \text{maximum height (mm)} \)

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Since its widespread introduction in the early 1980s, cerebral ultrasonography has occupied a central role in the evaluation of brain injury in preterm infants. Technological advances have brought about increasing clarity and detail in the images produced while other branches of neuroradiology have failed to match ultrasound in terms of accessibility and acceptability for the sick very low birthweight (VLBW) infant. Naturally, such a powerful tool has lead to a plethora of publications relating ultrasound to neurological outcome. During the counselling process, parents of a sick preterm infant often seize on the results of a scan, hoping for prognostic certainty. Despite the routine clinical use of cranial ultrasound much work still needs to be done to determine the cause and consequence of damage to the preterm brain.

This concise book is based around the Central New Jersey Brain Injury Study undertaken by the authors during the mid-1980s. It aims to re-examine the nature of brain insults affecting a population of preterm babies undergoing modern neonatal intensive care, providing a historical perspective to current neuroanatomical and pathological knowledge. Crucially, it also correlates ultrasound imaging with subsequent postmortem findings of the whole brain.

These objectives are achieved in a thoughtful and lucid way with meticulous methodological detail supplied for specialists in histopathology and radiology. Chapters on GM/IVH and white matter damage are presented in each case by explanations of terminology and pathogenesis.

On more than one occasion the authors unearth pertinent literature long since published and discarded by subsequent generations of pathologists. Some persistent misconceptions, such as regarding parenchymal haemorrhage as an extension of intraventricular bleeding, are exercised in an assured but scrupulous manner.

The text is superbly illustrated by colour photographs of pathological specimens adjacent to corresponding brain ultrasound and colour Doppler images. However, the educational impact is spoilt by excessive labelling of some images and overwhelming figure legends.

A splendid chapter dealing with the pathogenesis of GM/IVH presents many individual anatomical and pathological pieces of evidence from over the centuries to substantiate today’s concepts. The excellent final section on prognosis gives a refreshingly concise overview of the range and incidence of neuromotor development in VLBW survivors. This is followed by a succinct review of published data relating ultrasound abnormalities to outcome.

An important weakness in the original study group is acknowledged—namely, the bias to study haemorrhage by early scanning, now recognised as of lesser importance than white matter damage. I cannot, however, concur with the authors’ concluding statement that ultrasound imaging may act as a proxy for neurological disability, obviating the need for long term follow up studies. Undoubtedly this is a valuable book enabling budding neonatologists, experienced clinicians, and a range of allied disciplines, to improve their understanding of brain injury in preterm infants.

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The authors have set out to fill the gap between large textbooks and review articles, aiming to provide clinicians with detailed up-to-date information on the physiology of the fetus and neonate, and how this can be adversely affected. Unlike many monographs, they set out to provide a balanced view. They hope that this will be useful to investigators planning to explore the area in greater detail, and to provide those further on in their careers with state of the art information.

In this they have succeeded. They have followed the format adopted in the first volume on the cardiovascular system so that the first half of the book concentrates on the normal developmental physiology of the respiratory system in the fetus and newborn infant. I thought these review statements were excellent and I have already found them a very useful reference source.

The remainder of the book I have found less useful. It is loosely divided into a section...