ported 'failure to thrive' to some external criterion of developmental disadvantage, be that 'psychosocial deprivation' or organic disease. Otherwise a time honoured preventative activity may become a source of unjustified parental concern.

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Drs Edwards, Halse, and Waterston comment: The three points made by Drs Porter and Skuse cover the genetic influence on growth, the diagnostic validity of persistent centile deviation, and the need for additional criteria for psychosocial disadvantage. Definitive answers are not yet available on any of these points. Smith's paper indeed discusses length rather than weight, but the key statement we extracted from this work is that 'Those infants “catching up” after birth usually do so in early infancy (2-3 months).’ We have provided the evidence in our paper for the 4-8 week centile being a better predictor of future growth than the birth centile.

Our findings show that babies whose weight deviates downwards according to our definition are distinctly different in the second year from babies whose weight does not deviate, they are not only lighter, but also shorter and thinner. We therefore believe that we have identified a different population of babies and suggest that these are children who may be regarded as vulnerable and worthy of psychosocial assessment. We believe that these findings demonstrate the value of regular weighing of children. Concerning the meaning of the term 'failure to thrive', we agree with the Lancet that this term, with its connotations of emotional poverty, would be better abandoned and replaced by 'failure to gain weight appropriately'.

Providing definitions of failure to thrive which relate to growth below the 3rd or 10th centile do not take into account the growth trajectory, and are clearly unsatisfactory. We think that ours is a considerable advance, if still in need of further refinement.


Baby Check score card

DrS.—The series of articles by AJ Thornton, C J Morley, and S J Green et al on the Baby Check score card make very interesting reading and represent a timely attempt to provide parents with a diagnostic tool to grade their infant's severity of illness.1 2 3

As a general practitioner I must admit to some reservations a reader's parents' capability (particularly with first children) to document accurately their children's symptoms. I am frequently presented, as are my colleagues, with children whose parents cheerfully report that they have vomited everything they have been fed for the last 48 hours or longer, haven't passed urine for the last 24 hours, and are drowsy all the time ('He definitely isn't himself'). A set of symptoms belied by the fully hydrated and cheerful infant sitting in a wet nappy in the surgery (‘He must have just done it’).

This lack of reliability in parents' impressions of their children's symptoms is supported by a letter by Francy Pillo-Blocka et al from the Hospital of Sick Children in Toronto.4 They reported on the subject of mothers of infants with gastro-oesophageal reflux and their estimates of the quantity of fluid spilled on a baby's wash cloth. A spillage of 5 ml in volume produced a mean estimate of spillage of 35 ml (range 3-120 ml) and a 10 ml volume produced a mean estimate of spillage of 77 ml (range 7-240 ml). Of the 58 mothers tested, only one accurately assessed both volumes and the result was independent of education status or age of the mother. The authors advised caution in accepting parental impression of vomiting as a result.

I accept this finding only affects a small subsection of the Baby Check, but I would welcome ideas on the widespread application of the score card before more work is done on the accuracy of parental assessment of individual signs and symptoms included in the score card.

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Dr Morley comments: I would like to thank Dr Sowden for his interest in Baby Check. I understand his reservations about parents' ability to report their children's symptoms accurately. However, when mothers exaggerate their baby's symptoms one should consider whether they are really capable of reporting the symptoms accurately or whether they perceive that the doctor is disinterested in their baby's illness unless presented with florid and overt symptoms.

Dr Sowden uses mothers' lack of reliability in assessing the amount of fluid on a wash cloth as an indication of their inability to assess their baby's symptoms. This is a notoriously difficult thing to assess and I would be surprised if doctors could do better. However, assuming for the first time all doctors should do is ask the mother about the baby's symptoms. It is very difficult to come to an accurate diagnosis without the history. However, obtaining the possible symptoms and signs is a useful tool which might be used to grade the severity of a baby's illness. Inter- estingly, out of all the possible factors which might be considered important and useful, seven of the 19 factors selected by the analyses were symptoms. Despite any inaccuracy in the way these might have been reported by the mothers they were found to contribute significantly to the assessment of a baby's illness.

Although we were concerned that mothers might not be able to use Baby Check two field trials showed that they had few problems with the interpretation of the symptoms or signs. Most people who used Baby Check in the studies found it helpful, particularly if they were inexperienced at assessing babies' illness. I would like to suggest that Dr Sowden and his colleagues do some work on reliability and construct validity of the Baby Check. He might be pleasantly surprised to find how well parents can assess their babies when given a new tool for a difficult task.

Ischaemic brain lesions diagnosed at birth in preterm infants

SIR.—The observation by Sinha et al, that nine out of 232 newborn babies showed periventricular echogenicity two hours after birth requires clarification if inferences are to be drawn with regard to the timing of the insult which leads to periventricular leucomalacia.1 Confusion will exist as long as paediatricians continue to use the terms echogenicity, ischaemia, periventricular leucomalacia, and periventricular cysts as though the terms were synonymous.

Echogenicity from the authors' own observations is reversible, as is ischaemia, for at least some patients. Periventricular leucomalacia with or without cyst formation is as permanent as the disability which it may cause. Periventricular leucomalacia is a particular form of cerebral infarction which becomes cystic only after a few days when sufficient numbers of dead cells have been removed for a cavity to be detectable. Precisely how long this interval is before a cyst is seen is something of an imponderable but is probably of the order of 10 days.

It comes as no surprise that there were nine infants whose brain pathology may have been initiated in the intrapartum or immediate postnatal period. Changing the supply of oxygen from placenta to lungs is bound to be intrinsically hazardous. A more interesting question is how many babies sustained cerebral infarction from a hypoxic insult days before the mother's confinement. Cerebral hypoxia is so frequent and so widespread that it is a normal event in almost all cases. This is true of the intrapartum period of a baby's brain at two hours postnatal age would probably be surprising. It comes as no surprise to echogenicity, of the infant brain at two hours postnatal age would be convincing in that respect. The reader is not informed.

Dr Dis Child: first published as 10.1136/adc.66.7.906-c on 1 July 1991. Downloaded from http://adc.bmj.com/
The authors make it clear that late onset periventricular leucomalacia is three times more common than early onset cases. It is worth pointing out that this was not the situation that was apparent to perinatal pathologists in the days before ultrasonography existed. Only recently have significant numbers of small babies been kept alive long enough for periventricular cavitation to become manifest either by ultrasound or by direct observation postmortem. For these babies postnatal management is more likely to be relevant to their handicap than antepartum or intrapartum events. In historical terms antepartum and intrapartum brain damage has always afflicted the human race and is probably diminishing in western society. Late onset cerebral infarction in very prematurely born infants is a recent, iatrogenic problem whose increasing frequency lends greater urgency to its solution.

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Neonatal infections with coagulase negative staphylococci

Str,—In their recent leading article Millar et al state that resistance of coagulase negative staphylococci to penicillin, aminoglycosides, and other antibiotics is common.1 This is not our experience at Liverpool Maternity Hospital where coagulase negative staphylococci are responsible for most proved neonatal infections.2 When any infection is suspected, our practice is to treat initially with ampicillin and gentamicin until the results of cultures are available after which treatment may be adjusted according to antibiotic sensitivities. Occasionally the infant has a coagulase negative staphylococcal infection and has clinically improved despite in vitro resistance to ampicillin and gentamicin. If this is the case these antibiotics are often continued with good effect confirming a discrepancy between in vivo and in vitro sensitivities. Since 1986 we have used amoxicillin/clavulanic acid (Augmentin, Beecham) for coagulase negative staphylococcal infections caused by penicillin, methicillin, and gentamicin resistant organisms when the initial ampicillin and gentamicin combination has failed. In a series of 70 episodes of coagulase negative staphylococcal septicaemia occurring between January and July 1987 only one strain was found to be resistant to amoxicillin/clavulanic acid. The majority of strains were β-lactamase producers.

Coagulase negative staphylococcal infection cannot usually be differentiated clinically from other neonatal infection; using vancomycin to treat suspected coagulase negative staphylococcal infection as suggested by Millar et al means using it in virtually all episodes of suspected infection. From clinical and laboratory experience we do not agree that vancomycin, which is ototoxic, nephrotoxic, has to be given by infusion and is expensive, should be the drug of first choice for the treatment of suspected coagulase negative staphylococcal infection in the neonate.

Dr Millar comments:
Vancomycin is active in vivo and in vitro against 'methicillin resistant' coagulase negative staphylococci and side effects are well documented. Coagulase negative staphylococcal infection is uncommon in the first week of life, in the absence of an intravascular catheter, and in the mature neonate, so it is certainly not necessary to use vancomycin for the treatment of all episodes of suspected neonatal infection. The use of amoxicillin/clavulanic acid (Augmentin) for the treatment of neonatal infections as advocated by Shaw et al is indeed unusual. Neither of the two components are active in vivo or in vitro against 'methicillin resistant' staphylococci when used as single agents. The implication of Shaw et al (although not stated) is that resistance of 'methicillin resistant' coagulase negative staphylococci to amoxicillin/clavulanic acid is detected by β-lactamase and that the altered affinity of penicillin binding proteins for β-lactamase antibiotics does not contribute to amoxicillin resistance. If the hypothesis that amoxicillin/clavulanic acid is effective against 'methicillin resistant' coagulase negative staphylococci in vivo or in vitro can be substantiated then those data should be published.

Endotoxin induced cochlear damage

Str,—We read with interest the paper by Tarlow et al describing the cochlear damage in guinea pigs produced by endotoxin perfusion of the cochlea or injection into the cerebrospinal fluid. Two points are worth making. Firstly, the concentration of endotoxin in the cerebrospinal fluid or the cochlea is not estimated, but may be several orders of magnitude greater than that in human meningitis. For example, concentrations in cerebrospinal fluid compatible with survival in meningococcal meningitis were 85–250 pg/ml in one study. It would be valuable to know whether cochlear toxicity in the guinea pig occurs at endotoxin concentrations in the cerebrospinal fluid comparable with those found during human meningitis. Secondly, the cochlear mediators of cochlear damage might have important implications for children with Gram negative septicemias as well as those with meningitis. We have seen a child who first complained of unilateral tinnitus and deafness 12 hours after presenting with meningococcal septicaemia. He had no clinical or laboratory evidence of meningitis. Subsequent audiological confirmed complete leftsided hearing loss. This damage could have been mediated by endotoxin circulating in plasma.

To test the hypothesis clinically that