portion of HbA_2 was high in carriers and normal in controls. HbF determined by cellogel electrophoresis was present in three carriers. HbA_1 was measured by a column chromatographic method[6] in blood samples drawn after about 12 hours' fasting and immediately tested at 23°C.

The HbA_1 concentration in carriers of β thalassaemia trait was higher than that in controls (9·3 (SD 1·7)% *v* 7·1 (0·8)%; *t* test for unpaired data: $p<0.001$). This difference remained significant even when carriers with HbF were excluded from the comparison. We found no direct relation between HbA_2 and HbA_1.

The cause of our results remains a matter of speculation. Nevertheless, we think it is important to be aware that the usual column chromatographic method of measuring HbA_1 might give high results in carriers of β thalassaemia trait.

GIOVANNI SAITTA
CARLO CATALANO
ALFREDO CARDUCCI ARTENISIO

Cattedra di Malattie del Ricambio,

ORLANDO FILARDO

Istituto di Clinica Pediatrica prima
dell' Università degli Studi di Messina,
89100 Messina, Italy

1 Hall PM, Cadwell GM, Cook JGH, Gould BJ. Measurement of glycosylated haemoglobins and glycosylated plasma proteins in maternal cord blood using an affinity chromatography method. *Diabetologia* 1983;**25**:477-81.
2 Rucknagel DL, Chernoff AJ. Immunological studies of hemoglobins. III. Fetal hemoglobin changes in the circulation of pregnant women. *Blood* 1955; **10**:1092-9.
3 De Boer MJ, Miedema K, Casparie AF. Glycosylated haemoglobins in renal failure. *Diabetologia* 1980; **18**:437-40.
4 Franklin Bunn H. Evaluation of glycosylated hemoglobin in diabetic patients. *Diabetes* 1981;**30**: 613-7.
5 National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 1979;**28**:1039-57.
6 Walinder O. Determination of haemoglobin A_{1c} in diabetes mellitus: a simple method for routine clinical work. *Acta Endocrinol* 1978;**88**:219-20.

Spectinomycin as initial treatment for gonorrhoea

SIR,—The increasing numbers of strains of gonococci resistant to penicillin is a constant source of worry to genitourinary physicians. We agree with Professor C S F Easmon and others that a greater than 5% failure rate to the standard treatment regimens for uncomplicated anogenital gonorrhoea indicates a need for alternative first line treatment with penicillinase stable drugs (20 October, p 1032).

The 95% cure rate achieved by Professor Easmon and his colleagues with spectinomycin seems satisfactory, but we are anxious about the small but significant number of spectinomycin resistant strains of gonococci (at least 9 out of 22 developing after treatment), particularly because of the fear of spread into the community. As Professor Easmon and his colleagues mention, others have reported not only spectinomycin resistant β lactamase producing gonococci but also clinical failures in spite of in vitro sensitivity.[1 2] This has also been our experience over the past three years.

In a small study a single dose of Augmentin (3 g amoxycillin plus 250 mg potassium clavulanate) produced a cure rate of over 99% in 109 patients with uncomplicated anogenital gonorrhoea acquired either heterosexually or homosexually. There was only one β lactamase producing isolate. Subsequently a further 24 patients who had failed to respond to standard doses of talampicillin 2 g (22 patients) or spectinomycin (2 patients) all responded to Augmentin; two had β lactamase producing isolates. Unfortunately the number of β lactamase producers was too small to generalise on the effectiveness of Augmentin, and more work is needed to prove its efficacy with β lactamase producing strains. But our initial experience suggests that Augmentin in a single oral dose may prove an ideal first line drug to control spectinomycin resistant and β lactamase producing strains of *Neisseria gonorrhoeae*.

D D COELHO
A H DE SILVA
R BASU ROY

Royal Victoria Hospital,
Bournemouth BH7 6JF

1 Easmon CSF, Ison CA, Bellinger CM, Harris JW. Emergence of resistance after spectinomycin treatment for gonorrhoea due to β-lactamase producing strains of Neisseria gonorrhoea. *Br Med J* 1982;**284**:1604-5.
2 Thin RN, Barlow D, Eykyn S, Phillips I. Imported penicillinase producing Neisseria gonorrhoea becomes endemic in London. *Br J Vener Dis* 1983; **59**:364-8.
3 De Silva AH, Bashi S, Roy RB. Treatment of uncomplicated anogenital gonorrhoea with a single oral dose of Augmentin. *Br J Vener Dis* 1984;**60**: 132-3.

Nasopharyngeal tubes

SIR,—In Dr T H Hughes-Davies's review of *Hospital Paediatrics* by Milner and Hull he regrets the lack of mention of "Dinwiddie's tube to equalise pressure across the tongue in Pierre Robin syndrome" (27 October, p 1129).

The technique referred to is the use of a nasopharyngeal tube to provide a satisfactory airway in those babies suffering from severe upper airway obstruction complicating the Pierre Robin syndrome (micrognathia, glossoptosis, and cleft palate). When mild this obstruction can usually be relieved by nursing the infant prone, but when severe the tongue frequently falls back into the hypopharynx, resulting in upper airway obstruction, cyanotic attacks, respiratory failure, failure to thrive, and cor pulmonale. We have found that in these infants the use for several weeks of a 3·0-3·5 mm Portex endotracheal tube, shortened to end just above the epiglottis, provides a stable airway; splints the tongue forwards; and results in a considerable reduction of these complications, excellent catch up growth, and the possibility of early home management.[1]

We hope that this important advance will receive wider acceptance, will avoid the fate of an eponymous title, and might even be mentioned in Milner and Hull's next edition.

D J MATTHEW
R DINWIDDIE
P J HELMS
D HEAF

Hospital for Sick Children,
Great Ormond Street,
London WC1N 3JH

1 Heaf DP, Helms PJ, Dinwiddie R, Matthew DJ. Nasopharyngeal airways in Pierre Robin syndrome. *J Pediatr* 1982;**100**:698.

Non-specificity of surfactant deficiency in neonatal respiratory disorders

SIR,—It would be wrong to conclude from Dr D K James's paper (2 June, p 1635) and subsequent comment (11 August, p 383) on our letter (14 July, p 109) that the lecithin: sphingomyelin (L/S) ratio in pharyngeal fluid obtained within six hours of birth is of little value in diagnosing hyaline membrane disease. The table illustrates the sensitivity (number predicted to have hyaline membrane disease/number with hyaline membrane disease) and specificity (number predicted not to have hyaline membrane disease/number without hyaline membrane disease) of an immature L/S ratio as a test for hyaline membrane disease.[1]

Value of immature (<1·8) L/S ratio as predictive test for hyaline membrane disease

Gestation	Sensitivity (%)	Specificity (%)	
		Infants with respiratory disorders	All infants
≤34 weeks	33/41 (80)	15/16 (94)	54/55 (98)
>34 weeks	15/23 (65)	29/34 (85)	169/176 (96)

The L/S ratio is of particularly high specificity in infants of ≤34 weeks' gestation, the group in which previous trials of surfactant therapy have been conducted.[2-4] Prior estimation of the L/S ratio is therefore important to ensure comparability of treated and control infants, particularly when numbers are small. This conclusion is precisely opposite to that reached by Dr James.

P A JENKINS
W O TARNOW MORDI
J D BAUM

Department of Paediatrics,
John Radcliffe Hospital,
Oxford OX3 9DU

1 Jenkins PA, Baum JD. Respiratory distress syndrome. In: Wald NJ, ed. *Antenatal and neonatal screen.* Oxford: Oxford University Press, 1984:298-313.
2 Morley CJ, Bangham AD, Miller N, Davis JA. Dry artificial surfactant and its effect on very premature babies. *Lancet* 1981;i:64-8.
3 Halliday HL, McClure G, Reid MM, *et al.* Controlled trial of artificial surfactant to prevent respiratory distress syndrome. *Lancet* 1984;i:476-8.
4 Wilkinson AR, Jeffrey JA, Jenkins PA. Controlled trials of dry surfactant in preterm infants. *Arch Dis Child* 1982;**57**:802.

Cryptosporidiosis

SIR,—I read with interest the article by Dr D A Hunt and colleagues on cryptosporidiosis (29 September, p 814) and would like to report my findings.

During August and September diarrhoeal faecal samples submitted to the microbiology department of the Friarage Hospital, Northallerton, were examined for cryptosporidium oocysts. Two staining techniques were tried, one using Safranin,[1] the other a fluorescent stain (auramine/carbol-fuchsin),[2] before a modified Ziehl-Neelson method[2 3] was finally adopted. The Ziehl-Neelson method produced consistent results, although occasionally oocysts did not take up the stain. Extending the staining time in carbol-fuchsin to 15 minutes largely solved the problem.

One hundred and sixty six faecal samples were examined, and 12 (7·2%) contained oocysts of cryptosporidium. Thus cryptosporidium appeared to be nearly as common as salmonella (7·8%), slightly more common than campylobacter (6·6%), and more common than shigella, giardia, and enteropathogenic *Escherichia coli* combined. All age groups were affected, although most positive samples were from children aged under 13 (75%). The oldest patient was an 82 year old woman.

Contact with cattle or raw milk appeared to be common (7 out of 12 positive cases). One family had recently returned from a farm holiday in France: 11 of them were ill and five of the six members tested were found to be positive.

The hospital's catchment area is predominantly rural with a large farming community providing plenty of opportunities for zoonotic infection, thus helping to explain the high incidence in this area. Obviously a larger series over a longer period will be required before a more representative picture will emerge.

A S WYLLIE

Microbiology Department,
Friarage Hospital,
Northallerton,
North Yorkshire DL6 1JG

1 Baxby D, Blundell N. Sensitive, rapid, simple method for detecting Cryptosporidium in faeces. *Lancet* 1983;ii:1149.
2 Casemore DP, Armstrong M, Jackson B. Screening for cryptosporidium in stools. *Lancet* 1984;i:734-5.
3 Nichols G, Thom BT. Screening for cryptosporidium in stools. *Lancet* 1984;i:735.

"BNFs" for the Third World

SIR,—In his recent paper on essential drugs Professor P F D'Arcy mentioned the lack of reliable information in many Third World countries on the indications, contraindications, and side effects of individual drugs (13 October, p 982). In view of the notable lack of medical textbooks in many of these countries and of the "disposable" form now adopted for the *British National Formulary* (a new edition of which is supplied free to every doctor in the National Health Service four times a year) would it not be possible to divert three month "old" copies of the *BNF* to Third World doctors, nurses, and medical students?

Perhaps Oxfam or the Joint Mission Hospital Equipment Board (ECHO) would be able to arrange a collection depot, and a note of the relevant address could be prominently displayed by the publishers on the cover of future editions. Although distribution costs might be substantial, the small print used makes for extremely good value for money. Finally, some British drug manufacturers might be prepared to underwrite the exercise since the value of their products, in my own Third World experience, almost always exceeds that of those from other countries.

R D S FAWDRY

Milton Keynes Hospital,
Eaglestone,
Milton Keynes MK6 5LD

How to beautify your old hospital

SIR,—Sadly it is not only apathy or antipathy that deters the hospital beautifier (29 September, p 807). The physical task itself may be quite impossible.

Here we have no main hospital entrance, only a series of holes and doors into the corridors, which are festooned with loose cables in rows, loops, and straggles. The leaks in the various roofs appear to defy repair—and our redecorated postgraduate centre (which is hung with some lovely old photographs) was waterstained within a month. The positioning of strategic buckets in the ward above my office is a regular feature of rainy days.

What hope is there for the interior when the exterior cannot be maintained in a sound state, and what emotion for beautification other than despair? What is worse, the fine carved brickwork is ignored by Pevsner while the new concrete Greenwich District Hospital is almost eulogised.

ANDREW BAMJI

Brook General Hospital,
London SE18 4LW

Points

Time to push aside the diagnosis of infantile colic

Professor R ILLINGWORTH (Sheffield S11 9SD) writes: Dr Albert Massarano's letter (20 October, p 1072) suggests that he is neither conversant with the many published reports on evening colic nor had personal experience of the problem. He stated "no studies yet show a statistically significant improvement with infants treated with dicyclomine." I described a double blind trial,[1] and this was fully confirmed in Australia in a double blind crossover trial.[2] The word "colic" is used by many to explain any crying by an infant, and dicyclomine is commonly and ineffectively used for its treatment. Much crying merely represents the baby's desire to be picked up and cuddled (or at least is stopped by it), and dicyclomine is no substitute for love. But the typical rhythmical screaming attacks of colic, predominantly in the evening, which are not stopped by picking up or a feed, are nearly always prevented by the drug. It is true that meningitis is often initially labelled as colic; and it is often disastrously labelled as "teething." Teething does occur; so does evening colic. Both diagnoses are commonly wrong.

1 Illingworth RS. Evening colic in infants. A double blind trial of dicyclomine hydrochloride. *Lancet* 1959;ii:1119-20.
2 Grunseit F. Evaluation of the efficacy of dicyclomine hydrochloride syrup in the treatment of infantile colic. *Curr Med Res Opin* 1977;5:258-61.

Dr D M SYME (Killin, Perthshire FK21 8UH) writes: I can only assume that Dr Albert Massarano has had the good fortune not to be closely associated with any infants who have suffered from infantile colic. If he had I am sure he would be in no doubt as to the reality of this condition. His observation "that if the baby is removed from the vicious circle by admission to a paediatric ward the screaming or colic ceases" may well be explained, as Professor Illingworth has noted,[1] by the fact that "a busy nursery staff may not notice a baby's cries as much as a mother does at home."

1 Illingworth RS. *The normal child.* 7th ed. Edinburgh: Churchill Livingstone, 1979:27-31.

Too many doctors—or too few?

Dr J R W HANGARTNER (Chairman, Hospital Junior Staff Committee, London SE3 0HA) writes: Dr David Stevenson suggests that one's prospects of coping with unemployment are better if one has received higher education than if one has not (20 October, p 1070). He may well be correct, but he is not correct to suggest that training fewer individuals as medical students will deny those individuals higher education. True it will restrict their choice, but provided the resources remain devoted to higher education more individuals would be able to benefit from higher education—assuming their courses were for three rather than five or six years. It may be selfish to deny a medical education, but is it not cruel and callous to add increasing numbers of medical graduates to a career structure that cannot accommodate them?

Haemoglobinopathies and glycosylated haemoglobin estimation

Dr R J I BAIN (Claughton, Birkenhead L43 1TB) writes: Like Dr G Pulman and others (13 October, p 1001) I have recently diagnosed unsuspected heterozygous HbC trait in a longstanding diabetic after glycosylated haemoglobin (HbA_{1c}) estimation. A 54 year old black woman was diagnosed as having diabetes in 1958. Since then she has been reasonably well controlled on oral hypoglycaemic agents and diet. HbA_{1c} levels were determined by agar gel electrophoresis and analysed on a Corning 710 densitometer. This method determines the HbA_{1c} as a percentage of the nearby, but slower moving, HbA peak. In this case the autoanalyser gave HbA_{1c} levels of 15·5%. Visual analysis, however, revealed a large, slow moving peak far to the left of the HbA which accounted for 38·3% of the total haemoglobin. The HbA_{1c} level was therefore re-evaluated as the percentage of the total haemoglobin present and found to be 8·3%. Haematological investigation showed a normal full blood count, and 10% target cells were present in the peripheral blood film. Clearly it is necessary to recognise the presence of abnormal haemoglobin to determine correctly the HbA_{1c} value—which otherwise might be artificially high. All output cards should therefore be checked visually to exclude haemoglobinopathies, even when other haematological indices are normal.

Greenham Common

Dr ALAN DAVIS (Kyle, Ross-shire) writes: Dr Clare Hamon is to be congratulated on her courageous personal view (20 October, p 1069). Whatever one's views on Greenham Common women, I am sure that most National Health Service workers are appalled at the insidious increase in "defence" spending while hospital waiting lists lengthen, social services are stretched to the limit, and unemployment increases remorselessly. Like Gosport, this area relies heavily on the Ministry of Defence for jobs. Employees often express reservations about the nature of their work but, having families to feed and no real prospect of alternative employment, continue to contribute to the arms race. All the more reason, then, for the free individual to voice his protest. To remain silent is to condone what is happening.

How old is old?

Dr G D PIRRIE (Eastbourne, East Sussex BN20 7DA) writes: Dr Denis Gibbs is a little confused in his quotations from Shakespeare and the psalmists in his review of *Gastrointestinal Disorders of the Elderly* (20 October, p 1065). King Lear says: "I am a very foolish fond old man, Fourscore and upward, not an hour more or less: And to deal plainly, I fear I am not in my right mind." This is thus very close to the psalmist who says: "The days of our age are threescore years and ten: And though men be so strong that they come to fourscore years: Yet is their strength but labour and sorrow."

Metabolic effects of bicarbonate in diabetic ketoacidosis

Dr JULIAN M LEIGH (Royal Surrey County Hospital, Guildford, Surrey GU2 5XX) writes: Dr P J Hale and colleagues (20 October, p 1035) are to be congratulated on their contribution to the knowledge of this subject. Though one cannot dispute their findings, I think that their conclusion is overstated. Surely it is more realistic to say that, according to assessment by intermediary metabolites, sodium bicarbonate *administered according to the regimen of this study* appeared to be neither beneficial nor deleterious.

The case for not administering sodium bicarbonate *under any circumstances* is not made. The danger is that registrars who deal with patients with severe diabetic ketoacidosis and follow the saline only regimen may do so to the detriment of a few patients. As director of an intensive care unit I receive only the failures—that is, patients who have been rendered hypernatraemic with saline and are still acidaemic (pH $< 7{\cdot}0$) and hyperglycaemic with a huge insulin requirement. I then have "no room" for the bicarbonate anion and its obligatory Na cation and am always left with the feeling that a little less Cl^- and a little more HCO_3^- would have made all the difference.