CORRESPONDENCE

BCG in Britain J Lorber, FRCP..... 1066	Surveillance of food poisoning and salmonella infections F G Miskelly, MRCP, and R Orr, MB.... 1069	Hazards of surgical glove powders I D L Fraser, FRCS..... 1072
Collecting and banking human milk: to heat or not to heat J D Baum, FRCP..... 1066	Primary biliary cirrhosis: an epidemiological study W S Hislop, MRCP..... 1069	Breast cancer trials—a new initiative P A M Walden, MRCP..... 1072
Perinatal practice and compensation for handicap Gilliane M McIlwaine, MD, and others; P L C Diggory, FRCOG; R S Illingworth, FRCP..... 1067	Finger wrinkling after immersion in water S M O'Riain, FRCS; D J Williams, FRCS; G Alvarez, MD..... 1070	Parity and breast cancer E Trapido, MD..... 1072
The Steve Biko case: politics and medical ethics R Hoffenberg, FRCP..... 1068	Porphyria cutanea tarda and beta-thalassaemia minor with iron overload R W G Chapman, MRCP..... 1070	Mexiletine for supraventricular tachycardia S D Slater, FRCPGLAS, and others..... 1072
Feeding patients with advanced dementia C J Burns-Cox, FRCP..... 1068	Laboratory features of pleural effusions P P Sutton, MRCP, and S W Clarke, FRCP.. 1070	Risks of coronary arteriography C A Layton, MRCP..... 1073
Disturbed behaviour induced by high-dose antipsychotic drugs T R E Barnes, MRCPsych, and P K Bridges, FRCPsych..... 1068	Comparison of continuous and intermittent papaveretum after cholecystectomy J J Church, FFARCS; J G Jones, FFARCS, and others..... 1071	Confusion of filters: potential hazard from ultraviolet lamps E F J Ring, MSC, and others..... 1073
Allergy to insulins P Diem, MD..... 1068	Anthropometric and dynamometric variables and serious postoperative complications R W Parnell, FRCP..... 1071	Driving after anaesthesia J M Cundy, FFARCS..... 1073
Anaphylactic reaction to desensitisation Pamela W Ewan, MRCP..... 1069	Antibiotics in surgical treatment of acute abscesses D H Wilson, FRCS..... 1071	A rare case of atypical angina H A Cameron, MB, and others..... 1073
Unprecedented increase of infantile hypertrophic pyloric stenosis J A Dodge, FRCPED and others; T E Sensky, MB..... 1069		Vomiting as a diagnostic aid—or diagnostic pitfall A M N Gardner, FRCS..... 1074
		Women in hospital medicine Anne C MacLeod, MB..... 1074
		Restore the medical superintendent? S T H Jenkins, FRCP..... 1074
		Excluded from practice A E Ward, MB..... 1074
		Two lessons about rabies D Parker, MRCP..... 1074

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

BCG in Britain

SIR,—Your leading article (27 September, p 825) on BCG in Britain does full justice to the effect of BCG in mass vaccination programmes but barely mentions its importance for contacts; yet it is in this field that BCG has its greatest value and it is in this field that the highest number of individuals are likely to benefit.¹ Unfortunately, this does not seem to be realised by many doctors today, including some chest physicians—with disastrous results.

Although tuberculous meningitis is very uncommon almost every case that I have had in the last few years would have been easily preventable had the child had BCG. Within the last three years I had two children whose mother, grandmother, grandfather, or any combination had had tuberculosis and yet the child was not given BCG, even when the mother asked. These patients included one whose mother had recovered from tuberculous meningitis in her childhood and yet the child was not given BCG. Another tragedy is that as tuberculous meningitis is now, fortunately, unknown or uncommon, diagnosis is commonly delayed because people do not think of it. Although we have effective treatment to ensure recovery without sequelae in every patient who presents early enough, grave

sequelae can still occur in those who have been in a coma for days before they are referred to a proper centre.

It is essential that BCG should be given to every baby—which could be done on the first day effectively—with a positive family history, even if there are no active cases known at the time; and that BCG should be given to any

contacts of newly discovered cases. By contacts I mean even next-door neighbours and friends, not just relations.

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¹ Lorber J. *Br Med J* 1953;iii:1122-6.

Collecting and banking human milk: to heat or not to heat

SIR,—I wish to disagree with the recommendations of Dr Bengt Björkstén and others (20 September, p 765) in their paper on collecting and banking human milk.

The essential points in their paper can be summarised as follows: they have seen no ill effects from feeding donated raw human milk to newborn infants; pasteurisation is an inexact process which not only eliminates pathogenic bacteria but also damages the bacteriostatic mechanisms present in human milk; as a result pasteurised human milk will be more susceptible to later contamination; therefore nurseries should use raw breast milk.

These apparently reasonable arguments oversimplify, and in places misrepresent, some of the issues involved. Of key importance is the

fact that pasteurisation need not be an inexact and unreliable process. To my knowledge there are at least two commercially available machines specifically designed for the precise heat treatment of human milk. We have shown that such a machine *can* preserve the majority of the antimicrobial factors while reliably eliminating potential pathogens from pooled banked milk.¹ The milk is exposed in 100 ml bottles to a temperature of 62.5°C (±0.5°) for 30 minutes with a rapid and reproducible heating and cooling cycle. Furthermore, the machine is automated and adaptable to other time and temperature settings. Our recent studies (unpublished) have shown that at shorter time and lower temperature settings the bacteriocidal effect is retained with a

preservation of antimicrobial factors (other than milk cells) approaching 100%.

There is no simple correct way of running a human milk bank. However, great caution should be exercised in recommending the wholesale adoption of a policy of using raw bacteriologically unmonitored human milk for sick low-birthweight infants, especially in hospitals which have had little or no previous experience of human milk banking. I believe that, apart from the use of fresh milk from an individual mother for her own baby, it is safer to opt for a simple system based on precise pasteurisation,² which can be relied on to provide bacteriologically clean milk with the majority of its bacteriostatic properties intact.

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¹ Gibbs JH, Fisher C, Bhattacharya S, et al. *Early Human Development* 1977;1:227-45.

² Baum JD, Fisher C, Smith A. *A guide to human milk banking*. Basingstoke: Vickers Medical, 1980.

Perinatal practice and compensation for handicap

SIR,—Mr Peter Mitchell and Mr Iain Chalmers (27 September, p 868) are right to question the fashionable view of the "preventability" of perinatal deaths and the consequences it may have for obstetric practice. We have been particularly puzzled by the conclusions (paragraphs 4 and 5) of the Social Services Committee's report on perinatal and neonatal mortality,¹ which states: "(4) The total number of babies in England and Wales who die in the perinatal or neonatal period each year is 9-10 000. About one-third to one-half of the deaths are preventable, if modern knowledge and care were universally applied. This amounts to at least 3000-5000 avoidable baby deaths a year. (5) The number of children surviving each year with important handicaps that could have been prevented is probably at least 5000." We wonder about the basis on which their assertions are made.

In 1977 we analysed the case records of 1122 of the 1150 perinatal deaths which occurred in Scotland that year.² Of the 747 deaths of normally formed infants occurring in single births there were 408 deaths where the mother had no obvious obstetric abnormality. While this group may appear to offer scope for the reduction in perinatal mortality, the problem is more complex. Among these 408 babies, 106 weighed 2500 g or more, but only seven had a gestation length greater than 42 weeks. Induction prior to 42 weeks' gestation might have prevented those seven deaths, together with a further 40 occurring at 40-42 weeks' gestation, but this would also have required the induction of 37 000 Scottish babies delivered between these gestational ages. Such intervention would not have prevented the 59 deaths in this group which were delivered before term.

Although simpler measures such as fetal kick charts are likely to have their place, it is perhaps unrealistic to suppose that they can have a major role. In our study such charts may have been of value in the 106 cases mentioned above and the 72 cases of antepartum and intrapartum perinatal deaths—low-birthweight babies weighing 1500-2500 g. In order to reduce perinatal mortality, it would be necessary for all pregnant

women to keep fetal kick charts from 30 weeks onwards. In Scotland in 1977 this would have meant that 62 895 women would have kept charts, of whom 0.3% would have experienced perinatal death not related to obstetric complications. As Mr Mitchell and Mr Chalmers say, there is an important case for research in these methods, but it is also important that we preserve a perspective of their relevance and that we include in our reckoning the "negative" effects—in terms of the stress and anxiety imposed on pregnant women—of the techniques we employ.

In the low-birthweight group (less than 2500 g) 302 perinatal deaths occurred; 178 babies weighed less than 1500 g and 58% were born before 32 weeks' gestation. Thirty-three per cent of these babies were growth retarded.

Improved paediatric facilities in some areas where the current facilities are poor might have changed the prognosis for some babies. In general terms, however, prevention of deaths in this group of babies must depend on a better understanding of the reproductive mechanisms that are involved and more effective methods of diagnosis than are at present available.

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¹ Social Services Committee. *Perinatal and Neonatal Mortality. Second report from the Social Services Committee, 1979-80*. London: HMSO, 1980.

² McIlwaine GM, Howat RCL, Dunn FH, Macnaughton MC. *Scotland perinatal mortality survey 1977*. University of Glasgow: 1978.

SIR,—At a time when clinical practice is changing rapidly, Mr P Mitchell and Mr I G Chalmers (27 September, p 868) are wise to call for a medical audit of new techniques in perinatal care. Retrolental fibroplasia is only one example of failure to evaluate a changing technique that resulted in a long delay before the iatrogenic factors were established.¹

In the last decade there has been a revolutionary change in the practice of obstetrics, with far greater intervention than previously and increasing reliance on electronic fetal monitoring. These technological changes have not yet received adequate assessment. Obstetricians have seized on the instruments provided by scientific research but have failed to apply scientific discipline to their evaluation in clinical practice.

Only four small prospective clinical trials of fetal monitoring have been reported: two from Denver,^{2,3} one from Melbourne,⁴ and one from Sheffield.⁵ None has demonstrated the advantages of the new technique and the most significant finding, common to all four trials, was an approximate doubling in the caesarean section rate without demonstrable improvement in fetal outcome. If our new obstetrics raises the UK section rate to that of the US, we shall be doing 35 000 extra caesarean sections a year and some women will most certainly die.⁶

Because monitoring is a feature of the teaching and research hospitals, we should beware of giving the impression that it is superior to good clinical care with intermittent auscultation by doctor or midwife in

the management of straightforward, low-risk delivery. There is as yet no evidence to support such a postulate,⁷ and we may deceive a woman who did not receive monitoring in labour and whose child later developed a handicap into wrongly supposing that the handicap could have been avoided. On available evidence, truly perinatal events are relatively unimportant in the aetiology of handicap, social and prenatal factors being far more important.

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¹ Silverman WA. Retrolental fibroplasia: a modern parable. Monographs in neonatology. New York: Grune and Stratton, 1980.

² Haverkamp AD, Thompson HE, McFee JG, Cetrulo C. *Am Med J Obstet Gynecol* 1976;125:310-20.

³ Haverkamp AD, Orleans M, Langendoerfer S, McFee JG, Murphy J. *Am Med J Obstet Gynecol* 1979;134:399-412.

⁴ Renou P, Chang A, Anderson I, Wood C. *Am Med J Obstet Gynecol* 1976;126:470-6.

⁵ Kelso IM, Parsons RJ, Lawrence GF, Arora SS, Edmonds DK, Cooke ID. *Am Med J Obstet Gynecol* 1978;131:526-32.

⁶ Chalmers I. *Journal of Maternal and Child Welfare* November 1979:435.

⁷ Report of the Task Force on predictors of fetal distress in antenatal diagnosis. Washington: National Institutes of Health, 1979.

SIR,—I welcomed the letter by Mr P Mitchell and Mr I G Chalmers (27 September, p 868) and agree entirely with their comments. But the matter of compensation for handicap is a much wider one, and it is the unfairness which should concern us.

It is absolutely wrong that parents should be awarded a massive six-figure compensation for a child's handicap on the unprovable grounds that someone pulled too hard on the forceps or should or should not have done this, that, or the other, when the great majority of parents whose children have precisely the same handicap get nothing, either because no claim was made or because a barrister was not so persuasive that negligence had occurred or failed to arouse the judge's natural desire to help parents in their tragedy of having a handicapped child. It is most unfair that parents should receive massive compensation for a child who is spastic or mentally subnormal, and who was delivered with some difficulty, while the great majority of other parents having a child with exactly the same handicap but whose delivery was normal get no compensation at all. Most cases of cerebral palsy or mental subnormality are of multifactorial origin; it is irrational to pick on just one factor and pay compensation for it when there were several prenatal factors which were just as likely or more likely, alone or in combination, to have been responsible.

The same unfairness applies to compensation for so-called vaccine damage after whooping cough immunisation. It is totally irrational that some parents should receive compensation on the grounds that the child is found to be spastic or mentally subnormal or to have fits some time after immunisation when the great majority of parents whose children have exactly the same handicap get no compensation at all. There is no diagnostic test which indicates damage by whooping cough vaccine, neither are there specific clinical features. Exactly the same clinical picture can be caused by numerous other conditions, such as virus infections. It is wrong that compensation should be awarded or refused on no evidence at all, presumably