Letters

Website: **bmj.com**
Email: **letters@bmj.com**

Mercury sphygmomanometers: disposal has far reaching consequences

EDITOR—The papers by O'Brien and Beevers et al have documented the trend away from the use of the mercury sphygmomanometer.[1 2] But the ethical, legal, economic, and public health consequences associated with their disposal have not been so well documented. This lack of advice is surprising since mercury has well known toxic properties. Haphazard disposal of mercury inevitably leads to environmental recycling and bioconcentration, with ecological damage at best and human poisoning at worst.[3]

The Special Waste Regulations 1996 classify mercury and waste material containing more than 3% mercury as special waste and require extensive documented control of, and the use of approved contractors for, each stage of the disposal process. Noncompliance can result in a major fine, together with a custodial sentence.

Although technically only the mercury component is special, it would not be safe for staff to break sphygmomanometers open to access the mercury in uncontrolled conditions. In addition, we considered it likely that standard mercury containing sphygmomanometers used in general practice in England and Wales would contain more than 3% mercury and should therefore be considered special waste. We obtained four intact mercury sphygmomanometers from a general practitioner's surgery and drained and weighed the mercury under laboratory conditions. Each contained more than 3% mercury (average 6.5%).

The NHS must develop a policy for the storage and ultimate disposal of retired mercury sphygmomanometers that accords with the Special Waste Regulations 1996. Data from Birmingham health authority show that more than 30 000 mercury sphygmomanometers are still in active use by general practitioners in the UK. If plans are not made to dispose of these sphygmomanometers safely, some 1800 kg of mercury could enter the environment. The contamination that would result from the haphazard disposal of all mercury sphygmomanometers used in the NHS would be much greater, and the volumes involved suggest that collection and recycling may be commercially viable.

What should general practitioners, hospitals, and other NHS trusts do? Expecting each to negotiate a suitable system of collection and disposal would be burdensome, inefficient, and impractical, although it is their legal responsibility to do so. We are working with the environment agency in our region to develop an approved scheme that will reduce the bureaucratic demands on individual practices while protecting them from statutory action and, at the same time, provide a safe, efficient, and cost effective storage, transport, and disposal service.

Patrick Saunders *regional environmental health adviser*
p.j.saunders@bham.ac.uk

Andrew Rouse *consultant in public health*
Department of Public Health and Epidemiology, University of Birmingham, Birmingham B15 2TT

Shaukat Ali *clinical governance officer*
Birmingham South West Primary Care Group, Edgbaston, Birmingham B15 2TG

Since we performed our study the Environment Agency has revised its advice on the disposal of such equipment, with the result that effectively all sphygmomanometers (rather than those containing more than 3% mercury) must be regarded legally as special waste and be subject to the extensive and potentially expensive statutory and bureaucratic control of such wastes. The agency in this region has also, however, suggested a waste recovery scheme that would be cost neutral and would suspend the most bureaucratic elements of complying with legislation.

1 O'Brien E. Replacing the mercury sphygmomanometer. *BMJ* 2000;320:815-6. (25 March.)
2 Beevers G, Lip GYH, O'Brien E. ABC of hypertension: Blood pressure measurement. *BMJ* 2001;322:1043-7. (28 April.)
3 Takizawa Y, Sekikaw A. Epidemiology of Minamata disease. http://cisat.isciii.es/supercourse/lecture/lec0361/006.htm (accessed 28 August 2001.)

Advice to authors

We prefer to receive all responses electronically, sent directly to our website. Processing your letter will be delayed unless it arrives in an electronic form.

We are now posting all direct submissions to our website within 24 hours of receipt and our intention is to post all other electronic submissions there as well. All responses will be eligible for publication in the paper journal.

Responses should be under 400 words and relate to articles published in the preceding month. They should include ⩽5 references, in the Vancouver style, including one to the BMJ article to which they relate. We welcome illustrations.

Please supply each author's current appointment and full address, and a phone or fax number or email address for the corresponding author. We ask authors to declare any competing interest. Please send a stamped addressed envelope if you would like to know whether your letter has been accepted or rejected.

Letters will be edited and may be shortened.

bmj.com
letters@bmj.com

Duration of breast feeding and adult arterial distensibility

Humans are primates, designed to breast feed for years not months

EDITOR—With respect to the article by Leeson et al on duration of breast feeding and arterial distensibility in early adult life, of course the duration of breast feeding matters—the longer the better.[1]

Humans are animals, mammals, and primates. Research on correlates of weaning age in non-human primates, such as adult body size, length of gestation, timing of permanent tooth eruption, timing of sexual maturity, and growth rates during childhood, predict that modern humans should be breast fed for between two and a half and seven years.[2 3] Humans have slightly longer durations of all stages of the life span than our nearest relatives, chimpanzees. We have slightly longer gestation, later dental eruption, later sexual maturity, and therefore would expect slightly later ages of weaning. Chimpanzees breast feed for four to five years. Around the world, many children are breast fed for two and a half to seven years, including some in the United States, Canada, and Great Britain.

Maybe a healthy start in life of several to many years of breast feeding should be followed by a lifelong diet low in animal protein and fat and high in physical exercise, to maximise heart health in adulthood. But we will not know this until researchers study the effects on blood vessel flexibility of normal durations of breast feeding (2.5-7.0 years), and of the combination of normal durations of breast feeding with different post-weaning diets and amounts of exercise. I find it appalling that researchers would suggest that more than four months of breast feeding could be harmful to children, when research shows that 2.5-7.0 years is clearly the normal and natural duration for our species. On a final note, it is always good advice to question the credibility of research

and researchers funded by infant formula companies.

Katherine A Dettwyler *associate professor of anthropology and nutrition*
Texas A&M University, College Station, TX 77843-4352, USA
kadettwyler@hotmail.com

Competing interests: None declared.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)
2 Dettwyler KA. A time to wean: the hominid blueprint for the natural age of weaning in modern human populations. In: Stuart-Macadam P, Dettwyler KA, eds. *Breastfeeding: biocultural perspectives*. New York: Aldine de Gruyter, 1995: 39-73.

Explanation of findings and context before publication might have been helpful

Editor—Perhaps it is not surprising that the report by Leeson et al—that breast feeding followed by a high fat diet may later be associated with stiffer arteries—drew so many responses.[1] Breast feeding is a sensitive topic. The normalising of artificial feeding by formula companies and the media requires efforts to protect breast feeding. But we should not allow our protective stance to become blindly defensive. It is easy to fall into the teleological trap of believing that breast feeding was designed, by God or nature, to be perfect. It is possible that evolution could have had this result. During our evolution we have had much shorter life spans than we have achieved recently and eaten much less fat. This is a sound study that adds a piece to the complex puzzle of how early nutrition may influence adult disease risks. It does not prove that breast feeding increases the risk of heart disease.

In their attempts to counter the study's conclusions, many of the respondents tilt at windmills. Some point out that rates of heart disease are low in developing countries where breast feeding for two years or more is common. But with changing diets and a higher proportion of elderly people, rates of heart disease are increasing rapidly in developing countries.[2] Many have dismissed the findings because they are based on maternal recall. This is an important epidemiological issue, which can be studied by comparing mother's recall with clinic records. Such studies show that mothers can accurately recall breastfeeding duration for as long as 29 years, but they are less reliable at recalling age at introduction of formula.[3 4] A Queensland study found that the differences in breastfeeding duration as recalled by 75 mothers (over one to 10 years) and recorded by the clinic were less than one month for 79% of children, and less than two months for 95% of children.[5] They found no difference in accuracy of recall between mothers with different levels of education, or with numbers or ages of children.

Leeson et al described the limitations of their study and emphasised that it should not lead to any change in infant feeding recommendations. It is unfortunate but predictable that the media will sensationalise such research reports. If advocates of breast feeding fostered links with reputable infant nutrition researchers such as Lucas's team, perhaps an appropriate explanation of the findings and context could be prepared before publication. Midwives and breast feeding counsellors could then use this to reassure parents.

Wendy Holmes *women's and children's health specialist*
International Health Unit, Macfarlane Burnet Centre for Medical Research, Fairfield, Victoria 3078, Australia
holmes@burnet.edu.au

Competing interests: None declared.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)
2 Murray C, Lopez A. Mortality by cause for eight regions of the world: global burden of disease study. *Lancet* 1997;349:1269-76.
3 Troy LM, Michels KB, Hunter DJ, Spiegelman D, Manson JE, Colditz GA, et al. Self-reported birthweight and history of having been breastfed among younger women: an assessment of validity. *Int J Epidemiol* 1996;25:122-7.
4 Kark JD, Troya G, Friedlander Y, Slater PE, Stein Y. Validity of maternal reporting of breastfeeding history and the association with blood lipids in 17 year olds in Jerusalem. *J Epidemiol Community Health* 1984;38:218-25.
5 Eaton-Evans J, Dugdale AE. Recall by mothers of the birth weights and feeding of their children. *Hum Nutr Appl Nutr* 1986;40A:171-5.

Dose-response, cause and effect relation between breast feeding and heart disease seems unlikely

Editor—Once again epidemiological data from the United Kingdom are leading to a claim that extended breast feeding may lead to later adverse cardiovascular outcomes.[1 2] Leeson et al say that their findings are consistent with those of Fall et al, which were widely publicised in the media.[1 3] The causal mechanism postulated by Fall et al was not found to hold in this study, and the results do not support a hypothesis of deranged blood lipid profiles in adulthood. Will this failure to confirm the previous hypothesis receive attention, or will the media say that this study "confirms" the findings of the previous one?

We will never know the impact of breast feeding on human health because it is unethical to randomise. Thus we have to be very careful to look for confounders when we do associative studies such as this, and Leeson et al made an effort to do so. Presumably, however, families with children who breast fed for longer periods in the United Kingdom 20-30 years ago differed from those who fed their babies closer to the norm of the time. Slightly over a quarter of British babies were breast fed for longer than four months in 1980[4]; similar to the proportion of those contacted who agreed to participate in this study.

The demographic and health survey data for South Asian countries show that about half the children are breast fed for longer than two years in India, two and a half years in Nepal, and three years in Bangladesh.[5] Hundreds of millions of adults currently alive in that region were probably breast fed for even longer periods than this. If there were any dose-response, cause and effect relation between sustained breast feeding and heart disease, why is heart disease not at much higher levels there among those who reach old age than it is in rich countries? This study was conducted by a group that included the Medical Research Council childhood nutrition research centre, which has collaborated with the infant food industry for its outcome studies on nutrition. Their honesty in admitting this (or is it the exemplary *BMJ* insistence on such declarations?) may not allay our fears regarding the potential effects on the research of this kind of conflict of interest

Although Leeson et al point out that their findings cannot be interpreted as cause and effect, normally anything negatively associated with breast feeding quickly gets translated into just that by the media and receives wide dissemination. Let's see what happens with this one.

Ted Greiner *head, international nutrition research group*
International Nutrition Research Group, Department of Women's and Children's Health, Uppsala University, S-75185 Uppsala, Sweden
ted.greiner@kbh.uu.se

Competing interests: None declared.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)
2 Booth I. Does the duration of breast feeding matter? *BMJ* 2001;322:625-6. (17 March.)
3 Fall CH, Barker DJP, Osmond C, Winter PD, Clark PM, Hales CN. Relation of infant feeding to adult serum cholesterol concentration and death from ischaemic heart disease. *BMJ* 1992;304:801-5.
4 Martin J, White A. *Infant feeding*. London: Office of Population Censuses and Surveys, 1985.
5 Haggerty PA, Rutstein SO. *Breastfeeding and complementary infant feeding, and the postpartum effects of breastfeeding*. Calverton, MD: Macro International, 1999.

Authors did not discuss data from prospective studies

Editor—Leeson et al propose a complex mechanism to explain their observations linking a putative marker of vascular risk with the duration of any breast feeding.[1] They suggest a dose-response relation between duration of any breast feeding and brachial artery distensibility and that extending breast feeding by two months has an effect on arterial distensibility broadly equivalent to that produced by a 4 mm Hg increase in blood pressure. The discussion of their findings is, however, not systematic. It neglects (as does the editorial by Booth[2]) to review important evidence. The observational findings by Leeson et al should be placed in the context of other epidemiological data relating directly to factors (in this case, blood pressure) whose link to adverse health outcomes are more clearly established than that of arterial distensibility.

One of the most important pieces of evidence comes from the seven year follow up by Wilson et al of the Dundee infant feeding study.[3] In this study, systolic blood pressure at the age of 7 was found to be significantly raised in those children who had been exclusively formula fed for the first 15 weeks of life compared with those who had received any breast milk (mean 94.2 (95% confidence interval 93.5 to 94.9) mm Hg *v* 90.7 (89.9 to 917) mm Hg). These findings run counter to the observations by Leeson

et al on distensibility, from which the opposite findings would be expected—namely, that blood pressure would be higher in those children who had been breast fed. Further evidence against the hypothesis of Leeson et al comes from the work of Taittonen et al, who found that breast feeding after 3 months of age was associated with an average reduction in blood pressure of 6.5 mm Hg.[4]

We were surprised that Leeson et al did not refer to their own related research published earlier this year in the *Lancet*, in which they concluded that consumption of breast milk was associated with lower blood pressure at age 13-16 years.[5] This research was based on a unique opportunity afforded by a randomised trial to overcome some of the biases that are likely to be operating in observational studies, such as the one they report in your journal.

Jim Sikorski *general practice research fellow*
Department of General Practice and Primary Care, Guy's, King's, and St Thomas's School of Medicine, London SE11 6SP
jim.sikorski@kcl.ac.uk

Carol Dezateux *clinical senior lecturer in paediatric epidemiology*
Department of Paediatric Epidemiology and Biostatistics, Institute of Child Health, London WC1N 1EH

Competing interests: None declared.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)
2 Booth I. Does the duration of breast feeding matter? *BMJ* 2001;322:625-6. (17 March.)
3 Wilson AC, Stewart Forsyth J, Greene SA, Irvine L, Hau C, Howie PW. Relation of infant diet to childhood health: seven year follow up of cohort of children in Dundee infant feeding study. *BMJ* 1998;316:21-25.
4 Taittonen L, Nuutinen M, Turtinen J, Uhari M. Prenatal and postnatal factors in predicting later blood pressure among children: cardiovascular risk in young Finns. *Pediatr Res* 1996;40:627-32.
5 Singhal A, Cole TJ, Lucas A. Early nutrition in preterm infants and later blood pressure: two cohorts after randomised trials. *Lancet* 2001;357:413-9.

Does this study herald the return of national dried milk?

Editor—On the day the article by Leeson et al was published, and because of the national furore it created, I was asked to generate a response for midwives to use to allay the worries of mothers telephoning the 24 hour advice line.[1] We work in the wards and departments of a busy obstetric unit in the north east of England, supporting mothers in the initiation of breast feeding, but not one mother or any family members queried the health benefits of breast feeding. People in the north east of England do watch television and read newspapers, so it seems they disregarded what they saw as another conflicting message from health professionals.

We promote breast feeding in areas where there has been a traditional bottle feeding culture. Articles such as the one by Leeson et al do not make our work any easier, but I agree with Holmes in her response that we should not be defensive and that all research should be scrutinised, even if it does threaten conventional wisdom. I also agree with other respondents that further, large scale research may lead to different conclusions. My own local response questions the statistical methods used and the effects of confounding variables (such as weaning patterns, definitions of exclusive or partial breast feeding, etc), which have already been raised by other respondents. But another issue that should be considered in this debate is the type of formulas in use during the period studied, between 1969 and 1975. Before 1974, most types of formula milk were still comparatively unmodified. Most contained 100% milk fats, which were difficult for young infants to digest and absorb. In this area, a large proportion of the population was fed evaporated milk and national dried milk during this period. Presumably, although Cambridge is a more affluent area, the formulas available were still comparatively unmodified.

The 1974 report, *Present Day Practice in Infant Feeding* (first report), led to the withdrawal of national dried milk and stated that all artificial milk should approximate the composition of breast milk as nearly as is practicable. Formula manufacturers have since spent many millions (or billions) trying to meet this objective.

Given the time scale, it seems that many of the respondents in the reported study would have been fed unmodified infant formula. Do the findings of this study herald the return of national dried milk, as it seems from this study that these types of formula have benefits over breast milk? I do not think so; other factors need to be considered.

Anne Holt *senior lecturer and practitioner*
South Cleveland, Hospital, University of Teesside, Middlesbrough TS4 3BW
anne.holt@tees.ac.uk

Competing interests: Unrepentant mother of four children, all of whom have been breast fed for over a year; two have been breastfed for over four years.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)

Breast feeding: distension or distortion?

Editor—It seems that we live in an increasingly dangerous world: recent media scares have included calcium channel blockade, the oral contraceptive pill, and, latterly, long distance air travel. Now it seems that even breast feeding, promoted for its benefits by our grandparents, is not without risk, as described by Leeson et al in their paper.[1] But the scientific evidence on which such claims are based varies substantially.

Leeson et al set out to test the hypothesis that breast feeding is associated with a detrimental reduction in arterial distensibility. Why they sought to measure distensibility of the brachial artery as an early marker of cardiovascular disease is unclear. Although they say that arterial distensibility diminishes with age in relation to other risk factors, the references they cite concern changes in the carotid and femoral arteries and aorta, and not, as in their study, the brachial artery. This is an important distinction: although aortic distensibility does decrease with age, brachial distensibility does not change.[2] Moreover, despite careful application of the same methods employed by Leeson et al, others have shown that age and hypercholesterolaemia do not influence brachial distensibility.[3 4] Although we agree that aortic pulse wave velocity (a measure of distensibility) does predict cardiovascular outcome in hypertensive and normotensive people and those with renal disease, we are unaware of any data suggesting that the same is true of brachial distensibility.

Overall, Leeson et al could not show any difference in brachial distensibility between those who were breast fed and those who were not. Brachial pulse pressure, a surrogate measure of large artery stiffness that predicts outcome, did not differ significantly between the two groups. There was, however, an inverse association between the duration of breast feeding and distensibility, but this was significant only in women. This is surprising since their original hypothesis was based on the observation that boys who are breast fed up to 1 year of age have an increased risk of ischaemic heart disease in later life.[5]

As a result of the resulting media coverage, many mothers may choose not to breast feed their infants despite much evidence as to its benefits, including a reduction in cardiovascular disease in later life, as noted by Leeson et al. After the pill scare many women stopped taking the oral contraceptive pill, which resulted in a rise in unplanned pregnancies. Finally, there is the propensity for the infant food industry to use such data and media coverage out of context for commercial benefit.

Ian B Wilkinson *lecturer in clinical pharmacology*
Clinical Pharmacology Unit, University of Cambridge, Addenbrooke's Hospital, Cambridge CB2 2QQ

John R Cockcroft *senior lecturer in cardiology*
Department of Cardiology, Wales Heart Research Institute, University Hospital, Cardiff CF14 4XN
cockcroftjr@cf.ac.uk

Competing interests: None declared.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)
2 Nichols WW, O'Rourke MF. *McDonald's blood flow in arteries: theoretical, experimental and clinical principles*. London: Arnold, 1998.
3 Kool M, Lustermans F, Kragten H, Struijker BH, Hoeks A, Reneman R, et al. Does lowering of cholesterol levels influence functional properties of large arteries? *Eur J Clin Pharmacol* 1995;48:217-23.
4 Van der Heijden-Spek JJ, Staessen JA, Fagard RH, Hoeks AP, Boudier HA, van Bortel LM. Effect of age on brachial artery wall properties differs from the aorta and is gender dependent: a population study. *Hypertension* 2000;35:637-42.
5 Fall CH, Barker DJ, Osmond C, Winter PD, Clark PM, Hales CN. Relation of infant feeding to adult serum cholesterol concentration and death from ischaemic heart disease. *BMJ* 1992;304:801-5.

Statistical analysis was unclear

Editor—Leeson et al, in the conclusions in their paper, seem to rely in part on the statistical treatment of their limited observational data,[1] in particular, the use of multiple regression analysis and *t* tests. Multiple regression was used to determine regression coefficients as a measure of association between length of breast feeding and

non-invasive brachial artery distensibility. The *t* tests were used to test the null hypothesis (presumably) that there were no differences in brachial artery distensibility between those who were not breast fed, those who breast fed to age 4 months, and those who breast fed above age 4 months.

We are uncertain whether the regression analysis incorporated all subjects, but this is implied in the paper. Therefore, we are presented with a larger number of those adults who were either breast fed for a short period or not breast fed at all, and smaller numbers at longer periods of breast feeding (although numbers are not specifically given in the paper at each time grouping). We now need to interpret the regression coefficients (table 3), actually quite broad at the 95% confidence intervals. The paper tells us that the P values associated with these regressions are just significant, but no mention is made of the r^2 values that will tell us how much of the variability of arterial distensibility is explained by all variables, including duration of breast feeding. Furthermore, there seems to be no analysis of adults who had been breast fed alone and no r^2 value.

In addition, for the dichotomised groups, arterial distensibility is compared (*t* test) with the non-breastfed group, and a similar comparison is made between the dichotomised groups. Although two comparisons are reported, we suggest that these sort of comparisons should be conducted by using one way analysis of variance with appropriate testing afterwards (for example, Bonferroni), or if multiple *t* tests are used, then the level of significance (presumably set at $P=0.05$ here) should be reduced to account for multiple comparisons (we suggest three comparisons in this case). The low level of significance reported between the dichotomised groups ($P=0.02$) is unlikely to survive such conservative statistical treatment. These approaches are more conservative but give us greater confidence in the assertion that some arbitrarily determined time point could be important in determining future risk of cardiovascular disease.

We find little in this paper that will change our current personal habits or advice we give to other parents. We are delighted that the authors agree.

Paul Michael Dark *Medical Research Council clinical training fellow*
Emergency and Intensive Care Medicine, University of Manchester, Manchester
Paul.M.Dark@man.ac.uk

Marie-Josée Rölli *retainee, family medicine*
Greenmount Medical Centre, Bury BL8 4DR

Competing interests: Parents of breastfed infants and children. M-JR is a member of the breastfeeding network.

1 Leeson CPM, Katternhorn M, Deanfield JE, Lucas A. Duration of breast feeding and arterial distensibility in early adult life: population based study. *BMJ* 2001;322:643-7. (17 March.)

Authors' reply

EDITOR—Our paper on the duration of breast feeding and later arterial distensibility evoked much comment, especially from those who promote breast feeding. Unfortunately the media coverage may have deflected attention from the cautious way we framed our findings—and our clear recommendation that they should not change breastfeeding practice.

Some cast doubt on our work by implying that it was motivated or influenced by formula milk companies. This is certainly not so. The study was funded by the Medical Research Council and the university, with no industrial connection whatsoever. Our childhood nutrition centre is core funded by the government (MRC). Our longstanding research includes some of the strongest scientific evidence available favouring breast feeding, in terms of its beneficial effects on cognitive development, blood pressure, bone health, atopic disease, infection, gut disease, and catch-up growth[1 2]—evidence much used by professional organisations that support breast feeding. We have also researched the efficacy and safety of new advances in infant formula milks and, for transparency, cite this in our article. As an independent centre we publish what we find in the interests of public health, quite regardless of any pressures from either industry or advocacy groups.

Our findings have clearly seemed counterintuitive to many. Dettwyler cites an anthropological argument based on primate work, that humans were evolved to breastfeed for two and a half to seven years, as evidence that our results are biologically implausible. Life span was, however, much shorter when human lactation evolved, and we cannot assume that breast feeding, through past evolution, would now confer any advantage in terms of reduced adult degenerative disease or postreproductive survival. Nor can we assume that breast feeding evolved such that humans would necessarily be well adapted to a modern Western style post-weaning diet. Holmes affirms this view.

Our paper has stimulated comment on interpretation and methods. We agree with Greiner that it is difficult to interpret non-randomised outcome studies on breastfed infants, which of course also applies to the extensive and potentially confounded literature purporting to show benefits of breast feeding. This centre has been one of the only ones to conduct large scale randomised studies on breast milk versus formula in a circumstance in which this is ethical—in non-breastfed premature infants who can be assigned randomly to formula milk or donated banked breast milk. These few studies provide experimental evidence for long term effects of breast milk on health outcomes.[1] When randomisation is precluded (as, say, with smoking), however, causation must be established from a weight of epidemiological evidence, supported by animal experiments. We appraised the possible significance of our own data in such a context, although we accept that the research is at an early stage.

Some respondents imply that we were directly comparing formula feeding to breast feeding. This was not our intention. As Holt noted, formula milks used in the 1970s were different from those currently available, and study of formula fed subjects in our cohort would have had little contemporary relevance. In epidemiological and intervention studies, breast feeding seems to confer cardiovascular benefit over formula feeding.[1] Our interest focused solely on the duration of breast feeding in relation to vascular health in a Western population, in view of previous work we reference.

Our paper considers carefully our surrogate marker of arterial disease, brachial artery distensibility. Wilkinson and Cockroft note that much work on distensibility has been based on the widely used aortic pulse wave velocity. Oddly, their response entirely ignores more recent studies, including this paper, which consistently show an association between peripheral artery distensibility and concentrations of cholesterol and that the various methods for measuring distensibility in central and peripheral arteries are well intercorrelated.[3–6] Simple non-invasive vascular measures, as used in our study, provide unique opportunities to investigate early stages of disease development.

We used a statistically robust approach to data analysis and have been appropriately cautious in our interpretation, taking account of cohort size and significance level. We would reassure Dark and Rölli that the relation between breastfeeding duration and arterial distensibility persists whether analysis is performed on the entire cohort or solely on those breast fed. The r^2 for distensibility versus length of breast feeding is 0.22, suggesting the model accounts for around a quarter of the variability in distensibility.

Finally, we wish to re-emphasise why we would not suggest any current change in breast feeding practice. Firstly, our data are at too early a stage to be translated into health policy. Secondly, any risk-benefit analysis must include the many positive purported benefits of breast feeding on short and long term outcome.

If the hypothesis we raised proves correct, that more prolonged breastfeeding duration followed by a Western style diet explains our results, then future intervention policy might be better directed to our Western diet rather than breast feeding. We hope that the complex social issues that surround this subject will not cloud the need for dispassionate research to optimise infant nutrition in relation to long term health.

Paul Leeson *research fellow*
Alan Lucas *professor*
Medical Research Council Childhood Nutrition Research Centre, Institute of Child Health, London WC1N 1EH

Competing interests: The centre has collaborated with the infant food industry for its outcome studies on nutrition.

1 Singhal A, Cole TJ, Lucas A. Early nutrition in preterm infants and later blood pressure: two cohorts after randomised trials. *Lancet* 2001:357:413-9.
2 Lucas A, Morley RM, Cole TJ, Lister G, Leeson-Payne C. Breast milk and subsequent intelligence quotient in children born preterm. *Lancet* 1992;339:261-4.

3 Leeson CP, Whincup PH, Cook DG, Mullen MJ, Donald AF, Seymour CA, et al. Cholesterol and arterial distensibility in the first decade of life: a population-based study. *Circulation* 2000;101:1533-8.
4 Giannattasio C, Mangoni AA, Failla M, Carugo S, Stella ML, Stefanoni P, et al. Impaired radial artery compliance in normotensive subjects with familial hypercholesterolaemia. *Atherosclerosis* 1996;124:249-60.
5 Shiage H, Dart A, Nestel P. Simvastatin improves arterial compliance in the lower limb but not in the aorta. *Atherosclerosis* 2001;155:245-50.
6 Liang YL, Teede H, Kotsopoulos D, Shiel L, Cameron JD, Dart AM, et al. Non-invasive measurements of arterial structure and function: repeatability, interrelationships and trial sample size. *Clin Sci* 1998;95:669-79.

bmj.com

Summary of rapid responses

This paper and the accompanying editorial by Booth generated a great deal of heated argument.[1 2] We received a total of 51 responses for the two articles, all but eight within the first month of publication. We posted 10 rapid responses on the first day of publication on bmj.com, and over 60% (32) of responses were posted by the time the next issue of the printed journal was published, including a reply from the authors.

All but five of the responses were highly critical of the paper, largely for shortcomings in the methods and because it was funded by a manufacturer of formula milk. Others were concerned about the negative effects on breast feeding resulting from the media's treatment of the results.

Luis Gabriel Cuervo, a member of the *BMJ*'s editorial board, roundly criticised the *BMJ* in its management of the paper:

"The *BMJ* has a responsibility not only to publish evidence. It also has to foresee the effect of the published paper on global health and clearly address it. The breach that allowed the media to manipulate the results and jump to the conclusion that breast feeding for more than four months causes cardiovascular disease is inadmissible and will surely be commercially exploited for unscrupulous purposes, here and in the developing world, with terrible consequences. Later explanatory letters may not have the same impact in the media and may not compensate for the damage that has been done."

Three lone voices joined the authors' in the wilderness.

Allan Astrup Jensen, research director of a company in Denmark, thought that "the many critical responses try to kill the messenger because the message is unpleasant and may hurt common health policies. No paper is perfect, including this one. There will always be questions raised and criticism of methods, execution, and reporting."

Andrew Mimnagh, a general practitioner, and Timothy James, a university senior lecturer, were concerned about the demand by some respondents to ban research sponsored by companies as unethical. Mimnagh added: "I agree the finding is counterintuitive but so are many 'proven facts' in the natural world."

James was disturbed by the "low level of logic" in some of the responses: "[It seems that] the answer has been predetermined and only evidence that supports that answer is acceptable. This is contrary to the entire scientific approach to truth seeking, which demands that we go wherever the evidence takes us, whether it is where we wanted to go or not." He concludes that drawing "conclusions for our own environment is a complex multifactorial matter, which cannot be summed up in a simple slogan like 'breast is best'—however unethical the behaviour of sellers of breast milk substitutes."

Sharon Davies *letters editor, BMJ*

1 Electronic responses. Duration of breast feeding and arterial distensibility in early adult life: population based study. bmj.com 2001;322 (http://bmj.com/cgi/eletters/322/7287/643; accessed 13 Sep 2001).
2 Electronic responses. Does the duration of breast feeding matter? bmj.com 2001;322 (http://bmj.com/cgi/eletters/322/7287/625; accessed 13 Sep 2001).

Debate on screening for breast cancer is not over

EDITOR—In a news item, Mayor described a historically controlled study from Sweden that claimed that screening with mammography reduced deaths from breast cancer by nearly two thirds.[1] This was followed a week later by a comment in the Minerva section, which started off with the strong political message that the case for breast cancer screening programmes is now beyond debate and went on to speak about a meticulous report from Sweden, repeating the claim of a 63% reduction in breast cancer deaths in women screened.[2]

I am surprised that Minerva, who usually makes cautious reservations about the studies she quotes, on this occasion was not cautious, in particular as the quoted study is confounded and cannot reliably say anything about a possible effect of breast cancer screening with mammography.[3] For example, there was no significant difference in the decline in breast cancer mortality in the age group that had been invited to screening, compared with a younger age group that had never been invited to screening, and the study did not take account of introduction of effective treatments such as tamoxifen. Furthermore, it seems implausible that such a dramatic effect should be seen in Sweden, where the trend in mortality from breast cancer has not changed throughout the past 30 years. In contrast, for the United Kingdom, where an upward trend has changed into a downward one, a report tentatively ascribed only 6% of the decline in breast cancer mortality to screening—and the authors even found this estimate impossible to test.[4]

Whether one considers the results obtained in the randomised screening trials or what one could hope for on the basis of tumour biology, the result is the same: if there is any effect of screening, which we currently do not know with certainty, it is likely to be much smaller than the 20-30% reduction in mortality from breast cancer that has often been claimed.[5] It is therefore reasonable to continue the debate on screening for breast cancer.

Peter C Gøtzsche *director*
Nordic Cochrane Centre, Rigshospitalet, DK-2100 Copenhagen Ø, Denmark
pcg@cochrane.dk

Competing interests: None declared.

1 Mayor S. Study confirms that screening reduces deaths from breast cancer. *BMJ* 2001;322:1140. (12 May.)
2 Minerva. *BMJ* 2001;322:1256. (19 May.)
3 Gøtzsche PC. The alleged effect of mammography screening was not proved. *Cancer* (in press).
4 Blanks RG, Moss SM, McGahan CE, Quinn MJ, Babb PJ. Effect of NHS breast screening programme on mortality from breast cancer in England and Wales, 1990-8: Comparison of observed with predicted mortality. *BMJ* 2000;321:665-9.
5 Gøtzsche PC, Olsen O. Is screening for breast cancer with mammography justifiable? *Lancet* 2000;355:129-34.

Risks with continuous subcutaneous insulin infusion can be serious

EDITOR—The editorial by Pickup and Keen about continuous subcutaneous insulin infusion is worrying in advocating this treatment, albeit for a comparatively small proportion of the diabetic population in Britain.[1] Pickup and Keen do not highlight adequately the serious risks associated with it for doctors considering introducing this treatment to their patients.

Pickup and Keen acknowledge high rates of ketoacidosis with subcutaneous insulin infusion but attribute it to lack of experience, unsuitable pump insulin, and the less reliable devices previously available. They identify that rates of ketoacidosis fell as physicians' experience with the treatment increased,[2] but they do not acknowledge that a new generation of diabetes physicians considering using subcutaneous insulin infusion will be unfamiliar with it, as will their support staff and patients.

In the early years of subcutaneous insulin infusion there seemed to be a reluctance to identify ketoacidosis as a risk associated with it, and it would be a mistake to allow this understatement of the problem to occur again. In a large clinic based study of the treatment in Sheffield we observed that subcutaneous insulin infusion occurred at a 17.5-fold greater rate than in a group of patients treated by injection, although with experience this was reduced to a twofold increase during the second year.[3]

Clearly, experience gained was important, but a doubled rate of ketoacidosis in those infusing subcutaneous insulin was still observed. Despite Pickup and Keen's attribution of the development of ketoacidosis to insulin aggregation and unreliable pumps, of 18 observed episodes of ketoacidosis, none was associated with pump failure or insulin aggregation, although one occurred when a cannula became disconnected and another happened when an empty insulin reservoir was not detected. Most episodes (66%) were precipitated by intercurrent illness and were rapid in onset, presumably because there was no subcutaneous insulin reserve in patients with increased insulin requirements owing to physical stress. Two episodes of cardiac arrest occurred as a result of severe hyperkalaemia, one leading to death. Patients treated with subcutaneous insulin infusion with ketoacidosis presented with seemingly more severe hyperkalaemia than those having injection treatment.

Pickup and Keen emphasise the need to limit the availability of subcutaneous insulin infusion for use from specialist centres—although financial costs may not be comparatively high, the treatment is expensive in patient and professional time to ensure safety. Pickup and Keen do not define specialist centres—I suggest that physicians should avoid being coerced into dabbling in pump therapy by patients or pressure groups.

G Knight *consultant physician*
Rotherham General Hospitals NHS Trust, Rotherham General Hospital, Rotherham S60 2UD

1 Pickup J, Keen H. Continuous subcutaneous insulin infusion in type 1 diabetes. *BMJ* 2001;322:1262-3. (26 May.)
2 Bending JJ, Pickup JC, Keen H. Frequency of diabetic ketoacidosis and hypoglycaemic coma during treatment with continuous subcutaneous insulin infusion. *Am J Med* 1985;79:685-91.
3 Knight G, Jennings AM, Boulton AJM, Tomlinson S, Ward JD. Severe hyperkalaemia and ketoacidosis during routine treatment with an insulin pump. *BMJ* 1985;291:371-2.

Intracranial hypertension and nasal fluticasone propionate

Editor—Bond et al said that nasal fluticasone propionate caused benign intracranial hypertension in a 13 year old boy with a history of Crohn's disease and subsequently reported this to the Committee on Safety of Medicines.[1] We have numerous problems with this hypothesis. Firstly, Bond et al did not confirm the diagnosis of intracranial hypertension as the cerebrospinal fluid pressure, which should be markedly raised, was not measured on any occasion.[2] Their conclusions can thus at best be based only on papilloedema, headache, and backache.

The boy was seen by specialists from the ear, nose, and throat department, but no mention was made of the presence or absence of the otological manifestations, which include objective pulsatile tinnitus and low frequency hearing loss, which can be the major or only manifestation of this syndrome.[3] Other recognised associated conditions were not excluded, such as hypervitaminosis A, systemic lupus erythromatosis, hypothyroidism and its correction, and malnutrition and renutrition, which is not unheard of in patients with Crohn's disease; the patient was in remission, so both factors could have been at work.[4]

We have to assume that this teenage patient was not receiving any other drugs such as tetracyclines or isotretinoin (commonly used in the treatment of acne in adolescents), which also have been implicated in the development of benign intracranial hypertension. The condition tends to be self limiting, with a course of less than 12 months in most cases and recurrence in 10%.[5] We were therefore concerned that the temporal relation that Bond et al describe may just be the normal course for the condition. It is paradoxical that steroids are implicated as an aetiological factor and are also an accepted treatment. Would Bond et al suggest that nasal steroids could in different circumstances be a useful treatment?

In these days of evidence based medicine there are several tests of causation that a hypothesis or proposal should be subjected to, and Bond et al have not done this. All clinicians should be observant of adverse drug reactions, with particular reference to topical nasal steroids and children.

Michael Oko *specialist registrar in otolaryngology*
cybersurgeon@moko.freeserve.co.uk

Andrew Johnston *consultant otolaryngologist*
Monklands Hospital, Monkscourt Avenue, Airdrie ML6 OJS

Iain R C Swan *senior lecturer in otolaryngology*
Glasgow Royal Infirmary, Glasgow G31 2ER

1 Bond DW, Charlton CPJ. Benign intracranial hypertension secondary to nasal fluticasone propionate. *BMJ* 2001;322:897. (14 April.)
2 Susman JL. Benign intracranial hypertension. *J Fam Pract* 1990;30:290-2.
3 Sismanis A. Otologic manifestations of benign intracranial hypertension syndrome: diagnosis and management. *Laryngoscope* 1987;97(suppl 42):1-17.
4 Lessell S. Pediatric pseudotumor cerebri (idiopathic intracranial hypertension). *Surv Ophthalmol* 1992;37:155-66.
5 Cody CM. Benign intracranial hypertension. *Am Fam Physician* 1992;45.

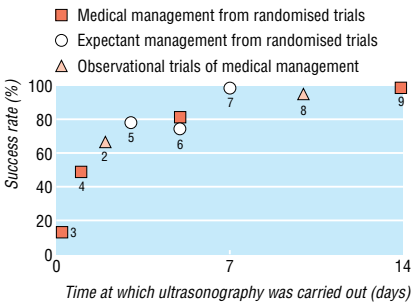
Ultrasonography may have role in assessing spontaneous miscarriage

Editor—Ankum et al's regular review on the management of spontaneous miscarriage suggests that medical management is of little benefit in the treatment of incomplete miscarriage.[1] The wide variation in reported success rates might, however, be responsible for differences in the use of ultrasonography.

The use of ultrasonography to determine whether there are retained products of conception will exclude about 30% of women from treatment as they will be found to have an empty uterus.[2] If ultrasonography is not used at this stage the success rate will be inflated by the inclusion of women having unnecessary treatment. Conversely, the use of ultrasonography to assess completeness after uterine evacuation may reduce the apparent success rates: the finding of intrauterine tissue usually leads to an assumption that the treatment has failed. The clinical course of this finding, however, is unknown, and most of these women will probably complete the spontaneous miscarriage without further intervention.

Analysis of the available studies shows a clear relation between the reported success rates and the time at which the ultrasonography was carried out after treatment (figure). The analysis suggests that the low reported success rates in some studies occurred as a result of an over-reaction to ultrasonographic findings that are of undetermined relevance. Initial medical treatment followed by expectant management may be the key to the effective management of incomplete miscarriages. These findings also add weight to the arguments in favour of using expectant management alone. These two regimens need to be compared.

The figure may represent the natural course of incomplete miscarriages, but this needs to be confirmed in a prospective ultrasonographic study of women having expectant management.



Comparison of reported success rates with time at which ultrasonography was carried out after treatment. Numbers refer to references

Andrew Weeks *lecturer in obstetrics and gynaecology*
Department of Obstetrics and Gynaecology, Makerere University, PO Box 7072, Kampala, Uganda
aweeks@doctors.org.uk

Godfrey Alia *senior house officer in obstetrics and gynaecology*
Mulago Hospital, Kampala

1 Ankum WM, de Waard MW, Bindels PJE. Management of spontaneous miscarriage in the first trimester: an example of putting informed shared decision making into practice. *BMJ* 2001;322:1343-6. (2 June.)
2 Cheung T, Leung P, Cheung LP, Haines C, Chang AMZ. A medical approach to management of spontaneous abortion using misoprostol. *Acta Obstet Gynecol Scand* 1997;76:248-51.
3 De Jonge ETM, Makim JD, Manefeldt E, De Wet GH, Pattinson RC. Randomised clinical trial of medical evacuation and surgical curettage for incomplete miscarriage. *BMJ* 1995;311:662.
4 Cheung TKH, Lee DTS, Cheung LP, Haines CJ, Chang AMZ. Spontaneous abortion: a randomized, controlled trial comparing surgical evacuation with conservative management using misoprostol. *Fertil Steril* 1999;71:1054-9.
5 Nielsen S, Hahlin M. Expectant management of first trimester spontaneous abortion. *Lancet* 1995;345:84-6.
6 Nielsen S, Hahlin M, Platz CJ. Randomised trial comparing expectant with medical management for first trimester miscarriages. *Br J Obstet Gynaecol* 1999;106:804-7.
7 Chipchase J, James D. Randomised trial of expectant versus surgical management of spontaneous miscarriage. *Br J Obstet Gynaecol* 1997;104:840-1.
8 Henshaw RC, Cooper K, El-Rafaey H, Smith NC, Templeton AA. Medical management of miscarriage: non-surgical uterine evacuation of incomplete and inevitable spontaneous abortion. *BMJ* 1993;306:894-5.
9 Hinshaw K. Medical uterine evacuation in the management of first trimester miscarriage. In: Prendiville W, Scott JR, eds. *Clinical management of early pregnancy*. London: Arnold, 1999:79-89.

Islam with the internet could do much to prevent disease

Editor—Modern public health has paid little attention to prevention tailored towards Muslims even though the Islamic population now numbers over 1 billion. Religion is a major component of the social life of many communities throughout the world, but in disease prevention we often use the concept of "one size fits all," with little recognition of religion or culture. Religion's positive influences can be incorporated into a strategy for health promotion and disease prevention by using recent developments in information technology.[1]

Our goal is to provide access to scientifically sound and culturally acceptable information on health promotion and disease prevention to all who need it, using the information superhighway. This resource will have a major role in the accumulation and dissemination of information to those who need it the most.[2]

Islam as a religion puts a considerable emphasis on health, and a wealth of scientifically sound information on different health issues is provided.[3] We have started to develop a network of professionals interested in Islam, the internet, and disease prevention from all parts of the world. This network is open to anyone interested in exploring the wealth of information that religious sources have on mental, physical, and social health and can be accessed from the homepage of the Islamic Global Health Network (http://islamicprevention.homestead.com/).

To improve global health we need to harness the new technology of the internet, as the largest factor in prevention is information sharing. Less than 5% of the world is connected to the internet, but Islam—like most religions—is good at reaching the poor, who have the highest disease burden. The internet alone cannot reach across the health and digital gap but when connected with Islam and other religions it could have a powerful effect in disseminating the most potent prevention systems worldwide.

An internet based Islamic supercourse (www.pitt.edu/~super1/ighn.htm) includes a variety of lectures on Islam and health and other topics. Since this is an exploration into the impact of religion on our holistic concept of health, research into this area will be important for all religions. We invite professionals of all faiths to join this global community.

Abdullatif Husseini *coordinator*
Epidemiology Unit, Institute of Community and Public Health, Birzeit University, PO Box 14, Birzeit, Palestine
islamicprevention@hotmail.com

Ronald E LaPorte *director*
Disease Monitoring and Telecommunications, WHO Collaborating Center, University of Pittsburgh, Pittsburgh, PA 15213, USA

This letter was prepared by Abdullatif Husseini and Ronald E LaPorte on behalf of members of Islamic Global Health Network (http://islamicprevention.homestead.com/).

1 LaPorte R, Randolph L, Sauer F, Sekikawa A, Aaron D. Information dominance over disease. *Lancet* 2000;355:1370-1.
2 LaPorte R, Akazawa S, Hellmonds P, Boostrom E, Gamboa C, Gooch T, et al. Global public health and the information superhighway. *BMJ* 1994;308:1651-2.
3 Al-Khayat M. *Health: an Islamic perspective.* Alexandria: World Health Organization Eastern Mediterranean Regional Office, 1997.

Removal of thioridazine from primary care formulary will result in prescribing vacuum

Editor—Recently the Committee on the Safety of Medicines recommended that general practitioners should not prescribe thioridazine.[1] It recommended that prescribing should be limited to second line treatment in schizophrenia and that prescription should be initiated only by a consultant psychiatrist. The reason was that the drug was thought to be a cause of very rare cardiac deaths by widening the QT interval in an electrocardiogram, leading to a greater risk of developing arrhythmias.

The evidence base to substantiate this decision is not, however, sufficient. Thioridazine is the most commonly prescribed phenothiazine in primary care.[2] Its implication in very rare deaths would not be substantiated if number of deaths were directly standardised to volume of phenothiazine prescribed. In 1997 the Royal College of Psychiatrists produced a council report on the association between phenothiazines and sudden death.[3] The report showed that sudden death was associated with all the phenothiazines prescribed in primary care. The phenothiazine associated with most deaths was not thioridazine but chlorpromazine. The report documented that abnormalities in an electrocardiogram are comparatively common in people receiving neuroleptics, occurring in around 25%. It also stated that such changes are commonly considered benign; even now consensus on the clinical significance of prolonged QT_c is lacking.

Removing thioridazine from the primary care formulary will lead to a prescribing vacuum, and it is unfortunate that more thought has not been given to which drugs will fill it. As thioridazine has become an adequate substitute for the prescribing of benzodiazepines in conditions of anxiety and irritability, its removal from the primary care formulary will increase the probability of benzodiazepines being overprescribed in primary care. Perhaps the Committee on the Safety of Medicines should have stood by its original advice in 1996 when it suggested that, whenever possible, drugs associated with QT interval prolongation should be avoided in patients who have underlying cardiac disease.[4]

Perhaps the committee singled out thioridazine as a result of the work by Reilly et al, who measured a prolonged QT interval in 495 patients.[5] The cohort had been prescribed a wide variety of psychotropic drugs, and no single drug was immune from being associated with a widening of the QT interval. The paper concluded that QT interval abnormalities are surrogate markers, and the link between psychotropic drugs, arrhythmia, and sudden death should be examined. It is unfortunate that this recommendation was not explored before the the Committee on the Safety of Medicines advised the removal of thioridazine from the primary care formulary.

Nat Wright *general practice consultant in substance misuse and homelessness*
No Fixed Abode Health Centre for Homeless People, Leeds LS9 8AA
nat.wright@virgin.net

1 Committee on the Safety of Medicines. *Thioridazine: restricted indications and new warnings on cardiotoxicity.* London: CSM, 2000.
2 Prescription Pricing Authority. *PACT standard report.* Newcastle upon Tyne: PPA, 1999.
3 Royal College of Psychiatrists. *The association between antipsychotic drugs and sudden death. Council Report CR57.* London: RCP, 1997.
4 Committee on the Safety of Medicines. Drug-induced prolongation of the QT interval. *Curr Probl Pharmacovigilance* 1996;22:2.
5 Reilly JG, Ayis S, Ferrier I, Jones S, Thomas S. QT_c-interval abnormalities and psychotropic drug therapy in psychiatric patients. *Lancet* 2000;355:1048-52.

"Coronary heart disease" is not tautologous

Editor—Gilroy repeats a common misconception in suggesting that the term "coronary heart disease" is a new term and that it is a tautology.[1] It is an established North American term, to be preferred to the British synonym "ischaemic heart disease," which dominates recent editions of the *International Classification of Diseases*.[2] Ischaemic is imprecise and could, as Friedberg pointed out 35 years ago, apply equally well to heart disease secondary to stenosis of the aortic valve, or even to anaemia.[3] "Coronary" does not mean cardiac but "resembling, or encircling, like a crown."[4] It was the 17th century anatomists' descriptive name for the arterial pattern around the heart.

Coronary artery disease is used, confusingly, to mean pathological findings confined within the coronary arteries. These changes are present in almost all adults and are usually symptomless. It is only when changes to the coronary arteries are severe enough to affect the myocardium, to cause coronary heart disease, that patients are truly diseased—from angina, myocardial infarction, sudden death, or heart failure. Coronary heart disease means that the heart as an organ is involved, necessitating secondary prevention, whereas we all have coronary artery disease for decades beforehand and try to slow its progression to heart disease by primary prevention. Heart disease and heart attacks are too non-specific as terms to get by without qualification. Twenty five years ago I tried to introduce "coronary heart attack" as a generic term to cover both cardiac infarction and coronary deaths (often unassociated with infarction), but without success.[5]

Hugh Tunstall-Pedoe *professsor of cardiovascular epidemiology*
Cardiovascular Epidemiology Unit, Ninewells Hospital and Medical School, University of Dundee, Dundee DD1 9SY
h.tunstallpedoe@dundee.ac.uk

1 Gilroy F. Tautology, or not tautology. *BMJ* 2001;322:1606. (30 June.)
2 World Health Organization. *International statistical classification of diseases and related health problems.* 10th revision. Geneva: WHO, 1992.
3 Friedberg CK. *Diseases of the heart.* 3rd ed. Philadelphia: Saunders, 1966:643.
4 *Concise Oxford Dictionary.* 7th ed. Oxford: Oxford University Press, 1982.
5 Tunstall-Pedoe H, Clayton D, Morris JN, Brigden W, MacDonald L. Coronary heart attacks in east London. *Lancet* 1975;ii:833-8.