

standards in intensive care units, as Atkinson and Bihari point out.

P. BADRINATH

Department of Community Medicine,
Kasturba Medical College,
Manipal-576 119,
South India

- 1 Atkinson SW, Bihari DJ. Selective decontamination of the gut. *BMJ* 1993;306:286-7. (30 January.)
- 2 Sacks HS, Berrier J, Reitman D, Ancona-Berk VA, Chalmers TC. Meta-analysis of randomized controlled trials. *N Engl J Med* 1987;316:450-5.
- 3 Vandembroucke-Grauls CMJE, Vandembroucke JJ. Effect of selective decontamination of the digestive tract on respiratory tract infections and mortality in the intensive care unit. *Lancet* 1991;333:859-62.

Gestational diabetes mellitus

EDITOR,—Several correspondents^{1,2} have commented on my review of gestational diabetes.³ Michael Maresh¹ chides me for not quoting the multicentre study by the European Diabetes Pregnancy Study Group.⁴ This was deliberate as it is difficult to know what to make of a study whose results differ so much depending on whether glucose concentrations were measured in venous plasma or capillary whole blood. Even so, I might have quoted the finding that pregnant women with two hour glucose concentrations (by either method) exceeding 8 mmol/l were on average older and fatter—as might be expected—but had a mean duration of pregnancy and gave birth to infants with mean weights no different from those of women with lower blood glucose concentrations.

Maresh also claims that the St Mary's study⁵ showed perinatal morbidity in association with glucose intolerance. In fact, the women with glucose intolerance were known to their attendants, were on average 5 kg heavier than their controls, were treated with insulin in two thirds of cases, and had a rate of caesarean section of 22% compared with 14% in the controls. In the absence of an untreated glucose intolerant group it is impossible to apportion responsibility for the morbidities (mostly trivial) among obesity, glucose intolerance, and iatrogenesis.

P S Sharp and colleagues seem to think that I am a stony hearted epidemiologist,¹ whereas I am a clinician fallen among epidemiologists. Epidemiologists, however, like public health colleagues, seem to be more aware of the ethical issues concerning screening: many clinicians cannot bear to think that their warm hearted actions are at best useless and at worst harmful.

Lindsay Edouard and Roland F Dyck,¹ like Craig McBride and colleagues,² draw attention to ethnic groups with relatively high rates of diabetes even in the reproductive years. There may be a case for screening for diabetes during pregnancy in these populations, but valid screening methods need to be developed. Unless some overriding reason for confining screening to pregnancy exists, non-pregnant women and even men might reasonably demand the same "benefit."

I thank Edouard and Dyck for drawing my attention to the Canadian Task Force review, which broadly agrees with my views and also answers the point made by Maresh. "It can be argued that such women could be offered anticipatory counselling to prevent the occurrence of overt diabetes. However, nothing more than good nutritional advice, which should be directed to all women, would be given, and most of the women would be unnecessarily worried and potentially penalised in future life insurance."³

R J JARRETT

London SE26 4PA

- 1 Correspondence. Gestational diabetes mellitus. *BMJ* 1993;306:581-2. (27 February.)
- 2 McBride C, Roberts A, Knox A, Cundy T. Screening for diabetes during pregnancy. *BMJ* 1993;306:858. (27 March.)

- 3 Jarrett RJ. Gestational diabetes mellitus: a non-entity? *BMJ* 1993;306:37-8. (2 January.)
- 4 Lind TA. Prospective multicentre study to determine the influence of pregnancy upon the 75 g oral glucose tolerance test (OGTT). In: Sutherland HW, Stowers JM, Pearson DWM, eds. *Carbohydrate metabolism in pregnancy and the newborn*. Heidelberg: Springer Verlag, 1989:209-26.
- 5 Maresh M, Beard RW, Bray CS, Elkeles RS, Wadsworth J. Factors predisposing to and outcome of gestational diabetes. *Obstet Gynaecol* 1989;74:342-6.
- 6 Canadian Task Force on the Periodic Health Examination. Periodic health examination, 1992 update. 1. Screening for gestational diabetes mellitus. *Can Med Assoc J* 1992;147:435-43.

Prenatal diagnosis of late onset diseases

EDITOR,—The discovery of genetic loci strictly associated with certain kinds of familial motor neurone disease¹ has again raised the prospect of prenatal diagnostic tests for affected fetuses in such relatively late onset conditions.

Apart from the ethical issues conventionally discussed in relation to decisions to test prenatally, further major factors are involved in consideration of late onset conditions. Not only is there likely to be a lengthy period of life which is generally asymptomatic for the disease concerned, but with late onset there is an increased probability that the person will die from other unrelated causes.² At advanced ages only a small proportion of susceptible people may have symptoms or will die from the specific condition for which prenatal diagnosis might be undertaken.³

In addition, the mean life expectancy of such individuals with certain late onset conditions is not far short of that of the population as a whole.⁴ Therefore the quality and type of death becomes as significant as its timing in the adjudication between actions that may allow a "good" as opposed to a "bad" death in susceptible older people.

These issues must be further considered before prenatal diagnostic tests are recommended for the increasing range of late onset conditions known to have a significant genetic aetiological component.

IAN ROBINSON
STUART NEILSON

John Bevan MND Research Unit,
Department of Human Sciences,
Brunel, The University of West London,
Uxbridge,
Middlesex UB8 3PH

- 1 Rosen D, Siddique T, Patterson D, Figlewicz DA, Sapp P, Heutani A, et al. Mutations in Cu/Zn superoxide dismutase gene are associated with familial amyotrophic lateral sclerosis. *Nature* 1993;362:59-62.
- 2 Neilson S, Robinson I, Hunter M. Longitudinal Gompertzian analysis of ALS mortality in England and Wales, 1963-1990: estimates of susceptibility in the general population. *Mech Ageing Dev* 1992;64:201-16.
- 3 Neilson S, Robinson I, Hunter M. Static and dynamic models of inter-disease competition: past and projected mortality from amyotrophic lateral sclerosis and multiple sclerosis. *Mech Ageing Dev* 1993;66:223-41.
- 4 Neilson S, Robinson I, Clifford Rose F, Hunter M. Rising mortality from motor neurone disease—an explanation. *Acta Neurol Scand* 1993;87:184-91.

Infertility linked to breast cancer

EDITOR,—Boukje M Zaadstra and colleagues¹ paper reporting an association between an increased waist:hip ratio and decreased conception rates¹ raises three questions: Do these results apply to women attempting to conceive naturally? Is luteinising hormone positively associated with waist:hip ratio? And does decreased conception account for our finding that women with a high waist:hip ratio have an increased risk of breast cancer?²

We previously reported that in a large population sample of postmenopausal women the waist:hip

ratio was positively associated with infertility, irregularity of the menstrual cycle, and late age at first live birth³—for example, the mean waist:hip ratio of women with no history of infertility was 0.838 compared with 0.841 for women with a history of infertility, adjusted for age and body mass index. In a subset of women luteinising hormone concentration was correlated negatively, not positively, with waist:hip ratio ($r = -0.35$);⁴ of course, this finding in postmenopausal women may not apply to women of childbearing age.

We recently reanalysed our data on breast cancer and found that infertility is a risk factor for breast cancer in older women⁵; moreover, in statistical models we have confirmed that infertility partly explains the association of waist:hip ratio with breast cancer. Further work is needed on the interrelations of abdominal adiposity, reproductive hormones, and risk of breast cancer.

AARON R FOLSOM
THOMAS A SELLERS
SUSAN A KAYE

Division of Epidemiology,
School of Public Health,
University of Minnesota,
Minneapolis, MN 55454-1015,
USA

- 1 Zaadstra BM, Seidell JC, Van Noord PAH, te Velde ER, Habbema JDF, Vrieswijk B, et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *BMJ* 1993;306:484-7. (20 February.)
- 2 Sellers TA, Kushi LH, Potter JD, Kaye SA, Nelson CL, McGovern PG, et al. Effect of family history, body fat distribution, and reproductive factors on risk of postmenopausal breast cancer. *N Engl J Med* 1992;326:1323-9.
- 3 Kaye SA, Folsom AR, Prineas RJ, Potter JD, Gapstur SM. The association of body fat distribution with lifestyle and reproductive factors in a population study of postmenopausal women. *Int J Obes* 1990;14:583-91.
- 4 Kaye SA, Folsom AR, Soler JT, Prineas RJ, Potter JD. Associations of body mass and fat distribution with sex hormone concentrations in postmenopausal women. *Int J Epidemiol* 1991;20:151-6.
- 5 Sellers TA, Potter JD, Severson RK, Bostick RM, Nelson CL, Kushi LH, et al. Difficulty becoming pregnant and family history as interactive risk factors for postmenopausal breast cancer. The Iowa women's health study. *Cancer Causes Control* 1993;4:21-8.

Clinical diagnosis of pyloric stenosis

EDITOR,—Joseph Macdessi and R Kim Oates highlight the importance of performing a careful abdominal examination as a first assessment to detect hypertrophic pyloric stenosis.¹ This condition should be diagnosed clinically but the presentation does not always allow the diagnosis to be made on initial abdominal palpation. Infantile pyloric stenosis is an acquired condition,² and hence the emerging tumour evolves at differing rates. The tumour should be palpated in all cases, but this may be difficult, particularly with slowly evolving tumours, which may take several days from the onset of symptoms to develop.

In a recent prospective study of 100 consecutive infants presenting with persistent vomiting, abdominal examination elicited a palpable tumour in 38 infants.³ Ultrasound imaging with use of the criteria for diagnosing pyloric stenosis^{4,5} showed the condition in these 38 infants and in six others; all 44 were subsequently documented as having pyloric stenosis at surgery. Diagnosis was hence made in 86% of the infants by clinical examination while ultrasound imaging was 100% accurate, with no false positive results. On reviewing those infants who did not have a palpable tumour but in whom ultrasound imaging gave a positive result we found that the dimensions of their pyloric muscle were significantly smaller than those in the infants in whom both abdominal palpation and ultrasound imaging gave a positive result. No moderate or large pyloric tumours were missed by clinical examination, but smaller degrees of muscle hypertrophy indicating early pyloric stenosis were,