suggestion of Körner[1] that Homer described the first case in history in the *Odyssey*[2] where Ares and Aphrodite are described as not being able to separate after illicit intercourse.

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[1] Körner, O, *Die ärtzlichen Kentnisse in Ilias and Odyssee*, München, Bergmannverlag, 1929.
[2] *Odyssey*, **8**, 267-361.

Antenatal prediction of fetal sex

SIR,—I would like to report another interesting case. In 1978 a multiparous woman who had previously given birth to a male child with X-linked muscular dystrophy had an amniocentesis during her third pregnancy and the fetal karyotype was 46,XY. As this fetus was apparently a male, with an even chance of being afflicted with muscular dystrophy, and the mother was unprepared to accept the risk, the pregnancy was terminated. The fetus delivered was phenotypically female but further investigations revealed testicular feminisation syndrome to be the cause.

It is certainly a strange coincidence that the parents are possibly at risk of yet another X-linked condition, but I am pleased to report that there has been a further pregnancy with a happy ending, with the birth of a normal girl this year.

I am grateful to Dr Marina Seabright, regional department of cytogenetics at the Salisbury group of hospitals, and Dr Martin Bobrow, department of medical genetics at Oxford, for their expert help.

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Screening for thyroid dysfunction in diabetics

SIR,—We were interested to read Drs John Feeley and T E Isles's report on the prevalence of thyroid dysfunction in diabetics (23 June, p 1678) and have recently completed a similar study on 347 randomly selected diabetics with no previous history of thyroid disease.[1]

We too have found the prevalence of primary thyroid failure as indicated by a raised serum thyrotrophin concentration to be surprisingly high at 10%, but, in contrast to Drs Feeley and Isles, showed that insulin-dependent diabetics accounted for the majority of patients with thyroid failure, of whom 13% had a raised serum thyrotrophin concentration, usually accompanied by thyroid microsomal antibodies (88%). Most (83%) of the insulin-dependent diabetics with raised serum thyrotrophin were aged over 50 years. Only 6% of insulin-independent diabetics of a similar age distribution had a raised thyrotrophin concentration, of whom 60% had thyroid microsomal antibodies. The higher prevalence of an elevated thyrotrophin concentration in insulin-dependent than in insulin-independent diabetics is in accordance with the prevalence of thyroid microsomal antibodies in these two conditions.[2]

Interpretation of Drs Feeley and Isles's findings would be helped by provision of details of serum thyrotrophin concentrations in patients of group 2, in view of the low upper limit of their normal range (4·5 mU/l). How many patients in group 2 had a serum thyrotrophin concentration greater than 10 mU/l? The age distribution of the three groups of patients categorised on the basis of diabetic treatment is also omitted and would probably indicate that the insulin-dependent are considerably younger than the insulin-independent diabetics. Furthermore, the criteria for selecting patients to be studied are unclear. Patients who have been treated for hypothyroidism or who have been previously treated for hyperthyroidism must surely be "suspected of having thyroid disorders."

Some of the patients in group 3 may have underlying renal insufficiency to account for a low free thyroxine index without coincident elevation of serum thyrotrophin or the presence of thyroid microsomal antibodies. Many patients in group 2 who have an elevated thyrotrophin concentration in the absence of microsomal antibodies (which we uncommonly saw) may be found at follow-up to have normal thyrotrophin concentrations.[3]

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[1] Gray, R S, *et al*, *Journal of Clinical and Laboratory Immunology*, 1979, **2**, 221.
[2] Irvine, W J, *et al*, *Lancet*, 1970, **2**, 163.
[3] Tunbridge, W M G, and Clark, F, *Annales d'Endocrinologie*, 1978, **39**, abstract 115.

**We sent a copy of this letter to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—The recent study by Dr R S Gray and others[1] supports our contention that screening for thyroid dysfunction in a diabetic population is worthwhile. Their main findings (almost 3% prevalence of previously unrecognised mild clinical hypothyroidism and a 7% at-risk group) are similar to ours but occur in different groups of patients. Many of the apparent discrepancies may be explained by the differences in the diabetic populations studied.

In the Edinburgh study 53% (and almost half of the over-50 group) of diabetics were treated with insulin as opposed to 43% in our study, where the insulin-dependent patients were considerably younger than our insulin-independent group. In the study by Gray *et al* the population was evenly divided between the sexes, whereas 62% of our population were female. More important, our diabetics were on average eight years older and there is evidence[2] that the prevalence of thyroid antibodies in insulin-dependent diabetics decreases with advanced years. Undoubtedly some of our elderly diabetics would have better physiological blood sugar control on insulin (and thus become classified as insulin-dependent) but the benefits of such therapy for this age group remains to be established.

The upper limit of our normal serum thyrotrophin (TSH) range (4·5 mU/l) is based on 350 control subjects, and although this is not greatly different from the Edinburgh limit of 5·7 mU/l it must be stressed that TSH values are not normally distributed but skewed to the right; thus mild elevation of TSH may reflect suboptimal thyroid function. In group 2 of our study 11 patients had TSH levels greater than 10 mU/l. We agree that TSH levels may vary from time to time and we stressed that it is those patients with both raised TSH and antibodies who are at particular risk of developing hypothyroidism. Our patients were selected as not suspected of having overt thyroid disorders and we thus included patients who had previously been treated for thyroid disease. Similarly Dr Gray and his colleagues, although selecting patients with no previous history of thyroid disease, must have suspected some thyroid disease in the insulin-dependent population, where they had previously shown thyroid antibodies occurring commonly.[3] We feel that patients with previous thyroid disorders should be included in a screening programme and that it is both unwise and impractical to exclude such groups.

It has long been recognised that insulin-dependent diabetics are at risk of developing thyroid disorders. Our screening has also identified elderly women (in part reflecting this group's propensity to develop hypothyroidism in the non-diabetic community), whether insulin-dependent or insulin-independent, as an additional at-risk group.

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[1] Gray, R S, *et al*, *Journal of Clinical and Laboratory Immunology*, 1979, **2**, 221.
[2] Whittingham, S, *et al*, *Lancet*, 1971, **1**, 763.
[3] Irvine, W J, *et al*, *Lancet*, 1970, **2**, 163.

Diagnosis of multiple pregnancy

SIR,—Mr G J Jarvis (8 September, p 593) considers in his summary that two ultrasonic examinations are necessary to confirm the clinical suspicion of multiple gestations, but in the text he suggests that two ultrasonic examinations *may* be necessary. He also states that since 1964 little has been reported on problems about diagnosing twins, and that no published reports state the percentage of twins diagnosed at various stages of the antenatal period. Taylor[1] and Sundén[2] reported ultrasound to be a good method of detecting twins in selected populations—that is, in the patients referred for screening as clinically suspected of carrying twins.

Since 1973 ultrasonic routine screening has been included in the antenatal care of all pregnant women attending the department of obstetrics and gynaecology at the University Hospital, Malmö.[3] Of 155 examined twin pregnancies from 1973 to 30 September 1979, two were mistakenly interpreted as singletons at the first screening (the faulty diagnosis being made by untrained operators in the beginning of the screening period). No false-positive diagnoses were made. This makes a methodological error rate of 1·3% compared with 21% (17/83) reported by Dr Jarvis. During the last two and a half years we have detected all twin pregnancies (54) by a single ultrasound examination in the 17th week. Thus our results do not support the apprehension of (among others) Powers[4] that accuracy might be reduced if these techniques were applied to a general population of pregnant women.

Since 1964 many authors have reported on physical[3-6] and biochemical methods[5 7-9] to detect twins. I reported in 1976 that the introduction of ultrasonic screening of pregnant women in Malmö increased the antepartum detection of multiple pregnancy and dimin-