ciate that the question posed could only be determined by means of an enlarged study.

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¹ Armitage, P, *Statistical Methods in Medical Research*.
Oxford, Blackwell, 1971.

Ten-minute abortions

SIR,—Mr W O Goldthorp (27 August, p 562) is correct when he says that the law on abortion in Britain refers to acts intended to interrupt pregnancy, whether or not a pregnancy exists. I submit, therefore, that he should notify the Chief Medical Officer after *all* his aspirations, perhaps using the complications section of the form to indicate if a woman was not pregnant.

If it becomes a common practice to withhold notification of abortions when there is no histological proof of the products of conception it will not be long before the diagnosis of pregnancy is ignored both after as well as before the procedure. This would produce both ethical and statistical confusion.

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Hyperglycaemia and complications of diabetes

SIR,—On the basis of their very impressive finding of a relationship, in rats with pancreatic islets damaged by streptozotocin, between mean plasma glucose value and renal basement membrane thickness Dr C J Fox and his colleagues (3 September, p 605) hope that doctors treating diabetics will be encouraged to pursue good control of glucose levels. This wish is strengthened by results from the Oxford diabetic clinic on patients observed for the first three years after diagnosis. Vibration sense was assessed with a biothesiometer and recorded as the sum of the mean of three readings at each of four sites, the two halluces and the two external malleoli. The results, now submitted for publication elsewhere, are summarised in the accompanying table. Although vibration sense did not change significantly over the three years when calculated for the whole group, it deteriorated in those with the higher plasma glucose values and improved in those with the lower values.

We had the advantage of dealing with natural human diabetes, here of "maturity-type onset," which certainly involves islet-cell deficiency but may not be exactly comparable in effect to streptozotocin-induced

damage, but at a disadvantage in that the two groups were not comparable initially in plasma glucose concentration, for those with higher mean fasting glucose levels tended to have higher values before treatment started. It is not surprising that those in this group were also much more likely to be treated with sulphonylureas, but this is comparable to administration of insulin to some rats in the experiments reported by Dr Fox and his colleagues.

We have examined a type of diabetic tissue damage different from the rat's renal micro-angiography, but large-nerve damage also causes much morbidity. It is most encouraging that Dr Fox and his colleagues have obtained a similar result from the speed of conduction in motor nerve fibres.

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Deafness due to Paget's disease of bone

SIR,—The involvement of the labyrinthine capsule of the inner ear by Paget's disease seems to be largely responsible for the severe sensorineural deafness that may occur. Treatment with calcitonin aims to retard and if possible arrest this process in the active phase in the same way that fluoride therapy is used for labyrinthine otosclerosis. The value of these protracted forms of treatment is uncertain, but the most recent report, by Dr L R Solomon and others (20 August, p 485), is favourable. They based the selection of patients for treatment on typical audiological changes and radiological evidence of Paget's disease of the skull but did not do temporal bone tomography "because of the risks to the eye."

This is an incredible statement, which presumably refers to the radiation dose to the eyes during temporal bone tomography. A previous publication from Manchester¹ showed a mean corneal radiation dose of 10 rads during temporal bone tomography, which was greatly reduced by using lead eyeshields. However, patients undergoing extensive neuroradiological procedures, including computerised tomography, may receive a cumulative corneal dose in excess of 60 rads. If lead eyeshields are used or tomography is done with the patient in the prone position the total radiation dose to the eyes may be less than that delivered for one Towne's view.²

In otosclerosis demineralised foci with blurring and thickening of the labyrinthine capsule are well shown by multidirectional tomography. The tomographic appearances are therefore used to select patients for fluoride therapy and then to assess the progress of the disease and the effect of treatment.³ In Paget's disease similar demineralisation and thickening of the labyrinthine capsule may be demonstrated as well as the more common narrowing

and distortion of the internal auditory meatus which may also be responsible for sensorineural deafness. In my opinion therefore tomography, while being no danger to the eyes, is necessary to show the presence and extent of Paget's disease of the temporal bone. This information, with the audiometric results and repeat tomograms to assess progress, would enhance the value of any survey of the use of calcitonin in deafness due to Paget's disease.

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¹ Isherwood, I, *et al*, *Neuroradiology*, 1975, **10**, 137.

² Ashley, A J, *Radiography*. In press.

³ Shambaugh, G E, and Causse, J, *Annals of Otolaryngology and Rhinology*, 1974, **83**, 635.

Antilipolytic effects of beta-blocking drugs

SIR,—I was interested to read the paper by Dr R J Newman (3 September, p 601) reporting the acute antilipolytic effects of acebutolol, metoprolol, and propranolol. Predicting the expected antilipolytic effect of β -adrenoceptor blocking drugs is difficult, for the precise physiological mechanisms controlling lipolysis have not yet been defined. In a similar study¹ I have suggested that basal lipolysis is mediated by β_1 -adrenoceptors and that stress-induced lipolysis is mediated by both β_1 - and β_2 -adrenoceptors. This is supported by studies showing that atenolol is as effective as propranolol in lowering basal plasma non-esterified fatty acid (NEFA) concentrations¹ and that, while atenolol¹ and practolol² reduce stress-induced lipolysis, they are not as effective as oxprenolol³ or propranolol.⁴ However, it is uncertain whether lipolysis is mediated by an increase in circulating catecholamines or by increased sympathetic nervous activity, or both. Other pharmacological properties of β -blocking drugs may influence their metabolic effects, and Dr Newman's general comments are open to criticism for he neglects to discuss the possible effects of relative β -adrenoceptor specificity, intrinsic sympathomimetic activity, and water-lipid solubility and also the possibility that more than one adrenergic mechanism may mediate lipolysis. For instance, pindolol, a non-selective β -blocker possessing intrinsic sympathomimetic activity, has been shown to produce a small but significant rise in basal plasma NEFA⁵ and not to affect the plasma NEFA response to insulin stress.⁶

While most β -blocking drugs do seem to have some acute antilipolytic effect, the effect of chronic administration might be different. Tanaka *et al*⁷ have shown that although propranolol initially lowered plasma NEFA levels, several weeks later increased levels were observed. Similarly, Waal-Manning⁸ had reported that raised triglyceride levels occurred following three months' administration of metoprolol, although Nilsson *et al*⁹ in a controlled study with metoprolol showed no significant effect. Likewise, Ghosh *et al*¹⁰ have shown long-term practolol therapy to have no significant lipolytic effects. It seems therefore that the net in-vivo lipolytic effects of β -blocking drugs may be dependent upon several pharmacological properties and there may be individual variation. Studies in normal subjects may not be applicable to patients, especially those with disorders of lipid metabolism, and as I suggested in a recent

Biothesiometer readings (mean $\pm$ SEM) as sum of mean of readings at four separate sites on feet (high value indicates insensitivity)

	Initial value	Change at 1 year	Change at 3 years
All diabetics (n = 55)	118 $\pm$ 6	+ 3 $\pm$ 5	- 1 $\pm$ 3
Diabetics with mean fasting glucose $\leq$ 8.5 mmol/l ($\leq$ 153 mg/100 ml) (n = 33)	114 $\pm$ 5	+ 1 $\pm$ 6	- 9 $\pm$ 5*
Diabetics with mean fasting glucose $>$ 8.5 mmol/l (n = 22)	124 $\pm$ 8	+ 6 $\pm$ 9	+ 12 $\pm$ 5*

*Difference between means, $P < 0.005$