The only feasible approach to this problem would seem to be a national survey such as the one conducted at the request of the Advisory Committee on Obstetrics and Gynecology of the Food and Drug Administration among the Fellows of the American College of Obstetricians and Gynecologists.[2] The response in this survey was good (76%) and it should be particularly noted that the Fellows were asked to report not just on their own experience but on any patient who died or was critically ill in a hospital in their community. As stated by Dr. Hawkins in his letter, 10 deaths associated with pelvic inflammatory disease and 12 non-fatal cases of intestinal obstruction following perforation were reported. The number of intrauterine devices inserted in the United States prior to the time of the survey (mid-1967) is not known, but it is known that the total number distributed by January 1968 was approximately three million.

The insertion and use of an I.U.D. are associated with a definite, albeit small, risk to life and health. The magnitude of this risk should be assessed against the known risks associated with the use of other highly effective methods of contraception as well as with pregnancy and childbirth.—I am, etc.,

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Hallucinations and Propranolol

SIR,—Propranolol is effective in the treatment of various cardiovascular conditions. However, it also has effects on the central nervous system. It has sedative and anticonvulsant properties,[1-4] and may reduce Parkinsonian tremor.[5] Sleeplessness and vivid dreaming have been reported. Prichard and Gillam[6] first reported a case of visual hallucinations.

A 53-year-old man was admitted to mental hospital suffering from nocturnal visual hallucinations. He described spirits appearing in his bedroom which stood still, worked spells, or copulated. This had commenced four or five months previously when the patient was taking 40 mg. four times a day of propranolol. The drug had been started three months before that at a dose of 10 mg. four times a day for severe angina. At the time of admission the dose had been slowly increased to 200 mg. four times a day and the patient had recently become aware of tactile hallucinations "like little darts dropped on the skin." He believed the spirits were about to take possession of his body, and to avoid this fate he had attempted suicide with aspirin and sleeping pills. He held the delusion that he must have unwittingly interfered with the spirit world in some unexplained way.

On admission the propranolol was tailed off over a period of three days, during which time chlorpromazine was given. Thereafter both propranolol and chlorpromazine were stopped. The hallucinations subsided and ceased after 10 days, but the patient's belief in the reality of the spirits remained. One month later further treatment with propranolol caused a recurrence at a dose of 40 mg. four times a day and this cleared up within two days of stopping the propranolol.

At no time was the patient confused or disorientated, there were no signs of a depressive illness, and no history of epilepsy or alcoholism. There was a six-year history of cardiac disease and he was obliged to cease work (a fitter) two years ago, at which time he was referred for a psychiatric opinion because of severe anxiety. He was described then as "a very anxious man who deals with his anxiety by excessive activity to reassure himself. This excessive activity is associated with the pain in his chest and it produces a really vicious circle." No psychotic symptoms were found and he was treated then effectively with diazepam.

It is likely that this illness started like other cases of visual hallucinations[7] with the patient retaining insight into the nature of the hallucinations. The patient admitted that initially he had suspected the propranolol; however, this insight was lost as the dose increased. This psychotic illness involving a suicide attempt seems to confirm Waal's view[8] that propranolol can cause serious psychiatric morbidity. It is also of interest in relation to the tranquillizing properties of propranolol that this patient was previously a very anxious man. In this case propranolol seemed to substitute different symptoms as though an alternative final common pathway came into operation when anxiety symptoms were blocked.—I am, etc.,

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Episodic Giddiness

SIR,—Your leading article on episodic giddiness (22 February, p. 457) does not mention that cigarette smoking may play a decisive part in precipitating attacks of true vertigo accompanied by increased intestinal peristalsis, sweating, weakness, and prostration.[1] In patients with high-tone deafness and attacks of periodic giddiness thorough eye testing is indicated. If perimetry reveals macular defects and defects for small red objects and ophthalmoscopic changes are absent nicotine poisoning should be suspected.

Nicotine stimulates peristalsis and may cause palpitations, weakness, prostration, and collapse. The eye changes are probably due to constriction of small blood vessels, which leads to degeneration of nerve fibres and cells. Vestibular branches of the labyrinthine artery are end arteries. Moderate smoking does not affect overall cerebral blood flow, but this does not exclude the possibility that nicotine-induced temporary vasoconstriction causes a localized hypoxia in a damaged vestibular system, which results in attacks of giddiness.—I am, etc.,

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Anticoagulants in Acute Myocardial Infarction

SIR,—The summary of the report of the working party on anticoagulant therapy in coronary thrombosis to the Medical Research Council (8 February, p. 335), concerning the assessment of short-term anticoagulant administration after cardiac infarction, is highly coloured, as appears from reactions in our country such as: Did you read the most recent report from the M.R.C., which demonstrates a failure of anticoagulant therapy in patients suffering from fresh cardiac infarction?

These reactions are based on a lack of important information in the summary of the report. This is to be deplored, because anticoagulants, if carefully and adequately administered, have proved to be a most powerful weapon in the prevention and treatment of almost any form of thrombosis, coronary thrombosis included.[1 2]

Fortunately, the careful reader is allowed to find the lacking information in the body of the report itself. In addition to the results reported in the summary (a not significantly lower mortality and a significant reduction in the frequency of clinically evident thromboembolic complications), the high dosage group showed less reinfarctions and less coronary thrombi at necropsy, though both not significantly.

Considered all together the results obtained by the different parameters demonstrate a more favourable effect of anticoagulant treatment than suggested by the summary. The difference between the two groups remains small, however. This fact is not surprising considering the basis on which the results were assessed—that is, the quality of anticoagulant treatment.

In almost 30% of the patients anticoagulant treatment was started later than 48 hours after cardiac infarction had occurred. Heparin was administered in a relatively low dose without monitoring hypocoagulability. The time of administration was too short (intrinsic hypocoagulability is insufficiently reduced by coumarin congeners 36 hours after the first dose is given, independent of the type of congener used).

The range of Thrombotest values aimed at and obtained are therapeutically insufficient: the range to be aimed at should be 5–10% instead of the 10–20% (5% Thrombotest is equivalent to about 10% P. & P. according to Owren and 15% according to Quick; 15% Thrombotest is equivalent to about 30% P. & P. or 45% Quick).

The stability of treatment obtained is low: only 50% of the values did lie in the range aimed at and 25% of the values were even higher than 20% Thrombotest.

In conclusion, the recently published results of the M.R.C. trial on anticoagulant treatment in coronary thrombosis do not detract from the value of carefully monitored intensive anticoagulant treatment.—We are, etc.,

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