abnormal (or even possibly abnormal) fetuses at any stage of pregnancy. I am particularly concerned that this may engender a change of attitude to the handicapped, somehow implying that they are less entitled to life than others.

Mr Victor Tunkel's legal analysis (p 253) is timely, but I feel that it would also be timely for all of us to look carefully into the moral question of abortion itself. Such a moral analysis may well give us the incentive to offer women the alternative of support and practical help throughout an unwanted pregnancy, and effective contraceptive advice afterwards.

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SIR,—Mr Victor Tunkel (28 July, p 253) points to some of the inconsistencies and absurdities in our abortion laws and appeals to the medical profession to exert pressure for reform. I would have more confidence that our laws might be realistic and responsive to the needs of the people they are meant to serve if legislators, doctors, and those enforcing the law listened to women rather than to themselves and to the rhetoric of intolerant bigots. I submit that, if they did, women would best be protected now by the repeal of all laws that make abortion a crime. Instead, I believe that the State should concern itself with the provision of the sort of abortion services that have been shown beyond reasonable doubt to protect the health of women. I am fed up with semantic arguments that ignore reality and with politicians, many of whom care more for their majority than for the health of half their constituents.

By concentrating on attempts to lower the legal limit for abortion your leading article in the same issue and some of your correspondents also miss the point. Mr John Corrie's Abortion Amendment Bill is going to affect far more women than would a reduction in the limit of viability. His Bill, by deleting the comparative clause in the present Act that allows the risks of abortion to be balanced against those of pregnancy continuing, together with the insertion of indefinable adjectives ("serious," "grave," and "substantial") to modify undefined nouns ("risks" and "health") could deprive up to 70 000 women each year of access to legal abortion. This provision would make a vast number of women even more dependent than they are now on the personal views, if not whims, of the doctors whose help they seek.

We profess to support sexual equality. But this can never exist while men, however noble their sentiments or erudite their arguments, ignore the right of women to choose freely whether or not they bear a child.

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SIR,—Dr Colin Brewer (11 August, p 389) wants your readers to have the pleasure—his taste in pleasure seems somewhat dubious—of pinning down anti-abortionists on the issue of the intrauterine contraceptive device.

I have a possibly heretical view that law ought to aim at keeping public order and preserving freedom, a view for which Dr Brewer may have little sympathy. However, Ms (presumably) Simms's letter in the same issue is an illustration of the callousness of the

pro-abortionist position, and might certainly arouse sympathy for the anti-IUD position as at least not lending support to the pro-abortion cause. One wonders how many decisions to use the IUD are not insufferably intolerant, as being a recommendation for people who are seen as incapable of controlling their fertility in any other way, made in the full knowledge that it is not reliable as a contraceptive anyway.

Perhaps Dr Brewer should be "made," to use his own delightful terminology, to state his own position on what he calls established human life. In what way is a fetus non-established—as opposed, for example, to a man on a respirator? The issues do not seem so clear cut as to allow the use of such coercive language as that of Dr Brewer. Perhaps also he might be invited to state his position on the morality, since he says abortion is a moral issue, of discrimination against would-be gynaecologists or others unwilling on conscientious grounds to perform, or assist at, abortions.

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SIR,—Dr Colin Brewer (11 August, p 389) is absolutely right. There seems little doubt that the intrauterine contraceptive device is abortifacient. Oral contraceptives also, besides preventing the production of fertile cervical mucus (contraceptive action) and inhibiting ovulation (sterilising action), interfere with implantation of fertilised ova by causing the endometrium to be out of phase (abortifacient action). Homicide should have no place in medicine, whether it be destruction of an embryo, fetus, infant, or adult.

I would go further. Mutilation, as practised in sterilising operations, and deliberate impairment of health, as when the endocrine system of the body is upset by oral contraceptives to impair fertility, should have no place in medicine. Such measures are inconsistent with the healing art. They are contradictory to our avowed purpose to promote health and prevent unnecessary loss of life. The medical profession has degraded itself by giving way to pressure to provide these "services." Some have gone only part of the way. Others have taken the road to its logical end—the destruction of human life on demand.

Is there an alternative? I think there is. Billings¹ has shown that women can be taught, through their mucus symptoms, to know their fertile and infertile times. Should we not as a profession be encouraging couples to have knowledge of and respect for their fertility? Would that not be constructive and positive rather than destructive and negative?

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¹ Weissmann, M C, *et al*, *Lancet*, 1972, 2, 813.

Rupture of the uterus during prostaglandin-induced abortion

SIR,—Although McKenzie *et al*¹ in their study in 1974 were unable to find in the literature a case of uterine rupture occurring as a complication of prostaglandin-induced abortion, several isolated cases have been reported since then,²⁻⁴ most recently by Dr Simon Emery and others (7 July, p 51). This complication has occurred only following intravenous or intra-amniotic administration of prostaglan-

dins. We now report a case in which uterine rupture occurred following the use of extra-amniotic prostaglandin E₂ (PGE₂).

The patient, aged 32, had had three previous vaginal deliveries at term and a spontaneous abortion at 12 weeks. An attack of rubella at 15 weeks was confirmed and termination of pregnancy was arranged at the 16th week. On admission, the size of the uterus was consistent with the period of amenorrhoea. A Foley catheter was introduced through the cervix into the extra-amniotic space and PGE₂ in solution, prepared in the usual manner, was instilled at a rate of 2 ml an hour. An intravenous infusion of oxytocin was commenced and after two hours the patient was having strong contractions. Twelve hours later vaginal bleeding occurred, at which time the patient's blood pressure had dropped to 95/60 mm Hg and her pulse was 120/min. On examination, a soft mass was found extending across the lower uterine segment, the uterine fundus being palpable above this at the level of the umbilicus. The cervix was dilated 4 cm. Uterine rupture was diagnosed and confirmed at laparotomy—it involved the right uterine artery, which was bleeding freely. After removal of the fetus, which had been extruded into the right broad ligament, subtotal hysterectomy was performed. The patient required seven units of blood and thereafter made an uneventful recovery.

Uterine rupture is a rare but sometimes fatal⁵ complication of therapeutic mid-trimester abortion, which has now been reported after prostaglandins administered by the intravenous, intra-amniotic, and extra-amniotic routes. We wish to draw attention to the fact that in most cases reported the patient also received intravenous oxytocin from an early stage in the procedure to facilitate abortion. We believe that early administration of intravenous oxytocin before prostaglandins have taken effect locally on the cervix may predispose to uterine rupture. In our unit therefore intravenous oxytocin is now used only after prostaglandins have been given for 12 hours, and then only if contractions are felt to be inadequate.

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¹ McKenzie, I Z, Hillier, K, and Embrey, M P, *British Medical Journal*, 1974, 4, 683.

² Propping, D, *et al*, *American Journal of Obstetrics and Gynecology*, 1977, 128, 689.

³ Borten, M, and Friedman, E A, *Prostaglandins*, 1978, 15, 187.

⁴ Smith, A M, *British Medical Journal*, 1975, 1, 205.

⁵ Grimes, D A, *et al*, *American Journal of Obstetrics and Gynecology*, 1978, 130, 591.

Lupus-type illness associated with labetalol

SIR,—We were interested to read the report of a lupus syndrome induced by pindolol, described by Dr J Bensaid and others (16 June, p 1603). We have recently seen a patient whom we considered to have a lupus-type illness, with strongly positive antinuclear antibodies, which was induced by another adrenergic blocking agent, labetalol, and which resolved on withdrawing the drug.

A 58-year-old white woman with a family history of malignant hypertension was found to have a blood pressure of 210/110 mm Hg in 1968. Renal function tests were normal and she was treated with methyldopa, initially alone and later with propranolol, from 1968 to 1976. From 1976 until February 1977 she received methyldopa and atenolol. She was then converted to labetalol only, initially 600 mg daily, increasing to 800 mg daily

in May 1977. She was receiving no other medication. At the end of 1977 she complained of pain and swelling in both wrists, which subsequently spread to involve the knees, ankles, hands, and elbows. In July 1978 there was clinically detectable synovitis of the proximal interphalangeal, metacarpophalangeal, and wrist joints. Her blood pressure was 150/80 mm Hg and her general examination was otherwise normal. There was no history suggestive of pleuropericardial disease or skin involvement. Investigations showed haemoglobin 9.6 g/dl, white blood cells $3.7 \times 10^9/l$, erythrocyte sedimentation rate 50 mm in the first hour, Coombs test negative, antinuclear antibody (ANA) test positive (titre 1/320), urea and electrolytes normal; the urine contained no protein, the Rose-Waaler test was negative, C3 and C4 levels were normal, DNA binding activity was <25 units/ml (normal), and the test for extractable nuclear antibodies was negative. Radiographs of the hands, feet, and chest were normal.

She continued to have evidence of a low-grade synovitis and in December 1978 she developed painful, pitting oedema of the right hand and left foot. The ANA titre remained at 1/320, the DNA binding was normal, and the leucopenia persisted. Labetalol was stopped in January 1979 and the patient was started on propranolol long-acting 160 mg daily. By February 1979 the symptoms had resolved; there was no evidence of synovitis and the ESR had fallen to 16 mm in the first hour. The ANA titre had then fallen to 1/80 and by April 1979 it was 1/20; and the patient remained asymptomatic.

We feel that this patient developed a lupus diathesis with synovitis, leucopenia, possible vasculitic oedema, and antinuclear antibodies during treatment with labetalol. There was resolution of the clinical signs and improvement of serological abnormalities on withdrawal of the drug. Our patient differs from the one described by Dr Bensaid and his colleagues in that pleuropericardial involvement was not present and at no stage was our patient's illness sufficiently severe to require corticosteroid therapy. Antibodies to double-stranded DNA were absent in both cases, as is usual in drug-induced lupus disorders. Although labetalol proved effective in controlling this patient's hypertension, it is probable that it induced a lupus syndrome and, as such, should be added to the list of drugs capable of provoking lupus-like illnesses.

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Sulphinpyrazone and warfarin after myocardial infarction

SIR,—Drs J A Tulloch and T C K Marr (14 July, p 133) ask for comments on their report of their experience with sulphinpyrazone (Anturan) and warfarin used early after myocardial infarction. I hope that the following remarks may be of some help.

At the present time evidence from controlled clinical trials of sulphinpyrazone following myocardial infarction is available only with respect to treatment being started approximately one month after the acute event and with a daily dose of 800 mg given in four divided doses. Indeed, the relevant current product licence for this product specifies both these conditions for its use.

The final results of the sulphinpyrazone reinfarction trial were presented at the recent

VIIth Congress on Thrombosis and Haemostasis in London and indicate a statistically significant reduction in "sudden death" in the sulphinpyrazone-treated group over the whole period of the trial (mean exposure time 16.1 months). No significant effect on reinfarction was noted. However, the benefit seemed to be due to an early effect which was maintained through the study, and it was therefore suggested that further controlled studies should be performed with earlier use of sulphinpyrazone following myocardial infarction.

At the present moment, however, little or no information is available on the use of this agent on admission of patients to the coronary care unit. Interpretation of results of therapeutic intervention in such patients is extremely difficult in view of the very labile natural history of the disease. For this reason, a comparative control group is essential in any trial attempting to assess new measures in this clinical setting. Preferably this comparison should be made on a double-blind basis. Unfortunately, it would seem that Drs Tulloch and Marr do not have such a comparative group and therefore an assessment of whether a particular event has occurred exceptionally frequently must have been made subjectively. Does "six recurrences . . . within a short period among 41 patients" represent a significant variation from the norm?

Even disregarding these methodological considerations, there are certain questions about this report which need to be answered before any attempt can be made to interpret the results. Did all patients admitted to the coronary care unit during the relevant period receive sulphinpyrazone? If not, how was the selection made? Similarly, were all patients admitted to the coronary care unit anticoagulated? What determined whether a patient received warfarin rather than heparin? Is any information available on the "coagulation status" of the patients following the withdrawal of warfarin? Were the recurrent episodes "fresh ischaemic episodes" or conventionally diagnosed myocardial infarctions?

We are not given any information as to the clinical course of patients during the hospital admission period, but it appears that there may have been only one death among the 78 patients following discharge from hospital. Subjectively, this figure would seem particularly encouraging.

Drs Tulloch and Marr have reported an interesting observation worthy of further investigation, but it would be unwise to draw conclusions from it regarding beneficial or adverse effects of sulphinpyrazone, alone or in combination.

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

SIR,—Dr Domenet's letter, while of interest, suggests that he has missed the reason for our letter. However, to answer the points he raises:

(1) It was and still is our intention to compare the progress over one year (initially) in patients admitted during the first half of 1979 who received sulphinpyrazone and in those admitted in the second half who will not receive it. (2) All patients admitted up to 30

June who had an acute infarct or acute ischaemic episode did receive sulphinpyrazone. (3) All of these patients were anticoagulated—those admitted under JAT receiving warfarin and those under TCKM subcutaneous heparin. No patient had an absolute contra-indication to anticoagulation. (4) We have no information on the "coagulation status" of the patients after warfarin was withdrawn. (5) No details of the clinical course of the patients were given as it did not seem relevant to the point we were raising. (6) The recurrent attacks were "ischaemic episodes" rather than major infarcts. In our experience six attacks among 41 patients in the course of a few months is unusually and alarmingly high.

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Sodium in peritoneal dialysis solutions

SIR,—We read with interest the paper by Dr P G Bisson and Mr K M Bailey on sodium in peritoneal dialysis solutions (19 May, p 1322). Dialysate sodium concentration is no doubt critical in the management of patients on regular dialysis treatment, whether they are on peritoneal dialysis or haemodialysis. Optimal dialysate sodium concentration in regular dialysis treatment remains a controversial subject. Whereas there are only two types of peritoneal dialysis solution available in the United Kingdom (Na 130 and 141 mmol(mEq)/l), we are alarmed by the fact that on our last count 21 haemodialysis solutions with varying sodium concentration (range 115–145 mmol/l) were commercially available in the United Kingdom.

The need for varying the type of dialysate for haemodialysis stems from the days when haemodialysis machines with fixed proportioning units were used. The modern generation of haemodialysis machines, and indeed the ones used during the past decade, have variable proportioning units, which deliver a desired dialysate sodium concentration, at site, and obviate the need for so many types of haemodialysis solutions.

In the light of these developments we suggest the reassessment of our requirements and standards of dialysis technology.¹

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¹ Ahmad, R, *Proceedings of European Dialysis and Transplantation Association*, 1979, 16 (in press).

Seizures after aminophylline by suppository

SIR,—A 19-year-old girl without a previous history of epilepsy presented in status asthmaticus. She had been ventilated on two previous occasions during the preceding nine years.

She weighed 36 kg and her arterial oxygen pressure (PaO₂) was 10.1 kPa and arterial carbon dioxide pressure (PaCO₂) 3.3 kPa. Within 12 hours of admission, following the failure of conventional treatment to prevent carbon dioxide retention, she was treated with positive-pressure ventilation for three days, intravenous salbutamol, hydrocortisone, and ampicillin. Off the ventilator on