stamps out, slamming the door behind him (locking it with older children). The child screams and shouts for hour after hour until, depending on his strength and determination, eventually he subsides in a lather of sweat and exhaustion. On no account must the parents enter the room until next morning, so it must be adequately warm.

This treatment will decondition most children in one night, and indeed if toddlers attend the consultation the treatment may not even be called into play. My most remarkable case was that of a 4-year-old, who, significantly, had remained an only child, having cried nightly since birth yet delighted his desperate parents by silence after the second night. Presumably owing to the superior intelligence of *Homo sapiens* compared with *Canis* he is easier both to condition and decondition.

One of the features of our age is the loss by authority of the self-confidence to assert itself, and nowhere is this more disastrous than in the home. It is unlikely that Victorian parents needed a doctor to tell them how to deal with such a simple domestic problem, but then they had no fearfulness of psychological trauma to displace the common sense handed down to them by genetic and cultural evolution as well as acquired by personal experience.

As a postscript, my daughter and niece both fit the jargon label: mature and well-adjusted adults.

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Beta-blockers and lipid metabolism

SIR,—We note that Dr A Nilsson and his colleagues (9 July, p 126) were unable to confirm our finding that plasma triglyceride levels tend to rise during treatment with metoprolol. They suggest that our results may have been due to the patients being ambulatory outpatients (and, perhaps, by inference less truly in a fasting state), or diabetic, or increasing in weight. We would be delighted to think that there was some such reason but have not been able to find evidence of this.

Our data were obtained in an open though systematic trial on 44 (20 male, 24 female) hypertensive outpatients aged 34-70 (mean 55) years. The patients received careful instructions to fast after 9 pm on the previous day, not to smoke, not to drink tea or coffee, but to take their antihypertensive medication with a little water on the morning on which blood for fasting lipids was to be taken, usually in association with a glucose tolerance test. The tests were done before metoprolol was started, after 3-4 months on metoprolol, and in many cases again after one year on metoprolol.

Of the 44 patients, 15 were untreated non-diabetic hypertensives, eight were non-diabetic hypertensives taking a diuretic alone which did not control their blood pressure adequately, and 21 were taking a non-selective β-blocker (17 of these on a diuretic also). Of these 21 patients, 15 had a diabetic tendency (blood sugar: fasting >5·6 mmol/l (>100 mg/100 ml); 2 h after 50 g glucose >6·7 mmol/l (>120 mg/100 ml) on non-selective β-blocker therapy and six were non-diabetics but had had side effects limiting the dose of the non-selective β-blocker. The dose of the diuretic was kept constant; non-selective β-blockers were replaced by an equivalent dose of metoprolol which was then increased if necessary for optimum blood pressure control. The daily dose of metoprolol ranged from 50 to 600 mg/day. Patients not previously treated with a β-blocker were started on metoprolol 25 mg twice daily and the dose was increased as dictated by blood pressure control.

As shown in the accompanying table the fasting triglyceride level showed a significant rise after three months' treatment with metoprolol in each of the four groups of patients. The rise was maintained after one year, though the numbers in two groups became too small for significance testing by the Wilcoxon signed rank test. Student's paired *t* test was significant at $P < 0.05$ levels for both these groups. Thus a diabetic tendency or diuretic therapy seemed to play no part in the findings.

The mean weight gain in the four groups shown in the table was of the order of 0·1-0·5 kg and was not statistically significant. Seven diabetic patients had a significant improvement in glucose tolerance after changing from a non-selective β-blocker to metoprolol,[1] but they nevertheless showed a significant rise in plasma triglycerides after three months (0·31 mmol/l (27·4 mg/100 ml)) and one year (0·56 mmol/l (49·6 mg/100 ml)) on metoprolol.

We have no reason to think that the patients were less careful over fasting while taking metoprolol. It may be of significance that our patients took their drugs at the usual time on the morning of the test—that is, 1-2 h before blood was drawn; possibly therefore we were observing an acute rather than a chronic effect. Dr Nilsson and his colleagues do not state whether their patients took the drug on the morning of the test. We hope that others also will make observations on triglyceride levels in patients treated with metoprolol and other β-blockers.

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[1] Waal-Manning, H J, *Drugs*, 1976, **11**, suppl 1, p 121.

Fasting plasma triglyceride concentrations before metoprolol and change after 3 and 12 months' treatment

Previous treatment	No of patients	Before metoprolol (mean; mmol/l)	Change with metoprolol (mean ± SE; mmol/l)		Dose of metoprolol at 3 months (mean and range; mg/day)
			3 months	1 year	
None	15	1·06	+0·24* ± 0·099	+0·39* ± 0·167 (3 dropouts)	159 (75-450)
Diuretic alone	8	1·78	+0·38* ± 0·150	+1·03** ± 0·360	219 (50-450)
Non-selective β-blocker; patients with diabetic tendency	15	1·56	+0·59** ± 0·149	+1·12 ± 0·578 (6 dropouts)	330 (50-450)
Non-selective β-blocker; non-diabetic patients	6	1·13	+0·87* ± 0·196	+0·31 ± 0·161 (2 dropouts)	275 (50-600)

Wilcoxon's signed rank test: *$P < 0.05$, **$P < 0.01$.
Conversion: SI to traditional units—Triglyceride (as triolein): 1 mmol/l ≈ 88·6 mg/100 ml.

Perinatal and infant mortality

SIR,—You published the official figures for perinatal and infantile mortality for 1976 (20 August, p 527). Some of your readers might be interested to see how they compare with the 1973 figures given in the Court Report,[1] (vol 2, table C3). This cannot be done on a national basis except for Wales, since in 1973 the figures were for England and Wales and in 1976 they were for England alone. However, the English regional figures are comparable. Scotland was left out of the Court Report.

	Perinatal mortality per 1000 births		Infantile mortality per 1000 births	
	1973	1976	1973	1976
Wales	21·45	19·0	16·36	13·6
Northern Ireland	—	22·3	20·90	18·3
England (RUAs)				
Northern	21·77	19·1	17·72	15·1
Yorkshire	22·89	18·4	19·95	15·0
Trent	21·05	19·2	16·05	14·7
East Anglian	17·37	13·9	13·66	11·6
NW Thames	19·20	16·9	15·31	13·9
NE Thames	20·40	16·6	16·57	13·4
SE Thames	19·55	17·4	15·02	14·8
SW Thames	18·80	14·8	16·17	12·7
Wessex	20·65	15·1	14·58	12·7
Oxford	17·04	13·8	13·74	12·9
South-western	18·99	16·1	15·04	12·9
West Midlands	23·63	21·1	19·14	15·7
Mersey	24·57	19·5	20·05	14·2
North-western	23·44	18·7	19·18	15·7

There is considerable overall improvement.

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Committee on Child Health Services, *Fit for the Future*, vol 2, table C3. London, HMSO, 1976.

Effect of calcium deposition on copper IUDs

SIR,—Recent work by Dr Christine Gosden (22 January, p 202) and Dr E Chantler (30 July, p 288) and their co-workers have suggested that progressive incrustation of copper intrauterine contraceptive devices (IUDs) by calcium or protein-containing deposits causes a decline in the rate of copper release and hence, presumably, in contraceptive efficacy. But the amount of such incrustation at a given time since IUD insertion shows much individual variation,[1 2] and metallic ions do diffuse through similar deposits on orthopaedic implants (Mr J R Newton, 6 August, p 387). What are the clinical implications of this work?

Unfortunately the paper by Mr Newton and his colleagues (22 January, p 197) is inconclusive. As Dr I Sivin showed (6 August, p 387), the non-random allocation of subjects at two years' copper IUD use to a "continuation group" and a "replacement group" led to differences between them which may have biased the comparison of the pregnancy rates. Moreover, the pregnancy rate among the 183 women who elected to have their Copper 7 replaced at 24 months was subsequently unbelievably low—no pregnancies at all for the next 21 months. Such a low rate has not been described in any previous IUD study. It cannot be entirely explained by the fact that some of the more fertile women initially recruited would have dropped out for reasons such as pregnancy during the first two years and hence been excluded from both the groups subsequently compared. However, the pregnancy rate in the third year among the "continuation group" was not significantly different from that of all the acceptors in the