contact is one of the most fundamental and important needs of an infant, on a par with feeding, changing, etc.

I was therefore surprised and perturbed to read Mr A W Fowler (29 August, p 614) suggesting that if picking up a baby stops it crying it is "only" seeking attention and can therefore "be put down immediately and left to cry it out." Firstly, this is fallacious, as cuddling a baby can often make it forget temporarily a pain or discomfort. Secondly, even if it is only seeking contact, has not the baby a true need for this contact, which in earlier days of our evolution (and not so very long ago, either) would have been continuous? I am glad Mr Fowler suggested a sound-proof separation for the parents; at least that will prevent their nerves being further tortured by the desperate pleas from their offspring, which all their instincts urge them to obey, but medical opinion urges them not to. For how much longer will the medical profession arrogantly advise mothers to go against every instinct which millions of years of evolution have given them?

And if mothers do get in such an exhausted state, as Dr H B Valman (8 August, p 422) suggests, might not the suggestion of a little helping hand from the father be in order? Or is that really much too progressive?

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Hyperglycaemic effect of nifedipine

SIR,—We were interested to read the report by Dr Stanislas Charles and his colleagues (4 July, p 19) of glucose intolerance in non-diabetic patients treated with nifedipine. Other investigators¹ reported improved glucose tolerance in normal subjects treated with nifedipine, but a further reduction in glucose tolerance in patients with impaired glucose tolerance.

We also studied groups of diabetics and non-diabetics before and during treatment with nifedipine and found no adverse effect on glucose or insulin secretion after a single 10-mg dose or after 30 mg daily for one month.² These various investigations, however, have used different types of patients, different doses of nifedipine, different methods of treatment, and different glucose loads and as such are not comparable. Further work is clearly needed before any definite significant adverse effect of nifedipine on glucose tolerance or insulin secretion can be assumed.

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¹ Gingliano D, Torella R, Cacciapouti F, Gentile S, Verza M, Varricchio M. *Eur J Clin Pharmacol* 1980;18:395-8.

² Donnelly T, Harrower ADB. *Curr Med Res Opin* 1980;6:690-3.

Effect of nifedipine on bronchomotor tone and histamine reactivity in asthma

SIR,—I wish to comment on the recent report by Dr D O Williams and his colleagues (1 August, p 348) on the effect of nifedipine on histamine reactivity in asthma.

Contrary to their observations, the calcium

antagonist verapamil given by inhalation (estimated dose 3 mg) failed to modify histamine- and methacholine-induced bronchoconstriction in 10 asthmatic patients I studied recently.¹ However, inhaled verapamil in identical doses was effective in preventing exercise asthma in nine of the 10 patients and its overall inhibitory effect was comparable to that of sodium cromoglycate both in the degree of protection offered and in relation to time.² Similarly, 20 mg of nifedipine given sublingually modified the exercise-induced fall in the forced expiratory volume in one second (FEV₁) in 13 of the 15 patients with exercise asthma.³ Although the inhibitory effect of nifedipine was statistically significant, this was considerably smaller than that reported by Cerrina *et al*⁴ and would be of little therapeutic significance.

In vitro, verapamil and nifedipine have been shown to inhibit anaphylaxis in airway smooth muscle⁵ and also to prevent release of platelet-activating factor (PAF) and slow-reacting substance of anaphylaxis (SRSA) from human neutrophils.⁶ Furthermore, Barnes and his colleagues have reported that nifedipine prevents postexercise rise in venous histamine levels in asthmatic patients (paper presented to spring 1981 meeting of the British Thoracic Association and the Thoracic Society). The observations on calcium antagonists in in-vitro studies and in exercise asthma would suggest that the effect is predominantly on the mast cell. Clearly more detailed studies are required to elucidate the therapeutic usefulness of calcium antagonists in asthma.

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² Patel KR. *Br Med J* 1981;282:932-3.

³ Patel KR. *Clin Allergy* (in press).

⁴ Cerrina J, Denjean A, Lockhart A, Douroux P. *Am Rev Resp Dis* 1981;123:156-60.

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SIR,—We read with interest the article by Dr D O Williams and others (1 August, p 348) regarding the effect of nifedipine on bronchomotor tone and histamine reactivity in 10 asthmatic patients. However, we were surprised that no side effects or changes in blood pressure or heart rate were observed.

Recently we have documented the haemodynamic effects of nifedipine in nine normal subjects by means of a double-blind placebo-controlled trial, and also measured blood pressure and heart rate changes as part of a larger study on the effect of calcium antagonists on peptide and steroid hormone release. The latter study was of single-blind placebo-controlled design and involved seven normal volunteers. The dose of nifedipine (20 mg) was that used by Dr Williams and his colleagues. Supine and erect blood pressure were measured before and at 30-minute intervals after nifedipine administration with a semi-automatic sphygmomanometer (Bosomat). The table shows

Supine diastolic blood pressure (BP; mm Hg) and pulse rate (PR/min) before (0) and 30, 60, and 120 minutes after nifedipine (20 mg) or placebo (mean $\pm$ SD, n = 16)

Time:	0	30	60	120
BP Nifedipine	71 $\pm$ 8	60 $\pm$ 10	64 $\pm$ 11	66 $\pm$ 9
Placebo	69 $\pm$ 8	70 $\pm$ 7	70 $\pm$ 10	70 $\pm$ 8
PR Nifedipine	57 $\pm$ 7	73 $\pm$ 11	72 $\pm$ 6	64 $\pm$ 7
Placebo	59 $\pm$ 7	59 $\pm$ 6	56 $\pm$ 5	57 $\pm$ 6

aggregate mean values ($\pm$ SD) from both studies for supine diastolic blood pressure and pulse rates before and 30, 60, and 120 minutes after nifedipine administration. We observed a consistent fall in supine and erect blood pressure, which was most marked 30-60 minutes after drug ingestion and lasted for two to four hours. The decrease was highly statistically significant ($p < 0.001$, Student's *t* test). We also noted a marked increase in heart rate ($p < 0.001$) and a significant negative correlation between the changes in blood pressure and pulse rate ($r = -0.81$, $p < 0.0001$, $n = 16$) at 30 minutes. The haemodynamic changes occurred in parallel with plasma nifedipine levels, which reached maximum values 30 minutes after administration. Finally, side effects consisting of facial erythema, a sensation of heat in the legs, and headache were present in most subjects, in contrast to the absence of side effects in the study of Dr Williams and others.

The pharmacological action of nifedipine (inhibition of transmembrane calcium flux) leads to relaxation of vascular smooth muscle, vasodilatation, and a fall in peripheral vascular resistance.¹ Blood pressure thus tends to fall, and it has been shown that both nifedipine and verapamil are potent antihypertensive agents.^{2,3} Agabiti-Rosei *et al* (paper presented to the 15th meeting of the European Society of Clinical Investigation, 1981), have also observed a fall in blood pressure in normal subjects after nifedipine ingestion, although Lederballe-Pederson *et al*⁴ observed only an increase in pulse rate without change in blood pressure. Our results strongly suggest that nifedipine at doses used in clinical practice does decrease normal blood pressure.

The differences in our results and those of Dr Williams and others may be due to technical factors, but may also indicate that asthmatic patients have a blunted response to nifedipine. It would therefore be appropriate to compare the effects of nifedipine on blood pressure and bronchial smooth muscle function in asthmatic patients and normal subjects. In addition, our results suggest that calcium antagonists may be indicated in the treatment of patients with hypertension and asthma, as well as those patients with combined asthma and angina pectoris as suggested by Dr Williams and his colleagues.

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² Olivari MT, Bartorelli C, Polese A, Fiorentini C, Moruzzi P, Guazzi MD. *Circulation* 1979;59:1056-62.

³ Leonetti G, Sala C, Bianchini C, Terzoli L, Zanchetti A. *Eur J Clin Pharmacol* 1980;18:375-82.

Neurosyphilis again

SIR,—Your interesting leading article on neurosyphilis states (25 July, p 263) that the postwar decline of neurosyphilis "is . . . partly attributable to the inadvertent treatment of unrecognised early stages of the disease by antibiotics given for other, unrelated diseases." This takes one back to controversy earlier this century concerning two questions. Does "adequate" antisyphilitic treatment prevent neurosyphilis? Does "inadequate" treatment of early syphilis hasten the onset of neurosyphilis?

The latter view seemed to be favoured by the findings of various observers that "inadequate" therapy of early syphilis (for example, by mercury¹ and in subsequent

years by neoarsphenamine) shortened the "incubation period" between syphilitic infection and onset of neurosyphilis. In the 1920s the view was expressed that the "modern" treatment of syphilis furthered the development of general paralysis of the insane (GPI).^{2,3} Our own review of the former antisyphilitic treatment received by 863 GPI patients (admitted to the neurological-psychiatric university clinic at the Charité, Berlin, under Professor Bonhoeffer during 1925-35) concluded that "modern" therapy of syphilis did not favour the onset of GPI.^{1,5} However, one was left with the feeling that no treatment known at the time was certain always to prevent later GPI, and anyway today's "adequate" therapy might prove in the light of later developments to have been "inadequate." That (possibly inadequate) penicillin therapy of early syphilis does not always prevent neurosyphilis has been demonstrated in the past^{6,7} and again by Hoffman's patient⁸ quoted in your leading article. One is therefore left wondering whether despite "excellent . . . response to adequate treatment" in the great majority, in certain cases even "excellent" antisyphilitic therapy may nevertheless prove inadequate in preventing neurosyphilis—as did yesterday's "adequate" treatment.

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¹ Jossmann P. *Monatsschrift für Psychiatrie und Neurologie* 1932;**84**:245-56.

² Gartner W. *Zeitschrift für Hygiene* 1921; **92**:341.

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⁴ Glatt MM. MD Thesis, University of Leipzig, 1937.

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Sulphonylurea and insulin combination in the treatment of diabetes

SIR,—I would like to comment on the interesting paper by Dr E A Ward and his colleagues (25 July, p 278) about the combination of a sulphonylurea with insulin in the treatment of insulin-dependent diabetes. This study showed no significant improvement in diabetic control when glibenclamide was added to insulin.

Only eight patients were studied, of whom only four showed residual beta-cell function, as judged by the presence of C-peptide in plasma. Of these, one patient showed a considerable fall in haemoglobin A_{1c} with the combined treatment (from 9.6% to 5.9%), indicating improved control; and the mean haemoglobin A_{1c} fell from 10.8% on placebo to 9.5% on glibenclamide. Perhaps a larger series of patients would have shown that this benefit was statistically significant. A longer period of study might also show more significant haemoglobin A_{1c} changes, for haemoglobin A_{1c} reflects diabetic control over a two-to-three month period¹ and the patients in this study were given glibenclamide for only four weeks.

The patients selected for the study were only mildly out of control, as judged by the mean haemoglobin A_{1c} of 10.8%, while on placebo and their plasma glucose profile, which showed normal values several times during the day, even when resting in hospital. A beneficial effect of this combination might have been more evident in patients who were grossly uncontrolled with insulin alone. Besides, it

would be only in such patients that the addition of another therapeutic agent would be considered. Again, the effect of sulphonylureas on insulin receptors might also have been more evident in patients less well controlled despite large doses of insulin.

Endogenous insulin secretion is suppressed by exogenous insulin while the plasma glucose is within the normal range.² The patients studied had normal values of plasma glucose on several occasions while receiving insulin, and this could explain the lack of a significant rise of plasma C-peptide concentration after the addition of glibenclamide.

The combination of a sulphonylurea with insulin has been found useful by other authors.^{3,4} I suggest that a larger and more prolonged study, in patients whose diabetes is difficult to control with insulin, is required before the combination therapy is dismissed as valueless.

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¹ Ditzel J, Kjaergaard JJ. *Br Med J* 1978;**i**:741-2.

² Service FJ, Nelson RL, Rubenstein AH, Go VLW. *J Clin Endocrinol Metab* 1978;**47**:488-92.

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Where do hunger strikers' rights end?

SIR,—It is the BMA's attitude not to force medical treatment on a hunger striker, even when he has fallen into a coma. This is justified by the hypothesis that suicide by starvation is a "considered decision," as opposed to "acute" suicide, where help to save the person's life must be given. Thus the hunger striker's right to choose his own fate is emphasised.

Other countries choose different procedures. In West Germany, for example, the medical organisation also respects the convicts' expressed will. This ends, however, when the patient falls into a coma, in which he cannot express his will or a change of it. Then everything to save his life will be done.

The ethics of saving the life of an unconscious person who previously wanted to die are debatable. Recent events in the Maze Prison, however, where through family intervention a life seems to have been saved, indicate that the course of the whole hunger strike campaign might have been altered by a different strategy of medical intervention. Bringing back a hunger striker from coma might not only mean saving his life—it might also (as in the above case) discourage further self-starvation, which in turn might contribute to ending the strike altogether.

Doctors have a key role in the past and future of the hunger strike. The BMA's decision should be reconsidered.

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*.*The Secretary writes: Medical ethics are always open to debate. The profession, however, has a plain view presently that it is wrong for a doctor to act towards a patient in a way that might make him—the doctor—an executive arm of the State. The doctor's job is not to alter the course of the hunger-strike campaign but to provide disinterested advice

when it is requested. An individual doctor may deplore or applaud a particular political action but when faced with a patient in distress it is his job to help that patient without regard to outside influences. And the patient has a paramount right to accept or reject treatment unless his power of consent has been legally removed.

Registrars will wait

SIR,—Recently the Minister of Health has suggested that the solution to the problem of the excessive number of registrars awaiting senior registrar posts would be to double the number of consultants. Halving the amount of work carried out by consultants and changing their posts from that of consultant to registrar—consultant at the district general hospitals would constitute a retrograde step. As mentioned in all the previous discussions most of the junior doctors are working in the teaching hospitals, where, certainly in London, the amount of work done is less than in the regional hospitals. No would-be consultant is intolerant of waiting for a proper consultant post, but at the moment owing to the monopoly control by the teaching hospitals of the senior registrar posts quite a number of them are forced to abandon their careers.

What is required is some fair way of ensuring that young doctors with their fellowship qualification who have received adequate practical training should at some time or other be granted the penultimate post to that of consultant. I am sure registrars would rather wait a few more years to achieve a consultant post such as exists today rather than have the consultant status half destroyed. The senior consultant does two days operating and two outpatient clinics and looks after approximately 30 patients. Anything less renders the job unacceptable. Introduce rationalisation and fairness in the young doctor's progress and the trouble will be at an end.

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What is a "soap opera?"

SIR,—Dr John Price's interesting and, to me, heartening review of *Maybury* (29 August, p 609) raises the question of whether such a programme is a "soap opera." While I appreciate that your columns may not be the place to define this term, as it was first used in your journal and has since crept into many reviews, perhaps you would allow me the space to say that *Maybury* does not fit any of my criteria for "soap opera."

A "soap opera" by custom and practice may be defined as a programme that follows the day-to-day story of a number of characters that appear in each episode. *Maybury* has only one running character, the consultant psychiatrist, Dr Roebuck, and does not attempt to follow day-to-day events.

Maybury is a drama series that has been divided into several anthologies; that is two three-part stories; three two-part stories; and a single play. A drama anthology would be nearer the mark in terms of definition.

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