labour the point that we should try harder to teach "organised neglect."

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Diet and weight reduction

SIR,—Your expert (10 June, p 1529) supports the views of the questioner that there are some individuals (presumably adults) who fail to lose weight on a daily intake of around 1000 kcal (4.18 MJ).

Certainly there are obese people who do not lose weight with an intake as low as 1500 kcal (6.27 MJ) a day, as has been demonstrated by Miller and Parsonage.¹ But an equally well-supervised study in which the diet supplied 1000 kcal a day resulted in weight loss in every one of the 49 subjects we studied.² They had all claimed that they did not lose weight when keeping strictly to low-calorie diets, and 12 of the subjects maintained that they had used diets of 1000 kcal a day or even lower and still failed to lose weight.

One reason for the discrepancy between what people say and what can be demonstrated if conditions are sufficiently well supervised is that, unless every item of food and drink is measured accurately, people are not at all good at assessing their own consumption, whether or not they are overweight. Nor is it good enough to assume that individuals really keep to a diet while in hospital. We have measured,³ for several days, the calorie intake in 14 hospital patients who had been prescribed low-calorie diets. In 10 of these the actual intake was substantially more than that prescribed and in six instances at least 50% more.

We cannot therefore subscribe to the common belief that there are obese individuals who will not lose weight when their intake is restricted to 1000 kcal a day.

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¹ Miller, D S, and Parsonage, S, *Lancet*, 1975, **1**, 773.
² Yudkin, J, *Practitioner*, 1972, **209**, 352.
³ Davies, G J, et al, *British Medical Journal*, 1975, **1**, 200.

Home or hospital care for coronary thrombosis?

SIR,—It is always interesting to read the arguments of consultants and general practitioners for and against the hospital treatment of myocardial infarction. In your recent "Medical Controversies" article (13 May, p 1254) Dr Peter Carson argues that patients with acute infarction are best managed in hospital because physicians and cardiologists are more experienced in rehabilitating coronary patients and getting them back to work. There is no evidence that this is true, and there is reason to believe that general practitioners are better acquainted with the problems of guiding postinfarction patients back to full activity. The vast majority of hospitals spend infinitely more interest and money on the equipping and running of coronary care units than on the rehabilitation of the patient when he is recovering from the acute episode. Having been taken through the elaborate ritual of coronary care, most patients are dropped back into the community with very little or no hospital follow-up or help with the convalescent phase. It is the GP who has to cope with the

day-to-day problems of lack of confidence, with odd chest pains, faintness, fatigue, and other symptoms, or of denial of illness with over-eager return to the old way of life which helped create the illness in the first place.

I run a course of graduated physical exercises for patients recovering from myocardial infarction, using the facilities of a local sports centre. I believe that it is most appropriate that coronary rehabilitation be conducted by GPs in the community for the following reasons:

(1) Hospitals are mainly involved with acute problems and are much less concerned with the long-term care of patients. They have little commitment to following up the patient once he is over the acute illness and tend not to release personnel to such mundane tasks.

(2) General practice rehabilitation is cheap, employing an inexpensive permanent form of labour and using equipment provided by the community. My course, when fully established and properly funded, will have cost a capital outlay of about £600 with a running cost of about £20 per patient, which compares very favourably with the cost of keeping him in hospital for one day.

(3) The course, being away from the hospital environment, does not seem so like treatment and encourages the customer to regard himself as a normal person and not a patient. He is more likely to see training as fun and more likely to keep it up.

(4) Many patients who suffer infarction are kept at home and would not be able to benefit from a hospital-based programme.

It is now recognised by most cardiologists and general physicians that supervised exercise training is of great value to the majority of patients after myocardial infarction, and such programmes are widely available in some parts of the world. Hospitals in Britain, however, have failed to provide adequate coronary rehabilitation facilities, and I believe that the time is ripe for GPs, using community sports equipment, to fill this gap. If we fail to meet this need it will be right for coronary patients to band together to demand that such a vital, but neglected, part of their treatment be provided by the Health Service for which they pay.

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Correlation between cytomegalovirus infection and HLA-BW15

SIR,—In the course of screening several "panels" of volunteer, HLA-matched, but otherwise unrelated donors in connection with a marrow transplant programme it was noted that two panels, both sharing the tissue type HLA-BW15, had higher than average numbers of individuals (9/10 and 3/4) with antibodies (complement-fixing and IgG-immunofluorescent) to cytomegalovirus (CMV). On further testing of donors possessing this tissue type CMV antibodies were found in 16 out of 22 (73%). The donor population was drawn from healthy adults in the Greater London area and the frequency of CMV antibody for all the donors so far tested (mean age 34.4, range 21-59 years) was 49% (40/82), which is comparable to the 54% reported for an age-matched population examined in London in an earlier survey.¹ The 22 donors (mean age 35.3 years) with the tissue type HLA-BW15 (16 of whom were CMV-antibody-positive) were compared with the 60 donors (mean age 34.1 years) possessing other tissue types (24 of whom were CMV-antibody-positive) in a Fisher exact probability test, one-

tailed since the direction of the difference was predicted in advance. The analysis indicated a low probability ($P=0.009$) that the different frequencies of CMV antibody were due to chance alone.

The findings suggest that normal donors with tissue type HLA-BW15 either have greater susceptibility to CMV infection or respond more readily with antibody production than does the general population. A similar association in the mouse between CMV susceptibility and the murine major histocompatibility complex has recently been reported.² The same donors were also examined for antibodies to other viruses (herpes simplex, varicella-zoster, EB virus, measles, mumps, parainfluenza type 1, influenza type A, rubella, adenovirus, and *Mycoplasma pneumoniae*), but the results failed to demonstrate any obvious relationship between these infections and tissue type HLA-BW15. Since CMV is a double-stranded DNA virus, the other reported association³ of HLA-BW15, with systemic lupus erythematosus, is particularly interesting in this context.

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¹ Stern, H, and Elek, S D, *Journal of Hygiene*, 1965, **63**, 79.

² Chalmer, J E, Mackenzie, J S, and Stanley, J F, *Journal of General Virology*, 1977, **37**, 107.

³ McDermott, H O, and Bodmer, W F, *American Journal of Medicine*, 1972, **52**, 1.

Diabetic complications in childhood

SIR,—I wish to comment on your leading article on this subject (15 April, p 941).

Concerning the late prognosis of juvenile diabetes the two more recent and important works on this subject published in French are often forgotten. Firstly, François *et al*¹ studied 204 patients with onset of diabetes before the age of 16 and duration longer than 10 years. The diabetics were treated with a normal diet and daily adjustments of insulin dosage. At regular ophthalmoscopy retinopathy was present in 14% of patients after a duration of diabetes of 10 years, in 38% after 15 years, and in 43% after 20 years. There was a correlation with the degree of control, retinopathy being present in 17% of patients with good control, 35% of those with fair control, and 47% of those with poor control. Secondly, Lestrade *et al*² studied the degenerative complications in a group of 114 subjects diabetic before the age of 15 years and followed up for 20 to 25 years. Retinopathy was present in 55% of the patients and proteinuria in 17%. In these two studies, the frequency of retinopathy is reduced when compared with that in children on a strict regimen.³

Concerning the early diagnosis of infantile diabetic retinopathy by fluorescein angiography, only a few such studies on retinopathy are available in the literature: those of Barta *et al*⁴ and of Malone *et al*,⁵ the latter being mentioned in your article. These authors consider the presence of microaneurysms to be the earliest finding. In 1974 we published a preliminary

Fluorescein angiogram (right eye): late venous phase. Fluorescein leakages near disc and foveola (arrowed).

study on retinopathy in 30 diabetic children⁶ and described, for the first time, fluorescein leakages (see figure) as the early lesions preceding the appearance of microaneurysms. These results have been confirmed in larger series of 87⁷ and 114 diabetics.⁸ Recently in juvenile diabetes the early breakdown of the blood-retinal barrier to fluorescein has been measured by quantitative vitreous fluorophotometry, confirming our work.^{9,10} We have proposed a new classification of diabetic retinopathy which takes the leakages of fluorescein into account.¹¹

As used by us angiofluorography doubles the frequency of diagnosis of incipient retinopathy. In 114 diabetic subjects who became clinically diabetic before the age of 14 years, with a duration of diabetes ranging from one month to 19 years, diabetic retinopathy was detected at regular ophthalmoscopy in 21 children but in 39 at fluorescein angiography. The influence of duration of diabetes is important.⁸ Retinopathy was detected at fluorography in 10.8% of the patients having diabetes for up to 5 years. After a duration of 5-10 years the frequency of retinal lesions increased to 45.0% and after more than 10 years to as much as 64.7%. In our experience insufficient or poor control considerably influences the frequency of retinopathy: good control—3.4% of retinopathies; insufficient control—34.4% of retinopathies; and poor control—70.8% of retinopathies.⁸ This is not in agreement with the report of Malone *et al.*,⁵ in which vascular abnormalities did not seem to be related to diabetic management and control.

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¹ François, R, *et al*, *Journées de Diabétologie de l'Hôtel-Dieu*, p 135. Paris, Flammarion, 1976.

² Lestrade, H, *et al*, *Nouvelle Presse Médicale*, 1976, **5**, 2297.

³ White, P, and Graham, C H, in *Joslin's Diabetes Mellitus*, ed A Marble *et al*, p 349. Philadelphia, Lea and Febiger, 1971.

⁴ Barta, L, Brooser, G, and Molnar, M, *Acta Paediatrica Academiae Scientiarum Hungaricae*, 1971, **12**, 343.

⁵ Malone, J I, van Cader, T C, and Edwards, W C, *Diabetes*, 1977, **26**, 673.

⁶ Toussaint, D, and Dorchy, H, *Bulletin de la Société Belge d'Ophtalmologie*, 1974, **168**, 783.

⁷ Dorchy, H, *et al*, *Nouvelle Presse Médicale*, 1977, **6**, 345.

⁸ Dorchy, H, *et al*, in *Proceedings of the 4th International Beilinson Symposium on "Nutrition and the Diabetic Child"*, Israel, 21-24 May, 1978.

⁹ Cunha-Vaz, J, *et al*, *British Journal of Ophthalmology*, 1975, **59**, 649.

¹⁰ Waltman, S R, *et al*, *Diabetes*, 1978, **27**, 85.

¹¹ Dorchy, H, and Toussaint, D, *Nouvelle Presse Médicale*, 1977, **6**, 2609.

Medical and nursing personnel and rubella embryopathy

SIR,—At present rubella is epidemic in Britain (Communicable Disease Report 78/21; Communicable Diseases in Scotland 78/22).

Because of the risk of rubella embryopathy in women who become infected with rubella during the first 16 weeks of pregnancy medical and nursing personnel attending women during this period should be immune to rubella. It is particularly important that general practitioners (men and women) and community nurses should be rubella-immune because women during early pregnancy are mainly in their care. Sadly, most general practitioners and community nurses do not know their immune status.

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Perinatal mortality and one-year infant morbidity

SIR,—In the article on this subject by Dr Carolyn E M Jones and Dr M Radford (11 February, p 325) the figures for perinatal mortality showed an admirable drop between 1974 and 1976, not between 1970 and 1976 as stated. However, it is difficult to interpret these numbers without better definition of the population studied. No lower limit for weight of livebirths or stillbirths is given. May we assume that all births in which the baby weighed more than 500 g were included? More importantly, what happened to the low-birth-weight rate during this time?

The implication in the article is that improved neonatal care has contributed to a recent decline in first-week neonatal deaths. However, it is clear that neonatal deaths are closely related to the number of low-birth-weight (500-2500 g) infants being delivered.¹ The low-birth-weight rate is increased in teenagers.² It is suggestive that there were few very-low-birth-weight (<1500 g) infants born in the years 1975 and 1976, since only 13 infants born in 1975 who weighed less than 1500 g survived the neonatal period.

Here in Vermont we have approximately 6500 births per year (compared with approximately 5000/year in Southampton). However, with a low-birth-weight rate of less than 7% we have three times the number of very-low-birth-weight babies. The survival figures shown in the table come from our intensive care nursery (ICN) which serves the majority of the State.

These figures suggest that much of the dramatic improvement in Southampton might have been related to the decline in the number of very-low-birth-weight babies being born. If this was indeed the case it would be im-

Survival of very-low-birth weight infants and perinatal mortality rate, Vermont, 1975-7

	1975	1976	1977
Admissions to ICN with birth weight <1500 g	54	59	50
Survivors with birth weight <1500 g ..	34	43	40
Perinatal mortality rate (State of Vermont) per 1000 births weighing >500 g) ..	17.4	13.8	14.6*

*Provisional

portant to try to understand why this might have happened. Questions include, "Are women delaying their pregnancies?", "Are teenagers availing themselves of contraceptives?", "Has illegitimacy decreased?", "Have obstetrical practices decreased the number of babies born significantly prematurely (before 32 weeks' gestation)?", etc.

The small number of pre-term babies contributing to one-year infant morbidity cannot be placed in perspective unless we know more about the sample from which this number was drawn. Perhaps the authors have this information available. I believe it should have appeared in their paper.

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¹ Lubchenco, L O, Searls, D T, and Brazie, J V, *Journal of Pediatrics*, 1972, **81**, 814.

² Chase, H C, *Clinics in Perinatology*, 1974, **1**, 3.

*We sent a copy of this letter to Drs Jones and Radford, whose reply is printed below.—Ed, *BMJ*.

SIR,—The perinatal mortality figures we quoted in our article certainly include all liveborn infants over 500 g weight, but we cannot be completely confident that all babies over 500 g without signs of life were included, since some of these could have been termed abortions. However, we were not aware of any babies in the latter category, and certainly have not excluded any deliberately. The proportion of babies born with a birth weight of less than 2.5 kg has remained at about 7% between the years 1970 and 1975 (range 6.5-7.7%).

In 1975 33 infants were born weighing less than 1500 g, of whom 13 survived, and in 1976 34 were born, of whom 28 survived. It would not be easy to extract from our data the numbers of infants with a birth weight of less than 1500 g born in earlier years.

We hope we have answered some of Dr Philip's questions. We would like to emphasise that the main point of our article was not to quote our perinatal mortality figures but to emphasise that most morbidity arose in the group of infants with abnormalities acquired in the antenatal rather than in the perinatal period.

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Translumbar aortography without pain

SIR,—As a radiologist I was very interested in Mr M L Obeid's comments about translumbar aortography as opposed to transfemoral catheter aortography for peripheral vascular disease in the legs (10 June, p 1530).

Translumbar aortography has traditionally been performed under a general anaesthetic, as he indicated. However, the problem of pain after the injection of contrast has now been solved and this procedure can now be done under a local anaesthetic with little more pain than often occurs with a lumbar puncture.

The method of eliminating pain after the arterial injection is to add lignocaine 1 g/l to the mixture injected. This amounts to 2 ml of 2% lignocaine per 40 ml of contrast injected. The maximum dose of lignocaine that can be injected is 200 mg. Up to this dose any effect