in Parliament who wish to know what can be done to ensure that G.P.s are not overwhelmed with social work to the detriment of those who come for the treatment of real illness.—I am, etc.,

T. D. RICHARDS

Pucklechurch, near Bristol

Patient Delay before Treatment of Myocardial Infarction

SIR,—Before accepting the findings of Dr. I. C. Gilchrist's interesting study (3 March, p. 535) as valid indications of some of the reasons why many patients who suffer myocardial infarction at home do not call medical help immediately, it is important to point out that the 50 patients interviewed were all in the recovery stage of their infarction. This being so, these 50 patients may be representative of a group of patients, who are different in many respects (including reasons for, and delay in, calling medical help) from the group whose members do not manage to survive to this stage. The possible differences between these two groups may explain the very short median delay of one hour in calling medical help reported in this study.—I am, etc.,

ARTHUR FURST

Hebrew University-Hadassah Medical School, Jerusalem

Pityriasis Rosea in Sisters

SIR,—My 11-year-old daughter returned from boarding school in Yorkshire after a full term's absence from home on 15 December 1972. On 19 December she showed a herald patch on her chest which was followed by the typical rash of pityriasis rosea.

On 29 January 1973 my 5-year-old daughter showed a herald patch below the right axilla which was in time followed by the typical rash of pityriasis rosea. The two girls were in very close contact during the Christmas holidays, as the younger girl lay in the older girl's bed having stories read to her.

Possibly this indicates an incubation period of six weeks.—I am, etc.,

WALLACE WHITE

Great Baddow, Essex

Chondromalacia Patellae

SIR,—The way to treat chondromalacia patellae is to treat the foot. I have come to this conclusion after three years' experience as medical advice columnist for *Runner's World*, a periodical with 10,000 subscribers, mostly distance runners. This has given me a "constituency" peculiarly prone to this disease and allowed me the opportunity to evaluate various forms of treatment. Contrary to the optimism of Drs. J. Darracott and B. Vernon-Roberts (24 February, p. 491), my patients have not had promising results from quadriceps exercise and corticosteroid injections, nor from any other remedies listed in the literature. They have, however, had very gratifying results from podiatric treatment (custom-moulded orthotics) of the biomechanical difficulties of the foot that supports and stresses the damaged knee.

It stands to reason that normal, well-conditioned runners have normal knees. What many of them do not have is normal feet. About 35% of a group recently surveyed had a short metatarsal (Morton's syndrome). Others had forefoot varus or narrow subtalar range. Determination of the exact pattern of foot abnormality causing chondromalacia will take more research; but my runners have convinced me that the principle is correct. The one-plane symmetry of the patella riding the groove between the condyles is altered to cause chondromalacia. This deviation is transmitted by structural abnormalities in the intricate architecture of the foot. And with 26 bones, three arches, and a complicated support system of tendons and ligaments there is much to go wrong.

Attention to the runner's foot has resulted in relief of symptoms (a reasonable test) and pain-free resumption of long-distance running (the only real test). The mystery of chondromalacia patellae seems close to solution.—I am, etc.,

GEORGE SHEEHAN

Red Bank,
New Jersey, U.S.A.

Mute of Malady

SIR,—In your comprehensive leading article on mutism (31 March, p. 755) it is mentioned that the condition is not uncommon among children up to the age of 3 or 4.

I have tentatively described a syndrome[1] in which mutism and withdrawal, not infrequently accompanied by rhythmical rocking movements, are the main features. It seems that this condition is confined to the young children of West Indian parents living in this country. Aetiologically, maternal depression, often aggravated by overwork and heavy material burdens, contributes to the young child being deprived from the beginning of emotional, verbal, and environmental stimulus. The prognosis appears to be poor.

These children are frequently misdiagnosed as suffering from mental defect, deafness, or infantile autism.—I am, etc.,

G. STEWART PRINCE

London W.1

1 Prince, G. S., in *Social Work with Coloured Immigrants and their Families*, ed. J. Triseliotis. London, Oxford University Press, 1972.

Drug Data Cards

SIR,—It seems very wise that the Medicines Act, 1968 should legislate for data cards giving all the relevant details regarding ethical products. What seems entirely illogical is that we should receive them (as we have been doing in the past few weeks) in different shapes and sizes and with different forms of heading so that it is impossible to file them for easy reference.

I would consider it incredible that standardization for data cards was not recommended beforehand but, if this is the case, may I hasten to suggest (to save unnecessary expenditure on new filing systems) that a suitable size would be that of the present N.H.S. medical card, so that the same filing boxes could be used and, if necessary, details of a particular drug which a patient might be taking could be kept in his or her medical record envelope.

I pray that the envisaged change in size of the medical record envelope will not materialize. All that is really required is that hospital letter paper should be standardized to a size which, folded once, would fit neatly into the present record envelope (convenient for use in the surgery and for carrying in one's hand or pocket when visiting patients). International size A5 paper, as has been suggested many times before, is suitable for this purpose and A6 paper (half the size of A5), used landscape fashion, would be convenient for short notes and pathology reports.

However, if there should be a change in the medical record envelope an "old size" filing box could at least serve a useful purpose for the storage of drug data cards if these were of the size I have suggested.—I am, etc.,

N. V. EDWARDS

Halstead, Essex

Assessment of Acid-base Disturbances

SIR,—The assessment of the acid-base status of patients in an intensive care unit described by Dr. A. W. Grogono (17 February, p. 381) was justifiably criticized by Dr. J. B. Stoker and others (31 March, p. 803) because the assessment was based on the Siggaard-Andersen nomogram, which is derived from the in-vitro CO_2 titration curve. On clinical grounds the article may also be criticized because of its apparent unquestioning trust in a biochemical analysis without reference to the clinical status of the patient.

Stoker *et al.*[1] proposed a new system for the analysis of acid-base disturbances on the ground that the current indices of base excess and standard bicarbonate were confusing and difficult for the average clinician to understand. This statment seems unfair when it is realized that the modern clinician has used these concepts in the day-to-day management of patients over the past 10 years. Instead of introducing yet another concept, "non-respiratory pH," it would be simpler to correct base excess from the in-vitro to the in-vivo situation as first described by Prys-Roberts *et al.*[2] and later elaborated by Siggaard-Andersen.[3]

The criticisms levelled at the in-vivo corrections suggested by Siggaard-Andersen can also be applied to the concept of "non-respiratory pH." Stoker *et al.*[1] stress that non-respiratory pH is only valid when acute changes of P_{CO_2} occur in a non-respiratory acidaemia. This condition excludes the majority of patients with acute non-respiratory acidaemia who develop a rapid swing to alkalaemia on treatment. Patients with acute exacerbations of chronic bronchitis may present with an acute hypercapnia superimposed upon a chronic hypercapnia and a chronic change in non-respiratory acid-base status. In these patients the slope of the in-vivo CO_2 titration curve is uncertain.[4,5]

We have found a close correlation between corrected in-vivo base excess measurements and "non-respiratory pH" in patients admitted to an intensive care unit who underwent marked changes of P_{CO_2} or non-respiratory acid-base status.[6] These patients included chronic bronchitics. These findings suggest that if errors exist in the titration curves they were equally reflected by both methods. Thus no merit would be achieved