stretching tolerance. It may well have been that Central Committee for Hospital Medical Services felt that as discussion in the periphery had taken place many weeks ago no second thoughts were acceptable. One wonders whether parliamentary democracy would survive for very long if a similar pattern of decision-making were to be followed. One was certainly reminded very forcibly of the term "lobby fodder." When an abortive attempt was made to reopen discussions on the matter by suspending standing orders I think my fellow consultants should know that the most revealing of the opposition came from a junior representative, who stated categorically that they did not wish the consultant contract to be discussed.

As a result of this the B.M.A. is saddled with an official policy which does not give much room for manoeuvre. This policy does not reflect the initially latent but now increasing concern regarding the position of the full-time consultant. I had hoped to have the terms of the Review Body clarified as to whether at any time this body, at its own discretion, could take total earnings into consideration. If this is the case then the ten-session consultant without private practice who wishes to remain full-time would appear to be at a loss. It therefore follows that each session could become undervalued with the result that all hospital consultants could be affected. In these circumstances I was, and still am of the opinion, that full-time and part-time contracts should remain.

The motions which were accepted as official policy and the pattern of events at the A.R.M. pose the possibility of this being negotiated as a "package deal." It would then be understandable that the Chairman of the Central Committee for Hospital Medical Services had no wish to accept any amendments, but again this is something which should have been discussed. The whole episode left much to be desired and the platform should certainly have misgivings about the whole affair.—I am, etc.,

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SIR,—May I, as one of the four members of the subcommittee of the Central Committee for Hospital Medical Services who drafted the proposals for the new consultant contract, reply to some of the critics in your recent columns? Dr. J. W. Paulley (5 August, p. 354) finds it sad that so few read the *Supplement* or attend meetings and yet goes on to write "that negotiators should tell the Review Body that consultants require pay commensurate with their responsibility . . ." and that "in the event of failure to persuade the Review Body, . . . the profession should be prepared to apply effective sanctions, yet not detrimental to patients."

If Dr. Paulley had bothered to read either the *Supplement* or the evidence to the Review Body over the past three years he would be well aware that Dr. R. M. Mayon-White (*Supplement*, 5 August, p. 127) has brilliantly presented exactly the case which he suggests, and yet has had to confess complete failure. That being the case, perhaps Dr. Paulley could share with us the secret of those sanctions which he suggests consultants could apply which would bring the Department of Health to its knees without being detrimental to patients.

Dr. N. F. Coghill (5 August, p. 354) doubts if there is great support for a 10-session contract, but as this is based on his own misunderstanding of both the existing contracts and the new draft proposals it may be that the Secretary's comment will have helped him to understand them. Like Mr. J. Kyle (5 August, p. 354), I am sorry that we could not achieve a debate on the new contract at Southampton, if only to set the minds at rest of those who have still not bothered to read it. It was perhaps not surprising, however, that the Representative Body insisted on voting overwhelmingly for the proposed contract without going over the same ground again, as the Representative Body contained a large number of hospital doctors who had attended the National Conference of Hospital Medical Staffs (*Supplement*, 22 July, p. 60), where a long and fruitful debate had resulted in a 95% vote for the new proposals, plus a great many general practitioners who well understand the advantages that accrued to them from the charter in which the component parts of their contract were identified.

So far as I am aware no one has ever claimed that the new draft contract will represent a Shangrila for consultants, but I will state categorically that it offers the only hope the hard-working consultant has of obtaining a fair reward for his services. If and when it is negotiated with the Department of Health each individual would be well advised to consult his solicitor, his accountant, his bank manager, and his conscience before he makes a change. One thing is quite clear: neither the Review Body nor the Department of Health has shown any wish to create increased differentials between us and the juniors, general practitioners, or pay prospects overseas. The sole remaining card, therefore, is the increasing work load, and I fail to see how this can be quantified unless we can state contractually what we are actually prepared to do as a basic commitment.

Faint hearts may take some courage from the fact that completely independent polling conducted by the Regional Consultants and Specialists Association on substantially the same issues has resulted in it adopting almost identical proposals as its association policy.—I am, etc.,

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Febrile Convulsions in Early Childhood

SIR,—A recent leading article on febrile convulsions in early childhood[1] has provoked a controversial correspondence. Doubts were expressed that a single febrile convulsion was sufficient indication for urgent admission to hospital, a view with which many general practitioners and paediatricians would concur provided the diagnosis is not in doubt. Several correspondents have questioned the recommendation that such seizures should be treated with paraldehyde, and one[2] tentatively suggested that continuous prophylactic anticonvulsant medication might have a place in the management of a child having "frequent convulsions." Recent studies have provided conclusive answers to some of these questions, and we would like to draw some of these to the attention of your readers.

Febrile seizures are common. About 1 in 20 of all children under 6 years have experienced one or more such convulsions. Clearly not all these children can be admitted to hospital, and Livingston's[3] criteria for admission, formulated in 1954, provide a useful guide. He suggested that admission is indicated when the child's parents are very anxious, when the child has had more than one seizure in a single episode, when there is a clinical suspicion of meningitis, when there are social problems, and if the interictal electroencephalogram is known to be abnormal. Many would not now regard the last of these criteria as sufficient indication for hospitalization.

The relation of febrile convulsions to the occurrence of epilepsy in adult life has long been problematical. Though a reluctance to diagnose idiopathic epilepsy is understandable, it is well known that a tendency to febrile convulsions may be inherited[4] and that about 30% of children who have had one febrile convulsion will have another before the age of 9. Less than 15% will have more than three such seizures, but some, perhaps as many as 20%, will later develop idiopathic epilepsy.[3 5–7]

Formerly intermittent anticonvulsants were advised, but several recent studies have deprecated this regimen,[6 8] since, firstly, the seizure usually occurs during a rapid rise in temperature and not when fever has been established for some hours, and, secondly, recurrent seizures usually occur during subsequent febrile episodes and not during the same episode. Thirdly, adequate blood levels of circulating anticonvulsants cannot be achieved for at least several days when oral phenobarbitone or phenytoin is prescribed in the doses usually advised.[1 9–11] Any possible rationale for treatment with intermittent oral anticonvulsants is thus invalidated.

If continuous anticonvulsants are to be prescribed, which drug should be used? Several Scandinavian groups of workers have examined possible continuous anticonvulsant treatment regimens in carefully controlled, prospective, double-blind clinical studies. These studies have shown that continuous prophylactic treatment with phenytoin is not effective.[8] Phenobarbitone, however, has produced striking results in the most recent study from the same centre.[11] Sixty children, all under the age of 3 years, who had presented with a febrile seizure were treated with phenobarbitone in a dose of 3-4 mg/kg, given in divided doses twice daily. Double this dose was given for the first 48 hours, since Svensmark and Buchthal[9] found that with this "loading dose" regimen effective plasma levels were usually achieved in two days, but if this "loading dose" was omitted effective plasma levels of phenobarbitone were often not reached for several weeks. Treatment was continued for six months and the results compared with those in 172 untreated children, of whom 20% had a recurrent seizure in a six-month follow-up period.[12] Of the treated children only one of 27 children whose blood phenobarbitone concentration, determined at monthly intervals, remained above 1·6 mg per 100 ml had a second seizure, but blood levels below 1·5 mg per 100 ml were found to be ineffective. There were no significant side effects. Both the Scandinavian workers[8 9 11 12] and others [6 13] have pointed out that the presence or absence of interictal E.E.G. abnormality should not be used as a guide

to the necessity for anticonvulsant treatment.

It may be concluded from these studies that continuous prophylactic treatment with oral phenobarbitone at these dose levels, as suggested some years ago by Hammill and Carter,[6] is an effective method of treatment which should therefore be given to all children with febrile seizures after their first episode. It should be continued for at least two years or until the age of 5 years, and it may be considered worthwhile during this time to make occasional estimations of the blood phenobarbitone concentration. That an effective continuous treatment regimen is worthwhile is supported by the occasional occurrence of irreversible brain damage sustained during a febrile seizure.[5 14 15] In addition febrile convulsions have been incriminated in the development of subsequent temporal lobe epilepsy.[16] It is therefore possible that effective prevention and treatment of febrile seizures could lead to a decrease in the incidence of epilepsy in adult life.

Some of your readers may not agree with the view that continuous anticonvulsant medication is of value in the treatment of patients with febrile convulsions, but at least we feel that they should be aware of the great volume of work continuing on this subject, both in this country and abroad.—We are, etc.,

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1 *British Medical Journal*, 1972, **1**, 608.
2 Lewis, B. W., *British Medical Journal*, 1972, **2**, 48.
3 Livingston, S., *The Diagnosis and Treatment of Convulsive Disorders in Children*. Springfield, Charles C. Thomas, 1954.
4 Lennox-Buchthal, M., *Epilepsia*, 1971, **12**, 147.
5 Millichap, J. G., *Febrile Convulsions*. New York, MacMillan, 1968.
6 Hammill, J. F., and Carter, S., *New England Journal of Medicine*, 1966, **274**, 563.
7 Van den Berg, B., and Verushalmy, J. *Pediatric Research*, 1969, **3**, 298.
8 Melchior, J. C., Buchthal, F., and Lennox-Buchthal, M., *Epilepsia*, 1971, **12**, 55.
9 Svensmark, O., and Buchthal, F., *American Journal of Diseases of Childhood*, 1964, **108**, 82.
10 Buchanan, R. A., Kinkel, A. N., Goulet, J. R., and Smith, T. C., *Neurology*, 1972, **22**, 126.
11 Faer, O., Kastrup, K. W., Lykkegaard-Nielsen, E., Melchier, J. C., and Thorn, I., *Epilepsia*, 1972, **13**. 279.
12 Frantzen, E., Lennox-Buchthal, M., Nygaard, A., and Stene, J., *Neurology*, 1970, **20**, 909.
13 Holowach, J., Thurston, D. C., and O'Leary, J. L., *New England Journal of Medicine*, 1972, **286**, 169.
14 Fowler, M., *Archives of Disease in Childhood*, 1957, **32**. 67.
15 Taylor, D. C., and Bowler, B. D., *Lancet*, 1971, **2**, 1136.
16 Ounsted, C., in *Recent Advances in Paediatrics*, ed. D. Gairdner and D. Hull. London, Churchill, 1971.

Subclinical Brucella Infection in Man

SIR,—Drs. R. J. Henderson and D. M. Hill (15 July, p. 154) draw attention to the booster effect on brucella antibodies of re-exposure to *Brucella abortus* infection.

Serological follow up of patients occupationally at risk in West Dorset shows that titres may not always remain high if the interval between exposures is a year or more. Booster effects producing a fourfold or greater rise in titre (direct saline agglutination) are common in those previously exposed and provide a trap for the unwary serologist. For example, a dairy farmer who had *B. abortus* infection with positive blood culture in November 1969 showed serological evidence of cure and disappearance of his symptoms, direct saline agglutination titres falling from 1,280 to 320 during the following six months, but follow up revealed that in April 1971 the titre had risen to over 2,580, the man being quite symptom free. If he had chanced to have an illness suggesting brucella in April 1971 an eight-fold rise in titre might have been interpreted as evidence of his symptoms being due to brucella relapse or active reinfection.

It is important to stress that the booster effect of symptomless reinfection may be accompanied by a rising complement fixation titre, as the following example shows. The patient is a dairyman who had an acute illness with positive blood culture in July 1971.

Date	*B. abortus* Saline Agglutination	Complement Fixation
July 1971	1,280	40
Aug. 1971	640	40
Sept 1971	320	less than 5
Jan. 1972	Over 2,560	20

In this instance the 2-mercaptoethanol titre was 640 in January 1972 suggesting that IgM antibody was present, yet this patient had no symptoms after the second week of his six weeks of treatment in the summer of 1971. He was well in January 1972 at the time of re-exposure.—I am, etc.,

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Faecal Flora after Prolonged Co-trimoxazole Treatment

SIR,—We were interested to read that Professor W. Brumfitt and Miss Rita Pursell (17 June, p. 673) did not find trimethoprim-resistant organisms in the faeces of patients treated with comparatively low doses (100 mg daily) of trimethoprim alone for periods of up to a year. We should like to report the changes in the faecal flora of a patient who received unusually high doses of co-trimoxazole for nearly a year.

As successful treatment of *Pseudomonas cepacia* endocarditis of a prosthetic mitral valve a 48-year-old woman received 0·64 g of trimethoprim and 3·20 g of sulphamethoxazole (8 tablets of Septrin) daily for seven weeks in hospital, and then 0·48 g of trimethoprim and 2·40 g of sulphamethoxazole (6 tablets of Septrin) daily for ten months at home. Kanamycin 1 g daily was also given for the first three weeks of treatment. She suffered no adverse effects and did not complain of any gastrointestinal disturbance.

We were interested to discover whether at the end of this long course of treatment her faecal flora included coliforms and other Gram-negative bacilli resistant to co-trimoxazole. A faecal specimen collected two days after she stopped taking the drug could not be made to yield any aerobic Gram-negative bacilli at all, even after heavy plating on selective media, and the organisms isolated were all of species generally resistant to co-trimoxazole. (We could not detect in the faeces residual antibacterial activity such as might have caused inhibition of sensitive strains in our cultures.) Aerobic Gram-negative bacilli were similarly undetectable in a specimen collected 10 days after the end of treatment, but after 5 weeks lactose-fermenting coliforms had reappeared and these isolates were all sensitive to co-trimoxazole and to trimethoprim alone. Approximate viable counts, per gramme of faeces, of the principal organisms found in the three specimens are given below; *Clostridia* and *Lactobacilli* were also present in all three.

Organisms	Time after Stopping Drug: 2 days	10 days	5 weeks
Lactose-fermenting coliforms	none detected	none detected	10^6
Bacteroides sp	10^9	10^9	10^9
Faecal streptococci	10^6	10^6	10^6
Candida sp.	10^4	10^4	10^3

It has been suggested[1] that in low dosage trimethoprim is almost completely absorbed and so does not upset the flora of the lower gut. The high dose given to this patient, however, appears to have eradicated or strikingly reduced her normal coliform flora, without bowel upset and without colonization by resistant strains. Administration of sulphonamides alone is rapidly followed by colonization of the gut by sulphonamide-resistant coliforms,[2] but it was to be expected that this would be prevented or delayed by the addition of trimethoprim. It is probable that in the domestic environment Gram-negative bacilli resistant to the combination are rare, and that even in hospitals resistant coliforms are infrequent at present,[3] although resistant pseudomonads are common and might be expected to colonize the gut of patients on treatment.—We are, etc.,

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1 O'Grady, F., *et al.*, *Postgraduate Medical Journal*, 1969, **45**, Supplement, p. 61.
2 Lincoln, K., Lidlin-Janson, G., and Winberg, J., *British Medical Journal*, 1970, **3**, 305.
3 Lacey, R. W., Gillespie, W. A., Bruten, D. M., and Lewis, E. L., *Lancet*, 1972, **1**, 409.

Rickets in Glasgow Pakistanis

SIR,—In the interesting article by Dr. J. A. Ford and others (17 June, p. 677) it seems that the authors noted but placed little weight on the possible role of skin pigmentation in the increased incidence of rickets and osteomalacia in the Pakistanis living in Glasgow. In doing so they were perhaps misled by their assumption that skins of Pakistanis living in Glasgow "are no darker than those of many southern Europeans."

In the Table are shown the percentage reflectance values (mean and standard deviations) for a sample of southern Europeans (Italians)[1] and a sample of Punjabis in Britain (Sikhs, personal observation, 1972). The Punjabi group is conspicuously darker than the Italians. This is especially clear at

Population	No. of Subjects	465 mμ (Blue) Mean	S.D.	515 mμ (Green) Mean	S.D.	655 mμ (Red) Mean	S.D.
Punjabi Sikhs	260	27·23	3·87	29·98	4·04	51·31	4·37
Italians'	703	30·99	4·70	37·97	4·86	61·56	4·65