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Streptokinase and Myocardial Infarction

SIR,—Dr. R. Heikinheimo (6 November, p. 361), in commenting on the report of the European Working Party on thrombolytic treatment in recent myocardial infarction (7 August, p. 325), remarks that more patients with recurrent infarction (44 of 357) belonged to the heparin treated group than the streptokinase treated group (35 of 373). This difference is, however, not significant ($P=0.066$), in contrast to the prevalence of arrhythmia on admission (10 of 373 streptokinase treated patients compared to 77 of 357 heparin treated patients).

It is indeed hard to resist the suspicion that there was some systematic reason why the eight deaths which occurred before the start of the infusion were all in the heparin group. An obvious explanation would be that there was, on average, more delay in starting heparin than streptokinase. This point was carefully checked; the time interval between admission to hospital and the start of the infusion was found to be similar in the two treatment groups. Moreover, the eight patients who died before the start of treatment were admitted to four different co-operating medical centres and were spread over the total duration of the trial. Six of them were admitted in shock. This trial is certainly a saga of great misfortune in that all preinfusion deaths were in the heparin group.

However, considering hospital mortality after the 24-hour infusion, there were 36 deaths in 340 streptokinase-treated patients (10.6%) versus 57 deaths in 320 heparin-treated patients (17.8%); this difference is significant, P two-tailed being 0.011 (and not 0.046 as stated in Dr. Heikinheimo's letter, who confused the overall death rate with an analysis of subgroups).

It is logical to expect some relationship between a favourable response to thrombolytic treatment and the time interval be-

tween the onset of symptoms of myocardial infarction. In the report of the European Working Party no significant difference between the two treatment groups was obtained when the patients were retrospectively grouped according to the time-interval between the onset of acute symptoms and the start of the infusion. In the first 12 hours subgroup the hospital mortality (21 days) was 17.1% in the streptokinase group versus 20.2% in the heparin group; in the 12 to 24 hours subgroup the corresponding figures were 22.7% versus 29.1%. These results are at variance with other trials conducted in patients with myocardial infarction admitted to general wards.^{1,2}

The weight of Dr. Heikinheimo's criticism is that though all patients received oral anticoagulants at the start of treatment the control group had a heparin infusion lasting as long (namely 24 hours) as the streptokinase infusion in the experimental group. The report clearly states that, at the time the European trial was planned, it was still the prevailing view that oral anticoagulants supplemented with heparin for the first few days was part of the conventional treatment in acute myocardial infarction. This opinion apparently was shared also by Dr. Heikinheimo, since one of the four treatment centres of the joint trial on thrombolytic treatment in myocardial infarction was co-ordinated in Finland by him in which oral anticoagulants and heparin were administered simultaneously to the experimental and control groups.³ More recently serious doubts have been raised as to the validity of using combined oral and intravenous (heparin) anticoagulants in these patients; this problem is being discussed elsewhere.⁴

Dr. Heikinheimo's final remark, which alleges that the rules of common sense were broken and that streptokinase is now recommended for recent myocardial infarction,

seems unfair. The discussion of the report indeed clearly indicates that the present evidence of the benefit of streptokinase in patients with acute myocardial infarction is too frail to permit a broad recommendation. Further stringent experimentation in patients with recent myocardial infarction admitted to general wards and to coronary care units should therefore be conducted by non-committed groups. There is also an ethical motivation for clinical investigators who are engaged in this field not to leave an issue of this importance unsettled in order to avoid imprecise usage of thrombolysis based on incomplete evidence.—I am, etc.,

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¹ Schmutzler, R., *et al.*, *Deutsche Medizinische Wochenschrift*, 1966, **91**, 581.

² Schmutzler, R., *et al.*, *International Congress of Haematology Abstracts*, Munich, 1970, 286.

³ Heikinheimo, R., *et al.*, *Acta Medica Scandinavica*, 1971, **189**, 7.

⁴ Verstraete, M., *Angiologica*, 1971, **8**, 43.

Treatment of Bell's Palsy

SIR,—Dr. D. Taverner with various colleagues has contributed much to our understanding of the factors governing the prognosis and treatment of Bell's palsy. In the latest paper (2 October, p. 20) Dr. Taverner and his colleagues conclude, "At present we believe that oral prednisolone is the treatment of choice in Bell's palsy and should be given in full doses from the day of onset." A nine-day course, starting with 80 mg daily for the first five days, is advised.

The practical problem arises, "Which patients with Bell's palsy should be so treated?" The possible side effects of prednisolone in certain patients have already