Dipyridamole in Acute Myocardial Infarction

SIR,—The results of oral treatment of myocardial infarction with dipyridamole (Persantin) reported by Dr. A. E. Gent and others (9 November, p. 366) do not quite agree with our own experience. Considering the mechanism of action of the substance—so far as it is known—one can expect only a limited effect in the first month after an acute infarction. In our series of 52 cases of myocardial infarction half the patients were treated with Persantin infusions (60 to 100 mg. per day). The electrocardiographic findings and the clinical course were more favourable in the Persantin group, and the mortality rate was lower (1 against 5).

Such results have to be evaluated critically because, owing to the relatively small number of cases, several risk factors in the course and prognosis of acute myocardial infarction cannot be taken into consideration. We did not observe any complications due to intravenous therapy with dipyridamole. In particular, the hypotension, which has frequently been mentioned, was never appreciable.

The large number of publications on clinical and animal experimental studies leave no doubt that Persantin has a favourable effect on the coronary circulation when it is administered in sufficiently high doses and treatment is continued for several months.¹⁻³ Hence the long-term treatment of coronary insufficiency (whether infarction has occurred or not) with dipyridamole is worth while in any case. We are at present studying the question of whether or not long-term Persantin treatment significantly increases the 5-year survival rate after myocardial infarction. The results so far suggest that the answer is yes. As the occurrence of an infarction is conclusive evidence of the presence of coronary insufficiency without sufficient collateral supply, it appears appropriate to start long-term Persantin therapy as early as possible and continue for at least six months. We do not yet know whether intravenous therapy is superior to oral administration in the acute phase.—I am, etc.,

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Sheepskins and Tetanus

SIR,—The use of sheepskins as bedpads in the prevention of bedsores is well established.¹⁻⁴ In a reply to "Any Questions" in the *B.M.J.* it is suggested that sheepskins as used in hospitals are a potential source of tetanus organisms. The answer offers no experimental evidence, but references are given to reports indicating the presence of *Clostridium tetani* on living sheep and greasy wool. It ignores the effects of tannery procedures. It also ignores the wider issue of the presence of spores of *Cl. tetani* and *Clostridium welchii* in hospital air and dust.

Because sheepskins have proved a valuable nursing aid we set out to determine whether

the process of tanning sheepskins was effective in destroying spores of *Cl. tetani*. The results will be published in detail elsewhere, but we wish to draw attention to a summary of the findings.

The experiments were conducted using small squares of dried raw sheepskin, sterilized by gamma irradiation and then heavily contaminated with tetanus spores. These were then put through various tannery procedures carried out under sterile conditions in the laboratory. After each stage in the process a number of samples of the squares were removed and cultured under appropriate conditions for *Cl. tetani*. The results are given in the Table.

Tanning Procedure	Result of Culture for <i>Cl. tetani</i>
Scouring and rinsing	Heavy growth
24 hours pickling	Heavy growth
4 hour chrome tanning	Occasional growth
48 hours chrome tanning	No growth
Combined chrome glutaraldehyde tanning	No growth
Chrome followed by glutaraldehyde tanning*	No growth

* Glutaraldehyde concentration a tenth that recommended by the manufacturer⁵.

It was also found that typical dyeing procedures were effective in markedly reducing the contamination with *Cl. tetani* spores.

We therefore make the following conclusions: Commercial tanning methods effectively sterilize sheepskins so that there is no risk of their conveying tetanus spores to hospitals; the sporidical effect of glutaraldehyde⁵ is confirmed when used on sheepskins; and chromeglutaraldehyde tanning is particularly suitable for sheepskins in hospital nursing practice. It also leaves a fleece with an attractive primrose colour.—We are, etc.,

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Fibrinolytic Activity and Sickle-cell Crises

SIR,—In an earlier study¹ a group of patients in severe crises of sickle-cell anaemia (SS) were shown to have significantly lower fibrinolytic activity in the blood than control groups consisting of "well" SS patients or hospitalized non-SS patients. Because that study compared activity in different patients rather than in the same one during periods of well-being and ill-health, it could not be claimed with certainty that the occurrence of a crisis is associated with disturbance of fibrinolytic activity in a given patient. A sequential study of four SS patients and 10 inpatient controls with haemoglobin phenotype AA is now reported.

All patients, except A and D who were studied as outpatients, were admitted to Mulago Hospital, Kampala, during their illness. The technique for dilute blood clot

The lysis times and plasma fibrinogen levels in SS patients are shown in Table I. All patients showed an increase in lysis time, compared to the precrisis level, during a crisis, returning to the precrisis range after recovery. There was no significant change in plasma fibrinogen level on recovery or with changes in lysis time. The clinical findings in these patients were as follows:

Patient A, a male aged 19 years, presented with a tender swelling over his tibia of three days' duration. He recovered by day 4.

Patient B, an 8-year-old female, presented with fever, abdominal pain, and vomiting of three days' duration thought to be due to an abdominal crisis. She recovered in three days.

Patient C, an 11-year-old female, presented with fever and pain in upper arms lasting a day. She grew worse and on day 3 developed a tender soft tissue swelling over her right elbow. On

TABLE I.—Lysis Time (Hours) in SS Patients

Patient	Pre-crisis	Days of Admission						Days before Postcrisis Tests	Postcrisis Days		
		1	2	3	4	5	6		1	2	3
A	3.5	23.2	9.2 (154.3)*	9.0	11.4	—	—	11	2.6 (149.6)*	6.8	4.8
B	9.3	24.0	24.0 (313.7)*	21.8	—	24.0 (184.2)*	—	9	8.0 (174.2)*	3.4 (104.6)*	4.4 (191.6)*
C	3.3	10.8	24.0	17.1 (256.3)*	14.3	—	11.9 (368.4)*	7	5.7 (293.8)*	4.5	14.9
D	—	24.0	8.9	6.3	—	—	—	9	10.5	—	—

* The values in parenthesis show plasma fibrinogen concentration in mg./100 ml.

lysis time was the same as used in our earlier study. In SS patients A, B, and C, a single random estimation of lysis time had been obtained when they were well, two to three months before the crisis ("precisis lysis time"). Lysis time was measured daily during crisis and again after the patient had been well for 7 to 11 days ("postcrisis lysis time"). The AA controls were studied daily for three days after admission. Plasma fibrinogen was measured using the method of King and Wootton.²

day 4 her condition was unchanged but she improved thereafter.

Patient D, a male aged 18 months, had fever and cough for two days. No infective cause was found and he was thought to have a febrile crisis. He became afebrile on day 2.

The diagnosis and lysis times in the control group are shown in Table II. There is no prolongation of lysis time with bed rest or infection.

The study confirms that disturbances of fibrinolytic activity are associated with crises in sickle-cell anaemia, although the nature of