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 ("precursor 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

this association is not clear. The disturbance does not appear to be due to infection or plasma fibrinogen changes as patients with an infection (G, J, M, and P) showed short lysis times, nor did the plasma fibrinogen levels change significantly with changes in lysis time or the occurrence of crises. Bed rest or hospitalization factors did not cause the changes, since, if these were operative, prolonged lysis times would be expected in most hospitalized patients.

TABLE II.—Lysis Time (Hours) in Control Patients

Patient	Diagnosis	Days of Admission		
		1	2	3
E	Rheumatic arthritis ..	3.3	10.8	7.2
F	Iron deficiency anaemia ..	24.0	19.3	16.0
G	Infective ulcers of toes ..	0.8	6.1	4.5
H	Iron deficiency anaemia ..	1.3	5.0	2.9
J	Pyogenic abscess of anterior abdominal wall ..	5.0	4.5	4.5
K	Pain in the neck, cause uncertain ..	24.0	19.1	—
L	Food poisoning ..	4.1	2.1	2.8
M	Lobar pneumonia ..	12.9	5.5	5.0
N	Acute falciparum malaria ..	5.0	3.3	5.1
P	Bronchopneumonia (non-tubercular) ..	4.3	1.7	—

The fibrinolytic disturbance could be the result or a contributing cause of thrombotic crises. In view of the latter possibility, further studies to elucidate the nature of the association and a trial of fibrinolytic therapy—for example, with a combination of phenformin and ethyloestradiol³ in the prevention of crises appear indicated.

I am grateful to Professor B. M. Laurance, Professor A. G. Shaper, and Dr. D. M. Macintosh for their advice, and to Mrs. I. Patel for plasma fibrinogen estimations. The work was carried out under a grant from the Medical Research Council.

—I am, etc.,

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Hiatus Hernia

SIR,—Your leading article on hiatus hernia (21 September, p. 691) and subsequent letters (12 October, p. 119) prompt me to make certain comments.

Your concluding paragraph stating that "Only a more troubled minority find their way there [to the hospital], and only a small number of these will ever require operative treatment" is a very fair statement. Why then do many surgeons feel that more cases should be operated upon? I am quite sure this is due to a misunderstanding of the basic facts. The symptoms of hiatal hernia depend upon the presence, or absence, of a functioning inferior oesophageal sphincter. In both cases primary conservative treatment often causes the symptoms to abate. If this treatment fails then the operative procedure must vary; in those with a sphincter reduction of the hernia and crural herniorrhaphy is all that is necessary, whereas in those without a functioning sphincter another procedure—

my own preference is for the Nissen's fundoplication—may be necessary.

In spite of these provisos there is still a very high postoperative incidence of recurrent symptoms, hiatal hernia, or both after prolonged follow-up, and then we are back to the preoperative status. In fact reflux is not as "virulent" a condition as has been made out in the past. What is refluxed is also not important, but what is important is the sensitivity of the mucosa. Stricture incidence is, likewise, much lower than is indicated in the literature. Many cases which have been diagnosed as strictures are really spasms of the oesophagus and are relieved by antispasmodics.

In essence, therefore, I feel that the majority of those hiatus-hernia cases referred to hospital should be treated conservatively, and only a very small percentage, because of associated disease or because of absolute failure of conservative treatment, should be subjected to surgery, with the proviso that one must be careful about the final prognosis.

—I am, etc.,

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Treatment of Skin Fissures

SIR,—In winter many older people suffer from painful fissures of the skin at the tips of the fingers and thumbs. In my experience the only certain cure is a thick application of a liquid cement, such as Durofix. When it is first applied it tends to sting, but only for a few minutes. It is allowed to dry. If necessary it must be renewed to keep the firm coat in place for at least five days. It stands washing and all ordinary use. I have never had to use it for longer than five days, but there would be no harm in doing so if the fissures were still present.

To prevent recurrence patients should be warned against using strong detergents without wearing rubber gloves.—I am, etc.,

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Tests for Hearing

SIR,—I entirely agree with Mr. R. J. Sellick's remarks in his letter (14 December, p. 706). Not infrequently children are referred to the ear, nose, and throat department at Great Ormond Street with school-clinic audiograms depicting hearing loss, but caused in actual fact by cerumen occluding the aural canals.

It is a waste of everyone's time to perform audiometry without a previous aural examination to exclude meatal obstruction.—I am, etc.,

London W.1.

H. S. SHARP.

E.C.G. in Diagnosis

SIR,—Dr. David Short in his well-documented paper (14 December, p. 673) rightly draws attention to the limitations of the electrocardiogram in the diagnosis of acute myocardial infarction. I would, however, disagree with his implied conclusion that an E.C.G. service for general practi-

tioners is undesirable. It is, after all, part of a doctor's training to assess and correlate x-ray, pathological, and other reports and sometimes, in the light of clinical findings, to disregard some of these reports and give due weight to others. It is not considered necessary in every case for a pathologist or radiologist to examine a patient in order that a diagnosis be arrived at.

A competent general practitioner, suspecting a myocardial disorder, should often himself be capable of "critical evaluation of history and clinical findings"; he may not necessarily be in need of consultant's advice but he should not, I submit, be denied the help of an E.C.G. report where this can be arranged.—I am, etc.,

St. Albans,
Herts.

J. HORTON.

Keloids

SIR,—I was interested to read your leading article on keloids (5 October, p. 3). As you so rightly emphasize, they are common in races with pigmented skin, and by and large people with pigmented skins do not live within easy reach of departments of radiotherapy.

Massive keloids in rural Nigeria are not uncommon. One aspect of the condition is seldom mentioned in the literature. The proliferating mass of collagen overgrows cutaneous hair, and eventually the tumour becomes a warren of pilonidal abscesses, constantly discharging rancid pus. The commonest site for such lesions, in my experience, is in the front of the neck. The results of treating the lesions without radiotherapy are appalling. But it is difficult to do nothing when a near-frantic patient pleads to have an offensive non-malignant tumour removed from right under his nose.

The lot of the tropical surgeon, like that of the policeman, is not a happy one.—I am, etc.,

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G. HOLMES.

Intravenous Penicillin

SIR,—We have read with interest the paper by Dr. F. P. Brunner and Dr. P. G. Frick on electrolyte imbalance during therapy with "massive" intravenous penicillin (30 November, p. 550), and would like to make the following comments.

The authors have made the point that a hyperosmolar infusion leading to water depletion was the cause of hypernatraemia. On one occasion we have experienced a similar complication, but this is easily prevented. It is worth notice that the method and vehicle of penicillin administration may be important. When patients were given their penicillin dissolved in 200 ml. of 5% dextrose administered 3- or 4-hourly through a continuously running physiological saline infusion hyponatraemia was often present.

We have also noted low serum-potassium values usually accompanied by low blood-urea levels and have ascribed this to urinary potassium loss. Although Brunner and Frick measured the urinary potassium they make no comment on urine volumes, and this could explain their rather low serum-potassium