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, 2, 3 Hence the long-term treatment of coronary insufficiency (whether the infection 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.,

MARTIN FRIEDEMANN.

REFERENCES
1 Friedemann, M., Schweiz. med. Wschr., 1966, 96, 156.

Sheepskins and Tetanus

SIR,—The use of sheepskins as bedpads in the prevention of bedsores is well established. 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. This reply was accompanied by the following statement: "A source of contamination is of course not to be excluded."

It is well known that adult sheep and lambs are occasionally infected with tetanus spores. In the field of industry it is a common practice to test sheepskins by culturing them; it is also known that Pasteur's studies of sheepskin tanning procedures have shown conclusively that sheepskins are not always free from tetanus spores and, consequently, that sheepskin tanning procedures have been suitable for use 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 study 1 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 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 ("precrisis lysis time"). Lysis time was measured daily during crisis and again after the patient had been alive for at least six days after the crisis. On 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 suggestion 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

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pre-crisis Days of Lysis Time</th>
<th>Days Before Postcrisis Lysis Time</th>
<th>Postcrisis Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>3-5</td>
<td>23-2 (194-2)</td>
<td>11</td>
</tr>
<tr>
<td>B</td>
<td>9-3</td>
<td>24-0 (317-2)</td>
<td>9</td>
</tr>
<tr>
<td>C</td>
<td>3-3</td>
<td>10-8 (256-3)</td>
<td>11</td>
</tr>
<tr>
<td>D</td>
<td>24-0</td>
<td>8-9 (368-3)</td>
<td>9</td>
</tr>
</tbody>
</table>

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

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 lysine times while 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, protracted lysis times would be expected in most hospitalized patients.

**Table II.—Lysis Time (Hours) in Control Patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Days of Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Rheumatic arthritis</td>
<td>1 2 3</td>
</tr>
<tr>
<td>F</td>
<td>Iron deficiency anemia</td>
<td>240 192 160</td>
</tr>
<tr>
<td>G</td>
<td>Infective ulcers of toes</td>
<td>08 61 45</td>
</tr>
<tr>
<td>H</td>
<td>Iron deficiency anemia</td>
<td>13 50 29</td>
</tr>
<tr>
<td>J</td>
<td>Pyogenic abscess of anterior abdominal wall</td>
<td>50 45 45</td>
</tr>
<tr>
<td>K</td>
<td>Pain in the neck, cause uncertain</td>
<td>240 191</td>
</tr>
<tr>
<td>L</td>
<td>Poiso. from</td>
<td>21 21 28</td>
</tr>
<tr>
<td>M</td>
<td>Lobar pneumonia</td>
<td>129 55 50</td>
</tr>
<tr>
<td>N</td>
<td>Acute fallopian tube</td>
<td>50 33 51</td>
</tr>
<tr>
<td>P</td>
<td>Bronchopneumonia (non-tuberculous)</td>
<td>43 17</td>
</tr>
</tbody>
</table>

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 ethyloestranol—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.

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4 January 1969

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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 is there a trend 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 minor reduction of the hernia and cural 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 “vulgar” a condition as has been made out in the past. What is refuxed 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 anti-spasmodics.

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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**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 mental obstruction.—I am, etc.


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**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 myocardial infarction. I would, however, disagree with his implied conclusion that an E.C.G. service for general practi-

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**Intravenous Penicillin**

Sir,—We have read with interest the paper by Dr. F. P. Brunner and Dr. F. G. Frick on “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 hypernatremia. On one occasion we have experienced a similar complication, but this is easily prevented. It is worth notice that the method and dose 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 hypotension 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 for such lesions in potassium they make no comment on urine volumes, and this could explain their rather low serum-potassium