LETTERS TO THE EDITOR

Scope
Heart welcomes letters commenting on papers published in the journal during the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation
Letters should be:
- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors

They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions in the January 1999 issue of Heart (page 104).

Diabetes and coronary artery disease: time to stop taking the tablets

EDITOR.—We write in response to the editorial “Diabetes and coronary artery disease: time to stop taking the tablets.”1 The authors highlight previous studies where diabetics treated with sulphonylureas have an excess cardiovascular mortality during myocardial infarction compared with diabetics treated by other means. As Connaughton and Webber point out, the concentrations of sulphonylureas required to activate cardiac KATP channels is between 100 and 1000 times higher than those required to induce pancreatic insulin release. These observations raise serious doubts as to whether glibenclamide used to treat diabetes will block ischaemic preconditioning.

In conclusion, Connaughton and Webber suggest the need for clinical trials to support the theoretical superiority of insulin. Such a trial has now been published—3867 newly diagnosed diabetics, randomized to glibenclamide or sulphonylurea, insulin or diet alone.2 Over a 10 year follow up the outcome of these treatments were compared and no difference was found in the rate of myocardial infarction or diabetes related death between participants assigned sulphonylurea or insulin treatment.

We agree with Connaughton and Webber that the time to stop taking the tablets is therefore “not yet”.

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Reduction in time delays in administering thrombolytic therapy in acute myocardial infarction

EDITOR.—Rao and Joseph’s correspondence in Heart highlighted the reduction in time to administration of thrombolytic therapy by direct administration of patients with suspected acute myocardial infarction to the coronary care unit (CCU) by ambulance staff who had been trained in reading ECGs.

There are four models for admission to hospital of patients with suspected acute myocardial infarction:

(1) The patient is evaluated in the A&E department where the first ECG is recorded, then the patient is admitted to CCU where thrombolytic therapy is administered.

(2) The patient is admitted to the A&E department, the ECG is recorded and thrombolytic therapy administered.

(3) The patient is admitted directly to the CCU after out-of-hospital ECG recording by paramedics or general practitioners.

(4) ECG is recorded before hospital admission (at home or in the ambulance) by paramedics and transmitted immediately by “telephone” to the receiving CCU where the attending cardiologist can analyse it; thrombolytic therapy may be administered before admission to the A&E department.

The last model is quite novel and does not require additional resources as large numbers of ambulance personnel will not require training in reading ECGs and the A&E department does not need to evolve a system for admitting suitable patients directly to the CCU. The ECG diagnostic accuracy in one study was 92% in the typical chest pain group with ischaemic ST segment changes. The time to ECG recording was shorter when done in the prehospital setting than when done after admission to the A&E department (mean (SD) 8 (6) vs 21 (12) minutes; p < 0.001).

Other factors may influence the delay to thrombolytic treatment and the method of administration is important as bolus administrations needs less time than an infusion. In addition, the overall “pain to needle time” is important in reducing infarct size and improving survival. Koren et al’s study first demonstrated that early administration of thrombolytics provided a gain in terms of left ventricular function and necrotic tissue mass if the “time to needle” was less than 90 minutes. The delay in administering thrombolytics, by infusion or bolus, was not as important as overall “pain to needle time” in reducing infarct size and ameliorating LV function. Therefore, greater use of ECG telephonic transmission and reporting, and prehospital bolus administration of thrombolytics may be significant in reducing infarct size and improving survival as they might shorten the “pain to needle time”.

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1 Rao AC, Joseph SP. Reduction in time delays in administering thrombolytic treatment [correspondence]. Heart 1998;79:422.


Figure 1 Absolute number of deaths avoided and absolute excess of strokes/1000 patients treated with different thrombolytic agents compared to prethrombolytic era on the basis of the result of the respective superiority or equivalence trials. Numerical data indicate point estimates and limits of 95% confidence intervals (horizontal bars). Mortality and stroke rate were assumed to be 10% and 1.5%, respectively, in patients not treated with thrombolytic agents.

<table>
<thead>
<tr>
<th>Thrombolytic Agent</th>
<th>Deaths avoided per 1000 patients treated with</th>
<th>Excess of strokes per 1000 patients treated with</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase</td>
<td>16</td>
<td>22</td>
</tr>
<tr>
<td>tPA</td>
<td>18</td>
<td>25</td>
</tr>
<tr>
<td>Reteplase</td>
<td>11</td>
<td>26</td>
</tr>
<tr>
<td>Saruplase</td>
<td>11</td>
<td>34</td>
</tr>
<tr>
<td>GISSI-I and ISIS-2</td>
<td>GISSI-2, ISIS-3, GUSTO-1</td>
<td>tPA</td>
</tr>
<tr>
<td></td>
<td>(14452) v no thrombolytic (14447)</td>
<td>(44888) v streptokinase (44420)</td>
</tr>
<tr>
<td></td>
<td>-3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>+5</td>
</tr>
<tr>
<td></td>
<td>+12</td>
<td>tPA</td>
</tr>
<tr>
<td>INJECT</td>
<td>tPA</td>
<td>Reteplase</td>
</tr>
<tr>
<td></td>
<td>(3004) v streptokinase (3006)</td>
<td>reteplase (1013B) v alteplase (4921)</td>
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<tr>
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<tr>
<td></td>
<td>+4</td>
<td>+10</td>
</tr>
<tr>
<td></td>
<td>+18</td>
<td>Reteplase</td>
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<td>GUSTO-3</td>
<td>reteplase (1542) v streptokinase (1547)</td>
<td>saruplase</td>
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<td>+1</td>
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<tr>
<td></td>
<td>+16</td>
<td>saruplase</td>
</tr>
</tbody>
</table>

Figure 1: Absolute number of deaths avoided and absolute excess of strokes/1000 patients treated with different thrombolytic agents compared to prethrombolytic era on the basis of the result of the respective superiority or equivalence trials. Numerical data indicate point estimates and limits of 95% confidence intervals (horizontal bars). Mortality and stroke rate were assumed to be 10% and 1.5%, respectively, in patients not treated with thrombolytic agents.
Nurse led, multidisciplinary intervention in chronic heart failure

EDITOR,—To complement the editorial by McMurray and Stewart,1 I present the results of a recent study from the Netherlands in which we randomized 179 high-risk patients (mean age 73 years), hospitalised with heart failure to intervention by a specially appointed nurse or to usual care. The intervention was intensive, systematized, and planned education by a study nurse about the consequences of heart failure in daily life, using a standard nursing care plan. During hospital stay, the study nurse assessed patients’ needs, provided education and support to patients (and family), gave patients a card with warning symptoms, and discussed discharge. Within one week after discharge the study nurse telephoned patients to assess potential problems and to make an appointment for a home visit. During the home visit the study nurse reinforced and continued education as warranted by the patient’s situation. If needed, home care was informed in writing about specific patient needs. Between discharge and home visit, patients could call the study nurse in case of problems. After the home visit, the patient was advised to call their cardiologist, general practitioner or emergency heart centre in case of problems. Therefore, the intervention lasted from hospital admission to 10 days after discharge from hospital. Data were collected on resource utilisation and a trend was described (p = 0.06) towards fewer readmissions and visits to the emergency heart centre in the intervention group.

The main focus of the intervention was education and support by a nurse and follow up of the intervention lasted to 10 days after discharge. The study provides insight in the particular effect of education and support by a nurse. Our results show that this limited intervention is effective to enhance self care, but more is needed to get statistically significant results on readmission. The information is valuable in determining the required “dose” of nursing interventions. This confirms McMurray’s and Stewart’s editorial that describes the importance of determining which aspects of the intervention work. I would like to add two points to the list of issues regarding implementation and achieving optimal cost–benefit mentioned by McMurray and Stewart.1

1. There is a huge difference in the populations in the published studies: Rich et al and Stewart et al investigated a high risk sample for hospital readmission.1,1 This means that a specific subgroup (high risk patients) of the very heterogeneous heart failure population can benefit from that specific intervention. Other researchers studied patients from a transplant clinic,1 which also had to be noted before generalising results to a general clinical heart failure population. Comparing all these studies in an overview as provided in the editorial can be helpful, but caution should be used when applying the results to practice.

2. End points in effect studies should be standardised as much as possible. There is a great difference between studies reported in the editorial. Some authors used rehospitalisation as a primary end point and others combined this with mortality. Accumulating end points to a “major” variable (such as rehospitalisation and mortality) may increase the power of studies, but it sometimes makes comparison with other studies difficult.

In addition, I would like to support the authors’ plea for inclusion of variables that explain the mechanism of (beneficial) effects of intervention (such as compliance or self care). In this way we might have a better insight as to which intervention (and which “dose”) is most appropriate for which heart failure patient.

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NOTICES

Inflammation in cardiovascular disease, a conference hosted by the Royal College of Physicians of London, will be held on 22 September in London, UK. For further details, please contact Royal College of Physicians, Conference Office, 11 St Andrews Place, Regent’s Park, London NW1 4LE, UK; tel: +44 (0)171 935 1174 ext 252/300/436; fax: +44 (0)171 487 5218; email: conferences@rcplondon.ac.uk.

The world congress on non-invasive and invasive cardiology will be held in Rajkot, Gujarat, India from 24–26 December 1999. For further details visit www.cardio99.com.

European conference on management of coronary heart disease will be held at the Acropolis Convention Centre in Nice, France from 17–19 April 2000 (abstract deadline 12 November 1999). For further details please contact Castle House Medical Conferences, 5 Linden Close, Tunbridge Wells, Kent TN4 6HH, UK; tel: +44 (0)1892 539666; fax: +44 (0)1892 517773; email: conferences@castlehouse.co.uk; web site: www.castlehouse.co.uk.

Seventh world congress on heart failure—mechanisms and management will be held in Vancouver, Canada from 9–12 July 2000 under the auspices of the International Society of Heart Failure (abstract deadline 29 February 2000). For further details please contact Dr Asher Kimchi, Chair, 7th World Congress on Heart Failure, PO Box 17659, Beverly Hills, CA 90209, USA; tel: +1 310 657 8777; fax: +1 310 275 8922; email: klimedico@ucla.edu; web site: www.cardiomyonline.com.
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