LETTERS TO THE EDITOR

Cardioesophageal Reflex: A Mechanism for “Linked Angina”

In the June issue of the Journal, Chauhan et al (1) reported a potentially very relevant study on the pathophysiology of “linked angina”. Their results show that infusion of hydrochloric acid into the esophagus dramatically decreases coronary blood flow and induces chest pain in a majority of patients with proven coronary artery disease. Because these effects were not seen in heart transplant recipients, the authors suggest a neural pathway for linked angina. Figures 3 and 4 of the report illustrate some of the key findings (i.e., the lack of both saline and acid to affect coronary blood flow in heart transplant recipients). However, we were surprised to see that the two figures are not only similar but identical. It is unlikely that the figures were simply mixed up because the numerical values (both the mean values and standard deviations) given in Table 3 for coronary blood flow for acid and saline experiments are also identical. Because this study may have important implications, we would be most grateful for an explanation of these statistically most improbable findings.

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Reference

Reply

In response to Koelz and Bertel, Figures 3 and 4 in our report (1) are indeed superimposable. However, the figures do hide some differences in individual patients. It can be seen from Table 3 of our report that there was no significant change in coronary blood flow after acid and saline infusions in the transplantation group. The actual and coronary blood flow (CBF) values in the transplant recipients before and after the saline infusion were virtually identical to the responses seen before and after the acid infusion except for four patients (Table 1): Therefore, Patients 1 and 2, and 3 and 4, are mirror images. However, this does not reflect in the scatterplot because the individual patients are not labeled. Although the response to saline infusion was different in these patients, the scatterplot is unable to demonstrate this difference, and the figures, in effect, are identical. The mean values are also therefore identical (as shown in Table 3 of our report [1]). Further, we previously demonstrated that coronary blood flow velocity measurements remain relatively constant under rest conditions of stable heart rate and arterial pressure (2). It can be seen from Table 3 that the rest heart rates and systolic pressures were similar before and after the infusions. In the absence of any effect of the infusions in the denervated heart transplantation group, one would indeed see very similar blood flow velocity measurements and hence the calculated blood flow under stable rest conditions.

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Table 1. Coronary Blood Flow Values in Four Transplant Recipients

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Rest Coronary Blood Flow (ml/min)</th>
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<tbody>
<tr>
<td></td>
<td>Before Acid Infusion</td>
</tr>
<tr>
<td>1</td>
<td>43</td>
</tr>
<tr>
<td>2</td>
<td>43</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
</tr>
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</table>

Pt = patient.

Doppler Evaluation of Patients With Constrictive Pericarditis: Use in Tricuspid Regurgitation Velocity Curves to Determine Enhanced Ventricular Interaction*

Building on the pioneer work of Hatle et al. (1), investigators, principally from the Mayo Clinic, have shown how Doppler echocardiography can be used to distinguish between constrictive pericarditis and restrictive cardiomyopathy. Of equal importance, these studies have elucidated the pathophysiology of these conditions. The publication by Klodos et al. (2) in a recent issue of the Journal adds significantly to this body of work and raises some important methodologic issues as well an interesting question concerning the pathophysiology of constrictive pericarditis. The first methodologic issue to which I would draw attention is the failure of many cardiologists, in their enthusiasm to demonstrate equalization of diastolic pressures, to obtain recordings at low gain of pressures simultaneously from the two ventricles or great arteries to enable examination of the phase relation of their respiratory variation in systolic pressure. Second, Klodos et al. evaluated respiratory variation of the pressure gradient between pulmonary wedge and left ventricular diastolic pressures, although accurate determination of respiratory variation in so small a pressure difference would require digitizing the pressures and subtracting electronically, preferably using critically damped pulmonary venous rather than pulmonary wedge pressures.

*The authors of the cited study have declined to prepare a response to this letter.
Katz and Gauchat (3) proposed that in cardiac tamponade, the decrease in thoracic pressure that accompanies inspiration is blocked from transmission to the right heart chambers by the elevated pericardial pressure, thereby preventing the normal increase in systemic venous return with inspiration. Subsequently, it was shown (4) that in tamponade, most of the drop in thoracic pressure during inspiration is transmitted to the heart and pericardium, and systemic venous return is augmented by the normal mechanism despite raised pericardial pressure. However, in constrictive pericarditis the heart is effectively insulated from respiratory fluctuation of thoracic pressure. This insulation prevents the development of an increased pressure gradient for right heart filling during inspiration and, as emphasized by Klodas et al. (2), diminishes the pressure gradient responsible for left ventricular filling. Consequently, because total cardiac volume remains constant throughout the respiratory cycle in constrictive pericarditis, right heart volume increases. These data lead to the conclusion that in cardiac tamponade, inspiratory increase in systemic venous return is a primary event leading to diminution of left heart volume, whereas in constrictive pericarditis, diminished pulmonary venous return is the primary event that leads to increased right heart volume. Had Katz and Gauchat postulated their proposed mechanism for constriction instead of tamponade, they would have been correct.

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References

Coronary Bypass Graft Surgery and Patient Outcome

FitzGibbon and co-workers have previously made important contributions to our knowledge about the results of coronary bypass surgery (CBS). In their recent study (1), they report the long-term results in 1,388 patients (all but 12 were men) who underwent CBS from 1969 to 1994. This study has important data, particularly with regard to the long-term fate of vein grafts (graft disease and graft occlusion) up to ≥15 years and especially about its effect on survival; the authors are to be congratulated in providing these data.

They have also compared their results in men with our data (2) in 5,468 men operated on from 1969 to 1988 (and in the late cohort 1974 to 1988); however, they inadvertently did not cite our more detailed study (3), based on gender, of the results of CBS in 6,927 men versus 1,979 women who underwent CBS from 1974 to 1991. They presented very few baseline (preoperative) characteristics of their patients; for example, the incidence of diabetes, previous myocardial infarction, three-vessel coronary artery disease or left ventricular dysfunction is not given. Therefore, we believe that one should be very careful about a comparison with our study because of possible baseline differences in the patients. They did provide data on the ages of the patients. Their patients’ ages ranged from 27 to 79 years (mean 48.9 years), and they emphasized the differences in the percent of patients ≤44 years old in their and our study. A higher percentage of patients in their study were ≤44 years old. However, an important age group is ≥55 years of age. In their study, only 267 (19%) of 1,388 patients were ≥55 years old. In our studies, of patients undergoing CBS from 1969 to 1988, the average age of the patients was 60.7 years, and 88% were ≥55 years old (2), and of male patients operated on from 1974 to 1991, the average age was 61 years, and 73% were ≥55 years old (3). In the years 1984 to 1988 and 1989 to 1991, the frequency of male patients ≥65 years old undergoing CBS was 45% and 52%, respectively (3). This is a very important difference because the survival of patients is lower in those who are older at the time of CBS. For example, the 10-year survival rate of patients aged 46 to 54, 55 to 64, 65 to 74 and ≥75 years old at the time of CBS was 86%, 75%, 60%, and 49%, respectively (3). However, the relative survival (age and gender matched to the population) is very favorable in the older patients, as they have shown (1) and which we had previously documented (4). Analysis of survival in our study by Cox regression model showed that the risks of mortality that were statistically significant were older age, previous CBS, previous myocardial infarction and diabetes, with a relative risk of 1.06, 1.57, 1.57 and 1.84, respectively (3). Besides an older age at time of performance of CBS, many baseline preoperative characteristics have changed in patients operated on from 1974 to 1991 (3). This is another reason why one should be very careful about comparing data from the different studies unless baseline preoperative patient characteristics are compared and shown to be very similar.

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References

Reply

We appreciate Rahimtoola’s kind and generous remarks about our work. The critique of the recent report (1), which he has admirably summarized, is important and requires a response. His objection to comparing incomparables is, of course, quite correct. However, we listed comparative information mainly to assure the reader that the survival results were not out of line with other long-term studies because this was not the major thrust of the report.

Rahimtoola draws our attention to the more comprehensive (2) of his recent reports (2,3), whose title does not do justice to its subject. However, the survival data for 6,927 men (2) with coronary artery bypass graft surgery (CABG) do not differ significantly from data for the cohort of 5,648 men (3), with which we compared our results. We reported a total 25-year experience. Our patients were insufficient in