LETTERS TO THE EDITOR

Guidelines For Thrombolytic Therapy

The article by Lengyel et al. (1) on guidelines for thrombolytic therapy of thrombosed prosthetic valves is thorough and practical. However, their conclusion that thrombolysis is not indicated as initial therapy of patients in functional class I or II does not appear to follow from the data they have presented. The authors state that “the reasoning against thrombolysis in patients in functional class I or II is based on the relatively low surgical mortality in this group as opposed to the embolic risk of 12% to 17% caused by thrombolysis.” However, in the next sentence, they point out that in 32 patients presenting in class I or II treated with thrombolytic therapy, there was an 88% success rate, 3% (one patient) major stroke rate, and zero mortality, whereas the lowest reported surgical mortality rate in a similar group is 5%. The major neurologic complication rate after cardiac surgery is about 3%, with a total neurologic complication rate of 6% (2). The authors then recommend use of heparin and warfarin but present no data on its efficacy. They do present data on the use of heparin alone and report a 63% success rate, two cases of cerebral embolism, and one fatal stroke. The complication rates for these three approaches as presented in this review and from the literature is summarized in Table 1.

Comparison of these data suggests that the lowest risk approach is to treat initially class I and class II patients with thrombolysis. Therefore, at our institution, we continue to favor the use of thrombolytic therapy initially in stable patients with St. Jude bileaflet valves (3). Heparin is used in stable patients with contraindications to thrombolytic therapy. Surgery is reserved for patients unresponsive to thrombolytic therapy or heparin therapy and for hemodynamically unstable patients. The primary reason for this latter decision is that patients must be stable enough to tolerate 24–72 h of thrombolytic therapy.

STEVEN S. KHAN, MD
Department of Cardiothoracic Surgery
Cedars-Sinai School of Medicine
Room 6215
8700 Beverly Boulevard
Los Angeles, California 90048-1865
E-mail: khan@csmc.edu

Table 1. Complication Rates of Three Therapies

<table>
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<tr>
<th>Therpay</th>
<th>Success (%)</th>
<th>Death (%)</th>
<th>Stroke (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombolysis</td>
<td>88</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Surgery</td>
<td>100</td>
<td>5</td>
<td>(3)</td>
</tr>
<tr>
<td>Heparin</td>
<td>66</td>
<td>5</td>
<td>10*</td>
</tr>
</tbody>
</table>

*Described as “minor cerebral embolism.”

References

Reply

We thank Khan for his letter regarding our guidelines (1). Based on his center’s experience and our own more recent experience of 18 patients with the treatment of nonobstructive prosthetic valve thrombosis (PVT), I agree with their conclusion advocating fibrinolytic therapy. The main message of our guideline was to extend the classical indications of fibrinolysis in left-sided PVT from the critically ill to the less sick patients.

I may remind Khan that our report summarized the results of a consensus meeting held in 1994. The data on nonobstructive PVT were scarce compared to the data on obstructive PVT at that time. The literature now clearly suggests a higher success rate but still a less evident complication rate in nonobstructive PVT. Embolic complications of thrombolysis are probably more associated with the amount of thrombus as defined by transesophageal echocardiography (TEE) than with its hemodynamic consequence. Khan and his group had not reported the use of TEE but they suggested that the high efficacy and safety of fibrinolysis would be confined to the St. Jude valve (2). However, we could not find such a correlation in the literature.

In some instances, heparin and warfarin may be a logical choice of treatment for patients presenting with inadequate anticoagulation in a stable hemodynamic state or having contraindications to thrombolysis. The latter is the case in most patients presenting with nonobstructive PVT: stroke or recent cardiac surgery (3–5). Interestingly, Khan and his center used heparin successfully even in obstructive PVT and they considered heparin as a sole agent (2). Unfortunately, the success and safety of heparin or warfarin is quite variable in different reports. The size of thrombus again appears to play a decisive role: thrombi smaller than 5 mm tend to resolve without complications in contrast to larger thrombi (3,4).

The possibility of using fibrinolysis either as a first choice in nonobstructive PVT or as a secondary treatment following unsuccessful heparin therapy after 48 h was not excluded in our guidelines. We showed that the decision as to which therapeutic modality should be used depends on several factors such as clinical presentation, anticoagulation status, local surgical expertise, contraindications to thrombolysis and importantly TEE findings. For example, a patient with prosthetic valve obstruction but no TEE evidence of thrombus should not be exposed to the bleeding complications or the deferral of surgery by using thrombolysis. Moreover, it seems to me unjustified to deny a patient the benefits of thrombolytic treatment who has PVT but not on a St. Jude valve.

In conclusion, the question of treatment of nonobstructive PVT is still open and more data are needed for the selection of patients for either thrombolysis or anticoagulant treatment.

MARIA LENGYEL, MD, FACC
Hungarian Institute of Cardiology
H-1450, Budapest, P.O. Box 88
Hungary

References
Passive Smoking and Heart Disease

The recent review article by A. Judson Wells on the association between heart disease and passive smoking in the workplace attempts to compile good data on a very important public health issue. The risks of passive smoke exposure in the workplace have recently received a great deal of attention, such as the recent class action suit by flight attendants. However, I found the data in this review article extremely hard to interpret as nowhere in the Methods or Results was heart disease defined.

The authors did spend some time explaining that the definition for passive smoke exposure varies from study to study, but the other part of the equation is the definition of outcome—in this case, heart disease. It is unclear whether heart disease was defined as chest pain, angina, nonfatal myocardial infarction or cardiac death.

Although the physiologic mechanisms for the association between passive smoking and heart disease have been eloquently explained (1,2), a lack of a definition for heart disease in the Wells study diminishes the usefulness of the findings.

RITA F. REDBERG, MD, MSc, FACC
Echocardiography Laboratory
University of California, San Francisco
505 Parnassus Avenue, Room M342A
San Francisco, California 94143-0214
E-mail: redberg@cardio.ucsf.edu

References

Reply

Dr. Redberg (1) has raised a question as to what the heart disease endpoints were in the various epidemiologic studies that were included in my report on heart disease and passive smoking at work (2). The abbreviated endpoints for the earlier studies are given in Table 1 of my 1994 report (3), and those for the later studies are given in Table A1 of the 1998 report (2). Because these details may be difficult to ferret out, a more descriptive statement of the endpoints for the workplace studies listed in Table 1 of reference 2 are: He, et al., nonfatal coronary heart disease, defined as myocardial infarction or coronary stenosis confirmed by coronary arteriography; Kawachi, et al., nonfatal coronary heart disease; Butler, fatal ischemic heart disease; Svendsen et al., fatal plus nonfatal coronary heart disease event; Jackson, nonfatal myocardial infarction; Muscat and Wynder, nonfatal myocardial infarction; Dobson et al., fatal or nonfatal definite or possible myocardial infarction or coronary death; Steenland, et al., fatal coronary heart disease, also denoted as ICD 410-414.

A. JUDSON WELLS, PhD
5 Ingleton Circle
Kennett Square, Pennsylvania 19348

Significant Arrhythmias During Pericarditis Are Due To Concomitant Heart Disease

Danias et al. (1) contribute an important set of observations regarding spontaneous conversion of atrial fibrillation to sinus rhythm. I have no quarrel with their excellent work but including pericarditis as a cause (“underlying systemic disorder”) of significant rhythm disturbances has been repeatedly shown to be erroneous. (The sterile pericarditis model for induction of atrial arrhythmias has artificial experimental conditions without clinical counterparts [2].) A prospective series of 100 consecutive patients with acute pericarditis continuously monitored for arrhythmia (conservatively defined as 6 ectopic beats per minute or anything worse) (3) and another series of 50 consecutive patients with acute pericarditis having 24-h Holter monitoring (4) each showed significant arrhythmia (same definition) only in patients with additional heart disease. Elegant postmortem investigations of the cardiac conduction system showed that all patients with pericarditis and significant arrhythmias had also had disease of the myocardium or valves (5). Of course, significant myocarditis in the syndrome of myopericarditis can be taken as sufficient heart disease to provoke arrhythmias, but in such cases myocardial involvement is evident (6,7).

These remarks are only to correct a traditional assumption that seems prevalent despite the evidence to the contrary. (This point will be discussed in a Core Curriculum session at the College meeting in Atlanta). The clinical pearl is: if your patient with arrhythmia has pericarditis, look carefully for heart disease.

DAVID H. SPODICK, MD, DSc
Cardiology Division
Saint Vincent Hospital
25 Winthrop Street
Worcester, Massachusetts 01604-4593

References