Letters

Complications of Cardiac Catheterization Versus Coronary Angiography

To the Editor: Questions have been raised about the frequency of complications of cardiac catheterization stated in our article on surgical aortic stenosis, which was published in the February 1996 issue of the *Mayo Clinic Proceedings* (pages 141 to 149). We think that the presentation of these events could be misinterpreted and thus deserves clarification. As we indicated in the article, the morbidity related to cardiac catheterization was low. We also indicated that the complications were more frequent among patients who underwent hemodynamic catheterization (7%) than among those who underwent coronary angiography only (4%). The percentages reported in the article, however, pertain to *all* the complications that occurred; these included hematomas, renal failure, and allergic reaction in addition to the complications specifically cited in the report—pulmonary edema, cardiogenic shock, and ventricular arrhythmias. The breakdown of all complications of hemodynamic catheterization versus those of coronary angiography only is as follows: ventricular arrhythmia, 1 vs 1; ischemia, 1 vs 0; hypotension, 1 vs 0; pulmonary edema, 1 vs 1; hematoma, 6 vs 7; renal failure, 1 vs 0; and allergic reaction, 0 vs 1. Thus, the frequency of *life-threatening* complications in this series was *very low*, and the results of the study remain unchanged.

In addition, we wish to reemphasize that our study was not designed to address the issue of catheterization-related morbidity and mortality of aortic stenosis. Our series *does not* represent the comprehensive experience of our catheterization laboratory relative to aortic stenosis; in particular, nonsurgical patients, whose baseline characteristics and outcome may vary greatly in comparison with those of a surgical series, were not included. Thus, generalizing the figures stated in this series to *all* patients with aortic stenosis undergoing cardiac catheterization would be erroneous.

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Has the Prothrombin Time Stood the Test of Time?

To the Editor: The editorial by Beyth and Landefeld, which was published in the August 1995 issue of the *Mayo Clinic Proceedings* (pages 806 to 808), provoked our concern because warfarin therapy must be monitored carefully to prevent hemorrhage or thrombosis. These conditions occur because monitoring of coagulation changes entails measuring the rate at which blood clots. We invented a modified recalcification time (MRT) test that can fulfill this need.[1]

Patients who are "targeted" for anticoagulant therapy have, in common, accelerated coagulation (hypercoagulability), resulting in thrombosis (intra-arterial, intravenous, or combinations of both in the antiphospholipid syndrome).

Normally, blood flows without the hindrance of clots. Homeostasis is achieved because of a balance between effects of mediators promoting and those opposing thrombosis. This balance maintains the rate at which blood clots within a predetermined, physiologically safe range. Excessive effects of one or the other can result in thrombosis or hemorrhage. Decisions to prescribe anticoagulants are based on clinical evidence of thrombosis and not on the prothrombin time, which has no capability to measure the rate of accelerated coagulation.

By correcting the shortcomings of the commonly employed prothrombin time, we invented the MRT. Unlike other tests, the MRT measures, besides the rate of clotting, the potential for accelerating coagulation. For this test, blood is used to incorporate both cellular and chemical participants in regulating coagulation. Blood must be incubated for 2 hours to promote release of latent mediators of coagulation. In another procedure with the MRT, blood *is* incubated with endotoxin to activate the monocyte to release the potent procoagulant, tissue factor. In each of these procedures, the recalcification time is determined instrumentally, and the results are depicted on a chart—the coagulation spectrum.[1] Details of this test, described in many publications, have been summarized.[1]

The capability of the MRT encourages us to suggest that the prothrombin time "has not stood the test of time."

Availability of a test with MRT capability opens up other diagnostic and therapeutic considerations in patients with accelerated or decelerated coagulation. Similar to the finding reported by Gitter and colleagues,[2] we have found that cancer creates a prothrombotic state that could lead to difficulties in regulating anticoagulation.[3] In our experience, an even more common entity that accelerates coagulation, resulting in similar difficulties caused by cancer, is an inflammatory or infectious process. Nonresponders to anticoagulant measures, as determined by the MRT, can be studied with an algorithmic approach to pinpoint the cause, such as congenital anticoagulant deficiencies.

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