Progression of Aortic Stenosis
Role of Age and Concomitant Coronary Artery Disease

To the Editor:

I enjoyed the recent article by Peter and colleagues from Basel in which they analyzed the rate of progression of Doppler-derived aortic valve gradient and of fractional shortening-velocity ratio in 49 patients. They observed a median rate of progression of 7.2 mm Hg/yr, similar to a number of other studies, including our own (median 6.5 mm Hg/yr in 65 patients studied by left heart catheterization). They also observed that rapid progression of aortic stenosis by ≥10 mm Hg/yr was more common in patients with coexisting coronary artery disease. In reference to our report, they mention that we found only aortic valve calcification to be associated with faster progression—in fact, we also noted that progression was faster in those with coexisting coronary disease (8.1 mm Hg/yr) than in those with rheumatic heart disease (4.1 mm Hg/yr). In addition, aortic regurgitation detectable at the time of the first catheter study was associated with faster rates of progression.

We now have data for 127 patients, with a median rate of progression of 5.4 mm Hg/yr, slightly lower than in the first 65 patients. Where more than two time-points are available for individual patients, progression is often strikingly nonlinear (Fig 1). There are statistically strong correlations between rapid rate of progression and valve calcification, valve regurgitation, and the presence of coronary artery disease. The magnitude of the correlation coefficients (typically 0.3 to 0.4), however, is such that most of the variability in rate of progression remains unexplained. Predicting which patients with mild or moderate aortic stenosis will progress, and how fast, is still a major clinical problem with potentially serious consequences for the patient if the risk is under-estimated. Repeated noninvasive studies with Doppler ultrasound may well prove the answer, and I look forward to further results from the Basel group.

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REFERENCES

Validation of New Pulsed Doppler Echocardiographic Techniques

To the Editor:

I read with interest the Sajkov et al article entitled “Validation of New Pulsed Doppler Echocardiographic Techniques for Assessment of Pulmonary Hemodynamics,” which appeared in the May issue of Chest, and I agree that Doppler methods are ideal for evaluation of pulmonary hemodynamics.

In this study, the authors mention a failure to reproduce contrast enhancement of tricuspid regurgitation jets. They compare this failure with the success we reported in measuring pulmonary artery pressure in severe COPD patients. Those measurements were made at rest and during exercise using contrast enhanced continuous wave (CW) Doppler tricuspid insufficiency jets with a high yield of satisfactory signals. Since the publication, this examination has become standard in our laboratory and is applied to a wide variety of cardiac diagnostic questions (prosthetic valves, congenital heart disease, mitral regurgitation, dyspnea of uncertain cause, etc.). The clinical acceptance of this approach by referring physicians has been gratifyingly enthusiastic.

Why then do we enjoy uniform success with this technique while other expert laboratories like yours are frustrated? The answer is that the most widely used echocardiographic instruments, the Hewlett Packard 500, 1000, and 1500 models (Andover, Mass), have Doppler circuits that are not equipped with automatic gain control. Thus, when the reflectance of the blood pool increases several fold, the receivers are saturated and produce noise. This characteristic makes contrast enhancement of weak Doppler signals difficult or impossible.

Our first studies were done with the now extinct Irex Meridian...
that used Doppler circuitry with automatic gain control. We have continued these studies with similarly equipped Acuson instruments (Sunnyvale, Calif.). Our three Hewlett Packard instruments, while otherwise superb, must sit idle when contrast enhanced CW Doppler examinations are performed.

While I ordinarily eschew highlighting or evaluating commercially available instruments, I am writing this letter to explain what to many investigators must seem a puzzling situation. I also hope that it will encourage Hewlett Packard to put the addition of automatic gain control a bit higher on their list of engineering priorities. With the imminent Food and Drug Administration approval of lung traversing contrast agents, echocardiographers will be anxious and able to enhance Doppler signals from both sides of the heart.

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REFERENCES


To the Editor:

We are very grateful for Dr. Schiller’s letter in which he draws our attention to the technological issues of continuous wave Doppler imaging of the tricuspid regurgitation jet. We were unable to replicate the results of his laboratory in estimation of pulmonary artery pressure with microbubble saline enhanced continuous wave Doppler measurement of tricuspid regurgitation flow (Circulation 1989; 79:865-71) and felt somewhat frustrated. Therefore, we chose the pulsed wave Doppler technique of Morera et al1 (Chest 1993; 103:1348-53). Dr. Schiller indicates that the gain adjustment after the saline injection was the major problem and the tricuspid flow could not be satisfactorily measured with the equipment we used at that time, an ultrasound imaging system (Hewlett Packard 77020a, Andover, Mass.). In fact, we had unsuccessfully tried manually to adjust the gain during Doppler imaging and after saline injection to enhance the signal. We are interested to know that new machines, such as the Acuson, equipped with automatic gain control circuits, overcome these difficulties and produce continuous wave Doppler signals of superior quality. This information should be of general interest to other investigators and laboratories trying to perform noninvasive Doppler measurements of pulmonary hemodynamics. The saline-enhanced continuous wave Doppler technique has some potential advantages over the technique by Morera et al in that it takes less time to obtain images and perform calculations. This and savings in on-line recording paper (often an expensive consumable) should make it a more economic investigation.

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REFERENCE


Oral Anticoagulants

To the Editor:

We read with great interest, the article by Hirsh et al1 published in the October, 1992 supplement to Chest. Their article is a useful review of oral anticoagulants and the new recommendations for anticoagulant therapy. We wish to bring your attention to some apparent confusion regarding the new standards for therapeutic ranges of oral anticoagulants. We believe the confusion resulted from the information in Tables 2 and 4. Although the text of the article discusses the standards for recommended therapeutic ranges for oral anticoagulant therapy as they appear in Table 2, at least one other publication has printed recommendations based on the information in Table 4.2 The differences between Tables 2 and 4 are in the international normalized ratio ranges for prevention of recurrent myocardial infarction and reduction of mortality post myocardial infarction.

We wish to bring this to the attention of your readers to avoid further distribution of two sets of standards. We would appreciate confirmation of which table (2 or 4) provides the information intended to be the current recommended therapeutic ranges.

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2 Karpman HL. INR—the new standard for monitoring oral anticoagulant therapy. Intern Med Alert 1992 (Dec 15); 178-79

To the Editor:

I thank Drs. Smith and Elkhathib for their comments on our article, "Oral Anticoagulants: Mechanism of Action, Clinical Effectiveness, and Optimal Therapeutic Range," which appeared in the October, 1992, Supplement to Chest. Table 2 provides the recommended indication for warfarin and the therapeutic ranges for these indications. Table 4 provides information on the minimum effective international normalized ratio (INR) values for each potential indication, based on the results of randomized studies. Although the use of warfarin was not recommended for the prevention of death or reinfarction in patients with acute myocardial infarction, the drug is effective for this indication. The right hand column of Table 4 provides the recommended INR based on hard evidence, if a decision is made by a physician to use warfarin to treat patients with acute myocardial infarction.

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