# Influence of Cardiac Motion on Doppler Measurements Using Ja Vitro and In Vivo Models

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*Objectives.* Using both in vitro and in vivo techniques, we investigated the extent to which cardiac motion alters Dopplermeasured blood flow velocity and thus potentially can alter the calculation of valve areas or pressure gradients.

*Background.* Blood flow velocity measured by Doppler ultrasound represents the net motion of the blood relative to the transducer. It is widely assumed that the measured velocity represents the actual flow. It has been demonstrated that cardiac motion generates regularly occurring low velocity Doppler signals that are commonly treated as artifact.

*Methods.* We used an in vitro model that allowed us to measure and independently control the flow of a liquid through a chamber and the motion of the chamber relative to the Doppler beam. A cornstarch-water slurry was driven by a pulsatile pump through tubing to simulate the blood flow within the heart, and the tubing was cyclically moved by a piston to simulate the heart motion. We

Blood flow velocity measured by Doppler ultrasound represents the net motion of the blood relative to the transducer. It is widely assumed that the measured velocity represents the actual flow; however, if the cardiac chamber surrounding the blood has independent motion relative to the transducer, the measured flow will be influenced by the motion of the heart and thus will be the vector sum of the chamber velocity and blood flow velocity. The upper panel of Figure 1 illustrates the reason for our interest. It demonstrates a typical pulsed Doppler recording from the left ventricular outflow tract from the apical five-chamber view. In addition to demonstrating the familiar pulsed Doppler signals, it also demonstrates low velocity signals that are seen repeating

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also measured cardiac motion using M-mode and two-dimensional echocardiography and compared the results with the Doppler signal derived from cardiac motion in subjects without cardiac disease.

Results. In the in vitro model, alteration in the motion of the tubing resulted in apparent changes in the measured maximal velocity of the fluid. The Doppler spectrum of the combined motion of the tubing and the fluid was the algebraic sum of their Doppler signals. In human subjects, the maximal slope of the M-mode tracing of the aortic annular motion and the peak Doppler signal due to cardiac motion were compared and were highly correlated.

*Conclusions*. Cardiac motion alters the Doppler signal derived from blood flow. This effect can be demonstrated in vitro and in vivo.

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throughout the recording. These signals are generated by cardiac motion and are often treated as artifact and ignored (1,2); however, these Doppler signals suggest that cardiac motion generates a Doppler signal and may therefore also affect the Doppler signal derived from blood flow.

The purpose of this study was to design and test an in vitro model to investigate the effect of cardiac motion on the Doppler signals generated by blood flow and to demonstrate the effect of cardiac motion on the Doppler signal generated by blood flow in normal subjects. Work done by others (3–9) has quantitated the motion of the aortic wall, the left ventricular posterior wall and the mitral and tricuspid annuli. Strunk et al. (3) have shown that changes in atrial filling and pulmonary venous flow alter motion of the mitral annulus in predictable ways. In this study we look at the Doppler signal generated by the cardiac structures themselves and their influence on the Doppler signal generated by blood flow patterns.

We designed an in vitro model that allowed us to observe and independently control the motion of a chamber containing a liquid and the motion of the liquid itself. This model enabled us to observe the effects of the movement of the chamber on the Doppler signal derived from the motion of the liquid. We tested our hypothesis that cardiac motion can alter the velocity of the Doppler signal generated by blood

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Figure 1. Upper panel, Pulsed Doppler signal obtained from the left ventricular outflow tract from the apical five-chamber view, showing the Doppler signals generated by cardiac motion during atrial systole (a), ventricular systole (s) and ventricular diastole (d). Lower panel, M-mode recording of the aortic annulus obtained from the same transducer position as the Doppler signal (upper panel), represent the motion of the aortic annulus during atrial systole, ventricular systole and diastole. The recordings were made sequentially, and then the M-mode recording was aligned with the Doppler recording using the electrocardiogram. Scale at upper left is in meters per second.

flow in the in vitro model. We measured cardiac motion with M-mode echocardiography in human subjects and compared the data with the Doppler signal generated by cardiac motion.

## Methods

Echocardiographic equipment. An Acuson 128 XP/10 with a 5- or 3-MHz transducer was used for the in vivo and

in vitro experiments. Images were recorded on high quality VHS tape and printed on a color page printer (Sony Color Video Printer UP-5050W). Machine factors were adjusted as follows: the pulsed Doppler transmit power was set at <100; the log compress was set at 35 dB; the length of the gate was 6.0 mm; the width of the gate or the focus was 3 mm. For the in vivo measurements, the incident angle, the depth of the gate and the depth of the two-dimensional image were dependent on the individual subjects. For the in vitro apparatus, the depth of the pulsed Doppler gate was 69 mm, and the gate was placed within the middle of the straight portion of the tubing, away from the walls.

In vitro model (Fig. 2). The in vitro model was used to independently control the flow of liquid through a chamber and the motion of the chamber relative to the Doppler beam. A 280-cm length of silicon rubber tubing with an inner diameter of 5 mm was connected to a variable pulse rate and flow velocity perfusion pump (Sorvall 49061 Peristaltic Pump). A 6% (6 g cornstarch/100 ml tap water) cornstarchwater slurry was driven by the pulsatile pump through the tubing to simulate the blood flow within the heart. Both the velocity of flow and the frequency could be independently controlled along a continuum. The flow rate for pulsatile flow through the pump was varied from 100 to 300 ml/min. corresponding to average linear velocities of 8 to 25 cm/s. A 6-cm section of the silicon tubing was fixed to a rigid plastic board, and the board was cyclically moved by a piston to simulate the heart motion. The tubing attached to the board was aligned with the echocardiographic transducer placed above it, and the motion of the board was parallel to the Doppler ultrasound beam as well. This eliminated possible artifacts from beam angulation. The pulsed Doppler gate was within the tubing, away from the wall and well above the curved portion of the tubing. The Doppler beam was parallel to the motion of the liquid and the tubing. The plastic board was coupled by means of gears, springs and connectors to a Harvard Rat Ventilator piston with a variable rate control



Figure 2. In vitro model. Thin arrows demonstrate the direction of flow of the fluid through the tubing and the motion of the plastic board to which the tubing is fixed.



Figure 3. Doppler signal derived from the motion of the tubing. The cornstarch-water slurry is within the tubing but is not being pumped. The Doppler signal is generated entirely by the motion of the tubing, reflected by the cornstarch-water slurry.

(Harvard Apparatus model 683). The board could be cyclically moved up and down to mimic cardiac motion. Both the rate and the excursion of the board could be controlled and varied independently of the flow within the tubing. The motion of the board could be continuously varied over a range of velocities from 0.5 to 40 cm/s, and its maximal excursion for these series of experiments was 1 to 2 cm. This is comparable to cardiac motion in humans (3,4). The tubing and board were placed within a water bath, with the echo transducer above the vertical portion of the tubing.

Human study. Sixteen normal subjects and patients with noncardiac diseases with good quality transthoracic images were identified (age range 8 to 60 years). Ventricular function, cardiac chamber dimensions and routine Doppler measurements were normal. This study was approved by the Human Subjects Review Committee of Harbor–University of California, Los Angeles Medical Center.

Measurements. The velocity of cardiac motion in human subjects was derived utilizing M-mode and pulsed Doppler recordings with a 3.0-MHz transducer placed at the left ventricular apex. For the Doppler portion of the study, the transmit power was set at <500, and the log compress was set at 35 dB with filter settings of 0 or 1. The gate size was 6 or 7 mm. The depth varied according to patient size. An M-mode echocardiographic cursor was placed through the aortic annulus in the apical five-chamber view during a period of stable heart rate. The pulsed Doppler signal was recorded with a 6-mm sample volume in the left ventricular outflow tract below the aortic valve orifice. The sample volume was placed within the blood pool away from the aortic wall and leaflets. The M-mode and pulsed Doppler signals were recorded sequentially within 1 to 2 min of each other, with the patient in the left lateral decubitus position and at a stable heart rate. The M-mode and Doppler recordings were aligned by superimposing the electrocardiographic R wave (Fig. 3). Alignment of the Doppler and M-mode recordings was possible because the heart rate remained constant, and the two recordings were made without change in patient position and over a short period of time. The M-mode signal generated by the aortic annulus (cardiac motion) was compared with the low velocity pulsed Doppler signal (labeled "a," "s" and "d" in Fig. 1). The direction of cardiac motion is opposite to the blood flow during systole. The slope of the M-mode recording of the aortic annulus and the maximal Doppler velocity of the cardiac motion were measured at the same part of the cardiac cycle in the aligned images. Each measurement was an average of 3 beats. The maximal slope (cm/s) was derived by drawing a line tangential to the M-mode recording of the annular motion. The slope of the motion of the aortic annulus is the velocity of the cardiac motion of the aortic annulus relative to the transducer. The maximal slope on the M-mode and the maximal velocity on the spectral Doppler recording were measured and compared. The Doppler signal and the slopes were measured at the following points in the cardiac cycle: at the time of atrial contraction, during ventricular systole and during early diastolic filling ("a," "s" "d," respectively, in Fig. 1).

Calculations and measurements of the velocity and amplitude of the tubing in the in vitro apparatus. The distance the tubing moved was determined by measuring the amplitude of the M-mode tracing of the tubing. It was compared with direct measurements of the distance that the apparatus moved. The slope of the M-mode measurements represents the velocity of the tubing. We compared the M-mode measurements with the Doppler velocities obtained with the apparatus maintained at a constant speed.

Statistics. Statistical calculations were performed using the STATS + statistical software package (StatSoft) for regression analyses. Coefficients of variation (SD/mean) for the triplicate in vivo data measurements were calculated using a hand-held calculator. In vivo data comparing M-mode and Doppler assessment of cardiac motion were analyzed using linear regression combining data for all time points in the cardiac cycle to find a single relation. We also analyzed the same data by multiple regression using dummy variables for the three time points in the cardiac cycle (atrial systole, ventricular systole and diastole). Because the correlation coefficient for the multiple regression was only marginally better than that obtained using simple linear regression, only the simple linear regression results are presented.

### Results

Figures 3 to 5 demonstrate the Doppler signals derived from the motion of the silicon tubing, the motion of the water-cornstarch slurry within the tubing during peristaltic pumping and their combined motions. The motion of the tubing was parallel to the flow of the slurry within the tube. Figure 3 shows the motion of the tubing being moved by the piston while the fluid is not being pumped. The apparent Doppler signal is a sinusoidal pattern with a maximal velocity of  $\pm 10$  cm/s. Figure 4 demonstrates a typical pulsed Doppler recording generated by the pulsatile motion of the fluid alone while the tubing is stationary. The maximal velocity is approximately 30 cm/s and occurs regularly with a frequency of approximately 4 Hz. The Doppler signals at the left of Figure 5 demonstrate the combined effect of motion of the tubing and flow through it. Although the velocity of the fluid through the tubing has not changed, its



Figure 4. Doppler signal derived from the pulsatile pumping of the fluid while the tubing remains stationary.

measured velocity has been altered by the motion of the tubing. The frequency of the pulsatile flow is twice the frequency of the tubing motion. When the peak velocities are in phase (arrow), the maximal velocities of both signals are additive, resulting in an increase in the measured velocity of the liquid. When the velocities are out of phase, they negate each other. The measured velocity is thus the vector sum of the two velocities. In Figure 5 the middle six waves (identical in profile and amplitude) are the Doppler signals generated by pulsatile flow of the liquid through the tubing (while the tubing remains stationary), similar to those seen in Figure 4. The low velocity waves at the right of Figure 5 are the result of periodic motion of the tubing recorded while the fluid is not being pumped. The Doppler signal derived from this motion is sinusoidal and similar to that seen in Figure 3.

The complexity of the interaction between the motion of the tubing and the liquid is further demonstrated in Figures 6 and 7, where the frequencies of the two motions are varied relative to each other. Flow of the liquid and motion of the tubing were controlled independently to create synchronized and desynchronized motion. The frequency of the motion of

Figure 5. Doppler signals generated by motion of the tubing alone (right) (similar to Fig. 3), by the motion of the cornstarch-water slurry being pumped through stationary tubing (middle) and by the combined motion of the fluid and the tubing (left). When the tubing is being moved and the fluid is pumped, the apparent or measured velocity changes, although the true velocity of the fluid within the tubing has not changed. Arrow indicates a cycle in which the two motions are in phase.





Figure 6. Doppler signals generated by fluid and tubing motion. The frequency of the motion of the tubing and the frequency of the peristaltic pump were varied. The frequencies of two motions were no longer integral multiples of each other and thus were desynchronized. The relative magnitude of the measured signals during the cycle varies even though the actual frequencies and velocities of motion and flow were constant. Arrow indicates a cycle in which the two motions are in phase, yielding the highest measured velocity.

the tubing was slower than the pulse rate of the peristaltic flow and is therefore slightly dissociated in Figure 6. This resulted in a continually varying alternans pattern of the peak amplitudes of the Doppler signal. The resultant waves change not only in amplitude (velocity) but also in apparent slope (acceleration and deceleration). Although flow through the tubing remains constant throughout, the desynchronized motion of the tubing (relative to the frequency of the pulsatile pump) results in an apparent twofold difference in velocity (20 to 40 cm/s) between adjacent peaks. Figure 7 demonstrates the Doppler flow pattern when the frequency of the motion of the tubing and the pulsatile flow are out of phase but are integral multiples of each other. When the flow and movement of the tubing are at about the same frequency but out of phase, the observed relationship is regular, and the Doppler velocity signals remain alternating but constant throughout the tracing. Although the flow of the liquid is unchanged in Figures 6 and 7, the velocity patterns are quite different. These patterns were achieved merely by changing the degree of phase synchronization of the motion of the tubing relative to the liquid. The resultant Doppler spectrum of the combined motion is the algebraic sum of their Doppler signals, resulting in apparent changes in maximal velocity as well as slope of the velocity curve.

Velocity measurements for the apparatus. Using a fixed frequency and amplitude, the M-mode and Doppler mea-

Figure 7. Doppler signals generated by fluid and tubing motion. The two motions were synchronized but out of phase; thus, the pattern remains constant, with the peak velocities representing the vector sum of the two motions. The slopes of the signals have changed because the peaks of the two motions no longer coincide. Arrow demonstrates the timing of the maximal velocity of the tubing.



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Figure 8. Relation between the peak measured velocity of cardiac motion by Doppler ultrasound and the M-mode measurement of aortic annulus velocity in human subjects. Each data point is the mean of three measurements, with a coefficient of variation within subjects that ranged from 2.2% to 4.4% for both Doppler and M-mode measurements. The solid line is plotted according to the following equation derived from linear regression analysis of the data:  $y = 2.27 \times + 1.03$  (r = 0.99).

surements for velocity of the tubing were compared. In three cycles the M-mode study yielded velocities of 75, 76 and 75 mm/s compared with Doppler velocities of 180, 190 and 180 mm/s. Thus, the Doppler/M-mode ratio is 2.4. An amplitude of 11 mm for the motion of tubing was measured from the M-mode tracing. Measuring the excursion of the tubing directly from the apparatus yielded the same result.

Patient data. Low amplitude pulsed wave Doppler signals from a sample volume placed just proximal to the aortic valve were compared with the M-mode-derived velocity of cardiac motion. Figure 1 is a representative example demonstrating the Doppler signal generated by blood flow and by the motion of the aortic annulus. Heart rate was constant throughout the measurement period. The Doppler-derived velocity due to cardiac motion is similar to that calculated by measuring the velocity of the motion of the annulus using the M-mode calculation of slope. Figure 8 shows the relation between the maximal slope of the M-mode tracing of the aortic annular motion and the peak Doppler signal due to cardiac motion at three time points in the cardiac cycle for 16 subjects. The Doppler measurements and the M-mode measurements are highly correlated (linear regression, r = 0.99; p < 0.001), but the estimates of cardiac motion from the Doppler signal are approximately twofold higher than the estimates from M-mode measurements.

#### Discussion

In vitro model. The motion of blood cells within the heart consists of the propulsion of blood cells driven by the contraction of the heart and the motion of blood cells as a component of the chamber, which is moving cyclically. In the past, the "flow" signal generated by cardiac motion was neglected. Two models were used in our study to demonstrate that cardiac motion can influence the measured Doppler signal generated by blood flow and to investigate the influence of the motion of the heart on measured velocity. In the in vitro model, the velocity of the slurry and the motion of the apparatus were varied to simulate cardiac motion and blood flow. This procedure allowed us to separate the two components of the measured velocities. By stopping the motion of the piston, we could observe the pure flow profile, which could be kept constant. Similarly, the flow through the apparatus could be stopped, allowing us to measure the motion of the apparatus. We illustrated the potential alterations in peak velocities as well as changes in the slope of the velocity curve (acceleration) and were able to cause apparent changes in cycle length. Although some of the relations between the motion of tubing and the flow of the slurry do not appear naturally on a recurring basis (i.e., Fig. 6 and 7), they illustrate the capacity of certain changes in timing to alter apparent flow patterns. Intermittent changes in cardiac motion relative to flow could be caused by atrial or ventricular ectopic activity or respirations, resulting in changes in cardiac motion. Because such changes might not be synchronized with flow alterations, changes in both the slope and the height of the Doppler signal might occur. The velocity of cardiac motion also varies throughout the cardiac cycle and therefore may not affect events at different parts of the cycle similarly. In measurements utilizing slopes, such as mitral valve half-time or pulmonary acceleration time, changes in cardiac motion might cause a significant change in a Doppler-derived measurement.

The motion of the base of the heart has been observed by many investigators (5.10.11). Atrioventricular and semilunar valvular annuli and the fibrous framework in the adjacent tissues should have similar patterns of motion because they move together as part of the base of the heart. The blood flowing through the chambers will have independent motion relative to the transducer. What the transducer observes, for example, in the apical five-chamber view, is motion of the annuli toward the transducer during systole while blood is flowing in the opposite direction from the aorta. We mimic this with our apparatus. By positioning the tubing and motion of the board along the ultrasound beam of the transducer, we were able to eliminate problems of angle correction from our calculations. The impact of the twisting motion that occurs in normal hearts was not included in our in vitro analysis, and by using the M mode study in the in vivo system we also only looked at motion in a single plane. Although the motion of the heart is complex, the ultrasound transducer observes only one-dimensional signals in both Doppler and M-mode studies.

Even though the Doppler sample volume had a focus of 3 mm within the central portion or bore of the tubing, we believe that the signals generated by the motion of the tubing are caused primarily by reflection of the ultrasound beam off the walls of the tubing and are a result of beam width. Some of this signal may also be generated by the cornstarch particles close to the wall, which move with the tubing as it accelerates. We placed the Doppler sample volume outside the tubing, in the water bath itself, and outside the cardiac

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chamber, within muscle (9) (data not shown), and we were still able to record the Doppler signals, although they were much weaker. Whether the source of the signal is reflectors adjacent to the wall of the chamber, the chamber itself or beam width artifact, these signals alter the measured signal generated by blood flow, and the direction and timing of the alteration correlate with the motion of the chamber.

In vivo model. In the in vivo portion of this study, we measured cardiac motion by two methods and showed the relation of the velocity of cardiac motion detected by M-mode echocardiography to the Doppler signal generated by the motion. The relation between the Doppler and the M-mode measurements is a temporal one and does not establish causality. The velocity of cardiac motion determined by measuring the Doppler signal generated by cardiac motion differed by a factor of 2 from the measured velocity based on M-mode measurements of the aortic annulus. This difference was similar to the findings obtained by using Doppler and M-mode signals to measure the velocity of the motion of the tubing in the apparatus. There are several possible explanations for the higher measured velocity obtained by using the Doppler signal. 1) The tissue or tubing disperses the Doppler signal, giving a wide spectrum. Because of beam width, the reflections causing the Doppler signal may be coming from different parts of the aortic annulus or tubing, therefore causing dispersion of signals. We used the maximal measured Doppler velocity for our comparison, which may not represent the true velocity at the point used to measure velocity on the M-mode recording (9). 2) Although we attempted to align the M-mode cursor with the pulsed Doppler cursor, this alignment might be imperfect. 3) The velocity measurement on the M-mode should be the slope of the tangent line at the point of the maximal velocity on the annular motion curve, which is difficult to obtain on the ordinary M-mode recordings.

The measured Doppler spectrum of blood flow velocity in clinical echocardiography always has the built-in error caused by cardiac motion. The resultant spectrum is different from the pure spectrum, not only in the amplitude of the peak velocity but also in the slope of the measured velocity. Thus, cardiac motion can alter the velocity signals used to measure hemodynamic variables. In mitral inflow, for example, the maximal velocity, the half-time and the relation between the E and A waves could be altered by movement of the annulus. Because the direction and the velocity of the motion of the annulus vary throughout the cardiac cycle, the slopes of the E and A waves and their relative heights might not be affected equally. This observation may in part explain the difficulty in utilizing mitral inflow to analyze diastolic events (12). The changes seen in diastolic filling that are attributed to changes in compliance and relaxation might be overshadowed by the changes due to cardiac motion. This might further obscure the already difficult to understand effects of diastolic relaxation on the Doppler signal. Measurements of mitral valve area might also be inaccurate

because of changes in slope caused by cardiac motion. Another example of problems that can be created by not considering the effect of cardiac motion on Doppler measurements is the use of the pulmonary artery flow acceleration time to diagnose pulmonary hypertension. Cardiac motion could alter the slope and therefore the acceleration time.

In our in vitro and in vivo analysis we did not consider the complexity of cardiac motion but measured it only along the axis of the blood flow. The heart is actually twisting while it contracts, and it exhibits a complex series of motions; therefore, it may not be moving entirely in the plane of the Doppler beam or the blood flow. We did not analyze the complexity of cardiac motion in our in vivo analysis and excluded it by design in our in vitro system. We believe, however, that one could partially correct for the error generated by cardiac motion by using the M-mode recording to measure cardiac motion or by measuring the Doppler signal derived from the cardiac motion and using one of these to correct the measured Doppler velocity signal.

**Conclusions.** Our experiments in vitro and in vivo provided important information about the interaction of cardiac motion and blood flow in the Doppler spectrum. The measured Doppler spectrum of blood flow velocity is modified by cardiac motion and could be corrected for more accurate measurement.

#### References

- Cao T. Duan Y. Yang B. The influence of cardiac motion on the measurement of Doppler spectra of blood flow velocity. Chin J Ultrasound Med 1991;7:84-6.
- Isaaz K, Munoz L, Lee E, Schiller NB. Quantitation by Doppler echo of the motion of the cardiac base in normal man (abstr). Circulation 1989;80(suppl II):II-169.
- Strunk BL, Fitzgerald JW, Lipton M, Popp RL, Barry WH. The posterior aortic wall echocardiogram: its relationship to left atrial volume change. Circulation 1976;54:774-50.
- Keren G, Sonneblick EH, Le Jementel TH. Mitral annulus motion: relation to pulmonary venous and transmitral flows in normal subjects and in patients with dilated cardiomyopathy. Circulation 1988;78:621–9.
- Hammarstrom E. Wranne B, Pinto FJ, Puryear J, Popp RL. Tricuspid annular motion. J Am Soc Echocardiogr 1991;4:131-9.
- Keren G, Sherex J, Medidish R, Levitt B, Laniado S. Pulmonary venous flow pattern—its relationship to cardiac dynamics. Circulation 1985;71: 1105-12.
- Ormiston JA, Shah PM, Tei C, Wong M. Size and motion of the mitral valve annulus in man I. A two-dimensional echocardiographic m ...d and findings in normal subjects. Circulation: 1981;64:113-20.
- Jones CJH, Song GJ, Gibson DG. An echocardiographic assessment of atrial mechanical behaviour. Br Heart J 1991;65:31-6.
- Isaaz K, Thompson A, Ethevenot G, Cloez JL, Brembila B, Pernot C. Doppler echocardiographic measurement of low velocity motion of the left ventricular posterior wall. Am J Cardiol 1989;64:66-75.
- Zaky A, Grabhorn L, Feigenbaum, H. Movement of the mitral ring: a study in ultrasonography (abstr). Cardiovasc Res 1967;1:121.
- Hamilton WF, Rompf JH. Movement of the base of the ventricles and the relative constancy of the cardiac volume. Am J Physiol 1932;102:559-65.
- Shapiro SM, Bersohn MM, Laks MM. In search of the Holy Grail: the study of diastolic ventricular function by use of Doppler echocardiography. J Am Coll Cardiol 1991;17:1145-7.