INTRODUCTION

One reason for the rapid growth in the use of Doppler echocardiography is its utility in detecting and assessing the severity of valvular stenosis. Detection of the presence of stenotic valvular heart disease using Doppler echocardiography was originally described over ten years ago. It has subsequently been demonstrated that Doppler blood velocity data could be used to estimate the severity of a stenotic lesion.

FORWARD BLOOD FLOW PROFILES

Aortic Valve Flow Velocity Profiles

During systole, blood is ejected through the open aortic valve cusps. In normal individuals, the velocity rapidly rises to a peak somewhere in the first one-third of systole and then falls back to baseline during the remainder of this portion of the cardiac cycle. A normal flow velocity profile and its relationship to electrocardiographic events is shown in Figure 3.1. Note that the rise in velocity begins just after the QRS complex.

There are various indices, or measurements, that can be made from the spectral recording which help to characterize systolic functional characteristics. These are demonstrated in the idealized spectral velocity in Figure 3.2. These flow profiles are characterized by a peak velocity (in cm/s), the maximum velocity reached during systole. This measurement is easily obtained from the inspection of the spectral tracing. The time to reach peak velocity (or “time to peak”) is another component of the systolic profile which helps to characterize systolic ejection and is measured in seconds. Left ventricular ejection time is the duration of the systolic flow velocity recording. These time durations are also easily measured directly from spectral recording.

The peak acceleration is a more complex measurement and usually requires the assistance of an automated, computerized device. It is found on the upstroke and is the maximum acceleration
expressed in centimeters per second. Likewise, measurement of the flow velocity integral usually requires computer assistance and is the area under the spectral flow velocity tracing.

**Ejection Rate Indices and Ventricular Function**

Some of these indices relate directly to the systolic function of the left ventricle in patients with normal aortic valves. The better the left ventricular contraction the more rapid the acceleration, and the higher the peak velocity. Conversely, the poorer the left ventricular ejection (as seen in patients with low ejection fractions), the less rapid the acceleration of blood flow in the aorta and the lower the peak velocity. These approximate relationships are illustrated in the idealized graphs in Figure 3.3. Thus, rapid peak acceleration and high peak velocities characterize optimum ejection fractions.

**Cardiac Output**

Doppler echocardiography is useful for the determination of cardiac output; this is the volume of blood pumped by the left ventricle every minute and is expressed in liters per minute. The volume of blood ejected every systolic beat is called the stroke volume (Fig. 3.4) and is the basis for calculation of cardiac output according to the following equation:

Cardiac output = stroke volume x heart rate

Doppler calculation of cardiac output is based on the assumption that the aorta is a cylinder during every systolic beat. This cylindrical flow volume may be determined if its area and its length are known. The area is determined from the two-dimensional echocardiographic image while the length is derived from the Doppler spectral recording (Fig. 3.5).

These relationships between area and flow velocity are shown schematically in Figure 3.6. Given identical volume of flow through a large cylinder (Fig. 3.6, left panel) and small cylinder (right panel), the velocities recorded by Doppler will vary considerably. The same volume through the larger cylinder will render
a lower peak velocity (and small flow velocity integral) in comparison with the flow recorded through the smaller orifice. The flow velocity integral reflects the average velocity of the red cells during systole. Because the red cells are moving faster through the smaller cylinder, they travel farther. Thus, the Doppler recording of velocity relates to distance traveled.

Conceptually, derivation of cardiac output begins with the recognition that the volume of blood ejected every time the heart beats is first limited by the area of the aortic root (the area of the cylinder). While there remains some argument as to the precise point where this area is best determined from the two-dimensional echocardiographic image, most Doppler users measure the narrowest diameter in systole at the bases of the aortic valve cusps. This is most reliably accomplished using the parasternal long-axis view. Dividing the diameter in half then results in the radius of the open aortic valve. This area is assumed to be a circle and is determined by the standard plane geometric equation: area = $r^2$.

Secondly, the distance the ejected blood travels may be calculated from the Doppler spectral recording. Since this is a measure of velocity over time, the flow velocity integral will result in the average velocity during systole.

As a result of knowing the average velocity, his may be normalized for one second and is an index of how far the blood has traveled. The method for calculation of cardiac output is demonstrated in Figure 3.7.

It is important to recognize that for proper calculation of cardiac output using this approach the beam must be as parallel to flow as possible. Alterations of a few degrees from parallel will result in lower Doppler velocity recordings and underestimation of cardiac output. Therefore, aortic outflow is best obtained from the apical or suprasternal approaches where the beam is nearly parallel to normal flow.

It is also important to remember that the Doppler estimate of cardiac output is based on the square of the measured radius of the aorta. Any error in this measurement will be multiplied and may profoundly affect the resulting calculation.
Doppler estimates of cardiac output compare quite favorably with those obtained by other methods. Comparisons have been made with cardiac output estimated by the Fick principle at catheterization, with thermodilution, as well as with a host of other approaches. In general, these studies show very good correlations, being within ±10% of the other method (Fig. 3.8). Cardiac output may also be determined from flow and diameter measurements through any of the other cardiac valves.

**Estimation of Pulmonary Arterial Pressures**

Many methods have been proposed for the estimation of pulmonary arterial pressures. *Unit 2* discusses the method based on the presence of tricuspid regurgitation. Other methods are based on the time to peak velocity of the pulmonary arterial flow velocity recording. These methods only work when there is no evidence for pulmonary stenosis.

Methods for estimating pulmonary arterial pressures are based on alterations in the capacity of the pulmonary vasculature to accept forward systolic flow. In normal individuals without pulmonary hypertension, the pulmonary vasculature is a very low resistance circuit and has a great capacity to accept the sudden increase in volume. The vessels are quite distensible and as blood is ejected from the right ventricle the time to peak velocity and acceleration time are accordingly relatively slow.

In pulmonary hypertension, however, resistance rises as blood vessels thicken and become less distensible. This results in a diminished capacity to accept the forward systolic flow out of the right ventricle. The sudden rush of blood into the main pulmonary artery in this setting results in a more rapid time to peak velocity as well as a more rapid acceleration time.

These relationships are illustrated in **Figure 3.9**, where idealized plots of mean pulmonary artery pressure and time to peak velocity are shown. Note that these relationships are curvilinear, making estimates of very high, or very low mean pulmonary pressures difficult. To overcome this problem, several investigators have pointed out that plotting time to peak velocity against the logarithm of the mean pulmonary artery pressures makes the correlations much better.

The clinical application of this approach for estimation of mean pulmonary arterial pressure remains controversial and many methods have been proposed. Our purpose is to present the general concept of these relationships and the reader should consult current literature for more
detailed descriptions of the continuing evolution of this principle. Many factors must be taken into account, an important one being heart rate. Adult patients with pulmonary hypertension may have normal heart rates of 60-70 beats/min, and infants and children >140 beats/min; this may significantly shorten measurements of time to peak velocity or acceleration times, and affect the reliability of these estimates of pressure.

What is most important is that time to peak velocity is significantly shortened in patients with pulmonary hypertension. **Figure 3.10** demonstrates both normal and rapid time to peak velocities in two idealized spectral recordings.

**ESTIMATION OF THE SEVERITY OF VALVULAR STENOSIS**

**Effect of Stenosis on Blood Flow**

The driving force for blood to move across any cardiac valve is the presence of a slight pressure difference normally found between the chambers (or chamber and great vessel) on either side of the valve. For example, systolic pressure builds within the left ventricle until it reaches a point where it exceeds the pressure in the aorta. The aortic valve is suddenly thrown open and blood is ejected into the aorta. In normal individuals, there is a very slight (1-2 mmHg) pressure difference between the left ventricle and aorta that helps drive the blood across the aortic valve.

Normal aortic valve blood flow is laminar (**Fig. 3.11**) and most of the red cells in the aortic root during systole are moving at approximately the same speed. Graphically, this translates into a narrow band of dark grey on the pulsed wave (PW) Doppler spectral recording (**Fig. 3.11, arrow**). Normal peak systolic velocity of blood flow across the aortic valve rarely exceeds 1.5 m/s.
When the aortic valve is diseased, the leaflets become thickened and progressively lose their mobility. Eventually, the valve itself becomes narrowed to the point where it begins to obstruct flow, and aortic stenosis is created. In the presence of aortic stenosis, systolic pressure in the ventricle must rise high enough to force the blood across the obstruction into the aorta. Thus, a pressure drop, or pressure gradient, is generated (Fig. 3.12). Severe degrees of aortic stenosis may result in aortic valve gradients that exceed 100 mmHg in systole. As discussed in Unit 1, the presence of such an obstruction results in both turbulent flow and increased velocity, two characteristics readily detected by Doppler echocardiography. Because of the large gradient, the pressures within the left ventricle rise significantly and left ventricular hypertrophy results.

Doppler detection and evaluation of the presence or absence of aortic stenosis is based on recording turbulence and increased flow velocity in the ascending aorta. In Figure 3.13 these characteristics are shown in a continuous wave (CW) spectral velocity recording of aortic systolic flow obtained from the apical window. Turbulent flow is represented by broadening of the velocity spectrum. There is also an increase in peak aortic velocity to 4 m/s. The Doppler audio in this case had a harsh, higher pitched quality during systole that was easily distinguished from the sound of laminar flow.

Similarly, the presence of these characteristics in the pulmonary artery during systole would indicate the presence of obstruction to right ventricular ejection. Figure 3.14 demonstrates a CW Doppler spectral recording from the left parasternal window in a patient with mild pulmonic stenosis and insufficiency. Turbulent diastolic and systolic flows are noted with a slight increase in the peak systolic velocity to 1.4 m/s (normal <1 m/s).

The PW Doppler examination shown in Figure 3.15 is from the same patient as Figure 3.1 and demonstrates the ability of PW to localize the level of the obstruction. With the cursor positioned on the ventricular side of the pulmonic valve (Fig. 3.15, A), the turbulent diastolic spectral recording of pulmonic insufficiency is noted, while systolic flow is undisturbed (laminar) with a peak systolic velocity of about 1 m/s. When the sample volume is positioned distal to the diseased pulmonic valve (Fig. 3.15, B) the systolic flow becomes
turbulent and the peak systolic velocity is elevated. The spectral recording of pulmonic insufficiency is lost because the sample volume is located distal to this lesion.

While PW Doppler is very useful for the localization of such obstructive lesions, it has limited value in establishing the severity of obstruction because most significant valvular obstructions result in velocities above 1.5 m/s. As emphasized previously, velocities above 1.5 m/s will usually cause aliasing of the PW recording. This prevents the faithful recording of peak velocities necessary for the calculation of valve gradients.

**Estimation of the Severity of Stenosis**

Use of Doppler ultrasound to estimated the severity of a valve stenosis is based principally on the fact that such obstructions result in an increase in the velocity of flow. In clinically significant mitral stenosis, the diastolic velocity of mitral flow usually exceeds 1.7 m/s. Systolic velocity of aortic flow in clinically significant aortic stenosis may reach 5-6 m/s (Fig. 3.16). Thus, CW Doppler is required for the detection of these increased velocities and for recording the full spectral profiles.

We have already noted that there is a relationship between the pressure increase (or gradient) across a valve and the velocity of blood flow across the valve. For any given pressure gradient there is a corresponding increase in velocity, as predicted by the simplified Bernoulli equation:

\[ p_1 - p_2 = 4V^2 \]

where \( p_1 \) = pressure distal to obstruction \( p_2 \) = peak velocity of blood flow across the obstruction.

As the stenosis becomes more severe, the valve orifice area will become smaller, and the velocity of flow across the orifice will increase as a function of the increased pressure gradient. Thus, by measuring the peak velocity in a systolic aortic jet with Doppler echocardiography, it is possible to estimate the pressure gradient that produced it using the above simple algebraic expression. The peak aortic velocity of the spectral recording in Figure 3.16 is approximately 5.8 m/s. Using the previous formula

\[ p_1 - p = 4(5.8)^2 \]
the pressure gradient is therefore 135 mmHg.

There are, however, three major technical requirements that must be satisfied if Doppler is to be used for this purpose. First, an adequate “window” into the chest for ultrasound propagation and reception must be found so that well formed Doppler profiles can be recorded. Second, as emphasized in Units 1 and 2, for the velocity measurement to be accurate, this window must allow orientation of the ultrasound beam so that it is as parallel as possible to flow through the valve. Third, the high velocities present in the disturbed jet often exceed the Nyquist limit of PW Doppler, so that CW or high pulse repetition frequency Doppler must be used.

**Expressing the Severity of Stenosis**

It should be recognized that knowledge of the gradient across a stenotic valve does not provide all the information necessary to assess the severity of obstruction. The gradient will vary with flow across the stenotic valve orifice and will increase in high flow situations and decrease in low flow situations. Thus, a patient with a fixed valve area will have a higher gradient during exercise when cardiac output is increased, than at rest when cardiac output is lower. Figure 3.17 shows the idealized relationship of valve gradient to flow. As flow across a valve rises, as with rapid tachycardia, the gradients will vary.

Valve orifice size is generally considered not to vary with the amount of flow across the valve and is, therefore, a preferred expression of the severity of a given stenosis. The following discussion will begin with estimates of gradients across stenotic valves and then review some of the simplified methods for estimating valve orifice area.

**Finding the Stenotic Jet**

The most common windows utilized for recording peak aortic systolic velocity are the apical, suprasternal, and right parasternal. While stenotic jets, like regurgitant jets, are often directed eccentrically, it is usually possible to find a fully formed aortic systolic profile from one of these windows. A comprehensive Doppler examination for aortic stenosis requires that the ascending aorta be examined from all possible windows in order to align the beam parallel to the jet. Figure 3.18 shows a CW examination of a patient with aortic stenosis from the suprasternal notch (left panel) and the apex (right panel). The spectral tracing from the apical window is
superior as judged by the presence of a fully formed profile with a discrete ascent, peak, and descent. While we have found the apical window to be most productive, we always examine the aorta from every possible view. Occasionally, the suprasternal window will be perfectly aligned to flow and will present the typical spectral profile of aortic stenosis. This is demonstrated in a patient with severe aortic stenosis in Figure 3.19. In this condition, there is marked spectral broadening, delayed systolic peaking, and a marked increase in velocity. In this patient, the peak systolic velocity is almost 5 m/s (100mmHg).

Considerable operator skill is required to obtain adequate spectral tracings for measurement of peak velocity. It is our experience that the Doppler examination for aortic stenosis will add an average of 15-30 minutes to the two-dimensional and routine Doppler echocardiographic examination, even with the experienced operator.

In Figure 3.20 (left panel) there is a recording from the apical window obtained by an operator with only modest experience. Both aortic stenosis and aortic insufficiency are recorded, but the systolic flow away from the transducer fails to show a fully formed profile. The spectral recording in Figure 3.20 (right panel) was performed by a more experienced individual, and the fully formed systolic profile is seen. Had a measurement been made on the upper panel, peak systolic velocity would have been approximately 2.8 m/s, while the true velocity shown on the panel below is 5 m/s. Use of the inadequate tracing would have severely underestimated the valve gradient.

Most experienced Doppler operators can obtain aortic systolic velocity profiles adequate for measurement of peak velocity in about 95% of patients. Figure 3.21 demonstrates combined aortic stenosis and insufficiency by CW Doppler from the apex of the left ventricle.

Note that at the left, the full profile of the aortic stenotic jet is encountered. With minimum transducer angulation, the aortic insufficiency...
profiling is readily encountered. At the far right, the systolic flow away from the transducer is lower in velocity and represents the velocity in the left ventricular outflow tract before the obstruction. The lower velocity proximal to the stenotic valve should not be confused with aortic stenosis.

Occasionally, only incompletely formed profiles are recorded. These should be considered inadequate and never used for estimation of gradient. An incompletely formed profile from an older patient is shown in Figure 3.22.

Another potential source for error is mistakenly interpreting the profile of mitral insufficiency for that of aortic stenosis. When recorded from the apical window, both occur in systole and are displayed as downward spectral velocity shifts. This is seen in the spectral tracings shown in Figure 3.23. These may be differentiated by remembering that the onset of ventricular systole and mitral regurgitation (3.23 arrow) occurs prior to aortic valve opening. In addition, mitral regurgitation is longer in duration.

At first, it may appear that the spectral profiles of aortic stenosis resemble mitral insufficiency and those of aortic insufficiency resemble mitral stenosis. These disease profiles may be differentiated by a knowledge of the various timing relationships of left-sided valvular opening and closing. Figure 3.24 shows the relationships between these various abnormal spectral velocities. The duration of mitral insufficiency is generally longer than that of aortic stenosis, partly because the time from mitral valve closing to opening is longer than for aortic valve opening to closing. Similarly, the duration of aortic insufficiency is longer than mitral stenosis because the time from aortic valve closing to opening is longer than for mitral valve opening to closing. Similar relationships are true of the pulmonic and tricuspid valve on the right side of the heart. Those experienced in phonocardiography will realize the advantage of using this technique to assist in the identification of the various valve profiles.

Figure 3.22 Inadequate recordings of aortic systolic velocity do occur and should not be used for estimation of gradient. Note that the profile is poorly formed on the upstroke, peak (arrow), and down stroke. (Scale marks = 2 m/s)

Figure 3.23 Aortic stenosis (left) should not be mistaken for mitral insufficiency (right). Mitral systole begins before aortic (arrow) and is longer in duration. (Scale marks = 2 m/s)

Figure 3.24 Relationship between abnormal systolic and diastolic flows through the left heart valves. For details, see text.
In patients with aortic valve disease and stenosis, a careful examination must be performed from all possible views as the abnormal jet may be directed anywhere in the aorta. In these cases, we are most interested in recording the highest peak systolic velocity present. As previously pointed out, the most faithful representation of flow will be obtained when the beam is parallel. The use of multiple positions for the recording of peak systolic aortic velocity is very important in aortic stenosis since this jet may be directed in a wide variety of orientations. This is especially true in older patients with acquired aortic stenosis. **Figure 3.25** demonstrates one such direction of flow and its relationship to various transducer positions for CW Doppler recording. In this case, peak flow was best recorded by CW Doppler from the right parasternal approach, rather than from the suprasternal notch. The velocity profile from the apex seems adequate but is slightly lower than the right sternal recording. The recording from the suprasternal notch is grossly inadequate and lacks a fully formed profile. When examining for aortic stenosis, all available acoustic windows should be utilized.

There will be times when the changing appearance of the spectral trace is not the result of an improper beam direction or misadjustment of system controls. **Figure 3.26** shows a CW recording from the suprasternal notch with the beam directed toward the ascending aorta. The differing appearances of the velocity profiles are a result of an irregular heart rate which leads to beat-to-beat changes in stroke volume and, consequently, aortic gradient. Stenotic jets, like regurgitant jets, readily change their configuration with cardiac rhythms.

**Aortic Valve Gradient at Catheterization**

Estimation of transvalvular aortic gradients in patients with aortic stenosis using Doppler has been in common use for some time. There is an abundance of papers in the literature discussing the relative merits and limitations of this approach. All are based upon correlations with pressure measurements obtained at catheterization.

In **Figure 3.27** three possible methods are shown for calculating pressure gradients across the aortic valve at catheterization. All depend upon the recording of pressure from the left ventricle and aorta, or some peripheral artery. If a peripheral artery is used, it takes time for the systolic pulse to
be conducted into the aorta and there is usually a short delay in the upstroke of the aortic pulse when compared with that of the ventricle. This requires the individual measuring these pressures to trace the aortic pulse and move it slightly backwards in time to adjust for the time delay. A peak to peak gradient is measured from the peak of the left ventricular pressure recording to the peak of the aortic. Peak gradient is measured as the largest difference between the two and occurs somewhere on the ascending pressure tracings. Mean gradient is estimated by summing the gradients measured at sequential time intervals during systole and dividing by the number of measurements made.

Appreciation of these various invasive methods for calculation of the aortic valve gradient is very important for a critical interpretation of the Doppler literature on aortic stenosis. Almost all catheterization laboratories report peak to peak and mean gradients since both are readily performed. Accurate measurement of peak gradient, however, is more difficult and requires precise alignment of ventricular and aortic pressures in time, and careful searching for the peak, or largest, instantaneous gradient.

Actual left ventricular and brachial arterial pressure tracings from a patient with aortic stenosis are shown in Figure 3.28. Note that the rate of rise of the left ventricular pressure is much more rapid than that of the brachial artery. Note also that there is a time delay in the onset of rise of the brachial artery. Note also that there is a time delay in the onset of rise of the brachial arterial pulse. This results because of the time required for the bolus of ejected blood to transit the aortic valve and aorta to the brachial artery. For proper measurement of peak gradient the brachial pulse must be “set back” to correspond to the rise of the left ventricular pressure tracing.

With the fluid-filled catheters commonly used, rapid changes in pressure, such as occur with ventricular ejection, occasionally create overshoot artifacts on the pressure recording. Peak gradients are often not reported from catheterization data, in part because of these artifacts and timing delays which can create large measurement errors.

**Doppler Estimation of Gradient in Aortic Stenosis**

It is most important to remember that the peak gradient is usually higher than the peak-to-peak or mean gradients. The spectral recordings resulting from Doppler interrogations of aortic stenosis reflect the highest gradients within the jet. This is frequently referred to as “peak instantaneous gradient”. Doppler estimates of the aortic peak gradient calculated using the simplified Bernoullis
formula generally overestimate severity when compared with catheterization peak-to-peak gradients. Accordingly, Doppler estimates of severity would be expected to correlate best with the peak gradient at catheterization.

In an attempt to evaluate the use of Doppler echocardiography in clinical practice, we studied sixty consecutive patients who were referred to the catheterization laboratory with clinical findings suggestive of possible aortic stenosis (aortic systolic murmur and peripheral pulse deficit). All patients underwent cardiac catheterization at a time remote from the Doppler study (usually after 24 hours). The Doppler examination and interpretation was performed without knowledge of the results of catheterization. Our patients were older than many of the groups reported from other centers (mean age 63 years), as one might anticipate in routine clinical practice, and 45% had a 2+ or greater aortic insufficiency.

As can be seen in Figure 3.29, Doppler over-estimation of catheterization peak-to-peak gradient in our series was sometimes quite significant. Indeed, overestimations are evident over the entire spectrum of gradient values and vary in magnitude between 1 and 53mmHg. In our view, this finding suggests that the clinician must be very careful in using catheterization laboratory criteria for estimating the severity of an aortic gradient with Doppler peak aortic gradient valves.

Doppler and catheterization estimates are more comparable when both laboratories report the peak aortic valve gradient (Fig. 3.30). The scatter of our data is obviously great because the studies were not performed simultaneously with catheterization. Altered hemodynamic states with different volumes of blood flow across the aortic valve could easily account for the differences between catheterization and Doppler gradients in nonsimultaneous studies. When Doppler data are collected simultaneously with catheterization, using micromanometer-tipped catheters, the correlations are much better.

In routine clinical practice, Doppler estimates of the severity of aortic stenosis are likely to be made at least 24 hours before catheterization. These Doppler measurements may find a role in selecting patients for invasive study. Eventually, they may be used to refer some patients to surgery without prior catheterization.

Figure 3. 29 Idealized relationship between Doppler peak gradient and catheterization peak-to-peak gradient. Comparisons show that there is overestimation of catheterization peak-to-peak gradient.

Figure 3. 30 Idealized relationship between Doppler peak gradient and catheterization peak gradient. Since Doppler reflects peak catheterization gradient, the comparisons are more favorable.
It is encouraging that even though comparative data were collected at different times in our study, peak gradients correlated favorably. In our experience, if heart rates vary more than 20 beats/min between the two studies, agreement between them will be reduced (due to differences in blood flow across the valve). Therefore, heart rate serves as one convenient index for assessing the similarity between hemodynamic states, when Doppler and catheterization comparison studies are performed at remote times.

There are some limitations inherent in using Doppler peak aortic gradient estimates. Few catheterization laboratories report peak gradient data, and a suitable frame of reference to judge severity of stenosis (as exists for peak-to-peak gradients) is not available. Clinicians have commonly used peak-to-peak gradients in excess of 50mmHg to identify severe aortic stenosis. There is no corresponding figure for peak gradients in current use.

The comparison of mean Doppler gradient with mean catheterization gradient also shows good overall agreement (Fig. 3.31). Mean gradients may be less sensitive to individual measurement errors since they reflect an average of multiple measurements. The calculation of mean Doppler gradient also has the advantage that such gradients are available from most catheterization laboratories and are familiar to most clinicians. When Doppler and catheterization measurements are collected simultaneously, the correlations are even better because of these factors.

Understanding the details of these various gradient comparisons is important for the beginner to Doppler echocardiography. We have seen a conflict of opinion develop between the echocardiography and catheterization laboratories when there is not a mutual understanding of the capabilities and limitations of both techniques. Despite these cautions, we do believe that Doppler comparison with catheterization data acquired with pressure transducer-tipped catheters are most satisfactory when obtained simultaneously.

**Other Aspects of Aortic Stenosis**

We occasionally encountered patients with a small gradient predicted by Doppler but with no gradient at catheterization. In most instances, heart rates were nearly identical at the time of Doppler and catheterization, making it unlikely that differences in hemodynamic status were responsible for the discrepancies. However, it is noteworthy that all of these patients had high cardiac outputs and, in most, significant aortic regurgitation was present. It is possible that the use of the simplified Bernoulli equation in such patients may be at least partly responsible for the overestimates. The full Bernoulli equation takes into account blood velocity on both sides of the valve, whereas the simplified form only uses peak velocity after the flow crosses the valve.

In Figure 3.32 the velocity of flow on the ventricular side of the valve ($V_1$) is simultaneous with flow in the aortic side ($V_2$) using CW Doppler from the apical approach. Patients with hyperdynamic circulatory states, such as those with aortic regurgitation, may have a significant velocity component below the aortic valve. We have also encountered a few patients with very
severe systemic hypertension (with high peripheral resistance) and high cardiac outputs where Doppler peak systolic velocities approached 5 m/s. In each, no gradient was found at catheterization.

Failure to take these experiences into account may lead the Doppler operator to attribute the elevated aortic velocity to an obstruction of the outflow tract, even in patients with pure aortic regurgitation or other reasons for the increased systolic velocities. In our series, the effect of aortic insufficiency was most evident when there was a minimal or no aortic valve gradient. It seemed to be less important in patients with combined stenosis and insufficiency.

Several observations may be helpful in recognizing such overestimated gradients. In patients with increased systolic velocities due to aortic insufficiency, early peaking of the aortic velocity profile suggests an insignificant gradient. In addition, patients whose aortic valve cusps open to the periphery of the root, on echocardiography will rarely, if ever, have significant aortic gradients at catheterization. Indeed, full mobility of the aortic valve cusps frequently indicates caution against assuming that an increased velocity is due to aortic stenosis. It may be best to use PW Doppler echocardiography with the sample volume located just before the aortic valve orifice to verify that $V_1$ is within the range of normal. If high velocities are encountered at this level, caution should be exercised and if $V_1$ is high it must be taken into account.

**Other Estimates of Severity of Aortic Stenosis**

Alternative methods are available for estimating the severity of aortic stenosis. One method uses the time to peak velocity in systole and examples are illustrated in Figure 3.33. Panel A shows a patient with less severe stenosis and a shorter relative time to peak than the patient in the panel B. A value of 0.50 or greater has been found to correlate with moderate-to-severe obstruction. The method is most accurate when clear evidence of aortic valve opening and closing is seen on the Doppler recording. If these are not evident, a phonocardiogram or Doppler signal amplitude tracing must be used to indicate the boundaries of the systolic ejection period.

One of the most promising methods makes use of the principle of the continuity of flow and may be used to estimate aortic valve area. The principle is simple: forward volume flow on the ventricular side of the valve is the same as forward flow on the aortic side. Whether or not an obstruction is present, these two flows are always equal.

**Figure 3. 32** Velocity below the valve ($V_1$) is not recorded as often as velocity on the aortic side ($V_2$). $V_1$ is ignored in the simplified Bernoulli equation. (Scale marks = 1 m/s)

**Figure 3. 33** The severity of aortic stenosis may also be judged by the relative proportion of total systolic time taken to reach peak velocity (stippled areas). Both time to peak and peak velocity are lower in panel A than in panel B. (Scale marks = 1 m/s)
Flow must first pass through a larger area (with low velocity) before entering the obstruction where the velocity will increase. Thus, there must always be a continuity of flow.

During the previous discussion of cardiac output calculation, it was shown that volume flow could be estimated from knowing the area of the aortic valve orifice and the flow velocity integral that crosses it in systole. This knowledge sets up the simple algebraic equation, shown in Figure 3.35, in which we want to find the aortic valve area ($A_2$).

In aortic stenosis, systolic flow first passes through the left ventricular outflow tract at one velocity ($V_1$) and then is rapidly accelerated to a higher velocity ($V_2$) through the narrowed area of the stenotic orifice. Both $V_1$ (using PW Doppler in the left ventricular outflow tract) and $V_2$ (using CW Doppler) can be determined. Both are usually obtained from the apical window.

The area of the outflow tract ($A_1$) may also be measured and requires the use of two dimensional echocardiography. This is best performed by measuring the radius at the base of the aortic cusps in the parasternal long axis, in a similar way as for calculation of cardiac output. Using the algebraic relationships given in Figure 3.35, the equation may then be solved for the area of the obstructed aortic valve ($A_2$).

Using this equation, it is not even necessary to perform the complex calculation of the flow velocity integrals for $V_1$ and $V_2$. The spectral recordings of each may be likened to triangles with bases that are equal in size but heights that are different. Their time durations (bases of the triangles) are nearly identical if the patient is in sinus rhythm. Therefore, the flow velocity integrals of both are nearly proportional to their height (or peak velocities). These concepts are brought together in diagrammatic form in Figure 3.36.

The Clinical Role for Doppler in Aortic Stenosis

Determining the severity of aortic stenosis by physical examination can be difficult, particularly in older patients. One useful clinical role for Doppler echocardiography would be to serve as a supplement to the history and physical examination in patients where the physical examination is confusing. In our experience it has been valuable in identifying
patients with significant aortic stenosis, particularly in the elderly. A perfectly normal Doppler examination, when aortic stenosis is clinically suspected, has been of extreme help in avoiding unnecessary cardiac catheterization in these individuals.

However, it is important for the beginner to appreciate that difficult patients do occur and considerable experience is required to perform these procedures properly. When recordings of poor quality are seen, they should be disregarded since they will generally underestimate the severity of the gradient. Even when good traces are obtained, it is possible to record falsely high gradients by Doppler, especially in the setting of aortic insufficiency. We have found that it takes most beginners a year of experience of examining reasonable numbers of patients to obtain reliable results.

### Mitral Gradient in Mitral Stenosis

The best window for examination of mitral valve diastolic flow is invariably apical. With the transducer at the cardiac apex, the ultrasound beam should be directed posteriorly and slightly laterally to intercept mitral valve flow. In normal individuals, PW Doppler is adequate for recording mitral valve diastolic flow. Mitral flow is typically laminar and biphasic (Fig. 3.37), peaking in early diastole (closed arrow) and rising again with atrial contraction in late diastole (open arrow).

![Figure 3.37](image)

*Figure 3.37* PW Doppler spectral recording from the mitral orifice taken from the apical window. Early diastolic flow is high (closed arrow), followed by a rapid descent and then peaks again after atrial contraction (open arrow) (Scale marks = 20cm/s)

The examination for mitral stenosis is usually much easier and more straightforward than that for aortic stenosis. The typical CW spectral recording of mitral stenosis demonstrates spectral broadening in diastole, with peak flow in early diastole and a progressive but slowed diastole descent (Fig. 3.38).

![Figure 3.38](image)

*Figure 3.38* Typical CW spectral velocity recording from a patient with mitral stenosis and insufficiency. From the apex, the diastolic flow of mitral stenosis is toward the transducer. There is a rise in velocity in early diastole followed by a slow diastolic descent.

The secondary increase in diastolic velocity due to atrial contraction is absent in patients with atrial fibrillation.

A mitral valve gradient is calculated using the modified Bernoulli equation, discussed previously. The spectral recording in Figure 3.39 shows a peak diastolic velocity of 2 m/s that is equivalent to a 16mmHg peak transmittal pressure gradient. As with aortic stenosis, the transmitral pressure gradient may be reported in several ways. Catheterization laboratories usually report the mean gradient. In order to compute a comparable mean gradient for Doppler data, multiple instantaneous peak gradients must be measured during diastole (such as 40-100 ms intervals) and the values averaged. At least 10 well
formed Doppler profiles should be averaged in this manner if the patient is in atrial fibrillation.

Results published to date have shown excellent agreement between CW Doppler estimates of the mitral valve pressure gradient, using the simplified Bernoulli equation, and simultaneous estimates derived from cardiac catheterization data. However, when the two studies are performed on separate days, the agreement between the two is reduced. This apparent discrepancy derives, in part, from the labile nature of the mitral pressure gradient. The value of this parameter at any particular instant is determined not only by the extent of mitral valve obstruction present but also by the flow across the valve (i.e., cardiac output) and the length of the diastolic filling period, which in turn is determined by the heart rate. Therefore, if the heart rate during catheterization differs from the rate during the Doppler study, the pressure gradients estimated by these two techniques would be expected to differ. In this situation, the higher gradient would be recorded in the study performed at the faster heart rate.

These relationships are pointed out in the idealized pressure recordings shown in Figure 3.40. The diastolic gradient is relatively low at rest (left panel) and rises significantly with exercise as heart rate and cardiac output rise (right panel).

**Mitral Valve Area Estimates**

The sensitivity of the mitral pressure gradient to changes in heart rate and cardiac output clearly makes it an incomplete description of the severity of mitral stenosis. As stated previously, valve area is generally considered not to vary with changes in cardiac output and is the preferred method for expressing the severity of mitral stenosis.

Mitral valve area may be calculated at catheterization using pressure gradient and flow information by the Gorlin equation. It may also be directly planimetered in diastole using the two-dimensional, echocardiographic image of the mitral valve orifice obtained in the short-axis view. In our laboratory, catheterization-derived mitral valve area correlates well with that measured by a two-dimensional echocardiography. We find both two-dimensional echocardiography and Doppler to be useful for noninvasive assessment of the severity of mitral valve obstruction.

A method has been described for estimating mitral valve area from Doppler measurements. The approach is based on the measurement of a parameter called the atrioventricular (AV) pressure half-time. This quantity was originally proposed as an alternative to the Gorlin formula for
estimating the severity of mitral stenosis from catheterization pressure data. It is defined as the
time interval in milliseconds (ms) required for the diastolic pressure gradient across the mitral valve
to fall to half of its initial value.

The original studies of atrioventricular pressure half-time carried out in the catheterization
laboratory showed it to correlate well with effective mitral valve area and to be relatively
insensitive to changes in heart rate or cardiac output. Doppler pressure half-time calculations
reflect the rate of diastolic descent of the spectral velocity recording.

In normal subjects, pressure half-time values range between 20 and 60 ms. In patients with mitral
stenosis, there is a marked delay and a strong correlation between the pressure half-time estimated
from Doppler data and the mitral valve area calculated from catheterization data using the Gorlin
formula. Pressure half-time is relatively insensitive to the effects of exercise, atrial fibrillation or
coexisting mitral regurgitation. Patients with mitral stenosis had values from 100 to 400 m/s, with
the higher values seen in subjects with smaller valve areas. Pressure half-times greater than 220 ms
correlate well with a valve area less than 1.0 cm². Patients with isolated mitral regurgitation have
values \( \leq 80 \) ms.

The pressure half-time is the time for the
diastolic pressure gradient in mitral stenosis to
fall to half of its original value. Figure 3.41
illustrates how the pressure half-time is
measured. The starting point is the time of
peak velocity (point 1), which in this case is 2.2
m/s. This corresponds to a peak pressure
gradient of 19mmHg by the simplified
Bernoulli equation. A line along the diastolic
descent of the mitral valve velocity spectrum is
drawn (step 2). A point is then found along this
line where the pressure has dropped to half of
its initial value (point 3). This point is rapidly
determined by dividing the initial velocity by
1.4 (square root of 2); that is, 2.2/1.4=1.6 m/s.
Thus, when velocity falls to 1.6m/s the pressure
is at half of its initial (point 1) value. The pressure half-time is simply the time interval between
point 1 and point 3, in this case 400 milliseconds (interval 4).

Beginners sometimes have difficulty relating
the spectral velocity tracings to pressure
changes on which this method is based. Most
of this confusion is based on the relationship
between pressure gradients and velocity. It is
important to remember that the spectral
recordings display velocity information.
Derivations of pressure are based upon the
modified Bernoulli equation where the gradient
equals 4V². Thus, the time taken for the
gradient to fall to half the original is always
shorter than the time taken for the velocity to decrease to half.

Subsequently, it was found that the pressure half-time could be used to estimate mitral valve area
from an empirical formula shown in Figure 3.42. In the case of the patient in Figure 3.41, the
predicted valve area would be 0.6cm². There have been several published correlations of this method for estimating mitral valve area with that derived by the Gorlin equation catheterization, with generally favorable results. The effect of heart rate and other factors that might influence the reliability of these calculations has not, however, been adequately addressed. We find that estimation of valve area by the pressure half-time method is always best confirmed by direct planimetry of the stenotic mitral valve orifice method on the two-dimensional echocardiogram. When the results are disparate, the reliability of estimates by both methods should be questioned.

**Tricuspid Stenosis**

Evaluation of tricuspid stenosis with Doppler poses problems similar to mitral stenosis but, since it is a rarer disease, the accumulated experience is less extensive. The presence of tricuspid stenosis is readily determined with either pulsed or continuous wave Doppler by detecting turbulence and increased velocities emerging from the tricuspid orifice into the right ventricle during diastole (Fig. 3.43). Since the pressure gradient across a stenotic tricuspid valve is fairly low, even with severe obstruction, peak Doppler velocities will be correspondingly reduced from those seen in severe mitral stenosis. Experience with both catheterization-derive and Doppler-derived estimates of tricuspid valve areas is limited. Consequently, assessment of the severity of tricuspid obstruction in the noninvasive laboratory consists of a two-dimensional, echocardiographic evaluation followed by Doppler measurement of the transvalvular mean pressure gradient.

**Pulmonic Stenosis**

Like all the other cardiac valves, stenosis of the pulmonic valve results in marked turbulence and an increase in velocity. A CW Doppler recording of pulmonic stenosis is shown in Figure 3.44. The peak velocity is nearly 4 m/s, indicating that a 64mmHg gradient is present. Gradients estimated by Doppler have favorable comparisons with those obtained at catheterization. In high right-sided flow states, such as those seen in patients with large left-to-right shunts, there may be a mild elevation in peak pulmonary arterial flows. This presumably results from the increased volume being ejected through the normal pulmonic valve orifice. In these cases it is sometimes
difficult to determine the presence of mild pulmonic stenosis.

OTHER OBSTRUCTIVE LESIONS AND GRADIENTS

Coarctation of the Aorta

These basic principles may be applied to almost any situation where pressure-gradient information is desired. Coarctation of the aorta may be readily identified with the transducer in the suprasternal notch and the Doppler beam angled slightly anteriorly and to the patient’s right side. Systolic flow from the ascending aorta is encountered moving toward the transducer using this window (Fig. 3.45). With angulation of the beam slightly posteriorly, the higher velocity jet through the coarctation in the descending aorta is then encountered moving away from the transducer. Doppler echocardiography has been shown to be a reliable method for determining the presence of this disorder.

Hypertrophic Obstructive Cardiomyopathy

The area of obstruction in hypertrophic obstructive cardiomyopathy (also called idiopathic hypertrophic subaortic stenosis) is thought to occur where the anterior mitral valve leaflet opposes the interventricular septum. This is a dynamic, rather than fixed, obstruction that usually becomes more severe as systole progresses.

In Figure 3.46 idealized aortic and left ventricular pressure tracings indicate that the peak gradient usually occurs in mid to late systole as a consequence of the changing relationships between the tip of the mitral leaflet and the septum. On the right is a typical CW spectral recording from a patient with this disorder. Note the late peaking of the jet as a result of the altered pressure dynamics.

Continued clinical practice indicates that patients with this disorder may have some of the highest velocities encountered by Doppler echocardiography. The peak velocity illustrated in
There are many other interesting phenomena that occur in patients with obstructive cardiomyopathy. Careful CW examination will frequently reveal multiple patterns of $V_1$ in relationship to $V_2$. One such unusual pattern is seen in Figure 3.47 where $V_1$ appears to fall during mid-systole, then rise again in late systole. These unusual patterns have not yet been fully correlated to the hemodynamic events in these patients, but differentiate CW spectral recordings of dynamic outflow obstruction from that due to fixed obstruction at the valvular level.

**Ventricular Septal Defect**

Ventricular septal defects are usually best interrogated using the parasternal long-axis approach. In this view, the abnormal flows between ventricles are normally parallel to the beam, and reasonably faithful velocities are recorded.

Doppler serves as a method for estimating right ventricular systolic pressures when ventricular septal defects are encountered. Figure 3.48 shows CW spectral recordings from two different patients with ventricular septal defects taken from the left parasternal approach. The peak velocity in the child in the left panel is slightly higher than 4 m/s, indicating a 64mmHg gradient between left and right ventricles. The peak velocity in the infant in the right panel is 2 m/s, indicating a gradient of only 16 mmHg.

Since aortic (and, therefore, left ventricular) systolic pressure can be measured by a blood pressure cuff, the difference between the cuff pressure and the gradient measured by Doppler results in the estimated right ventricular systolic pressure. Given the fact that both patients in Figure 3.48 had systemic systolic pressures of 110mmHg by cuff, the patient on the left had an estimated right ventricular systolic pressure of 46mmHg while the patient on the right had an estimated right ventricular systolic pressure of 94mmHg.

PW Doppler echocardiography has been used for the calculation of pulmonary-to-systemic shunt ratios in patients with intracardiac shunting. The approach is based upon the principles described earlier in the section on calculation of cardiac output. In brief, output through the pulmonary valve is related to output through the aortic valve.
DOPPLER EXAMINATIONS FOR VALVULAR STENOSIS

As seen from the many examples described in this Unit, considerable operator skill is necessary to acquire the critical quantitative data necessary by Doppler for the assessment of valvular stenosis. Points to keep in mind when carrying out an examination include:

**Hint 1:** An operator should never be satisfied by interrogating an abnormal jet by only one view. Stenotic jets may move in any direction. This is particularly true of aortic stenosis and apical, right parasternal, and suprasternal approaches must be used before an operator can be assured that the highest peak jet possible has been recorded.

**Hint 2:** Reliable quantitative assessments require that high quality spectral recordings are available for analysis. Measurement of peak velocities from poorly formed Doppler profiles may lead to serious underestimations of transvalvular gradients. When it is impossible to acquire a fully formed profile, it is best not to attempt quantitative assessment. Beginners are sometimes tempted to make some measurement with the expression that a gradient is “at least” a certain amount. In our experience, this only leads to confusion in later decision-making processes.

**Hint 3:** Much of the utility of Doppler for the assessment of valvular stenosis is based on the use of CW methods. Since this is frequently performed with a stand-alone transducer, there is no image available for anatomic reference. An operator must use frequent voice annotations on the video recording to assist in later interpretation and reference. We recommend voice notation of the valve being interrogated and the angulations being used as the beam is directed spatially. This frequently helps in eliminating confusion between the various abnormal flows that might be encountered during a Doppler examination.

**Hint 4:** When interpreting Doppler information for assessment of the severity of valvular stenosis all available information must be taken into account to avoid excessive dependence on any one quantitative index. For example, a patient with severe aortic stenosis, a dilated left ventricle and low cardiac output may only have a 3 m/s aortic stenotic jet since flow across the aortic valve is low. Without an estimate of aortic valve area, this would lead to the incorrect assumption that minimal aortic stenosis is present.
Figure Legends

Figure 3.1 CW Doppler recording of normal aortic systolic velocity taken from the suprasternal notch. Note that the onset of flow toward the transducer begins after the QRS complex of the electrocardiogram (arrow) and peaks in the first third of systole.

Figure 3.2 Many systolic velocity indices may be calculated from the flow in the ascending aorta, as indicated in these idealized spectral recordings.

Figure 3.3 The various ejection rate indices relate to left ventricular performance (ejection fraction).

Figure 3.4 Stroke volume is the volume of blood ejected from the left ventricle with each systole. Knowing stroke volume and heart rate provides a means for calculating cardiac output. Doppler may be used for calculating stroke volume. (CO = cardiac output; SV = stroke volume; HR = heart rate).

Figure 3.5 Echo-Doppler estimates of flow volume are based upon a knowledge of the area of flow (from echocardiogram) and the length (from Doppler). It is assumed that the aorta is a cylinder.

Figure 3.6 When stroke volumes are equal and areas remarkably different, the resultant velocities of flow may be quite different. The velocity for large areas would be less than for small areas.

Figure 3.7 Illustration of all the steps required for the calculation of cardiac output by Doppler.

Figure 3.8 Idealized representation of echo-Doppler calculations of cardiac output compared with that determined by thermodilution.

Figure 3.9 Idealized comparisons of mean pulmonary artery (PA) pressure to time-to-peak velocity (left) and the logarithm of mean pulmonary artery pressure to peak velocity (right).

Figure 3.10 Idealized spectral recordings demonstrating that time-to-peak velocity is very rapid in patients with pulmonary hypertension.

Figure 3.11 PW Doppler spectral recording of aortic blood flow (arrow) taken from the apical window. Note the laminar appearance of normal flow. (Scale marks = 20 cm/s)

Figure 3.12 Left panel – Without aortic valve obstruction, systolic pressures are almost the same in the ventricle and the aorta. Right panel – When significant aortic valve obstruction is present, left ventricular pressure rises much higher than aortic, and a systolic pressure gradient is present. The size of the arrows represents the magnitude of the pressures.

Figure 3.13 CW spectral recording from the apex in a patient with aortic stenosis. The velocity spectrum is broadened and systolic velocity is increased to 4 m/s. (Scale marks = 2 m/s)

Figure 3.14 CW Doppler spectral velocity recording of mild pulmonic stenosis and insufficiency. The abnormal diastolic flow toward the transducer of pulmonic insufficiency is easily recognized. (Scale marks = 1 m/s)

Figure 3.15 PW Doppler spectral recording from the same patient as Figure 3.24. At position A, pulmonic insufficiency is noted. At position B, on the distal side of the valve, the systolic velocity is elevated. (Scale marks = 20 cm/s)

Figure 3.16 Typical CW spectral velocity tracing from the apex in a patient with aortic stenosis and insufficiency. Peak systolic velocity is elevated to almost 6 m/s and peaking is delayed. (Scale marks = 2 m/s)

Figure 3.17 Idealized relationship between pressure gradient and flow. As flow increases, the gradient will also rise even though the valve area is fixed.

Figure 3.18 CW Doppler spectral recording of aortic outflow from the suprasternal notch with flow toward the transducer (left) and apex with flow away from the transducer (right). The spectral recording from the apex is better formed than the one from the suprasternal notch.
Figure 3. 19 Typical aortic systolic velocity recording from the suprasternal notch in a patient with aortic stenosis. Note that the peak velocity is almost 5 m/s. As gradient increases, so does the peak systolic velocity. (Scale marks = 1 m/s)

Figure 3. 20 Operator skill is important in obtaining an adequate systolic aortic jet profile in aortic stenosis. This figure shows a recording made by a less experienced operator (left panel) compared with one from a more experienced operator (right panel).

Figure 3. 21 The systolic jet of aortic stenosis and diastolic jet of aortic insufficiency often cannot be recorded at the same time. As the transducer beam is angled from the stenotic jet (closed arrow) to intercept the aortic insufficiency, the left ventricular outflow tract velocity is encountered (stippled arrow). Both outflow tract velocities are superimposed during the beam sweep (open arrow). (Scale marks = 1 m/s)

Figure 3. 22 Inadequate recordings of aortic systolic velocity do occur and should not be used for estimation of gradient. Note that the profile is poorly formed on the upstroke, peak (arrow), and down stroke. (Scale marks = 2 m/s)

Figure 3. 23 Aortic stenosis (left) should not be mistaken for mitral insufficiency (right). Mitral systole begins before aortic (arrow) and is longer in duration. (Scale marks = 2 m/s)

Figure 3. 24 Relationship between abnormal systolic and diastolic flows through the left heart valves. For details, see text.

Figure 3. 25 Left panel shows an aortic stenotic jet in relation to possible viewing directions using CW Doppler. Right panel shows spectral velocity tracings from each respective window. The best recording is from the right sternal window. (Calibration marks = 2 m/s)

Figure 3. 26 CW spectral velocity recording from the suprasternal window into the ascending aorta from a patient with severe stenosis. Note the varying peak velocities with varying R-R interval of the ECG. (Calibration marks = 1 m/s).

Figure 3. 27 Schematic representation of simultaneous left ventricular (LV) and aortic (Ao) pressure recordings obtained at catheterization with representation of different gradient measurement methods.

Figure 3. 28 Pressure recordings from the left ventricle (LV) and brachial artery (BA) from a patient with aortic stenosis. Note the delay in the upstroke of the brachial arterial pressure tracing and the delay in onset of the brachial pressure tracing when compared with that from the ventricle.

Figure 3. 29 Idealized relationship between Doppler peak gradient and catheterization peak-to-peak gradient. Comparisons show that there is overestimation of catheterization peak-to-peak gradient.

Figure 3. 30 Idealized relationship between Doppler peak gradient and catheterization peak gradient. Since Doppler reflects peak catheterization gradient, the comparisons are more favorable.

Figure 3. 31 Idealized relationship between Doppler mean gradient and catheterization mean gradient. Mean gradient comparisons are favorable because they average data over time.

Figure 3. 32 Velocity below the valve ($V_1$) is not recorded as often as velocity on the aortic side ($V_2$). $V_1$ is ignored in the simplified Bernoulli equation. (Scale marks = 1 m/s)

Figure 3. 33 The severity of aortic stenosis may also be judged by the relative proportion of total systolic time taken to reach peak velocity (stippled areas). Both time to peak and peak velocity are lower in panel A than in panel B. (Scale marks = 1 m/s)

Figure 3. 34 Continuity of forward flow. Flow that enters a cylinder is equal to the flow passing through an obstruction and exiting from the distal side.

Figure 3. 35 The continuity of flow equation. For details, see text.

Figure 3. 36 The continuity of flow relating the Doppler-determined velocity and area below the stenotic aortic valve ($V_1$ and $A_1$) to the Doppler-determined velocity and area at the stenotic site ($V_2$ and $A_2$). For details, see text.
Figure 3.37 PW Doppler spectral recording from the mitral orifice taken from the apical window. Early diastolic flow is high (closed arrow), followed by a rapid descent and then peaks again after atrial contraction (open arrow) (Scale marks = 20cm/s)

Figure 3.38 Typical CW spectral velocity recording from a patient with mitral stenosis and insufficiency. From the apex, the diastolic flow of mitral stenosis is toward the transducer. There is a rise in velocity in early diastole followed by a slow diastolic descent.

Figure 3.39 Typical diastolic pattern of mitral stenosis using CW Doppler. Note early diastolic velocity rises to 2 m/s. Mitral valve pressure gradients may be estimated using this technique. For details, see text. (Scale marks = 1m/s)

Figure 3.40 Idealized pressure gradients in mitral stenosis demonstrating that at rest the gradient may be low (left panel) but rises significantly with exercise (right panel).

Figure 3.41 Method for calculation of pressure half-time by Doppler. For details, see text. (Scale marks = 1m/s)

Figure 3.42 Formula for estimation of mitral valve area in cm² using the pressure half-time. The number 220 is an empiric constant.

Figure 3.43 CW Doppler recording of tricuspid stenosis. Note the similarity in appearance to mitral stenosis.

Figure 3.44 CW Doppler spectral recording from a child with pulmonic stenosis. Note that velocity approaches 4m/s, indicating a significant obstruction across the pulmonic valve. (Scale marks = 1m/s)

Figure 3.45 Schematic representation (left) showing direction of the CW beam from the suprasternal window into the ascending aorta (A) and descending aorta (B) in a patient with coarctation of the aorta. The resulting spectral velocity recording is shown on the right. (Scale marks = 1m/s)

Figure 3.46 In hypertrophic obstructive cardiomyopathy, the largest gradient occurs in mid-to-late systole (left panel). This usually shows a late peaking velocity across the left ventricular outflow tract (right panel). (Scale marks = 1m/s)

Figure 3.47 CW Doppler spectral recording from the apex across the area of obstruction from a patient with hypertrophic obstructive cardiomyopathy. Note the mid-systolic decreases in $V_1$. Many varied patterns to $V_1$ may be seen in this disorder.

Figure 3.48 CW spectral recordings from two different patients with ventricular septal defects. If both patients had similar systemic systolic pressures, the low gradient in the patient on the right would indicate nearly systemic right ventricular pressures. For details, see text. (Scale marks = 1m/s)