Hypertrophic Cardiomyopathy

Systolic Anterior Motion Begins at Low Left Ventricular Outflow Tract Velocity in Obstructive Hypertrophic Cardiomyopathy

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OBJECTIVES

The purpose of this study was to determine whether the dynamic cause for mitral systolic anterior motion (SAM) is a Venturi or a flow drag (pushing) mechanism.

BACKGROUND

In obstructive hypertrophic cardiomyopathy (HCM), if SAM were caused by the Venturi mechanism, high flow velocity in the left ventricular outflow tract (LVOT) should be found at the time of SAM onset. However, if the velocity was found to be normal, this would support an alternative mechanism.

METHODS

We studied with echocardiography 25 patients with obstructive HCM who had a mean outflow tract gradient of 82 ± 6 mm Hg. We compared mitral valve M-mode echocardiogram tracings with continuous wave (CW) and pulsed wave (PW) Doppler tracings recorded on the same study. A total of 98 M-mode, 159 CW, and 151 PW Doppler tracings were digitized and analyzed. For each patient we determined the LVOT CW velocity at the time of SAM onset. This was done by first determining the mean time interval from Q-wave to SAM onset from multiple M-mode tracings. Then, CW velocity in the outflow tract was measured at that same time interval following the Q-wave.

RESULTS

Systolic anterior motion began mean 71 ± 5 ms after Q-wave onset. Mean CW Doppler velocity in the LVOT at SAM onset was 89 ± 8 cm/s. In 68% of cases SAM began before onset of CW and PW Doppler LV ejection.

CONCLUSIONS

Systolic anterior motion begins at normal LVOT velocity. At SAM onset, though Venturi forces are present in the outflow tract, their magnitude is much smaller than previously assumed; the Venturi mechanism cannot explain SAM. These velocity data, along with shape, orientation and temporal observations in patients, indicate that drag, the pushing force of flow, is the dominant hydrodynamic force that causes SAM. (J Am Coll Cardiol 2000;36:1344–54) © 2000 by the American College of Cardiology

Systolic anterior motion (SAM) of the mitral valve is the cause of dynamic outflow obstruction in most patients with hypertrophic cardiomyopathy (HCM). There is agreement that SAM is caused by the action of left ventricular (LV) flow on the protruding mitral valve leaflet (1). The nature of the hemodynamic force on the leaflet remains a subject of unresolved debate (2–5). Initially, investigators hypothesized that anterior motion is caused by a Venturi mechanism, whereby high velocity flow in the outflow tract lifts the mitral valve toward the septum. More recent investigations indicate that drag, the pushing force of flow, is the dominant hydrodynamic force that initiates anterior motion, pushing the protruding mitral leaflet into the septum (6–9). Flow drag is the component of force on a body that is in the direction of the flow—examples are the familiar pushing force of rushing water or the wind. One of these studies used normal canine subjects and one was an in vitro investigation. Two were studies of HCM patients. Despite these investigations, prominent sources describe a Venturi mechanism for SAM (3–5). Thus, the present study of obstructed HCM patients was undertaken in an attempt to clarify this question.

Whenever flow traverses a surface such as the mitral valve, both lift and drag forces are generated. Pertinent to the present study is that lift-to-drag ratio falls with decreasing velocity (10,11). During an earlier study we noticed that color flow velocity in the left ventricular outflow tract (LVOT) seemed low when SAM began, as deduced by others previously (8). In the present study we reasoned that if SAM were caused by the Venturi mechanism, we should find high velocity flow in the LVOT at the moment of SAM onset. Conversely, if we found low velocity at that time, this would support a flow drag mechanism. These early systolic velocities have not previously been measured. Consequently, we have systematically studied the echocardiograms of patients with significant obstruction, specifically measuring velocity of early systolic outflow at SAM onset.
METHODS

Patients. We prospectively studied 25 consecutive patients with obstructive HCM who had resting LV outflow gradients greater than 36 mm Hg and technically adequate echocardiograms. Mean age was 63 years (27 to 86 years); 11 were women. Patients were referred for clinical indications; medications were continued at the time of the study. All patients had asymmetric LV hypertrophy with septal thickness >15 mm and had no apparent cause for the hypertrophy (12). None had hypertension or aortic valve disease. All had SAM and mitral–septal contact. The peak pressure gradient across the LVOT was determined using continuous wave (CW) Doppler in the apical five-chamber view (13). All patients were in sinus rhythm.

Abbreviations and Acronyms

CW = continuous wave
HCM = hypertrophic cardiomyopathy
LV = left ventricle, left ventricular
LVOT = left ventricular outflow tract
PW = pulsed wave
SAM = systolic anterior motion

Echocardiogram acquisition, selection and measurement. Studies were performed at rest in the left lateral decubitus position using Hewlett-Packard Sonos 1000 echocardiographs.

M-MODE ECHOCARDIOGRAM. The M-mode echocardiogram recordings were made from the parasternal window during the same examination and within 5 min of the Doppler tracings. A modified electrocardiographic (ECG) lead I was continuously recorded. The mitral valve area of interest was magnified and recorded at 100 mm/s sweep speed. Views showing the mitral coaptation point and the most SAM of the mitral valve were recorded on videotape. The M-mode views were correlated with the two-dimensional (2-D) view to avoid mistakenly recording chordal SAM.

Tracings of SAM with both a clear mitral coaptation point and continuity with mitral–septal apposition were subsequently selected for measurement using a Nova Microsonics analysis computer. We measured the time interval from Q-wave onset till the first abrupt systolic anterior movement of the mitral valve. We measured the RR interval for each M-mode beat selected. Figure 1 illustrates the M-mode and Doppler measurements.

Figure 1. (Left) Continuous wave (CW) Doppler tracings were recorded in the five-chamber view through the LVOT as shown by the dashed line. Pulsed wave (PW) Doppler tracings were recorded in the LV at a point 2.5 cm apical of the mitral coaptation point and 1 cm from the septum, in LV outflow, as marked with the X. This location is the AMV point, apical of the mitral valve. (Right) The time interval Q-SAM is the interval from Q-wave onset to SAM onset. The velocity V-SAM is the CW velocity in the outflow tract at the time interval Q-SAM. A1 = atrial reflected wave.
CONTINUOUS WAVE DOPPLER. Continuous wave (CW) Doppler was performed from the apex, through the area of mitral–septal contact. We avoided contamination of this signal by flow from mitral regurgitation and by flow from the aortic valve or aorta. Doppler flow velocity tracings were recorded on videotape at 100 mm/s sweep speed.

PULSED WAVE DOPPLER. In the apical five-chamber view, the pulsed Doppler sample volume was placed in the LV, 2.5 cm apical of the mitral valve coaptation point and 1 cm from the interventricular septum, near the centerline of ejection color flow. We refer to this point as the AMV point, apical of the mitral valve (14,15). The 2-D image apical of the mitral valve was magnified to assure proper placement of the sample volume. We recorded beats that had high peak velocities and minimal spectral dispersion. Small sample volume and low filter settings were used.

DOPPLER TRACE SELECTION AND MEASUREMENTS. Beats were excluded if they did not show laminar flow, or showed an attenuated envelope. We selected several M-mode, pulsed wave (PW), and CW Doppler beats matched for similar RR intervals; for individual patients there was <3% variation in mean RR intervals between modalities. Selected Doppler traces were digitized into the analysis computer. Doppler and M-mode measurements were made by different investigators. For Doppler traces, we measured the preejection period as the time from the beginning of the ECG Q wave to the onset of ejection.

THE LV OUTFLOW VELOCITY AT SAM ONSET. For each patient we determined the LVOT CW velocity at the time of SAM onset. This was done by first determining the mean time interval from Q-wave onset to SAM onset from multiple M-mode tracings. Next, we measured the CW velocity in the outflow tract at that same time interval following the Q-wave. The CW trace with the closest RR interval to the mean M-mode RR interval was selected. On this tracing we digitally magnified the early systolic area of interest. Using digital calipers, the CW velocity at the time of SAM onset was determined by two independent observers. This measurement is shown in Figure 1. The average velocity measurement of the two observers was determined, as was the interobserver variation.

As we found that SAM began before ejection onset in more than half the patients, we examined the Doppler LV intraventricular flow that occurred during this preejection period. During this time, the $A_v$ wave is a prominent preejection flow (16–19). Both peak $A_v$ velocity and the time from Q-wave onset to peak $A_v$ velocity were measured.

Statistics. Continuous data are presented as mean ± SEM (range). The Student group $t$ tests were calculated for comparisons of means. Relations between different variables were assessed by Pearson’s correlation coefficient. Significance level was based on a two-tailed test. A value of $p < 0.05$ was considered significant.

RESULTS

In the 25 patients, a total of 98 M-mode tracings, 159 CW Doppler tracings and 151 PW Doppler tracings were digitized and analyzed. There were an average of 4 M-mode, 7 CW Doppler and 6 PW Doppler tracings per patient. The mean peak LVOT gradient was 82 ± 6 mm Hg (range 40 to 150). The mean time of SAM onset was 71 ± 5 ms (range 34 to 155 ms) after the ECG Q wave. The mean CW Doppler velocity in the LVOT at the time of SAM onset was 89 ± 8 cm/s (range 43 to 193). In 67% of the patients, SAM began at velocities of less than 100 cm/s. Representative tracings in six patients are shown in Figures 2 and 3. Data on the 25 patients are shown in Table 1. There was little interobserver variability between the measured CW velocities at SAM onset. The mean interobserver difference was 8 cm/s (range 1 to 26); correlation between the two measurements was low, $r = 0.97$.

The RR intervals. In the 25 patients as a group, little difference was seen in the mean RR intervals of the analyzed M-mode, CW and PW Doppler tracings; the mean RR intervals were 868 ± 25, 872 ± 25, and 874 ± 25 ms, respectively. For individual patients, the mean difference between the average RR intervals of the M-mode and CW tracings was 24 ± 3 ms (range 2 to 48 ms). The mean difference between the average RR intervals of the M-mode and PW tracings was 20 ± 3 ms (range 0 to 48 ms).

Preejection SAM. In 17 patients (68%), the onset of SAM occurred before the onset of ejection flow recorded on both CW and PW Doppler. In these patients with preejection SAM, M-mode SAM began earlier than in the patients with SAM beginning after ejection onset, at 62 ± 4 compared with 92 ± 11 ms after Q-wave onset ($p = 0.03$). Examples of patients with early SAM are shown in Figures 2 and 3.

INTRAVENTRICULAR FLOW DURING THE PREEJECTION PERIOD. We observed preejection flow both within the LV and also within the LVOT in all patients. The predominant flow during this time period is the $A_v$ wave, seen as a flow directed away from the transducer (16–19), shown in Figures 1 through 3. In the LV at the AMV point, the mean peak velocity of the $A_v$ wave was 69 ± 4 cm/s (range 35 to 108); mean acceleration to peak of the $A_v$ wave was 1,217 ± 111 cm/sec². Peak $A_v$ wave velocity occurred 53 ± 4 ms after Q-wave onset.

DISCUSSION

In this study we have found that Doppler velocity measurements in the LVOT do not support the hypothesis that Venturi forces cause SAM. Whenever flow traverses a surface such as the mitral valve, both lift and drag forces are generated. Drag, the pushing force of flow, is the component of force that is in the same direction as flow. An example is the pushing force of water on the paddle of a
waterwheel. Lift refers to Venturi forces produced by high velocity flow over the surface of an object causing the object to move perpendicular to flow. An example is the force produced as a horizontal airplane wing moves through the air, lifting it vertically. For any object the relative importance of lift as compared with drag is dependent upon three factors: 1) velocity of flow, 2) shape, and 3) orientation relative to flow (10,11).

Velocity. It is the central premise of the Venturi theory of SAM that high velocity ejection flow causes a local underpressure in the LVOT, which pulls the protruding mitral leaflet toward the septum. In this study, we show that SAM actually begins very early in systole at a time when velocity in the LVOT is normal. Specifically, SAM begins at a time when mean outflow tract velocity is 89 cm/s (range 43 to 193), a velocity not unlike that routinely recorded in the LVOT of normal patients without SAM (20).

Decreased velocity significantly decreases lift in two ways, and decreases its importance compared with drag forces. First, lift falls because it is roughly proportional to the square of velocity. Second, for any given shape, the ratio of lift to drag falls with decreasing velocity (10,11). At lower velocity, profile drag increases because increased contact of the fluid with the surface leads to increased friction with flow. Lower velocity

Figure 2. M-mode (top), continuous wave (middle), and pulsed wave Doppler (bottom) tracings of two patients. Patient 3 is on the left and patient 14 is on the right. In both patients, the left dashed line indicates time of Q-wave onset. The right solid line indicates the time of SAM onset. In both patients SAM onset begins at a time of low velocity both in the outflow tract and in the LV. In both patients SAM begins before the onset of ejection, during the A, wave (16–19). Both M-mode and Doppler tracings were recorded on the same study, within a 5-min interval, and were selected for matched RR intervals. Vertical calibrations of CW and PW tracings are at 1-m/s and 20-cm/s intervals, respectively.
allows viscosity to do its work. The difficulty of flying at low speed is an example of falling lift/drag ratio at lower velocity.

The modest outflow velocities we have measured indicate that, at the time of SAM onset, the magnitude of the Venturi force is much smaller than previously assumed; high velocities needed to generate significant Venturi forces are, in fact, not present. Therefore, the Venturi mechanism cannot be the main cause of SAM.

Flow drag: Geometric shape and orientation relative to flow. Besides velocity, the other factors that determine the relative importance of lift and drag are shape and orientation relative to flow (10,11), in this case between the mitral valve and the LV intraventricular flow (6,8). Available data with regard to these geometric relations indicate that drag is the dominant hydrodynamic mechanism for SAM (6–9).

In obstructive HCM the mitral valve leaflets are anteriorly positioned, relatively large, and residual portions of leaflets extend past the coaptation point and protrude into the outflow tract (1,7,21–23). The anterior displacement puts the mitral valve into the edge of the flow stream of LV ejection, subjecting the mitral valve to the hemodynamic force of ejection flow (1).

This LVOT narrowing and anterior position of the mitral coaptation point can be cited as evidence for either Venturi or drag (pushing) mechanisms, as the narrowing could play a role in both mechanisms. On the one hand, flow velocity must increase as it enters the narrowed outflow tract producing Venturi (lift) forces (24,25). Such Venturi forces are necessarily present. On the other hand, narrowing of the LVOT and the anterior position of the coaptation point also places the protruding leaflet into the edge of the flow stream, subject to the pushing force of flow that strikes the undersurface of the leaflet (6–9,23). The flow drag mechanism is illustrated in Figure 4. Thus, the LVOT narrowing provides the substrate for, and is evidence to support, both theories. This has been a source of confusion in the debate.

Additional data show that drag forces are more important than Venturi for causing SAM.

Figure 3. Four patients with obstructive HCM. In all cases the M-mode echocardiograms showing SAM onset are on top and the Doppler tracings are below. For the two patients on the left, the M-mode is compared with the CW Doppler tracings in the LVOT. For the two patients on the right, the M-mode is compared with the PW Doppler tracings in the LV at the AMV point. In all, the left dashed line indicates Q-wave onset. The right solid line indicates SAM onset. In all four patients SAM onset occurs at a time of low velocity, before the beginning of ejection. In the two patients on the right, SAM onset begins during the A wave. Both M-mode and Doppler tracings were recorded on the same study, within a 5-min interval, and were selected for matched RR intervals. Vertical calibrations of CW and PW Doppler tracings are at 1-m/s and 20-cm/s intervals, respectively.
In HCM patients: Orientation, shape, temporal and Doppler velocity evidence. In patients, a high angle of attack of Doppler color flow relative to the protruding leaflet of the mitral valve has been found; this orientation precludes significant Venturi effects and implicates drag (6). In the apical five-chamber view, local flow direction comes from an angle lateral of the protruding leaflet. The mean angle at time of mitral coaptation was lateral by 21°; mean angle just before septal contact increased to 45° (6). At these high angles relative to flow, drag is the dominant hydrodynamic force on the leaflet. This is illustrated in Figure 5. Flow pushes on the underside of the protruding leaflet (6–9), as drawn in Figure 4 and imaged in a patient in Figure 6. Critical overlap exists between the inflow and outflow portions of the LV that allows flow drag to catch the leaflet (6,27).

The leaflet is swept by the pushing force of flow into the septum. As it moves toward the septum the angle relative to flow increases and constricting drag forces are correspondingly increased as the leaflet presents a greater surface relative to flow (Fig. 5) (6). An example of this phenomenon is a widely open door in a drafty corridor. The door starts moving slowly; it then accelerates as drag increases because the door presents a larger area, until it slams shut (28).

Shape of the mitral valve. The mitral valve was not designed for lift. To the contrary, the valve has a sharp anterior edge with no streamlining, and there is a concavity under the cowl of the protruding leaflet. It resembles other biologic structures with high drag coefficient (Fig. 5) (10). The midseptal bulge. Besides the anterior position of the mitral valve, another contributor to the initial positive angle of attack is the midseptal bulge. In patients with obstructive HCM and resting gradients, hypertrophy of the midventricular septum is the rule, occurring in 92% of patients (29). This midseptal bulge is often associated with basilar or whole septal thickening (29,30). Resting gradients are uncommon in patients with only subaortic basilar septal thickening; in these patients resting gradients are found in just 12% (29). If it were Venturi forces from acceleration into a small outflow tract that caused resting obstruction, then isolated basilar hypertrophy and subaortic narrowing should commonly cause resting obstruction. But this is

| Table 1. Doppler and M-Mode Measurements in 25 Patients With Systolic Anterior Motion (SAM) |
|---------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Patient | Age (yrs) | Gender | LVOTG (mm Hg) | Q to SAM (ms) | PEP/PW (ms) | PEP/CW (ms) | v at SAM (cm/s) |
| Early SAM | | | | | | | |
| 1 | 72 | M | 64 | 34 | 59 | 51 | 91 |
| 2 | 65 | M | 47 | 51 | 109 | 99 | 75 |
| 3 | 52 | M | 40 | 41 | 83 | 64 | 80 |
| 4 | 40 | M | 100 | 78 | 88 | 79 | 48 |
| 5 | 83 | F | 75 | 62 | 68 | 81 | 60 |
| 6 | 57 | F | 68 | 57 | 84 | 78 | 44 |
| 7 | 77 | M | 64 | 62 | 74 | 85 | 71 |
| 8 | 48 | M | 125 | 96 | 116 | 106 | 81 |
| 9 | 69 | M | 58 | 46 | 81 | N/A | N/A |
| 10 | 74 | F | 100 | 54 | 84 | 108 | 170* |
| 11 | 42 | M | 77 | 73 | 91 | 80 | 44 |
| 12 | 85 | F | 80 | 51 | 62 | 60 | 61 |
| 13 | 66 | M | 68 | 90 | 100 | 110 | 63 |
| 14 | 77 | M | 45 | 75 | 84 | 81 | 92 |
| 15 | 87 | F | 78 | 66 | 87 | 75 | 100* |
| 16 | 68 | F | 120 | 42 | 65 | 61 | 74 |
| 17 | 86 | F | 54 | 71 | 112 | 90 | 53 |
| Average | 68 | | 74 (±6) | 62 (±4) | 85 (±4) | 82 (±4) | 75 (±8) |
| Late SAM | | | | | | | |
| 18† | 32 | F | 75 | 67 | 68 | 57 | 44 |
| 19† | 27 | M | 40 | 82 | 91 | 74 | 126 |
| 20† | 42 | M | 75 | 58 | 55 | 62 | 43 |
| 21 | 49 | M | 150 | 103 | 92 | 91 | 114 |
| 22 | 62 | F | 144 | 87 | 81 | 79 | 133 |
| 23 | 78 | M | 120 | 155 | 116 | 103 | 167 |
| 24 | 75 | F | 70 | 75 | 60 | 55 | 118 |
| 25 | 70 | F | 125 | 110 | 73 | 56 | 193 |
| Average | 54 | | 100 (±14) | 92 (±11) | 80 (±7) | 72 (±6) | 117 (±19) |
| Total Average | 63 | | 82 (±6) | 71 (±5) | 83 (±4) | 78 (±4) | 89 (±9) |

*A velocity ≥100 cm/s was measured in two patients with “Early SAM.” In these patients, SAM coincided with the A r-wave.
†In three patients, onset of ejection in PW/CW occurred around the beginning of SAM; those patients were considered as “Late SAM.”
LVOTG = left ventricular outflow tract gradient; Q to SAM = onset of O wave to beginning of systolic anterior motion; PEP = preejection period (onset of Q wave to ejection); PW = pulsed wave Doppler; CW = continuous wave doppler; v at SAM = velocity at beginning of SAM.

In HCM patients: Orientation, shape, temporal and Doppler velocity evidence. In patients, a high angle of attack of Doppler color flow relative to the protruding leaflet of the mitral valve has been found; this orientation precludes significant Venturi effects and implicates drag (6). In the apical five-chamber view, local flow direction comes from an angle lateral of the protruding leaflet. The mean angle at time of mitral coaptation was lateral by 21°; mean angle just before septal contact increased to 45° (6). At these high angles relative to flow, drag is the dominant hydrodynamic force on the leaflet. This is illustrated in Figure 5. Flow pushes on the underside of the protruding leaflet (6–9), as drawn in Figure 4 and imaged in a patient in Figure 6. Critical overlap exists between the inflow and outflow portions of the LV that allows flow drag to catch the leaflet (6,27).

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Shape of the mitral valve. The mitral valve was not designed for lift. To the contrary, the valve has a sharp anterior edge with no streamlining, and there is a concavity under the cowl of the protruding leaflet. It resembles other biologic structures with high drag coefficient (Fig. 5) (10). The midseptal bulge. Besides the anterior position of the mitral valve, another contributor to the initial positive angle of attack is the midseptal bulge. In patients with obstructive HCM and resting gradients, hypertrophy of the midventricular septum is the rule, occurring in 92% of patients (29). This midseptal bulge is often associated with basilar or whole septal thickening (29,30). Resting gradients are uncommon in patients with only subaortic basilar septal thickening; in these patients resting gradients are found in just 12% (29). If it were Venturi forces from acceleration into a small outflow tract that caused resting obstruction, then isolated basilar hypertrophy and subaortic narrowing should commonly cause resting obstruction. But this is
LVOT flow. In light of this and the previously mentioned little or no area of the posterior leaflet that is exposed to LVOT cannot be lifting the posterior leaflet because there is the cowl of the anterior leaflet (Fig. 4). Venturi forces in the posterior leaflet is separated from the flow in the LVOT by the leaflet moves anteriorly as well (31,32). However, the 89% of patients with SAM and obstruction, the posterior mitral leaflet is evidence for the drag mechanism. In Posterior leaflet SAM.

Measurements of the LV were thickened rather than the distal half. Posterior leaflet SAM. The anterior motion of the posterior mitral leaflet is evidence for the drag mechanism. In 89% of patients with SAM and obstruction, the posterior leaflet moves anteriorly as well (31,32). However, the posterior leaflet is separated from the flow in the LVOT by the cowl of the anterior leaflet (Fig. 4). Venturi forces in the LVOT cannot be lifting the posterior leaflet because there is little or no area of the posterior leaflet that is exposed to LVOT flow. In light of this and the previously mentioned uncommon; patients with just basilar outflow tract hypertrophy usually just have provicable obstruction.

Instead, data indicate that midventricular septal thickening is generally a necessary condition for resting SAM with mitral–septal contact (29). We have observed that this occurs because the midseptal bulge forces the outflow to sweep from a relatively posterior and lateral direction in the LV, as shown in Figures 4 and 6. When viewed in the echocardiographic apical five–chamber view, flow comes from “right field” or “one o’clock” direction. This contributes to the high angle of attack relative to the protruding leaflets. Also note that the posterior mitral leaflet is shielded and separated from outflow tract flow by the cowl of the anterior leaflet. Venturi flow in the outflow tract cannot be lifting the posterior leaflet because there is little or no area of this leaflet exposed to outflow tract flow. Venturi forces cannot be causing the anterior motion of the posterior leaflet.

Figure 4. The pushing force of flow. Intraventricular flow relative to the mitral valve in the apical five–chamber view. In obstructive HCM the mitral leaflet coaptation point is closer to the septum than normal (1). The protruding leaflets extend into the edge of the flowstream and are swept by the pushing force of flow toward the septum. Flow pushes the underside of the leaflets (arrow) (6–9). Note that the midseptal bulge redirects flow so that it comes from a relatively lateral and posterior direction; on the five–chamber view, flow comes from “right field” or “one o’clock” direction. This contributes to the high angle of attack relative to the protruding leaflets. Also note that the posterior mitral leaflet is shielded and separated from outflow tract flow by the cowl of the anterior leaflet. Venturi flow in the outflow tract cannot be lifting the posterior leaflet because there is little or no area of this leaflet exposed to outflow tract flow. Venturi forces cannot be causing the anterior motion of the posterior leaflet.

In summary, geometric data indicate that the necessary conditions for SAM are anterior position of the mitral coaptation point (which puts the valve into the edge of the outflow stream), positive angle of attack between outflow and the protruding leaflet, and chordal slack.
The geometric conditions for SAM are met in another condition where obstruction occurs, despite the absence of septal hypertrophy. Following mitral annuloplasty certain patients develop, as a complication, SAM. The mitral coaptation point has been shown to be anteriorly displaced into the outflow tract by the ring (33). Consequently, surgical techniques have been developed to ensure that the postoperative mitral coaptation point is posterior in the LV, explicitly out of the way of the outflow stream with its attendant drag (34,35).

In the present work, we have shown that rapid ejection flow velocity in the LVOT is not a necessary condition for SAM. In addition, we found that SAM begins before ejection onset in two-thirds of the patients. That SAM often occurs before ejection begins was first reported in 1987; in 10 patients on 2-D echocardiography SAM was seen to begin before the aortic valve opened (8). These temporal data, confirmed in the present work, support the drag hypothesis, rather than Venturi. In patients with preejection SAM, the early motion has been observed to be associated with a prominent LV preejection flow (36).

Figure 7 summarizes the evidence in the debate between Venturi and drag forces as the cause of SAM.

Therapeutic implications. Recently, new methods to relieve obstruction have been developed: revised surgical techniques (37–40), dual chamber pacing (41–44) and percutaneous transluminal septal myocardial ablation (45,46). Interventions are not always successful (47), and the reason for heterogeneity in response is not clear (43). Despite active research in treatment, the hemodynamic mechanism of obstruction has hitherto remained controversial (2–5). By clarifying the circumstances and the nature of the dynamic force that causes obstruction, the present study should help explain how novel treatments work and also could permit their refinement. This understanding is of crucial importance for the development of future treatment strategies.

New surgical approaches address the problem of the large
protruding mitral valve and its contribution to obstruction; the operation described by McIntosh reduces by plication the size of the anterior mitral leaflet, adding this to myomectomy (37). Another frees the bound papillary muscles from the surrounding LV muscle. This allows more normal posterior mitral coaptation, explicitly out of the outflow stream and its drag forces (38,39). As Messmer states: “This relieves the obstructive component of the mitral valve, which is rarely due to the often cited and never proved Venturi effect but has its origin rather in pathologic insertion of subvalvular apparatus” (38).

As discussed above, the impact of the midventricular septal bulge is to redirect LV flow so that it comes from a relatively postero-lateral direction. Consequently, an important goal of myomectomy must be to extend the myomectomy resection far enough down toward the apex to allow flow to track more anteriorly and medially along the surgically reduced septum and away from the mitral valve (23,38,39). Adequate extent of the myomectomy past the tip of the anterior mitral leaflets was stressed by Morrow (48) and others (23). A large decrease in the angle of attack of flow relative to the mitral valve has been shown after successful myomectomy; flow is made more parallel to the mitral valve (49). A recent modification extends resection to the deepest portion of the septum, to ensure complete resection of the midseptal bulge (38,39); 90% of patients had no postoperative SAM. Conversely, failure of myomectomy to alleviate SAM is often due to inadequate excision; in many such cases only the basal septum has been resected without resection farther down to the midventricle (47,50,51). Such considerations also apply to site selection for percutaneous alcohol ablation procedures. Targeting the basal septum alone will likely be inadequate; the midventricular bulge must also be addressed for complete relief of obstruction.

Dual chamber pacing with complete ventricular preexcitation through short atrioventricular delay significantly reduces outflow tract gradients (41–44). However, therapeutic effect is often incomplete; SAM persists with mean gradients of 30 to 55 mm Hg after three months of pacing (43,44). The mechanism by which pacing benefits SAM is unclear at this time (52). One hypothesis is that pacing might cause asynchronous or paradoxical septal motion, widening the outflow tract and decreasing Venturi forces (42). However, septal paradox is only rarely seen (41,52). Jeanrenaud (52) did find a modest decrease in regional septal wall motion but there was no uniform correlation between the magnitude of decreased septal motion and percent gradient reduction. Also, the present research indicates that a decrease in Venturi forces can only play a minor role in SAM improvement. Therefore, both direct observations and pathophysiology indicate that the mechanism by which pacing reduces SAM is more complex than just a widening of the outflow tract (52).

**Study limitations.** The M-mode tracings of SAM and Doppler tracings were not done simultaneously. Rather, they were done sequentially, within 5 min. This could have influenced results. However, tracings for M-mode, CW, and PW Doppler analyses were selected for matched RR intervals, and many tracings were selected and measured (4 M-mode and 7 CW Doppler beats). The mean RR intervals between modalities were quite close. For individual patients, the mean difference between the average RR intervals of the M-mode and CW tracings was 24±63 ms (range 2 to 48 ms). No patients with atrial fibrillation were examined in this study. Interventricular pressures were not directly measured. In the parasternal M-mode view, as the mitral valve moves anteriorly, the point with SAM onset and the point of mitral–septal contact may not be on the same M-mode line. For this study, clear registration of SAM onset was deemed essential, sometimes to the detriment of the point of mitral–septal contact.

**Conclusions.** In patients with obstructive HCM we have shown that the onset of SAM of the mitral valve is a low velocity phenomenon. At SAM onset, though Venturi forces are necessarily present in the outflow tract, their magnitude is much smaller than previously assumed. This velocity data, combined with other experimental, geometrical and temporal observations, indicate that drag, the pushing force of flow, is the dominant hydrodynamic mechanism for SAM.

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<td>Posterior leaflet SAM.</td>
<td>Posterior leaflet SAM.</td>
</tr>
<tr>
<td>SAM begins at low Doppler outflow tract velocity</td>
<td>SAM begins at low Doppler outflow tract velocity</td>
</tr>
<tr>
<td>SAM occurs without ASH</td>
<td>SAM occurs without ASH</td>
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<tr>
<td>Septal myomectomy improves SAM</td>
<td>Septal myomectomy improves SAM</td>
</tr>
</tbody>
</table>

**Figure 7.** Evidence in the debate between Venturi (lift) and drag (pushing) force as the dynamic cause for SAM. References are enumerated in the text. ASH = asymmetric septal hypertrophy.
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