


Turbulent Blood Flow in Humans

Its Primary Role in the Production of Ejection Murmurs

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SUMMARY To clarify the postulate that turbulence may produce ejection murmurs, point velocity and sound were measured in the ascending aorta of 13 subjects: six with normal aortic valves, six with aortic valvular disease, and one with a Björk-Shiley prosthetic aortic valve. Velocity was measured with a catheter-tip hot film anemometer probe, and sound was measured with a catheter-tip micromanometer. Ejection murmurs detected intra-arterially were always found to be associated with turbulent or highly disturbed flow. Conversely, in the absence of intra-arterial sound during ejection, only minor disturbances of flow were detected. A linear relation between the sound energy density and turbulent energy density was shown (r = 0.92) and a linear relation between the acoustic power output (sound intensity) and turbulent power supply (r = 0.87) also was shown. Studies in vitro and sound and point velocity distal to a porcine valve inserted within a cast of the aorta, which permitted precise centering of the transducers along the axis of flow, confirmed these observations. When the power generated by the turbulence exceeded 3 ergs/sec per cm², the murmurs were audible at the chest wall. The clinical gradation of the intensity of the murmurs increased as the power of turbulence increased. In conclusion, in this study we have demonstrated a clear association between turbulent blood flow and systolic ejection murmurs.

TURBULENT blood flow has been postulated to be the cause of ejection murmurs.1,2 Although alternate mechanisms for the origin of ejection murmurs have been proposed,1 prominent among these is periodic vortex shedding productive of Aeolian tones.2 Even the question of the existence of turbulent flow in the human body has been a matter of differing opinion. However, studies with point velocity sensors in animals support the likelihood of its existence.4-6 Studies in humans,1 however, establish that turbulent flow occurs above the aortic valve under some circumstances in normal humans.7 and it occurs routinely in the presence of aortic valvular disease.2 The purpose of this investigation was to determine the role of turbulent blood flow in the production of ejection murmurs.

Methods

Thirteen subjects were studied during diagnostic cardiac catheterization. Six subjects had normally functioning aortic valves, six had aortic valvular disease, and one had a Björk-Shiley prosthetic aortic valve. The study was approved by appropriate human experimentation committees, and informed consent was obtained from each subject. All subjects received light sedation with diazepam (Valium), which was taken orally.

Instantaneous point velocity in the ascending aorta was measured with a catheter-tip hot film velocity probe, used in combination with a TSI-1050 constant temperature anemometer (Thermo-Systems). The dynamic frequency response of hot film probes has been stated by Clark8 to be
The profile of flow in the ascending aorta of subjects with a normal aortic valve is relatively flat, with a boundary layer of less than 0.2 cm throughout the cardiac cycle. Therefore, peak velocity at the site of measurement with the hot film probe would be representative of velocity across the cross section, if the sensor were located anywhere except in the immediate vicinity of the wall of the vessel. The profile of flow would not be flat in the presence of aortic stenosis. In these patients, the tip of the catheter was manipulated and multiple recordings were made; velocities with the highest amplitude were presumed to be nearest to the midline axis of the jet.

Pressure and sound in the aorta and innominate artery were measured simultaneously with a catheter-tip micromanometer (frequency response 20 kHz) (Millar Instruments). The measurements were made in sequence after the measurements of point velocity. In each case care was taken to record the pressure fluctuations productive of sound in close proximity to the site of the previous recordings of point velocity, as judged by the position of the radiopaque markers.

Sound within the vessels was measured with the same high frequency catheter-tip micromanometer used for the measurement of arterial pressure. A high impedance audio output was connected to the same transducer. Fluctuations of pressures which are productive of sound and frequently referred to as "intra-arterial sound" were thereby recorded. It is important to note that only a small portion of the fluctuating pressures actually result in acoustic pressure. The turbulent fluctuating pressures that we recorded with the catheter-tip micromanometer perhaps could be considered as potentially productive of sound. Throughout this study, these turbulent fluctuating pressures which are potentially productive of acoustic pressure will be termed "sound pressure" or "sound."

Pressure, point velocity, and sound were recorded on a photographic recorder (Electronics for Medicine) (frequency responses of the recorder, amplifier, and filter system were uniform to 350 Hz) at a paper speed of 200 mm/sec. Velocity and sound pressure tracings were digitized by electronic digitizer (Numonics Corp.) on line with a Hewlett-Packard model 2100-A computer. Signals were digitized at intervals of 0.25 mm (0.00125 sec). Point velocity and sound were digitized over the period of ejection, as judged by aortic pressure tracings, from the upstroke of pressure to the dicrotic notch. Both velocity and sound signals were subjected to Fourier analysis by use of a fast Fourier transform in order to determine the frequency spectrum of these signals (Fig. 1). Signals above 320 Hz were considered invalid because of the magnitude of the interval at which signals were digitized, the method of digitization, and the frequency response of the recorder. Included data which lie in the upper range of frequencies may be somewhat in error. Component velocities below 30 Hz were considered intrinsic in pulsatile velocity signals and, therefore, were excluded from considerations relative to measurement of the energy and power of fluctuations. Low frequency fluctuations due to turbulence, if they existed, were of necessity eliminated in this process. Because sound is a nonpulsatile signal, it was not necessary to exclude low frequency components in its analysis. Amplitudes of fluctuating components of velocity less than 0.25 cm/sec and fluctuating components of sound pressure.
pressure less than 300 dynes/cm² were considered insignificant and probably due to artifact; this conclusion was based on tests of the digitizer and Fourier program with a known sine wave input.

Cardiac output was measured by the indicator-dilution technique prior to measurement of point velocity, pressure, and sound. The viscosity of blood was measured at 37°C with a cone-in-plate viscometer (Brookfield Engineering). Density of the blood was measured with a hydrometer.

At the conclusion of all sound and point velocity measurements, the diameter of the root of the aorta was measured by aortography. A scale was positioned at the level of the ascending aorta in order to determine amplification.

The peak Reynolds number in the root of the aorta was calculated as:

\[ \text{Peak Reynolds number} = \frac{\rho V D}{\mu} \]  

where \( \rho \) = density (g/cm³), \( V \) = peak instantaneous aortic velocity (cm/sec); \( D \) = diameter of aorta (cm); and \( \mu \) = viscosity (poise).

The sound energy density was calculated as:

\[ \text{Sound energy density (ergs/cm³)} = (\frac{\rho u_{\text{rms}}^2}{\rho c^2}) \]  

where \( u_{\text{rms}} \) = root mean square value of the magnitude of the fluctuating Fourier components of sound pressure (dynes/cm²), over a range of frequencies from 0 Hz to 320 Hz at intervals of 3.6 Hz to 4.6 Hz; \( c \) = speed of sound in blood (assumed to be 15.3 \times 10^4 cm/sec, the same as sea water at 1.025 g/cm³ at 25°C); and \( \rho \) = density of blood (g/cm³).

The spectral energy distribution of the sound energy density was plotted for frequencies between 0 and 320 Hz at intervals of 3.6 Hz to 4.6 Hz; \( c \) = speed of sound in blood (assumed to be 15.3 \times 10^4 cm/sec, the same as sea water at 1.025 g/cm³ at 25°C); and \( \rho \) = density of blood (g/cm³).

The peak Reynolds number in the root of the aorta was 2.5 cm. The cast was attached to a rigid Plexiglas chamber which simulated the left ventricle. A catheter or motion of the tip of the catheter, were eliminated occurred in studies on humans because of the position of the line of the axis of now. Errors or artifact which may have occurred in studies on humans because of the position of the catheter or motion of the tip of the catheter, were eliminated in this system.

A porcine aortic valve mounted on flexible stents (Hancock Laboratories), was used in the system. It was first employed at its fullest central opening of 3.0 cm². Then stenosis was produced by suturing the leaflets, causing a reduction of the area of the orifice to 1.2 cm². After the completion of studies with a 1.2 cm² orifice, the leaflets were sutured further, producing severe stenosis with an orifice of 0.7 cm². Studies were then repeated. The porcine valve was inserted in a pliable plastic cast of a human aorta, which included the ascending aorta, aortic arch, descending aorta, and great vessels (Cooper Scientific Corp.). The diameter of the root of the aorta was 2.5 cm. The cast was attached to a rigid Plexiglas chamber which simulated the left ventricle. A
mixture of glycerin in 0.9 g/100 cm³ of saline was pumped in a pulsatile fashion through the system. The viscosity of this mixture was 0.05 poise, and the density was 1.04 g/cm³; both of these values are comparable to those of blood.

Aortic flow was measured by means of a cannulating electromagnetic flow transducer (Carolina Medical Electronics) located in the descending aorta. All proximal vessels were occluded, causing the entire flow to pass through the transducer. Pulsatile flow of 80 cm³/beat, at a rate of 70 beats/min, was produced by a pulsatile pump (Harvard Apparatus Company). Aortic pressure, sound, and point velocity were measured simultaneously along the center line of the axial stream of flow, using the same basic instrumentation that was used to study humans. Measurements were made consecutively at eight sites along the aorta. Aortic pressure and sound pressure fluctuations were measured with a No. 7 catheter-tip micromanometer. Point velocity was measured with an L-shaped hot film probe (Fig. 3). The ratios of the sensing devices to the cross-sectional area of the root of the aorta were 0.0064 and 0.0023, respectively. The tip of the sound pressure transducer was located just distal to the point of the hot film probe and 1 mm above it (Fig. 3). Particular care was taken to be certain that the inner surface of the cast of the aorta was smooth throughout its entire length proximal to the site of the measurements. All perforations of the wall of the aorta which had been produced to permit entry of the sensing devices during previous measurements were sealed and smoothed.

Pressure was maintained at 120/80 mm Hg by a combination of laminar resistors, a compression chamber, various lengths of corrugated rubber tubing, and a variable clamp (Fig. 2). The magnitude of systolic pressure was controlled by varying the fluid level in the compression chamber. The level of diastolic pressure was controlled by varying the adjustable clamp proximal to the compression chamber. The laminar resistor (synthetic sponges enclosed within a Plexiglas chamber) and the corrugated rubber tubing permitted control of the resistance and compliance of the simulated arterial system. The size of the laminar resistor and the length of the corrugated tubing were adjusted to achieve configurations of pressure and flow comparable to those seen in humans (inset, Fig. 2). All data from the in vitro
system were recorded and used in calculations in a fashion identical to that used for humans.

**Results**

In all subjects the intensity of intra-arterial sound (acoustic power output) was highest at peak flow, that is, during peak Reynolds numbers. This corresponded to the time of peak fluctuations of flow. The peak Reynolds numbers of subjects with normal aortic valves ranged between 5,700 and 10,000 (Table 1). Turbulent energy density, sound energy density, turbulent power supply, and acoustic power output for these subjects are also shown (Table 1).

Low amplitude, inaudible fluctuating pressures indicative of sound, were recorded in the region of normal aortic valves coincident with disturbances of flow which occurred during peak ejection (Fig. 4). In the midportion of the ascending aorta and proximal portion of the arch of the aorta, flow in such subjects was virtually undisturbed and intra-aortic sound was negligible. In the presence of a high cardiac output, turbulence was observed in the region of a normal aortic valve during ejection (Fig. 5). Audible sound pressure fluctuation occurred in association with the turbulence and intra-arterial sound was recorded at lower amplitudes in the mid-ascending aorta, where turbulence was reduced. In some subjects with aortic insufficiency, turbulent flow during ejection was measured throughout the ascending aorta and within the proximal portion of the aortic arch (Fig. 6). Sound within the aorta was recorded at the site of turbulent flow. The amplitude of the sound diminished with distance from the aortic valve, just as the turbulence diminished with distance from the valve (Fig. 6).

Ejection murmurs with the highest magnitude were observed in subjects with aortic stenosis. As in subjects with aortic insufficiency, sound and turbulence were recorded throughout the ascending aorta and within the innominate artery; and the magnitude of both diminished with distance from the valve (Fig. 7). The association of sound with turbulent flow also was observed distal to a prosthetic aortic valve (Fig. 8).

The spectral energy distribution of sound was smooth and showed no discontinuities, regardless of whether the sound was associated with a normal or a stenotic valve (Fig. 9).

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**TABLE 1 Hemodynamic, Sound, and Turbulence Measurements in Subjects**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Aortic valve</th>
<th>Cardiac output (liters/min)</th>
<th>Heart rate (beats/min)</th>
<th>Peak velocity (cm/sec)</th>
<th>Peak Reynolds number (dimensionless)</th>
<th>Sound energy density (ergs/cm²)</th>
<th>Turbulence energy density (ergs/cm²)</th>
<th>Acoustic power output (ergs/sec per cm²)</th>
<th>Turbulence power supply (ergs/sec)</th>
<th>Murmurs grade (I-VI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>12.9</td>
<td>144</td>
<td>181</td>
<td>10,000</td>
<td>980 x 10⁻⁴</td>
<td>2.9</td>
<td>15.0 x 10⁻²</td>
<td>4.2</td>
<td>I</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
<td>5.3</td>
<td>92</td>
<td>96</td>
<td>5,700</td>
<td>270 x 10⁻⁴</td>
<td>1.2</td>
<td>4.3 x 10⁻²</td>
<td>1.2</td>
<td>O</td>
</tr>
<tr>
<td>3</td>
<td>Normal</td>
<td>5.3</td>
<td>72</td>
<td>118</td>
<td>6,200</td>
<td>30 x 10⁻⁴</td>
<td>0.7</td>
<td>0.6 x 10⁻²</td>
<td>0.6</td>
<td>O</td>
</tr>
<tr>
<td>4</td>
<td>Normal</td>
<td>5.4</td>
<td>100</td>
<td>117</td>
<td>5,900</td>
<td>4 x 10⁻⁴</td>
<td>0.6</td>
<td>0.1 x 10⁻²</td>
<td>0.4</td>
<td>O</td>
</tr>
<tr>
<td>5</td>
<td>Normal</td>
<td>5.6</td>
<td>120</td>
<td>109</td>
<td>6,900</td>
<td>1 x 10⁻⁴</td>
<td>0.8</td>
<td>0.02 x 10⁻²</td>
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</tr>
<tr>
<td>6</td>
<td>Normal</td>
<td>4.7</td>
<td>60</td>
<td>141</td>
<td>8,900</td>
<td>130 x 10⁻⁴</td>
<td>1.1</td>
<td>2.0 x 10⁻²</td>
<td>1.0</td>
<td>O</td>
</tr>
<tr>
<td>7</td>
<td>Prosthetic</td>
<td>4.7</td>
<td>80</td>
<td>216</td>
<td>2,000 x 10⁻⁴</td>
<td>2.0</td>
<td>8.0 x 10⁻²</td>
<td>11.3</td>
<td>II</td>
<td></td>
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<tr>
<td>8</td>
<td>A1</td>
<td>6.5</td>
<td>78</td>
<td>238</td>
<td>*</td>
<td>1,300 x 10⁻⁴</td>
<td>2.5</td>
<td>19.9 x 10⁻³</td>
<td>3.4</td>
<td>II</td>
</tr>
<tr>
<td>9</td>
<td>A1</td>
<td>5.3</td>
<td>66</td>
<td>238</td>
<td>*</td>
<td>1,500 x 10⁻⁴</td>
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<td>22.9 x 10⁻²</td>
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<td>10</td>
<td>A1</td>
<td>6.5</td>
<td>70</td>
<td>256</td>
<td>*</td>
<td>1,700 x 10⁻⁴</td>
<td>3.2</td>
<td>26.2 x 10⁻²</td>
<td>5.1</td>
<td>II</td>
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<tr>
<td>11</td>
<td>AS-A1</td>
<td>4.4</td>
<td>80</td>
<td>308</td>
<td>*</td>
<td>5,300 x 10⁻⁴</td>
<td>14.0</td>
<td>81.0 x 10⁻²</td>
<td>43.9</td>
<td>III</td>
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<tr>
<td>12</td>
<td>AS-A1</td>
<td>4.4</td>
<td>80</td>
<td>352</td>
<td>*</td>
<td>4,100 x 10⁻⁴</td>
<td>11.8</td>
<td>62.7 x 10⁻³</td>
<td>20.3</td>
<td>III</td>
</tr>
<tr>
<td>13</td>
<td>AS-A1</td>
<td>4.5</td>
<td>54</td>
<td>459</td>
<td>*</td>
<td>11,100 x 10⁻⁴</td>
<td>14.6</td>
<td>169.8 x 10⁻²</td>
<td>34.9</td>
<td>IV</td>
</tr>
</tbody>
</table>

AI = aortic insufficiency; AS = aortic stenosis.

* The presence of an abnormal valve caused the Reynolds number to be of different significance and, therefore, not comparable.
The highest energy density of sound pressure fluctuations at each frequency was observed in subjects with aortic stenosis. The spectral energy density distribution of the fluctuating components of point velocity also was smooth and showed no discontinuities. As with sound, the highest energy density of fluctuations of velocity at each frequency occurred with aortic stenosis. Sound energy density diminished more gradually with increasing frequency than did turbulent energy density. The absolute magnitude of the sound energy density was lower than the turbulent energy density at all frequencies. The association with turbulent flow of sound pressure fluctuations comparable to ejection murmurs was also observed in vitro. Sound and turbulent flow were recorded in the region of the fully opening porcine valve (Fig. 10), and with the stenotic porcine valve (Fig. 11).

The sound energy density in our subjects was linearly related to the turbulent energy density ($r = 0.92$). This relation was valid for normal and diseased valves and in the presence of a prosthetic valve; the relation also was valid at various sites in the aorta and innominate artery. The relation was confirmed in vitro, and values measured in vitro were comparable to those measured for our subjects (Fig. 12).

The acoustic power output (sound intensity) in the subjects also was linearly related to the turbulent power supply ($r = 0.87$). As was the case for energy density, this relation was shown for all valves at all sites of measurement. Values measured in vitro could be superimposed upon values measured in the subjects (Fig. 13).

Maximal sound and maximal turbulence occurred at some distance distal to stenotic aortic valves both in the subjects (Fig. 14), and in the in vitro model of the cardiovascular system (Fig. 15). The distance from the valve to the site of the maximal sound energy density and the maximal turbulent energy density increased with increasing severity of the stenosis. Maximal turbulence and sound, in the absence of significant stenosis, occurred in close proximity to the valve (Fig. 15). The clinical gradation of the intensity of the ejection murmurs increased with the turbulent power supply (Fig. 16) and with the acoustic power output (Fig. 17).

**Discussion**

The results of this study indicate that it was specifically turbulent flow which initiated the pressure fluctuations which can produce sound. This was apparent from the following: (1) intra-arterial fluctuating pressures transduced as sound were uniformly associated with turbulent flow at all...
Highly turbulent flow (turbulent energy density = 14.6 ergs/cm$^3$) was recorded distal to aortic valve in subject 13 with predominant aortic stenosis. Fluctuations in pressure due to turbulence are apparent on the upstroke of the aortic pressure curve. Turbulence persisted in innominate artery with a turbulent density of 4.0 ergs/cm$^3$. A clearly defined ejection murmur was recorded at this site within the innominate artery.

Sites of measurement, whether in the aorta or innominate artery, (2) Intra-arterial sound was absent in the absence of turbulent or highly disturbed flow. (3) The energy density of the intra-arterial sound was linearly related to the energy density of turbulent flow. (4) The acoustic power output of sound (sound intensity) was linearly related to the turbulent power supply. (5) The turbulent power supply correlated with the clinical gradation of the intensities of the murmurs. Turbulence was indicated by the appropriate frequency spectrum. Point velocity fluctuations in one plane which

\[ 16 \text{ Point velocity fluctuations in one plane which} \]

\[ \text{FIGURE 7} \text{ Highly turbulent flow (turbulent energy density = 14.6 ergs/cm}^3\text{) was recorded distal to aortic valve in subject 13 with predominant aortic stenosis. Fluctuations in pressure due to turbulence are apparent on the upstroke of the aortic pressure curve. Turbulence persisted in innominate artery with a turbulent density of 4.0 ergs/cm}^3\text{. A clearly defined ejection murmur was recorded at this site within the innominate artery.} \]

\[ \text{FIGURE 8} \text{ Turbulent flow occurred in the region of a prosthetic aortic valve in subject 7, and highly disturbed flow was recorded as far distal as the innominate artery. The amplitude of intra-arterial sound diminished coincidently with the reduction of disturbances of flow.} \]
FIGURE 9  Turbulence energy density (left) and sound energy density (right) in subject 13 with aortic stenosis, subject 1 with normal valve and high cardiac output, and subject 7 with normal valve and normal output. No discontinuities of the spectral distribution of sound or velocity were noted.

FIGURE 10  Point velocity and sound measurements at the indicated positions distal to a porcine aortic valve in vitro. Area = 3.0 cm²; distance in centimeters from valve to various positions is shown in Figure 15.
TURBULENT FLOW AND MURMURS/Sabbah and Stein

FIGURE 11  Point velocity and sound pressure at indicated sites distal to stenosed porcine aortic valve in hydraulic model of circulatory system. Area = 0.7 cm². The turbulent energy density, 2.6 ergs/cm³, and sound energy density, 900 \times 10^{-9} \text{ ergs/cm}^{3}, were higher in the mid-ascending aorta (position 3) than in close proximity to the valve (position 1). At the valve, turbulent energy density and sound energy density were 0.8 ergs/cm³ and 70 \times 10^{-9} \text{ ergs/cm}^{3}, respectively. The turbulent energy density and sound energy density were less than values measured in humans because peak velocity along the center line was less, presumably because of a smaller stroke flow than in our subjects.

have an appropriate frequency spectrum can be accepted as evidence of turbulence. An energy distribution which decreases smoothly with increasing frequency and shows no discontinuity would be expected with turbulent flow, and this was observed. The characteristics of point velocity in these subjects, therefore, satisfied definitions of turbulence that state that turbulence is an irregular condition of motion in which velocity and pressure show a random variation in relation to time and space coordinates.

Because it has been demonstrated in living subjects that turbulent flow causes ejection murmurs, one can now apply hydraulic theories of turbulent flow in pipes and in jets to better understand the clinical characteristics of murmurs. The reason for the occurrence of murmurs during various high output states is documented. Characteristics of ejection murmurs can be explained, such as the time of their occurrence in the cardiac cycle, their duration, shape, intensity, location, transmission, and quality. During circumstances which increase flow (fever, exercise, hyperthyroidism, and anemia), turbulence would increase. Higher magnitudes of turbulent energy would be associated with higher levels of sound energy. With higher flows, turbulence would be present throughout a larger portion of the ejection period.

Anemia would increase the peak Reynolds number and, therefore, turbulence, both by its effect on the cardiac output and by its effect on viscosity. Anemia also augments turbulence by virtue of hemorheological effects related to the hematocrit. Turbulent flow distal to a stenotic valve would occur at Reynolds numbers well below the critical Reynolds number which applies to steady flow in smooth pipes. Even so, the mean Reynolds number at the valve of these subjects with combined aortic stenosis and aortic regurgitation ranged between 10,000 and 20,000. These values were based on the estimated diameter of the valvular orifice, as derived from hemodynamic measurements of the cross-sectional area of the valve. Such calculations used ventriculographic estimates of stroke volume in order to include the regurgitant fraction of flow. The

FIGURE 12  Relation of sound energy density to turbulent energy density in humans and in vitro. Data points are included for all valves at all sites of measurement. A linear relationship appears to be present.
 intensity of turbulence of blood flowing through a stenotic orifice in a pulsatile fashion increases as the Reynolds number increases. Therefore, high magnitudes of turbulence would be expected distal to stenotic valves with the magnitude of peak Reynolds numbers estimated to have occurred in these subjects.

The cause of the reduced intensity of organic ejection murmurs in subjects with congestive heart failure can be explained in terms of fluid mechanics related to the observations of this investigation. In congestive heart failure the maximal rate of flow diminishes. It was previously shown in human subjects that the turbulent energy density decreased with reductions of peak velocity. The sound energy density was shown in this study to be linearly related to the turbulent energy density. Therefore, a reduction of the issuing velocity caused by reduced ventricular performance would cause a reduction of sound energy density. In the region of unbounded jets, the acoustic efficiency itself diminishes with a reduction of velocity. Therefore, a reduction of velocity would certainly produce a reduction of sound.

FIGURE 13 Acoustic power output in relation to turbulent power supply. Data points apply to all valves at all sites of measurement both in subjects and in vitro. Relationship appears to be linear.

FIGURE 14 Turbulent energy density and sound energy density recorded at the indicated sites in the aorta and innominate artery (inset, lower illustration) in subject 11 with aortic stenosis and aortic regurgitation. Both the turbulent energy density and sound energy density reached maximal values in the mid-ascending aorta, some distance downstream from the valve.
The time in the cardiac cycle of innocent murmurs usually is during early systole or midsystole; the duration of such murmurs is short. This corresponds to the time of maximal turbulence. During the late portion of ejection, disturbances of flow in subjects with normal aortic valves may be insufficient to produce sound. The murmur of aortic stenosis, on the other hand, is a long ejection murmur. Distal to a stenotic valve, turbulence occurs at most levels of flow and, therefore, sound extends throughout the major portion of ejection. In terms of the Reynolds number, its critical value would be exceeded and sound due to turbulence would be produced at all but the lowest velocities of flow distal to a stenotic orifice.

The cause of the diamond shape of ejection murmurs also can be explained on the basis of the Reynolds number and magnitudes of turbulence. As velocity increases the turbulence energy density increases; and the turbulence energy density was shown to be linearly related to the sound energy density. Velocity increases during ejection, reaches a peak, then diminishes. Sound energy density, therefore, would be expected to follow the same curve, which results in a diamond-shaped murmur.

The intensity of innocent ejection murmurs rarely exceeds grade 2 on a scale of grades 1 to 6; whereas the intensity of the murmur of aortic stenosis is frequently louder. Distal to a stenotic orifice (in a jet), magnitudes of turbulence would occur at identical levels of flow which, in the absence of a stenotic orifice, would result in less turbulence or possibly no turbulence. A direct relation of turbulent energy density to sound energy density, and of turbulent power input to acoustic power output, now has been shown in humans. Therefore, it is clear that higher turbulence caused by the jet distal to a stenotic valve would produce a louder murmur than would be produced by low amplitudes of turbulence distal to a smooth inlet (normal valve). Since turbulence both in a jet and in a smooth pipe increases with velocity, an increased intensity of both innocent and organic ejection murmurs would be expected with increased velocity. This has previously been observed.

Observations related to the distance from the valve of the occurrence of maximal turbulence indicate why an innocent murmur is usually heard at the 3rd or 4th left intercostal space, whereas the murmur of aortic stenosis usually is heard at the 2nd or 3rd left intercostal space.
best heard at the 2nd intercostal space, to the right of the sternum. That is, an innocent murmur is heard closer to the anatomical location of the aortic valve, whereas the murmur of aortic stenosis is best heard distal to the valve, at the aortic auscultatory listening post. In the presence of a normal valve, if velocity were high enough to exceed the critical Reynolds number, such a velocity would be expected close to the valve. Measurements in this study showed that maximal turbulence, in fact, occurred in close proximity to the aortic valve when the valve was normal. In contradistinction, jet theory indicates that turbulence distal to a stenotic orifice would occur some distance from the orifice.

Innocent murmurs, because of the site of their maximal intensity, are sometimes termed pulmonary ejection murmurs or innocent pulmonary systolic murmurs. The results of this investigation suggest, however, that such murmurs may be flow murmurs originating in the region of the aortic valve. Maximal turbulence and sound associated with high flow in the presence of normal valves occurred in close proximity to the valve. Therefore, aortic flow murmurs would be of maximal intensity at the anatomical location of the aortic valve (3rd intercostal space, left sternal border). In contradistinction, maximal turbulence and sound due to aortic stenosis occurred downstream from the valve, which corresponded to the aortic auscultatory listening post.

The quality of organic murmurs has been thought to reflect turbulence, as indicated by a wide spectrum of frequencies inherent in these murmurs. Murmurs caused by turbulent flow should contain a broad spectrum of frequencies, whether they are innocent murmurs or murmurs caused by stenotic valves. Some investigators postulated that the cause of innocent murmurs is different from that of organic murmurs. This was based on spectrograms of innocent murmurs which showed a compact type of output with the majority of frequencies below 250 Hz, whereas organic murmurs (mitral regurgitation) showed a high range of frequencies. Our study showed that turbulence of lower energy density (as in the presence of a normal aortic valve) would produce sound with lower energy components at the higher bands of frequency than turbulent flow of a higher energy density (as in aortic stenosis). The latter caused an increased energy density at all measured frequencies, and particularly showed significant levels at the higher frequencies. True acoustic pressure is estimated to be six orders of magnitude (130 dB) less than the turbulent pressure fluctuations. This estimated acoustical pressure would be well within the threshold of audibility. Some of these murmurs were heard at the chest wall with intensities of grade 3 and grade 4.

The fluctuating velocities and pressures due to turbulence presumably produce local vibrations at the wall of the vessel. The vibrations of the arterial wall then would appear to be transmitted to the surface of the chest. This mechanism for the transmission of murmurs to the chest wall has been postulated by others. Vibration of the walls of distensible tubes may occur with turbulent flow distal to an orifice. The failure of a phonocatheter to detect sound pressure vibrations upstream from a jet suggests that the conduction of sound through the blood by compression waves, if any occurs, is negligible. Measurements of the rate of transmission of sound from the heart to the chest wall indicated that there is transmission by vibration of overlying tissues. Clinical observations, such as auscultation of loud ejection murmurs at the olecranon process of the elbow in subjects in whom the brachial artery was occluded by a blood pressure cuff, support the concept that sound is transmitted by the vibration of tissues in juxtaposition to the site of the origin of the murmur.

Among the postulated mechanisms for the genesis of cardiac murmurs is periodic vortex shedding (Aeolian tones). If periodic vortex shedding were productive of sound in these subjects, point velocity measurements should have shown components of velocity which occurred periodically. Spectral analyses failed to show periodicity. A wide spectrum of velocity components was shown with no irregularities; the velocity fluctuations were completely random in nature.

In conclusion, the results of this study indicate that turbulence or highly disturbed flow is the cause of organic and innocent murmurs. Furthermore, it should be emphasized that the vibration of the walls of vessels, if it occurs, is caused by the turbulent flow. Specifically, pressure and velocity fluctuations inherent in turbulence would produce such vibrations. The vibrations themselves do not constitute the cause of the murmurs, but rather are a consequence of turbulent flow.

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SUMMARY In conscious dogs static pressure-flow relationships (I-P curves) were obtained both for the normal autoregulating kidney and under conditions of maximal vasodilation induced by infusion of papaverine or acetylcholine into the renal artery. Concentration-response curves for the substances infused showed the plateau; under maximal vasodilation the I-P curves were pressure-passive up to a perfusion pressure of 50 mm Hg, following a power function (I = a·P^b) with an exponent greater than 1. Under the influence of acetylcholine, renal blood flow was significantly higher than under control conditions even at a perfusion pressure of 20 mm Hg. This indicates that there is an appreciable vascular tone even at a low pressure. The I-P curves under acetylcholine showed a break at about 50 mm Hg, above which the I-P curves were straight lines.

THE SIGNIFICANCE of blood vessel distensibility in regard to the pressure-flow relationship was realized early. 2,3 Green et al. 3 described the pressure-flow relationship (I-P curve) by a power function with an exponent greater than 1. Weizler and Sinn 4 showed the physical and mathematical background of this power function. In a previous study, 4 the dynamic I-P curves for the autoregulating kidney vasculature were described as following a power function. These curves were obtained within a few seconds and thus fulfilled the conditions for a pure pressure-flow relationship as emphasized by Wetterer and Kenner. 6 The exponent was found to increase when mean arterial pressure that of a dynamic I-P curve obtained at a low mean arterial pressure was increased pharmacologically. It was expected that under these experimental conditions the static I-P curve would follow a power function with an exponent comparable to the previous study, 4 the dynamic I-P curves for the autoregulating kidney vasculature were described as following a power function. These curves were obtained within a few seconds and thus fulfilled the conditions for a pure pressure-flow relationship as emphasized by Wetterer and Kenner. 6 The exponent was found to increase when mean arterial pressure that of a dynamic I-P curve obtained at a low mean arterial pressure was increased pharmacologically. It was expected that under these experimental conditions the static I-P curve would follow a power function with an exponent comparable to that of a dynamic I-P curve obtained at a low mean arterial pressure, when smooth muscle tone also is low. The literature concerning smooth muscle tone in the kidney is contradictory. The view that there is no vascular tone in the

**Basal Vascular Tone in the Kidney**

Evaluation from the Static Pressure-Flow Relationship under Normal Autoregulation and at Maximal Dilation in the Dog

Rainer Gross, M.D., Hartmut Kirchheim, M.D., Ph.D., and Kurt Brandstetter, M.D.

**SUMMARY** In conscious dogs static pressure-flow relationships (I-P curves) were obtained both for the normal autoregulating kidney vasculature and under conditions of maximal vasodilation induced by infusion of papaverine or acetylcholine into the renal artery. Concentration-response curves for the substances infused showed the typical S-shape. Control I-P curves exhibited an autoregulatory plateau; under maximal vasodilation the I-P curves were pressure-passive up to a perfusion pressure of 50 mm Hg, following a power function (I = a·P^b) with an exponent greater than 1. Under the influence of acetylcholine, renal blood flow was significantly higher than under control conditions even at a perfusion pressure of 20 mm Hg. This indicates that there is an appreciable vascular tone even at a low pressure. The I-P curves under acetylcholine showed a break at about 50 mm Hg, above which the I-P curves were straight lines.

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