Turbulent Blood Flow in the Ascending Aorta of Humans with Normal and Diseased Aortic Valves

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SUMMARY Turbulent blood flow may contribute to a variety of pathophysiological effects. Because of its postulated importance, this study was undertaken to determine whether turbulent flow does in fact occur in the human body. In 15 persons (seven normal, seven aortic valvular disease, one prosthetic aortic valve), point velocity was measured in the ascending aorta with a hot-film anemometer probe. In one normal individual with a high cardiac output, turbulent flow occurred above the aortic valve during peak flow which corresponded to a peak Reynolds number of 10,000. In the other six normal subjects (peak Reynolds numbers of 5,700–8,900), flow was highly disturbed during peak ejection. Each of the subjects with aortic valvular disease and the subject with a prosthetic aortic valve showed turbulent flow during nearly the entire period of ejection, with Fourier components of velocity of significant magnitude up to 320 Hz (the maximum frequency we could evaluate with the equipment available). The turbulence energy density was higher in subjects with abnormal valves (3.2–14.6 ergs/cm²), than in normal subjects (0.6–2.9 ergs/cm²). In subjects with aortic stenosis, turbulence was observed throughout the ascending aorta and in the innominate artery. In others, the turbulence dissipated more proximally. The results of this study indicate that turbulent flow can occur in the ascending aorta of subjects with normal cardiac function; and it occurs consistently in the ascending aorta of individuals with abnormal aortic valves.

THE QUESTION of the existence of turbulent flow in the human body may be of importance in regard to several pathophysiological states. Disturbed flow is thought to be a factor in the initiation of platelet deposition. One form of disturbance which contributes to thrombosis has been identified as turbulence, and a quantitative relation between turbulence-induced thrombosis in dogs and the magnitude of turbulence subsequently has been shown. Studies with pumps have shown turbulence in the region of aortic ball valve prostheses. These investigations suggest the possibility that turbulent flow may contribute to thrombus formation in the region of prosthetic valves. This mechanism for the initiation of platelet deposition also may participate in atherogenesis according to the encrustation theory of atherosclerosis. Turbulent blood flow in the region of prosthetic valves is thought to be the cause of intravascular hemolysis. Turbulent flow has been shown to augment the sickling process, possibly through modification of stabilizing factors of the colloidal solution of hemoglobin, which thereby contributes to sol-gel transformation of the abnormal hemoglobin. Cardiac murmurs are thought to relate to turbulent flow. Shear stresses due to turbulent flow may induce damage to the intimal surface of blood vessels, and intimal thickening, apparently due to turbulence in the region of prosthetic valves, may extend to the proximal portion of the coronary arteries.

Because of the possibility that turbulent flow may participate in a variety of pathophysiological processes, it is important to determine whether turbulent flow, in fact, occurs within the human body. Therefore, we undertook this study of the nature of flow in the ascending aorta of subjects with normal and diseased aortic valves.

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Methods

Fifteen persons were studied during diagnostic cardiac catheterization. All investigations were performed with informed consent, the protocol having been approved by the University Human Experimentation Committee and the Veterans Administration Research and Education Committee. Seven subjects had normally functioning aortic valves, as determined by the absence of a pressure gradient across the valve, only a grade I/VI systolic murmur (one subject) or no systolic murmur (six subjects), no diastolic murmur, and no aortographic evidence of aortic insufficiency. Seven subjects had aortic valvular disease and one had a Björk-Shiley aortic prosthetic valve. All subjects received light sedation with diazepam (Valium) taken orally. Pressures in the left ventricle and aorta were measured with a catheter-tip micromanometer (Millar Instruments). Cardiac output was measured by the indicator-dilution technique. 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.

Instantaneous point velocity in the ascending aorta was measured with a catheter-tip hot-film velocity probe in combination with a TSI-1050 constant temperature anemometer (Thermo-Systems). The platinum thin-film sensors were formed on a quartz substrate, then coated with interstitially bonded quartz. The probe was conical with a pointed tip and a cone angle of 40 degrees. The thin-film sensor itself was approximately 0.06 mm wide and was placed circumferentially around the cone at a distance of 0.5 mm from the tip (Fig. 1). The constant temperature anemometer was operated at an overheat ratio of 1.02. This relatively low overheat ratio limited the temperature of the heated film to 5°C above the ambient temperature and was, therefore, not high enough to cause the deposition of fibrin upon the film when used in blood. Slight changes of the zero flow level were sometimes noted with this application of thin-film gauges. The direction of flow past the transducer...
The dynamic response of a hot-film probe is limited by the heat-exchange characteristics of the sensor and the thermal capacity of the fluid around the sensor. Under conditions of unsteady flow, fluctuating thermal gradients may be set up in the substrate. The time course of these is different from the fluctuations of flow and, therefore, they may distort the flow signal. Heat transfer from the film ultimately is dictated by the form of the boundary layer over the surface of the probe. The limiting factor for transient response becomes the transfer of heat through the fluid boundary layer. Ling and associates reported a dynamic frequency response of 0–10 kHz at a mean velocity of 100 cm/sec. The range of the dynamic response will increase or decrease with a corresponding increase or decrease in flow velocity. Peak velocity in the present study ranged between 96 and 459 cm/sec. A comparable frequency response, therefore, would be expected. However, the amplitude of the fluctuating components has been found to diminish as the Strouhal number increases.

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:

$$ \text{Peak Reynolds number} = \frac{\Delta V D}{\mu} $$

where $\rho$ = density (g/cm$^3$); $V$ = peak instantaneous aortic velocity (cm/sec); $D$ = diameter of aorta (cm); $\mu$ = viscosity (poise).

The unsteadiness parameter, $\alpha$, was calculated as:

$$ \alpha = \frac{D}{2 \sqrt{\frac{\rho V}{\mu}}} $$

where $\omega = 2\pi \times$ heart rate/60; $\rho$, $\mu$, and $D$ are as previously defined.

Pressures and velocities were measured on an Electronics for Medicine photographic recorder (frequency response uniform to 300 Hz) at a paper speed of 200 mm/sec. Tracings were digitized, utilizing an electronic digitizer (Numonics Corp.) on-line with a Hewlett-Packard 2100-A computer. The reproducibility of digitized data from a single beat was shown to be within 2% with this method of digitization. About 30 beats were recorded at each site of measurement and shown to be consistent in appearance. Signals from two or three beats were then digitized at intervals of 0.25 mm (0.00125 second) over the period of ejection (as judged by aortic pressure tracings from the upstroke of pressure to the dicrotic notch). The root mean square value of the velocity components at various frequencies throughout the range of analysis was observed to be nearly the same for each of the beats. A value from one beat was arbitrarily selected as being representative. Velocity signals were subjected to Fourier analysis by use of a fast Fourier transform. All frequencies below 30 Hz were considered intrinsic in pulsatile velocity signals and, therefore, were excluded from considerations relative to the measurement of the energy of fluctuations. Signals above 320 Hz were excluded because of the magnitude of the interval at which signals were digitized, because of

*A conservative estimate of frequencies that can be obtained by Fourier analysis is calculated as $0.4/\Delta t$, where $\Delta t$ is the sampling interval (in this study, 0.00125 second).
inaccuracies that may have been introduced with hand operation of the electronic digitizer, and because of the frequency response of the recorder. Included data in the upper range of frequencies may be somewhat in error. Amplitudes of fluctuating components of velocity below 0.25 cm/sec were considered insignificant and possibly due to artifact based upon tests of the digitizer and Fourier program with a known sine wave input.

The root mean square value of the fluctuating components of velocity has been defined as the intensity of turbulence. In analogous fashion, we measured the amplitude of the fluctuating components as the root mean square value of the Fourier components of velocity. This is possible since the Fourier components of velocity are descriptive of the frequencies inherent in the original signal. (The first 30 Hz must be eliminated from consideration, because these frequencies are inherent in pulsatile flow, and may not necessarily represent random fluctuations.) To achieve a measure of the energy of flow, we calculated the energy of the fluctuations per unit volume of blood. This would be the product of the turbulence intensity squared and the density.

\[ \text{Turbulence energy density (ergs/cm)} = \rho (u_{rms})^2 \]  

where \( u_{rms} \) = root mean square value of the magnitude of the Fourier components of velocity (cm/sec) at each frequency taken at intervals of 2.6–3.6 Hz between 30 Hz and 320 Hz; and \( \rho \) = density of the blood (g/cm\(^3\)).

To measure \( u_{rms} \), each component of velocity, measured at intervals of 2.3–3.6 Hz over a range of frequencies from 30 to 320 Hz, was squared and summed, and then the square root was taken. This gave the root mean square value of the magnitude of the fluctuating components, as judged by Fourier analysis.

The spectral energy distribution was plotted. The average turbulence energy density was calculated at intervals of 15 Hz throughout the range of measured frequencies. Data points were plotted at the central frequency of each interval. This spectral energy distribution is comparable to those described by others, although absolute rather than fractional and nondimensional values were utilized. The turbulence energy density (Eq. 3) is comparable to the area under this curve.

The techniques of analysis used for each subject are illustrated in Figure 2. This particular subject (subject 14) had aortic stenosis. Instantaneous velocity in the region of the aortic valve over the period of one complete cardiac cycle is shown. The magnitude of the fluctuating velocity components between 30 Hz and 320 Hz during ejection is shown. The root mean square value of the magnitude of these velocity components was then calculated. The square of this value, times density, represents the turbulence energy density (Eq. 3).

Results

Data for a subject (Table 1, subject 5) with a normally functioning aortic valve and normal cardiac output are shown in Figure 3. Disturbances developed at peak velocity and were maintained throughout ejection. The amplitudes of velocity components to 100 Hz were significant (>0.25 cm/sec). The turbulence energy density at the point of measurement was 0.6 ergs/cm\(^3\). In the mid-ascending aorta, and at the arch of the aorta, the velocity signal was virtually smooth. No significant disturbances of flow were present. Frequencies above 50 Hz in the mid-ascending aorta and above 38 Hz in the proximal portion of the aortic arch were of negligible magnitude, as was the energy density (0.3 erg/cm\(^3\)) and 0.1 erg/cm\(^3\), respectively).

Data from a subject (No. 1) with a normal aortic valve who had a high cardiac output (12.9 liters/min) are illustrated in Figure 4. Turbulent flow was shown in the region of the aortic valve. Disturbances occurred on the ascending portion of the velocity curve and continued through the early portion of diastole. Amplitudes of fluctuating velocity components were of significant magnitude to 320 Hz, the highest frequency measurable by the methods of analysis available. The turbulence energy density was 2.9 ergs/cm\(^3\).

The turbulence energy density in the mid-ascending aorta was 1.2 ergs/cm\(^3\), less than one-half that above the valve. Velocity components to 320 Hz were observed.

In subjects with normally functioning aortic valves, the peak Reynolds number ranged between 5,700 and 10,000 (Table 1). The turbulence energy density, as it related to the cardiac output, stroke volume, peak Reynolds number, and the alpha number, is shown (Fig. 5 and Table 1).
TABLE 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Aortic valve</th>
<th>Cardiac output (liters/min)</th>
<th>Stroke volume (ml/beat)</th>
<th>Heart rate (beats/min)</th>
<th>Viscosity (poise)</th>
<th>Peak velocity (cm/sec)</th>
<th>Peak Reynolds number (dimensionless)</th>
<th>Unsteadiness parameter, α (dimensionless)</th>
<th>Energy density (ergs/cm³)</th>
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</thead>
<tbody>
<tr>
<td>1 Normal</td>
<td>Normal</td>
<td>12.9</td>
<td>90</td>
<td>144</td>
<td>0.058</td>
<td>181</td>
<td>10,000</td>
<td>26</td>
<td>2.9</td>
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<td>2 Normal</td>
<td>Normal</td>
<td>5.3</td>
<td>57</td>
<td>92</td>
<td>0.054</td>
<td>96</td>
<td>5,700</td>
<td>21</td>
<td>1.2</td>
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<tr>
<td>3 Normal</td>
<td>Normal</td>
<td>5.2</td>
<td>72</td>
<td>72</td>
<td>0.051</td>
<td>97</td>
<td>5,800</td>
<td>18</td>
<td>0.8</td>
</tr>
<tr>
<td>4 Normal</td>
<td>Normal</td>
<td>5.3</td>
<td>74</td>
<td>72</td>
<td>0.053</td>
<td>118</td>
<td>6,200</td>
<td>16</td>
<td>0.7</td>
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<td>50</td>
<td>100</td>
<td>0.055</td>
<td>117</td>
<td>5,900</td>
<td>19</td>
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<tr>
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<td>Normal</td>
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<td>55</td>
<td>120</td>
<td>0.051</td>
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<tr>
<td>7 Normal</td>
<td>Normal</td>
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<td>141</td>
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<td>1.1</td>
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<tr>
<td>8 Prosthetic</td>
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<td>52</td>
<td>90</td>
<td>0.059</td>
<td>216</td>
<td>†</td>
<td>†</td>
<td>†</td>
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<tr>
<td>9 AI</td>
<td>Normal</td>
<td>6.5</td>
<td>80</td>
<td>80</td>
<td>0.055</td>
<td>238</td>
<td>†</td>
<td>†</td>
<td>†</td>
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<tr>
<td>10 AI</td>
<td>Normal</td>
<td>5.3</td>
<td>66</td>
<td>66</td>
<td>0.038</td>
<td>238</td>
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<td>11 AI</td>
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<td>64</td>
<td>64</td>
<td>0.054</td>
<td>231</td>
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<tr>
<td>12 AI</td>
<td>Normal</td>
<td>6.5</td>
<td>70</td>
<td>70</td>
<td>0.047</td>
<td>256</td>
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<tr>
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<td>80</td>
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<td>0.051</td>
<td>352</td>
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<td>†</td>
<td>†</td>
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<tr>
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<td>Normal</td>
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<td>80</td>
<td>80</td>
<td>0.051</td>
<td>352</td>
<td>†</td>
<td>†</td>
<td>†</td>
</tr>
<tr>
<td>15 AS-AI</td>
<td>Normal</td>
<td>4.5</td>
<td>54</td>
<td>54</td>
<td>0.049</td>
<td>459</td>
<td>†</td>
<td>†</td>
<td>†</td>
</tr>
</tbody>
</table>

AI = aortic insufficiency; AS = aortic stenosis.

* In the presence of aortic regurgitation, total flow across the valve could not be calculated from the cardiac output.
† The presence of an abnormal valve caused the Reynolds number and α number to be of different significance and, therefore, not comparable.
aortic valve, appeared to be directly related to peak velocity, regardless of the valvular configuration (Fig. 11). In the presence of aortic stenosis, higher turbulence energy density was observed than for normal valves at comparable stroke volumes (Fig. 12). (In subjects with aortic regurgitation an approximate stroke volume was estimated from the ventriculograms as the difference between the end-diastolic volumes (Fig. 12). (In subjects with aortic regurgitation an approximate stroke volume was estimated from the ventriculograms as the difference between the end-diastolic volume and end-systolic volume.) The turbulence energy density did not appear to be affected by the alpha number, although the range of heart rates was small.

Discussion

The existence of turbulent blood flow in the human body would seem to be likely on the basis of studies using pumps and laboratory animals, although there is some evidence to the contrary. Turbulence immediately downstream from branches, S curves, and bifurcations has been shown in models of the vascular system in which a steady flow of water was produced at Reynolds numbers which allowed overall laminar flow. Turbulence also has been observed immediately downstream from the entrance of branches in plexiglass models of the arterial system in which there was a pulsatile flow of human blood. Turbulence downstream from the aortic bifurcation has been shown in rabbits at peak velocity. Fully developed turbulence has been measured in the ascending aorta of one dog, and highly disturbed, probably turbulent flow has been measured in the aorta of other dogs and of horses. In one subject with normal cardiac function, point velocity was measured above the aortic valve during cardiac catheterization, and turbulence was noted near peak velocity (150 cm/sec), which corresponded to a peak Reynolds number of 11,000. In another subject with an apparently normal valve, laminar flow was noted throughout ejection, although when remeasured during open heart surgery (for mitral valve replacement), turbulence appeared to be present during peak systole. Turbulence during open heart surgery was observed above the aortic valve of one subject with aortic stenosis. One subject with aortic regurgitation also was studied and showed turbulence. These same investigators also showed turbulent flow during open heart surgery in the pulmonary artery distal to a stenosed pulmonic valve in one subject.

A strict definition of turbulent flow is elusive. Hinze defined it as "motion in an irregular condition of flow in which the various quantities show a random variation with time and space coordinates so that statistically significant average values can be discerned." Perhaps a more encompassing definition is: "Turbulence is an irregular eddying motion in which velocity and pressure perturbations occur about their mean values; these perturbations are irregular or random, even chaotic, in time and space with components extending smoothly over an extensive continuous hierarchy of scales or frequencies so that they must be characterized by statistical means."

According to strict definition, the presence of turbulence can be proved only by the simultaneous demonstration of random velocities in more than one plane. However, the process has been so well characterized in many fluid dynamic problems, that fluctuations in one plane which have an appropriate frequency spectrum can be accepted as evidence of turbulence. It would seem that flow in the ascending aorta of at least one of the subjects with a normal aortic valve, and all of the other subjects with abnormal aortic valves, satisfies these definitions.

The critical Reynolds number, beyond which the amplification of small disturbances will occur under conditions of steady flow in smooth, straight pipes is about 2,300. This value can be regarded as the lower limit for the critical Reynolds number, below which even strong disturbances do not cause the flow to become turbulent. With an arrangement which is as free from disturbances as possible, critical Reynolds numbers of 10,000 can be attained. Since disturbances of flow in arteries occur near peak velocity, a peak Reynolds number based upon peak velocity has been found to be a meaningful expression. The effects of nonsteady flow upon the critical Reynolds number are unclear, since contradictory results have been reported. Sinusoidal flow, according to some, causes a reduction of the Reynolds number of transition from laminar to turbulent flow. On the other hand, others have reported that...
Pulsatile flow is more stable than corresponding steady Poiseuille flow. The concentration of erythrocytes also affects turbulence. Blood with a hematocrit between 20% and 30% flowed with a higher intensity of turbulence than equally dense and viscous plasma at the same Reynolds number. This augmentation of turbulence produced by the erythrocytes was less apparent when the hematocrit was increased to 40% or diminished to 10%.

The Reynolds number at a constricted orifice does not have the same significance as the Reynolds number for a smooth pipe. Turbulent flow would occur at much lower values in the former case. For example, turbulent flow occurred distal to a constriction in a tube at Reynolds numbers below 500. Calculations of the order of magnitude of the Reynolds number for the valvular orifice of subjects with diseased valves, nevertheless, were even higher than the Reynolds numbers measured in subjects with normal valves. (These values were based upon the estimated diameter of the valvular orifice as derived from hemodynamic measurements of the cross-sectional area of the valve. Such calcula-
tions used ventriculographic estimates of stroke volume in order to include the regurgitant fraction of flow.) The approximate peak Reynolds number at the valvular orifice of these subjects with various combinations of aortic stenosis and regurgitation ranged between 10,000 and 20,000. Since the magnitude of the intensity of turbulence with pulsating flow through a jet increases as the Reynolds number increases, high magnitudes of turbulence would be expected distal to stenotic valves with the magnitudes of peak Reynolds numbers estimated for these subjects.

The frequency content of turbulence, expressed in the form of a power spectrum, has been used to describe the structure of the disturbance. Schlichting used a spectral distribution based upon the fractional content of the root mean square value of the longitudinal fluctuations which belong to small frequency intervals. The spectral distribution of the fractional root mean square values shows that the highest value occurs at the lowest measured frequency. This was observed in this study from the analysis of the magnitude of the various fluctuating components of velocity and their energy distributions (Figs. 2 and 10).

A plot of the spectral distribution of the fractional energy of fluctuation does not lend itself to simple numerical characterization. Therefore, comparisons between subjects would be difficult with data presented in this form. We found it more meaningful to describe the absolute energy of fluctuations averaged throughout the band of frequencies which were identified by Fourier analysis. This gives a

**Figure 9** Energy density of fluctuations (turbulence energy density) measured at aortic valve, mid-ascending aorta, proximal portion of aortic arch, and innominate artery. (For some subjects data were not obtained at each site.) NL = normal valves; AI = aortic insufficiency; AS-AI = aortic stenosis and insufficiency; PAV = prosthetic aortic valve.

**Figure 10** Spectral energy distribution of flow above the aortic valve of subject 5 with a normal valve, subject 1 with a normal valve and high cardiac output, and subject 15 with predominant aortic stenosis. Point velocity in these subjects was illustrated in Figures 2, 3, and 6. The spectral energy distribution showed no discontinuities in any of the patients.

**Figure 11** Relation of turbulence energy density measured just above the aortic valve to peak point velocity. Symbols and abbreviations as in Figure 9.
Numerical value which permits comparisons between subjects (Eq. 3).

Polymeric additives have been shown to effectively reduce turbulent flow in blood as indicated by a reduction of drag, and a diminished intensity of turbulence. In view of possible pathological processes which may relate to turbulent flow, such as thrombosis in the region of prosthetic aortic valves, it may be that nontoxic forms of such materials may be of potential therapeutic value.

In conclusion, highly disturbed flow occurs in the ascending aorta of normal subjects under some circumstances, such as during states of high cardiac output. Turbulent flow in the ascending aorta of normal subjects was highest in subjects with aortic stenosis. In such individuals, turbulence was observed throughout the ascending aorta and in the innominate artery.

References


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doi: 10.1161/01.RES.39.1.58

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