Relationship between Intraventricular Flow Patterns and the Shapes of the Aliasing Area in Color M-mode Doppler Echocardiograms* —A CFD Study with an Axisymmetric Model of the LV—

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Spatiotemporal maps of the velocity of intraventricular blood flows obtained with color M-mode Doppler (CMD) echocardiography are used to assess the diastolic function of the left ventricle (LV). However, theoretical basis for that is unclear. Hence, we studied the relationship between flow patterns in the LV and the shapes of aliasing areas that appear in CMD echocardiograms by means of computational fluid dynamics using an axisymmetric model of the LV. The results showed that a ring vortex formed in the early stage of expansion and grew larger while shifting its center towards the apex of the LV, occupying a large annular space between the mainflow along the long axis and the lateral wall of the LV and constricting the mainflow. Due to that, fluid elements in the mainflow increased their velocities and proceeded further deeper into the LV with high velocities, which appeared to be an elongated shape of the aliasing area in the CMD echocardiogram. From these results it was concluded that the shape of the aliasing area in a CMD echocardiogram shows the change in the velocity of the mainflow affected by the growth and migration of a ring vortex formed in the LV.

Key Words: Bio-fluid Mechanics, Computational Fluid Dynamics, Vortex, Left Ventricle, Color M-mode Doppler Echocardiography, Intraventricular Flow, Diastole, Aliasing Area

1. Introduction

Transmitral velocity of blood obtained with pulsed Doppler echocardiography has been widely used clinically to assess the diastolic function of the human left ventricle (LV) for its non-invasive nature

and handiness⁽¹⁾. However, limitations of this method have also been pointed out⁽²⁾ that the transmitral velocity is easily affected by heart rate⁽³⁾ and preload⁽⁴⁾, and that it shows a pattern similar to that of normals in some pathological cases where an atrioventricular pressure gradient increases, i.e., a case called pseudonormalization⁽⁵⁾.

As a new method that overcomes drawbacks of the pulsed Doppler echocardiography, color M-mode Doppler (CMD) echocardiography was proposed. In this method, distributions of blood velocity along the long axis of the LV are measured as a function of time by transmitting an ultrasound beam from the apex of the LV, and measured velocity distributions are presented as a spatiotemporal map in which the magnitude of blood velocity is expressed by a color and brightness. Using this method, Jacobs et al.⁽⁶⁾

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measured times at which the peak velocity of filling flow appeared at different levels from the mitral valve to the apex, and found that propagation of the peak velocity of the ventricular filling flow was delayed in the LV with dilated cardiomyopathy. The validity of using the propagation rate of the peak velocity of the ventricular filling flow as an indicator of the diastolic function was confirmed by comparing it with invasively measured parameters such as a time constant of LV pressure decay in isovolumetric relaxation^{(7)–(9)}. Takatsuji et al.⁽⁸⁾ intentionally changed the aliasing limit of the color Doppler signals in a CMD echocardiogram and observed an aliasing area where blood velocity is higher than 70% of the maximum velocity during diastole. They postulated that the shape of the aliasing area would reflect the diastolic function of the LV and described that it was related to the isovolumetric time constant which was determined invasively in clinical patients. However, since it is difficult to grasp the characteristics of the flow in the LV as a whole and wall motions of the LV only from a CMD echocardiogram, theoretical bases of the relationship between the shape of the aliasing area and the diastolic function of the LV are not well understood yet. Therefore, in order to improve the accuracy of the diagnosis of the diastolic function of the LV with color M-mode Doppler echocardiography, it is necessary to analyze the relationship between intraventricular flow and wall motion of the LV during diastole, and to clarify how this relationship is reflected on the shape of the aliasing area that appears in a CMD echocardiogram. Hence, we attempted to study intraventricular flow patterns by a computer simulation of the filling-flow of the LV.

2. Method

2.1 Computational model of the wall motion of an expanding LV

A two-dimensional axisymmetric model of the LV shown in Fig. 1 was used to study the characteristics of diastolic flows in the LV. It was assumed that the volume of the LV at its maximum expansion or at the end of diastole including the phase of atrial contraction was 120 cm³ and the geometry at that point was expressed by an ellipsoid with a long and short axis of 4.96 cm and 2.60 cm, respectively. One end of the ventricle model was cut off perpendicularly to the long axis so as to provide a space for the left atrium with a radius of 1.63 cm. The other end was also cut off perpendicularly at a location at which the radius was 0.5 cm to mimic the shape of the apical curvature of the LV. The location of the inlet of the mitral orifice (the annular level) was assigned to the origin of the x -axis that was laid along the long axis of the

LV. The radius of the inlet of the mitral orifice was set to be 1.48 cm. It was also set that the angle of opening of the valve leaflet (1.48 cm in length) was fixed at 85 degrees with respect to the inlet of the mitral orifice, and remained unchanged during the whole phase of diastole.

Based on clinical data on the magnitude of the intraventricular blood pressure⁽¹⁰⁾, we considered that intraventricular pressure during this phase was not high enough to push out the wall of the LV. In addition to this, it is generally considered that the deformation of the LV wall during an early diastolic phase is not initiated by blood pressure but by a spontaneous relaxation of myocardium that has contracted during a systolic phase. Thus, it was assumed that the geometry of the LV at each time step was not affected by the intraventricular flow. In the present study, the deformation of the wall of the LV due to the relaxation of the myocardium was modeled by moving the wall independently of the intraventricular flow and pressure.

The geometry of the model of the LV during diastole was determined as follows. Each nodal point on the wall of the LV was assumed to move in a direction normal to the luminal surface of the LV. Then, the radius of the LV at $t + \Delta t$ is given by

$$r(X, t + \Delta t) = r(x, t) + v(x, t)n_r\Delta t \quad (1)$$

where $r(x, t)$ is the radius of the LV, $v(x, t)$ is the velocity of a moving wall at time t , X is the x coordinate of the nodal point at $t + \Delta t$ expressed as $x + v(x, t)n_x\Delta t$, and n_r and n_x are the components of a unit vector normal to the wall of the LV. Therefore, the volume of the LV $V(t)$ can be expressed as

$$V(t) = \pi \int_0^{x_L(t)} \{r(x, t)\}^2 dx \quad (2)$$

where $x_L(t)$ is the distance from the inlet of the mitral orifice to the apex of the LV at time t . The volume increment $\Delta V(t)$ during Δt is expressed by

$$\Delta V(t) = V(t + \Delta t) - V(t). \quad (3)$$

Factoring the wall velocity $v(x, t)$ into a function of

Fig. 1 The geometry of an axisymmetric model of the left ventricle at its maximum expansion and at the beginning of diastole

time $\tau(t)$ and a function of space $\sigma(x)$, $v(x, t)$ is given by

$$v(x, t) = \tau(t) \cdot \sigma(x). \quad (4)$$

Once the function $\sigma(x)$ and the volume increment $\Delta V(t)$ are provided, the value of $\tau(t)$ is determined by solving Eq.(1) to Eq.(4), and then the radius $r(X, t + \Delta t)$ at a new time step can be obtained.

In this study, we used a function of space

$$\sigma(x) = \sin(0.5\pi \cdot x/x_L) \quad (5)$$

so as to maintain the radius of the inlet of the mitral orifice constant. The geometry of the LV model at the end of systole was obtained from its geometry at the maximum expansion by decreasing its volume from 120 ml to 60 ml based on the model of the wall motion described in this section.

2.2 Procedures for computation

It was assumed that blood was an incompressible Newtonian fluid with a density of 1.05 g/cm³ and a viscosity of 0.035 g/(cm·sec). The Navier-Stokes equation and the equation of continuity were solved for the case of an axisymmetric expansion using a finite element method for a turbulent flow (k - ϵ model) given by ANSYS-FLOTRAN ver. 5.5 (distributed by Cybernet Systems Co. Ltd., Tokyo). The convergence of the solution was assessed at each step of the 248 steps of expansion that were assigned to 0.248 sec of a diastolic phase.

Computation of intraventricular flow was initiated from the point of maximum contraction. The model of the LV was divided into 100 and 60 elements in the direction of the long and short axes, respectively. As an initial condition, it was assumed that there was no movement of blood in the LV. Boundary conditions on various parts of the LV are

$$\begin{aligned} P &= 0 && \text{at } \Omega_1 \\ u_x &= 0, \quad u_r = 0 && \text{on } \Omega_2 \\ u_r &= 0 && \text{on } \Omega_3 \\ u_x &= v(x, t)n_x, \quad u_r = v(x, t)n_r && \text{on } \Omega_4 \end{aligned}$$

where u_x and u_r are the fluid velocity in the x - and r -directions, respectively, P is a pressure, Ω_1 is the inlet of the mitral orifice, Ω_2 the leaflet and the ring of the mitral valve, Ω_3 the long axis of the LV, and Ω_4 the wall of the LV including the apex of the LV. If the convergence of the solution was confirmed at a time step, a further expanded model was constructed for a next time step and divided into finite elements again. However, since the nodal points created in the new LV model did not coincide with those old ones due to re-division of the LV model, physical quantities, namely a new velocity, a pressure, a turbulent energy and its dissipation rate, on new nodal points were interpolated by the use of the Lagrange interpolation with data obtained in the previous computation. Computation for a new time step was then carried out.

Fig. 2 Temporal changes of the mean velocity of the inflow or transmittal flow velocity curve at the annular level for the LVs with a normal diastolic function (solid line) and with a diastolic dysfunction (dashed line), which were used to calculate the volume change of the left ventricle

2.3 Volume change of the LV used for the present study

Prior to computer simulations of intraventricular flow, we calculated the volume change to be imposed on the LV model. Parameters necessary to calculate the volume change of the LV during this period were determined based on a transmittal velocity clinically measured with pulsed Doppler echocardiography at the annular level. Figure 2 shows a time course of the mean velocity at the annular level or a transmittal velocity during early diastole. For the LV with a larger volume change (LV with a normal diastolic function), times from the beginning of diastole ($t=0$) to the peak of transmittal velocity and to the end of early diastole were set to be 0.086 sec and 0.248 sec, respectively, and the peak value of transmittal velocity was set to be 52 cm/sec. Clinically, diastolic function of the LV was evaluated based on the peak value of transmittal velocity, and it is considered that the higher the value of the peak velocity, the better the diastolic function is up to some extent⁽¹⁾. Then, for the LV with a poor diastolic function (LV with a diastolic dysfunction), the value of transmittal velocity was set to be a half of the normal value as shown by a dashed line in Fig. 2.

The rate of volume change of the LV is obtained as a product of mean value of transmittal velocity ($u_m(t)$) and cross-sectional area of the inlet of the mitral orifice (πr_0^2). Thus, the volume of the LV at t is expressed by

$$V(t) = V(0) + \int_0^t \pi r_0^2 u_m(t) dt \quad (6)$$

where $V(0)$ is the initial volume of the LV and r_0 is

Fig. 3 Temporal change of the volume of the LV with a normal diastolic function (solid line) and with a diastolic dysfunction (dashed line) used for computer simulations. These volume changes were calculated based on the temporal change of the mean velocity shown in Fig. 2.

the radius of the inlet of the mitral orifice. Figure 3 shows the time course of the volume change of the LV calculated using the above equation with a mean velocity shown in Fig. 2 assuming that the volume of the LV at the beginning of diastole is 60 cm³. This gives a volume change of 45 cm³ and 22 cm³ for the LVs with a normal diastolic function and a diastolic dysfunction, respectively.

In the present study, we carried out computer simulations of intraventricular flow during early diastole for the cases of the LVs with a normal diastolic function and a poor diastolic function (a diastolic dysfunction) by giving a large and small volume change to the LV as shown in Fig. 3.

3. Results

Figure 4 shows a temporal change of intraventricular blood pressure at two different locations on the long axis of the LV, that is, the apex of the LV, and the midpoint between the inlet of the mitral orifice and the apex. Here, the values indicate the changes from an initial value of $P=0$ given at the inlet of the mitral orifice. It was shown that in both cases the intraventricular pressure fell suddenly at the beginning of the diastole, and thereafter it decreased slowly until it changed to an increase. Then, the pressure rose steeply and attained stable positive values in the early stage of the diastolic phase. Comparison of the intraventricular pressures evaluated at the two sites showed that the absolute value of pressure at the apex was always greater than that at the midpoint. Temporal change in the intraventricular pressure was much larger in the LV with a normal diastolic function than

Fig. 4 Plots of the intraventricular pressure at the midpoint (circles) between the inlet of the mitral orifice and the apex, and at the apex of the LV (triangles). Empty and filled symbols indicate the results for the LVs with a normal diastolic function and with a diastolic dysfunction, respectively.

that in the LV with a diastolic dysfunction.

Figure 5 shows contour lines of intraventricular blood pressure drawn at an interval of 20 Pa (0.150 mmHg) and corresponding flow patterns expressed by velocity vectors respectively in the upper and lower halves of each longitudinal cross-section of the LV at each time step during early diastole. Here, the pressure at each point was expressed as a relative value to that evaluated at the inlet of the mitral orifice. As shown in the uppermost figure of Fig. 5, as soon as the LV started to expand ($t=0.01$ sec), a pressure gradient with the lowest and highest pressures locating at the apex and the inlet of the LV, respectively, was created within the LV which was filled with blood. As a result, the blood started to flow into the LV through the inlet of the mitral orifice. Since the contour lines of pressure were dense in the entrance region surrounded by mitral valve leaflets, the velocity of blood took the largest value in this region. At $t=0.05$ sec, the pressure gradient became further steeper as evident from dense contour lines of pressure in the entrance region, resulting in a rapid increase in the velocity of the blood flowing into the LV. After passing by the tip of the valve leaflet, a lump of fluid elements (blood) headed straight ahead to the apex, becoming a mainflow. There were also peripheral flows that diverged to the lateral wall of the LV beyond the tip of the valve leaflet. Furthermore, since the blood pressure on the lateral wall of the LV just behind the valve leaflet became negative, fluid elements located in the vicinity of the ventricular wall

Fig. 5 Plots of the contour lines of the intraventricular pressure (upper half) and the velocity vectors of the flow (lower half) at each time step during early diastole. **A**: LV with a normal diastolic function, **B**: LV with a diastolic dysfunction. The number on each contour line shows the magnitude of pressure (Pa) on the line.

behind the valve leaflet started moving backward. In this way, a tiny ring (annular) vortex was formed in the space between the valve leaflet and the lateral wall of the LV. Then, at $t=0.086$ sec when the velocity of expansion of the LV reached its maximum, a part of dense contour lines coiled up towards the lateral wall, creating a very steep negative pressure gradient along the lateral wall. Due to that, the tiny ring vortex formed in the previous sequence grew larger and stronger. As the expansion of the LV proceeded with gradually diminishing velocities, the region of dense contour lines shifted towards the apex of the LV, and the ring vortex further grew in size and increased its intensity while shifting its center towards the apex, occupying a large annular space between the mainflow and the lateral wall of the LV. A further expansion of

Fig. 6 Color M-mode Doppler echocardiographic images of the velocity distribution on the long axis of the left ventricle obtained with CFD. The magnitude of an instantaneous velocity is expressed by a color according to the scale of the color bar presented on the right hand of each echocardiogram. Note that the original figure is printed in color. The color bar changes from black to red corresponding to the velocity range from 0 to the aliasing limit and it changes from blue to light blue above the aliasing limit. The gray region in the center of each figure appears in blue.

the LV with diminishing velocities at the late stage of the diastolic phase resulted in the decrease in the velocity of fluid elements in the mainflow. The Reynolds number at the inlet of the mitral orifice in this case varied from 0 to approximately 8,000.

A similar pattern was observed in both the LVs with a normal function and a diastolic dysfunction. However, in the latter case, the ring vortex did not grow much larger, and the vortex center did not shift so much towards the apex of the LV.

Figure 6 shows CMD echocardiograms constructed from a series of time-dependent velocity distribution along the long axis of the LV obtained for the LVs with a normal diastolic function (left) and a diastolic dysfunction (right) which were shown in Fig. 5. Here, the horizontal and vertical axes show the time lapsed from the onset of the diastolic phase and the distance from the inlet of the mitral orifice, respectively. The magnitude of the velocity at each point was expressed by a color according to the scale of the color bar presented on the right hand of each echocar-

diogram. The area that appeared in blue in the middle of the abscissa indicates the region where the velocity of fluid elements is larger than 70% of the maximum velocity in the diastolic phase, and it is called an aliasing area. The width of the aliasing area at the bottom of the CMD echocardiograms shows the duration of time in which the velocity of fluid elements passing through the inlet of the mitral orifice was larger than 70% of the maximum velocity. Comparison of the CMD echocardiograms between the LVs with a normal diastolic function and a diastolic dysfunction showed that, despite the large difference in the rate of volume change, the width of the aliasing area at the bottom was almost the same. However, the shape of the aliasing area was quite different. In the case of the LV with a normal diastolic function, the aliasing area was elongated and sharpened at the tip, reaching the midpoint of the long axis of the LV. By contrast, in the case of the LV with a diastolic dysfunction, the aliasing area was short and rounded at its tip, and the whole area was confined within one-third (approximately 2.5 cm) of the distance from the inlet of the mitral orifice to the apex of the LV.

4. Discussion

In the present study, it was shown that the intraventricular blood pressure dropped quickly as soon as the expansion of the LV started, and it continued to drop until the velocity of expansion changed to a decrease. As soon as the velocity of expansion or the rate of volume change of the LV changed to a decrease, the intraventricular pressure rose abruptly from negative to positive values with the highest value locating at the apex of the LV. A similar trend for the temporal change of the intraventricular pressure was found in a clinical study⁽¹¹⁾. It was also observed that a time-dependent pressure gradient was created in the LV during the diastolic phase. At the beginning of the expansion of the LV, the pressure was the highest at the annular level and the lowest at the apex of the LV until the velocity of expansion of the LV reached its maximum, and this provided a driving force to draw the blood stored in the left atrium into the LV. In this study, the values of the intraventricular pressure were obtained under the condition that the pressure at the inlet of the mitral orifice stays unchanged ($P=0$ mmHg) throughout the diastolic phase, although, in fact, the atrial pressure changes during diastole. Thus, a direct comparison of our results with those found clinically is difficult. Nevertheless, it is worthwhile to mention that the spatial distribution of intraventricular pressure which we observed was similar to that found clinically by Courtois et al.⁽¹²⁾ who measured the

intraventricular pressure in the human heart *in vivo* using micromanometers. Net change in blood pressure in the apical region of the LV with a normal diastolic function during the diastolic phase was approximately 930 Pa (7 mmHg) which was close to the value obtained by Courtois et al.⁽¹¹⁾ in the LV of a human heart *in vivo*. The highest pressure difference observed in our present study in the LV with a normal diastolic function in an early stage ($t=0.05$ sec) of expansion of the LV was approximately 665 Pa (5 mmHg) which is too small to cause the deformation and expansion of the thick wall of the LV composed of myocardium. This implies that our assumption that the expansion of the LV occurs independently of the flow and the pressure difference between the left atrium and the LV is reasonable.

It was shown that an inflow of blood caused a formation of a vortex around the tip of mitral valve leaflet and it grew as the LV expanded. Formation and development of such vortices in the LV during diastole has been demonstrated theoretically in other computer simulations of the intraventricular flow^{(13)–(15)} and experimentally *in vivo*^{(16),(17)} and *in vitro*^{(18)–(20)}. In the case of the LV with a normal diastolic function, the vortex grew both in size and intensity while shifting its center towards the apex of the LV, occupying a large annular space between the mainflow located along the long axis and the lateral wall of the LV. However, in the case of the LV with a diastolic dysfunction, the vortex did not grow so much, and the center of the vortex stayed close to the tip of the valve leaflet. Here, if we consider the phenomena that take place in the LV from a viewpoint of energy balance, they could be explained as follows. First of all, relaxation of myocardium causes expansion of the LV, generating momentum energy of a movement of the LV wall. Then the momentum energy is transmitted to blood in the LV and is converted to pressure energy, creating a pressure gradient in the LV. The pressure energy is then converted again to momentum energy of fluid elements so that they flow in the LV. The velocity of the fluid elements depends on the amount of the momentum energy generated by the expansion of the LV (relaxation of myocardium), and the larger the amount of the momentum energy, the larger the velocity of the fluid elements in both the mainflow and the ring vortex. Therefore, the growth and migration of the ring vortex that affect the mainflow are largely dependent on the diastolic function of the LV which could be evaluated by the velocity and the extent of expansion of the LV.

The CMD echocardiograms constructed using the results of the present CFD study showed that the

aliasing area which appeared in the CMD echocardiogram was relatively long and sharpened at the tip in the case of the LV with a normal diastolic function, and it was short and rounded at its tip in the case of the LV with a diastolic dysfunction. These results are qualitatively consistent with those found clinically^{(6),(8),(21)}. An aliasing area that shows spatiotemporal change in the velocity of the mainflow on the long axis of the LV is largely affected by intraventricular flow dynamics. At the beginning of the diastolic phase, the inflow of blood through the mitral orifice diverges after passing by the tip of mitral valve leaflet because of enlargement of the flow channel from the mitral orifice, resulting in the decrease of the velocity of fluid elements. However, as the vortex formed in the early stage of expansion grows in size and occupies a large annular space in the LV, it narrowed the passage of the inflow of blood. Due to that, the path of the fluid elements in the mainflow was locally constricted, and the fluid elements located in the portion of the mainflow surrounded by the ring vortex increased their velocities while passing through the vortex region. Furthermore, since the center of the vortex migrated towards the apex of the LV as the vortex grew in size and intensity, the region of the constriction of the mainflow also shifted towards the apex. This helped the fluid elements in the mainflow travel with high velocity and reach the points much closer to the apex of the LV in the case of the LV with a normal diastolic function. Therefore, it was considered that the elongation of the aliasing area in the color M-mode Doppler echocardiogram obtained in the LV with a normal diastolic function resulted from the fast growth of the vortex and the shifting of its center towards the apex. In contrast to this, if the growth rate of the vortex is small as seen in the LV with a diastolic dysfunction, the velocity of inflow beyond the tip of the mitral valve leaflet decreases sharply as a result of flow dispersion, resulting in a short aliasing area. Based on these findings, it was suggested that the CMD echocardiogram shows the spatiotemporal change of the translational velocity of the fluid elements in the mainflow along the long axis of the LV that was affected by the growth and migration of a ring vortex formed in the LV.

5. Conclusion

Computer simulation of intraventricular flow dynamics was carried out to understand the physical meaning of the shape of the aliasing area that appears in a color M-mode Doppler echocardiogram. It was shown that a ring vortex formed in the LV and grew larger as the expansion of the LV proceeded. The vortex constricted the mainflow along the long axis of

the LV and increased its velocity. Also, the shifting of the vortex center towards the apex caused the migration of the constriction point, helping the fluid elements with high velocities reach the points much close to the apex of the LV. Therefore, it was concluded that the shape of the aliasing area that appears in the color M-mode Doppler echocardiogram reflected the formation and the growth of a ring vortex which occurred as a result of expansion of the LV in early diastole.

The present study provides a fluid mechanical background for interpretation of color M-mode Doppler echocardiograms. The vortex formed in our present study appeared as a concentric ring with respect to the long axis of the LV due to an axisymmetric nature of the model. However, we are not sure if it occurs in the same manner in the LV of a natural heart which is known to exhibit a complex 3-dimensional shape. Thus, further investigation of intraventricular flow in realistic 3-dimensional models of the LV and its effects on CMD echocardiograms are required to confirm the validity of our findings.

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