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Spatiotemporal complexity of the aortic sinus vortex as a function of leaflet calcification

Hoda Hatoum, PhD¹ and Lakshmi Prasad Dasi, PhD^{1,2}

¹Department of biomedical engineering, The Ohio State University, Columbus, Ohio, USA

²Division of cardiac surgery, The Ohio State University, Columbus, Ohio, USA

Abstract

Several studies have shown the variation of aortic sinus structures' hemodynamics with different flow and geometric characteristics. They have also correlated aortic sinus hemodynamics with the progression and evolution of calcific aortic valve disease (CAVD). This study aims at visualizing aortic sinus fluid structure variations as functions of different leaflet calcification degrees and assessing their potential relationship with CAVD. A degenerated 23mm Carpentier-Edwards Perimount Magna valve extracted from a redo-surgery patient was implanted in an aortic root model and tested in a pulse duplicator left heart simulator. The valve has 3 leaflets with 3 different levels of calcium distribution: mild, moderate and severe. High-speed imaging and particle image velocimetry were performed to assess sinus vortices, leaflet tip position and velocity along with shear stress. Results have shown that (a) aortic sinus vortices initiation, entrapment and evolution varied with different calcified leaflet exposure; (b) higher velocities in the sinus were calculated with the mildly calcified leaflet compared to the moderately and severely calcified ones; (c) during systole, the mildly calcified leaflet sinus case shows the most spread-out and higher ranges of shear stress probabilities and highest magnitudes going from (−1.5 to +1.8 Pa) compared with (−1.0 to +1.0Pa) for moderately and severely calcified leaflets. The higher the calcification degree the lower the shear stress range and likelihoods of having higher shear stress. This holds in diastole as well. This study shows the impact of calcification on the aortic sinus flow structures.

Introduction

The aortic sinus vortex is one of the most important and prevalent fluid dynamic features in the aortic sinuses[1]. While the existence of a vortical pattern in the sinus may have first been hypothesized by Leonardo da Vinci in the 1500s, studies in the past century have confirmed the existence and correlated its important role in valve closure[2]. Bellhouse and Talbot[3] and Bellhouse [4] have reinforced this suggestion through measurements of pressure changes from the sinus vortex adjacent to the open leaflets. Talukder et al[5] demonstrated that valve closure can be achieved primarily by the adverse pressure gradient and does not necessitate a trapped sinus vortex. Given all these studies, it is now

Address for correspondence and reprints: Lakshmi Prasad Dasi, PhD, Associate Professor, Department of Biomedical Engineering, The Ohio State University, 473 W 12th Ave., Columbus, OH 43210, TEL: (614) 247-8313, lakshmi.dasi@osumc.edu.

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acknowledged that the aortic sinus vortex plays a role not only in the opening and closing mechanisms of the leaflets, but also has a crucial role in the context of sinus washout and overall energy efficiency of the aortic valve system [6–10]. Recent studies by Hatoum et al demonstrated how the evolution of the aortic sinus mechanism can correlate with aortic valve efficiency and energy dissipation[8]. In addition, it was demonstrated that aortic sinus vortices initiation, entrapment and evolution play an important role in sinus stasis and washout [7, 11–14].

Nonetheless, the factors impacting the spatio-temporal structure of the aortic valve sinus and how perturbation of this vortex can impact function, washout, and energy losses have not been completely addressed. Moore et al [15] provided an experimental study highlighting the importance of heart rate and fluid viscosity on the spatio-temporal structure in the aortic sinus vortex. Experimental studies by Hatoum et al[10] and numerical studies by Fukui et al[16] highlighted the dependence of aortic sinus vortex spatio-temporal topology on the morphology and geometry of the aortic sinuses. Coronary flow presence was also deemed important in changing the sinus vortices topologies, intensities and evolution [7, 14, 17]. However, the stiffness of the leaflets themselves and how that could impact the vortex formation, propagation and the sinus vortex topology has not been investigated. Thus investigating the aortic sinus dynamics in the sinuses of Valsalva is still crucial to understand their variations based on different conditions.

Leaflet stiffening and its impact on the sinus vortex is critical from the standpoint of progression of calcific aortic valve disease. This is because, in addition to genetic characteristics along with predisposition for particular inflammatory pathways[18, 19], fluid shear stresses have emerged as one of the major regulators for initiation and progression of CAVD[15, 20]. Later studies have highlighted hemodynamics in the sinuses of Valsalva as crucial factors impacting calcific aortic valve disease (CAVD) progression[21, 22]. While it is not yet fully understood whether hemodynamics initiate calcific valve disease or help in the progression of disease, it has been demonstrated that hemodynamics and disease are tightly correlated [23, 24]. CAVD impairs the normal function of the aortic valve through preventing the valve from fully opening leading to higher jet velocities, larger space for sinus vortex, and consequently non-physiological flow[25]. In the sinus, low shear stress has been considered a metric linked with CAVD[15, 23].

The objective of this study is to visualize and characterize the variations of the aortic sinus coherent structures as they form and evolve throughout the cardiac cycle as a function of leaflet calcification degrees (with more stiffer leaflets with calcification). Following this, we offer fluid dynamic mechanisms for the noted changes in coherent structures.

Methods

This study is part of an Institutional Review Board (IRB) approved study. Two-dimensional in-vitro particle image velocimetry (PIV) experiments were conducted to visualize aortic sinus hemodynamics for three different degrees of valve leaflet calcification.

Hemodynamic assessment

A degenerated 23mm Carpentier-Edwards Perimount Magna SAV of bovine pericardial leaflet material extracted from a patient who underwent a redo surgery (Fig.1a,b) was implanted in an aortic root model as per Moore et al[15]. The valve is degenerated and characterized by three leaflets with three significantly different degrees of calcific nodules: mild calcification (volume of 0.001 cm^3), moderate calcification (volume of 0.0592 cm^3) and severe calcification (volume of 0.275 cm^3). The calcification volume was assessed using a HeliScan 3D Micro Computed Tomography (Micro-CT). Given that the degree of stenosis in practice and literature is not assessed by calcification volume[26], the terms mild moderate and severe are only relevant to this study to easily characterize the valve leaflets. The sinus radius of the aortic valve chamber is 19 mm, sinus height 21.6 mm, and aortic radius 12.7 mm. The chamber was set up into the aortic position of a left heart pulse duplicator flow loop as previously described in other publications [7, 14, 27–30]. Sinus hemodynamics were assessed for each of the three leaflets through positioning the valve in all 3 possible positions in the aortic root model keeping the commissures aligned with those of the representative native model. Hemodynamic parameters for all conditions were maintained with a systolic to diastolic pressure of 120/80mmHg, a 1 beat per second heart rate and a cardiac output of 5l/min. The working fluid in this study was a mixture of water-glycerine producing a density of 1080 Kg/m^3 and a kinematic viscosity of 3.5 cSt similar to blood properties. The aortic flow rate was measured with an ultrasonic flow probe (Transonic Inc., Ithaca, NY) and pressures were measured just upstream and downstream of the valve using Validyne pressure sensors (Validyne Engineering Corp., Northridge, CA). Sixty consecutive cardiac cycles of aortic pressure, pressure difference across the aortic valve (or pressure gradient as clinically recognized and as will be adopted in the sections below), and aortic flow rate data were recorded at a sampling rate of 100Hz.

High-speed imaging and Particle Image Velocimetry (PIV)

Videos of *en-face* views imaging were acquired throughout the cardiac cycle at 1000 frames/s to record the opening and closing dynamics of the leaflets using a Photron Fastcam SA3 high-speed video camera (Photron, San Diego, CA, USA) and a high-speed controller (HSC) (LaVision, Ypsilanti, MI).

For PIV, the flow was seeded with fluorescent PMMA-Rhodamine B particles with an average diameter of $10\mu\text{m}$. This involves illuminating the sinus region using a laser sheet created by pulsed Nd:YLF single cavity diode pumped solid state laser coupled with external spherical and cylindrical lenses; while acquiring high-speed images of the fluorescent particles within the sinus region. Time-resolved raw PIV images were acquired with a resulting spatial and temporal resolutions of 0.159mm/pixel and 4000Hz respectively. Refraction was corrected using a calibration in DaVis particle image velocimetry software (DaVis 7.2, LaVision Germany). Velocity vectors were calculated using adaptive cross-correlation algorithms. The commercial PIV software, DaVis (LaVision, Germany) was used for data acquisition and processing. Velocity vectors were calculated using advanced PIV cross-correlation approaches with a 50% overlap multi-pass approach starting from one 32×32 pixel interrogation followed by two 16×16 pixel interrogation passes. Post processing

was executed using adaptive median filtering. Further details of PIV measurements can be found in our previous publications [7, 8, 14, 27–29].

Using the velocity measurements from PIV, vorticity dynamics were also evaluated for the sinus region. Vorticity is the curl of the velocity field and therefore captures rotational components of the blood flow shearing[31]. Regions of high vorticity along the axis perpendicular to the plane indicate both shear and rotation of the fluid particles. Out of plane vorticity in the z direction was computed using the following equation:

$$\omega_z = -\left(\frac{dV_x}{dy} - \frac{dV_y}{dx}\right) \quad (1)$$

Where ω_z is the vorticity component with units of s^{-1} ; V_x and V_y are the x and y components of the velocity vector with units of m/s. The x and y directions are axial and lateral respectively with the z direction being out of measurement plane. In addition to vector and vorticity fields, streak plots were generated.

Circulation Γ was also computed as per equation (2) below. Circulation around a closed boundary of contour Γ is defined as the line integral of the velocity along that contour and by Stokes theorem it is evaluated as the integral of the normal component of vorticity over the area S enclosed by that vorticity – which is the sinus for this study.

$$\Gamma = \oint \vec{u} \cdot d\vec{l} = \int_S \vec{\omega} \cdot d\vec{S} \quad (2)$$

Where Γ is the circulation (m^2/s), $\vec{u}$ and $\vec{\omega}$ are the velocity and vorticity vectors respectively.

Viscous shear stress field was evaluated as follows:

$$\tau = \mu \left(\frac{dV_x}{dy} + \frac{dV_y}{dx} \right) \quad (3)$$

Where τ is the shear stress in Pascal (Pa) and μ is the dynamic viscosity in $N.s/m^2$.

Leaflet kinematics tracking

Leaflet tip motion was tracked manually across 6 different takes (repeats) for each leaflet case. Radial distance from the aorta centerline was recorded and averaged across the six trials. Results were reported $\pm$ standard deviation. Velocity at the position of the leaflet tip when the leaflet is fully open was plotted versus time.

Results

The goal of the study is to visualize the flow structures within the aortic sinus when different calcification distributions are present on the aortic valve leaflets in order to understand how the spatiotemporal effects of the flow structures change and interact with the different leaflet kinematics secondary to the respective calcification intensities. The flow visualization results below are separated into qualitative and quantitative sections. The qualitative sections encompass streak plots generated (Video 1) along with still snapshots that highlight the most important points to investigate and track. The quantitative section includes instantaneous vector fields. The still frames are chosen based on the most important topological changes in the sinus flow patterns for each of the three cases.

High-speed imaging and leaflet kinematics

Video 2 shows the *en-face* views of the valve opening and closing throughout the cardiac cycle. Fig.1c and 1d show the valve configuration when it is closed and when it is open respectively. It is clear that the mildly calcified leaflet almost completely opens while the severely and moderately calcified leaflets do not. Leaflet tip tracking shown in Fig.1e displays the differences among the 3 leaflets measured from a common point near the centerline. As expected, the mildly calcified leaflet opens the most ($4.38 \pm 0.037\text{mm}$) followed by the moderately calcified ($2.23 \pm 0.07\text{mm}$) and the severely calcified ($2.16 \pm 0.02\text{mm}$). It is also noteworthy to highlight that the leaflet opening duration before deceleration begins also varies among the three leaflet cases. The severely calcified leaflet valve peak opening time is the slowest followed by the moderately calcified one and finally the mildly calcified leaflet. Data from PIV measurements captured 0.1s additional time of complete leaflet opening time prior to deceleration for the mildly calcified leaflet, and 0.05s additional time of total leaflet opening time prior to deceleration for the moderately calcified leaflet compared to the severely calcified leaflet.

Qualitative description of the sinus flow structures

Severely calcified leaflet sinus—Fig.2 shows snapshots of the streak plots in the sinus adjacent to the severely calcified leaflet from the moment the leaflet starts opening to its closure time. Given that the leaflet is severely calcified, its opening is impaired as shown in the *en-face* imaging in Fig.2b. The forward flow in fact is not captured in the snapshots as the flow accelerates downwards away from the sinus in these images. This leaves significant room for the recirculation regions to take over and propagate slowly into the sinus as peak systole is achieved (Fig.2e). As the valve starts closing, the adverse pressure gradient upon closure causes some vortices to form and collide (Fig.2g) until total valve closure (Fig.2h). Video 1 shows clearly the onset of the starting vortex and how quickly it is transported towards the sinotubular junction (STJ) before recirculating flow takes over. A main clockwise (CW) vortex takes place in the sinus, however at the interface between the CW vortex and the reverse flow, several counterclockwise (CCW) vortices form.

Moderately calcified leaflet sinus—Fig.3 shows snapshots of the streak plots in the sinus adjacent to the moderately calcified leaflet from the moment the leaflet starts opening to its closure time. Similar to the severely calcified leaflet, the moderately calcified leaflet

does not fully open either thus letting a large portion of the recirculatory flow occupy the sinus cavity. This recirculation disturbs the relatively stagnant flow in the sinus prior to opening of the valve and causes a dominant CW rotation in the whole area (Fig.3c). This aspect of the flow persists in a way that one CW vortex is clearly shown in (Fig.3d) and (Fig. 3e). The back flow as the valve remains open disturbs the singularity of the CW vortex and splits it into smaller CW and CCW vortices (Fig3.f–h). As the forward flow intensity decreases with deceleration and valve progressive closure, randomly oriented (CW and CCW) vortices form in the sinus.

Mildly calcified leaflet sinus—The streaks snapshots in the sinus adjacent to the mildly calcified leaflet from the moment the leaflet starts opening to its closure time are shown in Fig.4. At the beginning of valve opening, two successive CCW vortex form one that quickly dissipates into the forward flow (not shown) and another shown in (Fig.4a) that evolves downstream to hit the sinus ridge and curl back into the sinus cavity causing intense rotation (Fig.4c) that occupies the whole sinus area. As the valve stays open and forward flow gains momentum, a clear main CCW vortex and rotation keep occupying the sinus region (Fig.4d–i). When the valve starts closing, recirculatory flow caused by the adverse pressure gradient collide with the CCW vortex as shown in (Fig.4j) till the vortex splits leading to small pockets of vortices obvious at (Fig.4k) and (Fig.4l) until the valve closes completely.

Quantitative description of the sinus flow structures

Severely calcified leaflet sinus—Vorticity contours and velocity vector fields are shown in Fig.5 for the severely calcified leaflet sinus. The time points are those depicted in the qualitative descriptions above. The vector fields' direction mirrors the streaks of Fig.2. Time point $t = 0$ represents the onset of leaflet opening and $t = 0.40$ s represents the time when the leaflets are closed. The time points ranging from 0.12525 to 0.26875 s fall within the completely open leaflet configuration after acceleration and prior to deceleration. The maximal velocity in the sinus reached during acceleration is 0.11 ± 0.03 m/s, during peak systole 0.37 ± 0.05 m/s and during deceleration 0.28 ± 0.03 m/s. Peak vorticity magnitude in the sinus during acceleration is 92.2 ± 2.6 s⁻¹, during peak 152.7 ± 5.8 s⁻¹ and during deceleration 122.1 ± 4.1 s⁻¹.

Moderately calcified leaflet sinus—Fig.6 shows the velocity vectors and vorticity contours in the sinus adjacent to the moderately calcified leaflet. The time point at 0.17525s clearly shows the clockwise rotation that englobes the whole sinus region as noted in the qualitative results. At $t = 0.2230$ s, the disturbance of the reverse flow shows clearly and evolves to destroy the main CW vortex into smaller vortices. The duration through which the leaflet remains completely open (after acceleration and prior to deceleration) is captured in frames $t = 0.17525$ s to $t = 0.34775$ s. The maximal velocity in the sinus reached during acceleration is 0.11 ± 0.025 m/s, during peak systole 0.38 ± 0.04 m/s and during deceleration 0.26 ± 0.05 m/s. Peak vorticity magnitude in the sinus during acceleration is 104.4 ± 5.1 s⁻¹, during peak 191.4 ± 4.3 s⁻¹ and during deceleration 135.4 ± 5.9 s⁻¹.

Mildly calcified leaflet sinus—Vorticity contours and velocity vector fields are shown in Fig.7 for the mildly calcified leaflet sinus. As described qualitatively, the first vortex that

gets dissipated as the forward flow starts is clearly shown in frame at $t = 0.06125s$, the other vortex at $t = 0.085s$. The duration through which the leaflet remains completely open (after acceleration and prior to deceleration) is captured in frames $t = 0.1455s$ to $t = 0.4s$. The maximal velocity in the sinus reached during acceleration is 0.34 ± 0.03 m/s, during peak 0.62 ± 0.02 m/s and during deceleration 0.27 ± 0.01 m/s. Peak vorticity magnitude in the sinus during acceleration is 122.3 ± 6.5 s⁻¹, during peak 270.9 ± 8.2 s⁻¹ and during deceleration 154.1 ± 6.1 s⁻¹.

Circulation assessment in the sinuses

Evaluating the circulation in the sinus bound area, Fig.8a shows the alternating positive and negative circulation values in the severely calcified sinus with an average circulation of -1.30×10^{-4} m²/s. Fig.8b shows a prevalent negative circulation with the moderately calcified leaflet with only positive variations at the beginning and the end of the cycle. The circulation found is -9.47×10^{-4} m²/s. Fig.8c shows a prevalent positive circulation of total magnitude of 1.85×10^{-3} m²/s.

Shear stress probability distribution

Fig.8d and 8e show the probability density function in log scale of shear stress values along a sub-region near the different leaflets in the sinus during systole and diastole. During systole, the mildly calcified leaflet sinus case shows the most spread-out and higher ranges of shear stress probabilities and highest magnitudes going from $(-1.5$ to $+1.8$ Pa). While the shear stress range is similar for the moderately calcified leaflet sinus flow compared to the severely calcified one going from -1.0 Pa to 1.3 Pa, notably higher probabilities to develop higher shear stress magnitudes were found with the moderately calcified case compared to the severely calcified one. The severely calcified case has the highest shear stress probabilities around 0 Pa shear stress, followed by moderate then mild.

During diastole, the severely calcified leaflet case is characterized by the smallest span of shear stress. The moderately and mildly calcified cases show almost similar probability distribution range going from -0.8 Pa to 1.0 Pa with similar likelihoods on the positive shear stress distribution side and higher likelihoods for higher shear stress for the mild on the negative range side.

Discussion

The results reported above emphasize the sensitivity and complexity of the flow structures in the sinus when degrees of calcification at the leaflet (and consequently corresponding leaflet kinematics) vary between cases. The highly stenotic valve characterized by severe calcification impairs the opening function of the valve through narrowing the vena contracta. This directs the jet radially away from the sinus and the sinotubular junction edge allowing for the recirculation region and the backward flow to be the dominant features driving any flow in the sinus. Thus, the degree of calcification impacts valve operation and function leading to differences in sinus flow hemodynamics.

It is important to recognize the differences between the valve assessed in this study and a normal human aortic valve. This study constitutes a more idealized study given that native

human aortic valves are not symmetric with the three leaflets of different sizes and shapes. In addition, calcification in native versus bioprosthetic valves is different although bioprosthetic valves are prone to calcification just like native aortic valves. The process of calcification of native aortic valves is not well understood and that of bioprosthetic valves is even less well understood[24]. The root of the calcification process (initiation and progress) is still not identified yet. However, it is believed to be an active process that involves initiating factors, endothelial dysfunction, inflammatory responses, oxidative stress leading to remodeling of the valves, and mineralization, in addition to abnormal hemodynamics[32]. In both bioprosthetic and native aortic valves, calcification happens on the aortic side of the leaflet and similarities in calcium embedment in and on the leaflet exist between the two types[25]. It was also demonstrated that endothelial progenitor cells localize in the zona fibrosa of calcified native and bioprosthetic valves, indicating that cells of extra-valvular origin contribute to CAVD[34]. Newer porcine and pericardial bioprosthetic valves are treated with various agents that are found to be effective in decreasing calcification of the cusps, and the failure mode of these newer valves might not be the same as in first-generation bioprostheses[24]. Both valve types end up with compromised orifice area that lead to left ventricular outflow tract obstruction[33] thus an increased jet velocity and transvalvular pressure gradient. Despite changes in geometry and anatomy, the decrease in the flow orifice area in both types of valves leads to similar sinus flow patterns and hemodynamics given the same valve opening extent in addition to the same surrounding geometry, as patient-specific surrounding geometry may have an important impact[10].

Aortic sinus dynamics and impact of leaflet calcification

The starting vortex of the severely calcified leaflet resulting flow gets washed out once the valve opens and fails to entrap into the sinus. When the leaflet is severely calcified, one does not observe a single main vortex as the significant topological feature in the adjacent sinus. In fact, the overall motion onset in the sinus only starts at peak systole and prior to that, the flow is relatively stagnant. Overall, the sinus flow patterns are a combination of colliding small vortices resulting from the backward flow interaction with the relatively stagnant flow in the sinus. This is because the main jet flow is radially too far from the sinus (oriented downward), resulting in a failure of the starting vortex to be entrapped within the sinus as it misses the ridge (intersection of sinus and STJ)[35]. Instead the starting vortex simply “washes out” and is transported downstream. The clockwise vortices driven from the recirculation (separated flow surrounding the main jet) formed collide with the flow in the sinus leading to disturbances and to sign change in the resulting small vortices. This was also captured through the oscillatory circulation variations throughout the cardiac cycle. The impact of the CCW and CW is captured through the many positive and negative alternations (respectively) in the overall variations. This is illustrated in Fig.9a.

The moderately calcified leaflet opens further than the severely calcified however not enough to change the topology of the sinus flow completely albeit only a little earlier. Similarly to the severely calcified leaflet case discussed above, recirculation region effects are dominant in the sinus. The collision between the forward flow that is also oriented downwards given that the leaflet is not fully open and the recirculation region (shear force interaction between flows) cause a single prevalent clockwise vortex topology throughout

the sinus until vortex fission takes place with more backflow disturbance. Contrary to previous studies, the vortex does not keep growing until late systole[15]. As expected and because of the further valve opening, higher velocities, vorticities and shear stress spans and likelihoods are captured within the sinus compared to the severely calcified leaflet sinus along with less negative peaks at the point of fully open tip of the leaflet. Higher circulation (in absolute value) is also captured compared to the severely calcified leaflet case as discussed above. The majority of the variations were negative emphasizing the prevalence of the CW vortex and the higher flow activity in that sinus. Fig.9b captures these findings.

The mildly calcified leaflet almost fully opens allowing for the generation of the more well-known counterclockwise aortic sinus starting vortex and for its successful entrapment after reaching the sinus ridge. The vortex migrates to the middle part of the sinus and takes over the whole cavity space as long as the feeding shear layer continues to develop[7, 35]. For this case, the vortex is a large dominant and coherent structure easily identified. The larger orifice area allowing for the flow to pass with minimal resistance throughout systole yielded more velocity, vorticity and shear stress distributions and probabilities in the sinus compared to the highly stenotic valve cases. The circulation variations reinforce these observation through a highly positive evolution throughout the cardiac cycle with a magnitude that exceeded both the moderately and the severely calcified leaflet cases by almost one order of magnitude. These findings are summarized in Fig.9c.

The different degrees of calcification affect the jet properties including instabilities, entanglement and mixing rates. Knowing this information depends on the jet and vortex entrainment rate and based on studies by Tennekes and Lumley on entrainment rate of jets, it was highlighted how entrainment rates are controlled by the interaction of large scale structures and their movement into the surrounding fluid[36]. Understanding jet and vortex entrainment rate properties leads to understanding the rate of propagation of the interface between rotational and irrotational fluid[37]. With mild calcification, the vortex is characterized by a high vorticity core that entrains the surrounding fluid around to rotate. Moderately calcified leaflet clockwise sinus vortex being formed by a shear force interaction between forward and backward flows also entrains the surrounding fluid into a clockwise rotation that stays stable throughout almost all systole. In the severely calcified leaflet sinus, and despite not having a one-main-sinus topology, rotation in the sinus is also an induced motion similar to the other two cases.

Aortic sinus dynamics and correlation with calcific disease

Cell expression profiles studied by Butcher et al[38], suggest that valvular endothelial cells are protected from oxidative, inflammatory stress and calcification beginning and propagation by shear stress. Calcification disease process is thought to be initiated by several factors, low shear stress is one of the most important ones[39]. In this study, when the shear stress is quantified in each of the three cases, during systole and probably intuitively, the larger shear stresses and the larger probabilities to obtain high stress with the smaller probabilities for near-zero shear stresses were obtained when the leaflet has mild calcification (almost fully opens). The higher the calcification density on the leaflet, the

notably smaller the shear stress distribution becomes. This is tightly correlated with the flow propagation observed in the streaks (video 1 and figures) and discussed above.

In summary, in this study, we explicated complex spatiotemporal sinus hemodynamics as dictated by different calcification degrees: severe leaflet calcification, moderate leaflet calcification and mild leaflet calcification. The degree of calcification impairs valve opening and closing mechanisms and thus it directly affects the coherent vortex structure development in the sinus. We found that there exist drastic differences in the initiation and the evolution of aortic sinus vortex and vortices. Different jet and vortex entrainment characteristics led to developing an understanding regarding the nature of the vortex in the aortic sinus with different leaflet calcification types. In relation to aortic valve disease, high shear stress is found to be compromised as the degree of calcification increases specifically.

Limitations

A few limitations exist in this study. First, the use of a rigid sinus chamber is not physiological. Second, the use of 2D PIV does not allow us to capture the 3D effects that add to the complexity of sinus flow. Another limitation of the study is that the STJ diameter was maintained at a physiological level and this dimension significantly depends from one patient to another. Therefore the results of this study must be viewed as only related to the isolated effect of leaflet calcification alone for one specific sinus root configuration.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1: Calcification distribution on the (a) aortic and the (b) ventricular side of the valve. The severely calcified leaflet is shown on the bottom left side in the aortic view, the moderately calcified leaflet is on the top side and the mildly calcified one is on the right; (c) Closed configuration and (d) open configuration of the valve as the flow goes through from high-speed imaging acquisition. (e) Leaflet tip position from a common point above the centerline as a function of normalized time (0 = opening; and 1 = closing).

Figure 2:
Streak plots for qualitative sinus flow description adjacent to the severely calcified leaflet. Flow is from left to right. STJ denotes sinotubular junction. The white pattern shown represents the fully open leaflet.

Figure 3:
Streak plots for qualitative sinus flow description adjacent to the moderately calcified leaflet. Flow is from left to right. The white pattern shown represents the fully open leaflet.

Figure 4:
Streak plots for qualitative sinus flow description adjacent to the mildly calcified leaflet. Flow is from left to right. STJ denotes sinotubular junction. The white pattern shown represents the fully open leaflet.

Figure 5:

Velocity vectors and vorticity contours within the sinus region adjacent to the severely calcified leaflet at specific points throughout the open leaflet phase.

*Length ratio of sinus vectors to jet flow vectors is 2:1 with the jet vectors being half of the unstarred ones.

*Length ratio of sinus vectors compared to the other unstarred sinus figures is 2:1.

Figure 6:
Velocity vectors and vorticity contours within the sinus region adjacent to the moderately calcified leaflet at specific points throughout the open leaflet phase.

*Length ratio of sinus vectors compared to the other sinus figures is 2:1.

Figure 7:

Velocity vectors and vorticity contours within the sinus region adjacent to the mildly calcified leaflet at specific points throughout the open leaflet phase.

*Length ratio of sinus vectors to jet flow vectors is 2:1 and compared to the unstarred sinus schematics, the vector length is 2:1 as well.

**Length ratio of sinus vectors to jet flow vectors is 1:1.

Figure 8:

Variations in circulation versus time for the 3 different sinus cases with (a) severe leaflet calcification, (b) moderate leaflet calcification and (c) mild leaflet calcification along with Probability density function in log scale of varying shear stress distribution values along a sub-region near the different leaflets in the sinus during (a) systole and (b) diastole.

Figure 9:
Cartoon of sinus flow pattern differences at peak systole for the (a) severely, (b) moderately and (c) mildly calcified valve.