Keynote Paper

MODEL STUDIES ON THE CLOSURE MECHANISMS OF HEART VALVES

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Abstract Until two decades ago there was a lot of controversy regarding the roles of different fluid mechanical factors in the closure process of the heart valves. The main question was how regurgitation or back flow through a closing valve is minimized. Vortices behind valve leaflets were once believed to play an important role in minimizing back flow by causing an early partial valve closure. Later experimental studies demonstrated that an adverse pressure gradient associated with flow deceleration can accomplish efficient valve closure, even in absence of vortices, and confirmed the possibility of valve closure without back flow. It was also recognized that a closed or nearly closed valve could undergo backward movement along with the fluid in its vicinity, giving rise to an apparent back flow or pseudo-regurgitation. Recent model experiments have revealed the occurring of pseudo-regurgitation during the valve closure process, i.e., before valve closure. Pseudo-regurgitation should be regarded as a characteristic of the natural heart valves. For a membrane valve model, pseudo-regurgitation was estimated to be approximately 25% of the cube of the valve diameter.

Keywords:Back flow, adverse pressure gradient, flow deceleration, pseudo-regurgitation

INTRODUCTION

The mechanisms of closure of the natural heart valves have been of great interest to many investigators. Although it was widely believed that the heart valves open and close passively under the forces exerted on the valve cusps (or leaflets) by the blood flow around them, until two decades ago there was a lot of controversy regarding the roles of different fluid mechanical factors in the closure process. The main question was how regurgitation or back flow through a closing valve is minimized. Although early experimental studies [Henderson and Johnson, 1912] indicated the possibility of valve closure under flow deceleration without any regurgitation, the proposed mechanism being breaking of the jet and lateral inrush of fluid, the importance of these findings was not fully appreciated by many of the later investigators. A partial valve closure before onset of back flow was widely assumed to be the basic mechanism behind an efficient valve closure. The controversy was mainly regarding the mechanism of this early partial valve closure. Some investigators [Bellhouse and Bellhouse, 1968, 1969, Bellhouse, 1972] attributed the crucial role of partial valve closure to strong vortices generated behind valve leaflets during forward flow. From their model experiments they concluded that without vortices the heart valves would close after considerable back flow or regurgitation. Some other investigators [Talukder et al., 1977, Reul et al., 1981] regarded the adverse pressure gradient associated with decelerated flow to play the main role in efficient valve closure without appreciable back flow. Their model experiments demonstrated that valve movement remains largely unaffected in absence of strong vortices behind the leaflets, provided the proper pulsatility of the flow is maintained. They concluded that adverse pressure gradient causes flow deceleration and valve closure simultaneously, any back flow recorded being due mainly to backward movement of a closed or nearly closed valve along with the surrounding fluid and thus indicating apparent back flow or pseudo-regurgitation.

It became clear that, despite the morphological differences between the heart valves, their closure mechanisms are the same. However, only recent investigations [Talukder et al., 1995, 1996, 1998] demonstrated clearly the presence of forward velocity through a closing valve model and revealed the possibility of pseudo-regurgitation during the valve closure period, i.e., when a closing valve is still partially open. The possibility of real regurgitation, i.e., backward flow relative to a receding and diminishing valve opening was found to be very slim. Pseudo-regurgitation should thus be regarded as an important characteristic of the flow associated with the closure of natural heart valves.

The present paper reviews some of the earlier and recent investigations and discusses ongoing effort to determine the volume of pseudo-regurgitation.

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MECHANISMS OF CLOSURE OF THE AORTIC VALVE

The formation of strong vortices behind the aortic valve cusps during systole [Bellhouse and Bellhouse, 1968] was based on the assumption that the cusps on opening move into the sinuses to allow a part of the jet through the aortic valve to impinge on the sinus wall [Bellhouse and Talbot, 1969], as illustrated in Fig. 1. This hypothesis was untenable since it was recognized that the morphology and flexibility of the aortic sinuses should cause the commissures to move outward under rising aortic pressure and make the valve aperture nearly triangular, implying that the cusps stay away from the sinuses. This phenomenon may be explained as follows.

When the aortic valve is closed, forces with components towards the center are applied by the cusps on the aortic wall which must have the sinuses so that the wall tension at the commissures can have radial (outward) components to balance the inward acting forces [Swanson, 1975], as illustrated in Fig. 2a. When the aortic valve opens, the inward forces vanish while the outward forces not only persist but also increase with rising aortic pressure and move the commissures outward (Fig. 2b), as confirmed by other investigators [Brewer et al., 1976]. Due to this the free edges of the cusps must be stretched giving rise to an almost triangular valve orifice (Fig. 2c). It was also observed in an animal experiment [Padula et al., 1968] that in a beating heart, in which the blood was replaced for a short time with a transparent blood substitute, the aortic valve aperture becomes almost triangular shortly after valve opening. The triangular shape of the normal aortic valve orifice was also confirmed by other investigators [Stein, 1971, Robel, 1972].

There would be no necessity for any special mechanism for early partial valve closure if the dynamic pressure-flow characteristics of the pulsatile aortic flow were taken into consideration. Measurement of transvalbular pressure gradient [Spencer and Greiss, 1962, Driscol and Eckstein, 1964] revealed adverse gradient during flow deceleration. The pressure forces should be enough to cause efficient valve closure.

Another important observation was the occurring of a very short phase of negative flow at the end of flow deceleration. It remained to be examined whether this implied real back flow through a diminishing valve

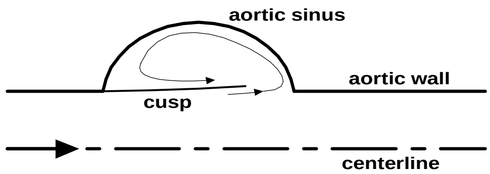

Fig. 1 Mechanism of systolic vortex generation in an aortic sinus [Bellhouse and Talbot, 1969]

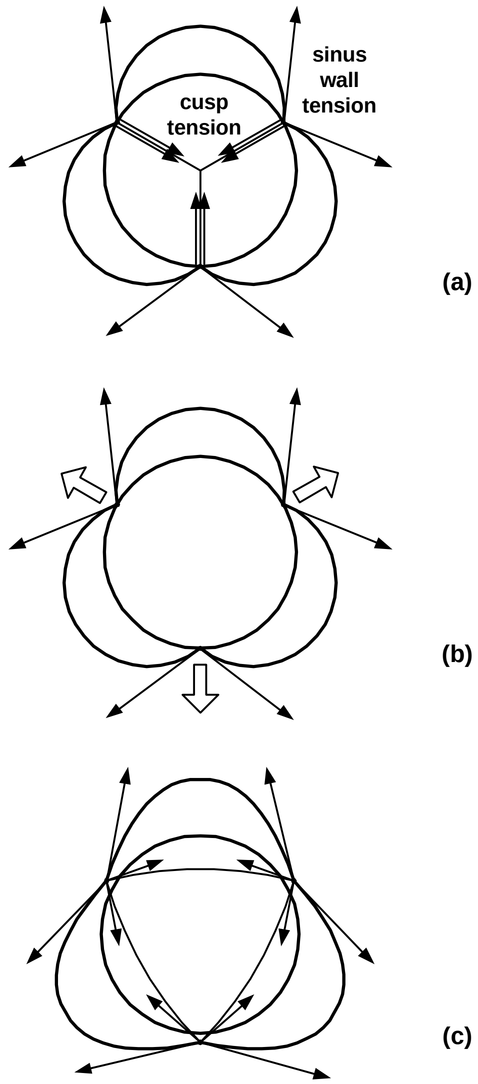

Fig. 2 Morphology and function of the aortic sinuses. (a): Balance of wall forces and cusp forces in the diastole [Swanson, 1975], (b): Force unbalance in early systole, (c): Stretched cusps giving rise to a triangular valve opening

aperture or just backward displacement of a closed valve along with the adjacent fluid.

In order to investigate if valve motion is affected by the absence of strong vortices in the aortic sinuses and to detect back flow through a closing aortic valve, in vitro experiments were carried out [Talukder et al., 1977] using flexible models of the aortic tract and aortic valve mounted in a mock circulation system. An aqueous glycerin solution (40% glycerin) was used as model fluid in which neutrally buoyant amberlite beads were suspended for flow visualization. Hydrogen bubbles were generated close (downstream) to the valve, to detect back flow, if any. Fig. 3 illustrates the aortic model and the relative position of the hydrogen

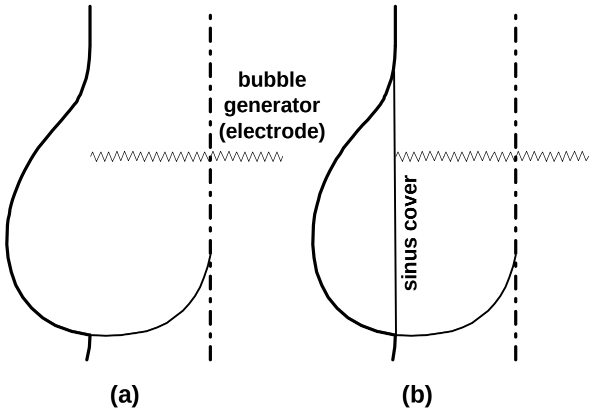

Fig. 3 Position of hydrogen bubble generator installed inside the aortic model with open (a) and covered (b) sinuses

bubble generator. A second set of observation was performed with the sinuses covered as illustrated in Fig. 3b.

The flow visualization studies showed hardly any vortex formation in the sinuses during systole as illustrated in Fig. 4a. Vortices in the sinuses appeared after valve closure (Fig. 4b) due to cessation of backward aortic flow. These vortices, which rotate opposite to the speculated [Bellhouse and Bellhouse, 1968] systolic vortices (Fig. 1), however, play no role in the valve closure process. No back flow through the valve could be detected (no hydrogen bubble came to the ventricular side). Therefore the small amount of back flow at the end of systole, as also found by other investigators [Spencer and Greiss, 1962], was thought to be mainly due to backward movement of a closed or nearly closed valve until its complete stoppage and regarded as pseudo-regurgitation.

With the sinuses covered, no significant difference was found in valve motion nor was there any indication of back flow through the valve. It was concluded that sinus vortices play no significant role in valve closure and that adverse pressure gradient should be enough to

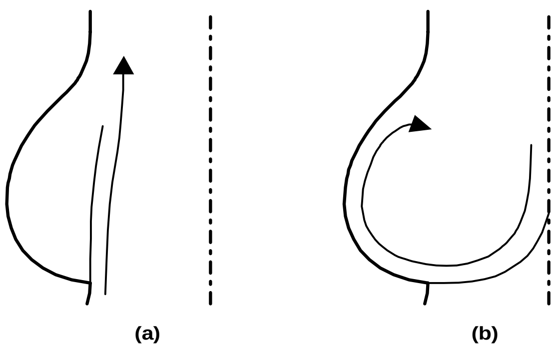

Fig. 4 Schematics of (a) systolic and (b) diastolic flow near the aortic valve [Reul and Talukder, 1979]

accomplish efficient valve closure without appreciable regurgitation. This important finding was later confirmed by other investigators [Steenhoven et al., 1981]

MECHANISMS OF CLOSURE OF THE MITRAL VALVE

Although formation of strong vortices behind the mitral valve leaflets was almost obvious, the role of the vortices in the subsequent valve closure process remained controversial. Model experiments designed to demonstrate that without strong ventricular vortices there would be considerable back flow though the mitral valve before its closure [Bellhouse, 1972] were considered misleading [Reul et al., 1981], since in those experiments, while eliminating the vortices the strong deceleration of mitral flow and the associated adverse pressure gradient during forward flow were also eliminated.

Like the reversal of pressure gradient across the aortic valve during systole [Spencer and Greiss, 1962], there is reversal of pressure gradient across the mitral valve at mid diastole and late in diastole [Brockman, 1966]. These adverse pressure gradients seemed to be related to mid diastolic flow deceleration and partial valve closure, and late diastolic flow deceleration and valve closure, respectively. It was clearly demonstrated [Laniado et al., 1973] that at the beginning of the late diastolic mitral flow deceleration, the mitral valve was wide open and that valve closure took place during flow deceleration under a rising vetricular pressure, i.e., after the onset of ventricle contraction. These findings indicated that adverse pressure gradient associated with flow deceleration should play a significant role in mitral valve closure too.

In order to investigate the role of ventricular vortices in the closure of the mitral valve, model experiments were performed [Reul et al., 1981], using a porcine mitral valve mounted inside a polyurethane ventricular model in the same mock circulation system as used for aortic valve studies [Talukder et. al., 1977]. Two sets of flow visualization and valve motion studies were carried out under the same physiological flow pulsation, with and without the ventricle model. The ventricle model was mounted inside a relatively large rigid chamber as illustrated in Fig. 5a. When the ventricle model was removed for the second set of observations, the rigid chamber served as a large ventricle, as illustrated in Fig. 5b. Vortices were generated inside the ventricle model early in diastole (Fig. 5a) and might play some role in the mid diastolic partial valve closure. However, the valve opened again after mid diastole (atrial contraction) and the vortices seemed to die out or get weaker before the actual valve closure began late in diastole. When the ventricle model was absent, there was no vortex generation in the vicinity of the valve

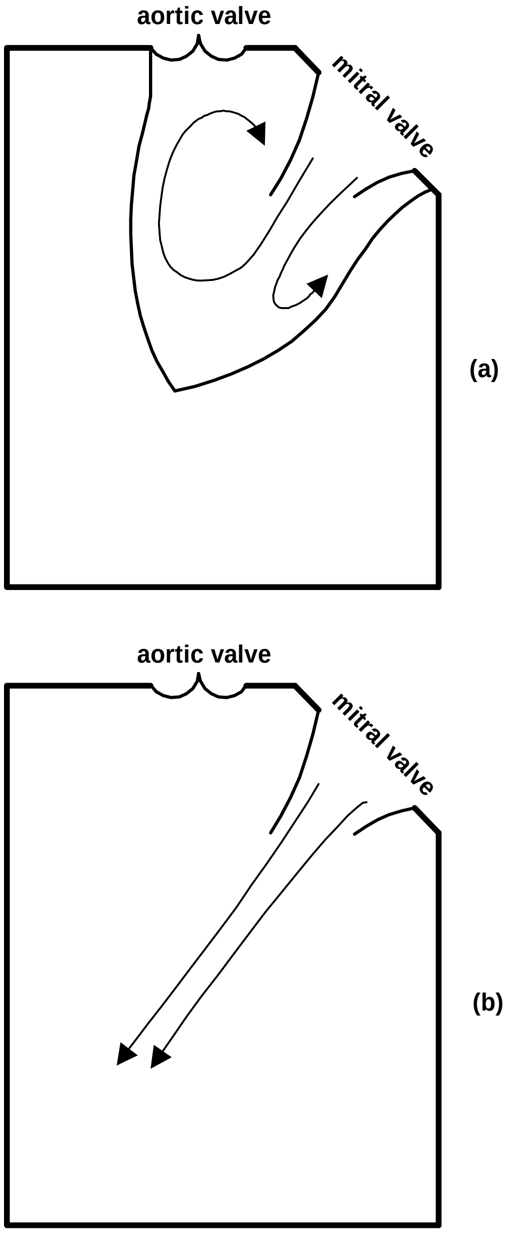

Fig. 5 Schematics of early diastolic mitral flow into (a) a ventricle model and (b) a larger chamber

(Fig.5b). Comparison of valve motions with and without ventricle showed no significant difference between the two cases. Therefore it was concluded that the mitral valve closure is governed primarily by the adverse pressure gradient associated with the late diastolic flow deceleration. This confirmed the findings by other investigators [Lee and Talbot, 1979].

PSEUDO-REGURGITATION DURING HEART VALVE CLOSURE

As explained in the foregoing two sections the primary mechanism of closure for both the aortic and the mitral valve is basically the same, viz., adverse pressure gradient associated with flow deceleration. During deceleration of flow through a valve, valve aperture and flow velocity at the valve aperture were thought to be coupled and it was speculated that the two quantities might vanish at the same time [Talukder et al., 1977]. This, however, remained to be verified. Although the instant of valve closure was shown to coincide with the instant of zero flow measured at the valve annulus [Laniado et al., 1973], flow velocities at the valve orifice and relative to the free edges of the valve leaflets were not known. In case the valve remains partially open at the instant of zero relative flow through the diminishing aperture, the subsequent relative flow must be investigated before concluding whether or not regurgitation occurs before complete closure of a valve. Unless there is back flow relative to the valve aperture, any back flow recorded at the valve annulus is only apparent back flow or pseudo-regurgitation. While backward movement of a closed valve was easily recognized as a possible origin of pseudo-regurgitation, the possibility of pseudo-regurgitation in the last phase of valve closure (before the valve aperture vanishes) could also be imagined [Talukder et al., 1977]. Recent investigations showed clear indication of pseudo-regurgitation before valve closure [Talukder and Komerath, 1995, 1996, Talukder and Funk, 1998], as described below.

To explore the possibility of back flow in the last stage of valve closure, the pulsatile flow phenomena and accompanying valve movement were investigated using an atrio-ventricular model as illustrated in Fig. 6 and applying video image analysis. The experimental setup and methods were described in earlier papers [Talukder and Komerath, 1995, Talukder and Komerath, 1998].

The observations were restricted to the deceleration phase till the threads representing valve cusps started prolapsing into the tube in a swift whipping motion.

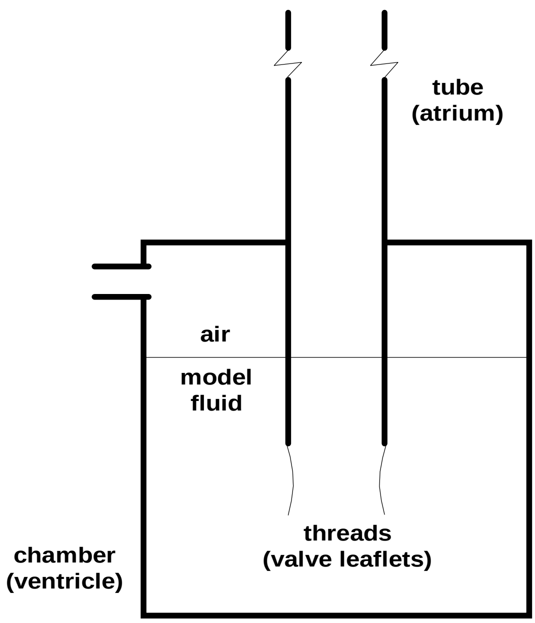

Fig. 6 Atrio-ventricular model

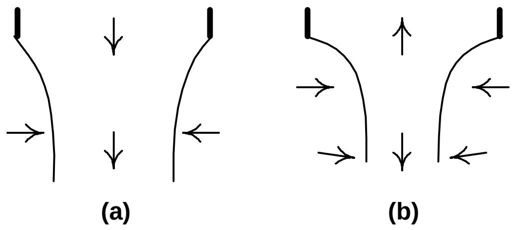

Fig. 7 Schematics of flow velocity vectors and valve motion during flow deceleration. (a): early stage (b): later stage (breaking of the jet)

The flow velocity vectors in the vicinity of the moving threads clearly showed simultaneous occurring of inward motion of the fluid with the threads and forward flow in the region between the thread tips, as illustrated in Fig. 7a, followed by breaking of the jet [Henderson and Johnson, 1912], as illustrated in Fig. 7b.

It was also observed that the fluid column in the tube reaches its lowest level and move upward (backward) before the threads start prolapsing [Talukder and Komerath, 1996]. This back flow into the tube (through valve annulus) starts at a time point when the flow in the region between the thread tips (valve aperture) is still forward and thus clearly indicates pseudo-regurgitation occurring during valve closure, i.e., before vanishing of valve aperture. It was called Pseudo-regurgitation-B [Talukder and Funk, 1998], to distinguish it from the possible pseudo-regurgitation after valve closure (Pseudo-regurgitation-A due to backward displacement of a closed valve). Pseudo-regurgitation-B is due to inward and backward movement of fluid and valve leaflet around a narrowing zone of forward flow or forward flow relative to valve aperture, as explained below.

Since the orientation of the free ends of the threads should indicate the direction of the relative flow, it appeared that there was no relative back flow even when the flow near the thread tips reversed, implying that the thread tips were moving backward faster than the adjacent fluid. There was no indication of real regurgitation (backward relative flow) even in a worse case in which the chamber pressure was suddenly raised when the fluid was practically at rest [Talukder and Funk, 1998]. Although there was backward flow in the vicinity of the threads, suspended particles near a thread tip were found to have a forward relative motion as also indicated by the orientation of the free end of the thread.

To verify the above observations, a cylindrical membrane was used as valve model. The collapsing movement of the membrane was found to be similar to the whipping movement of the threads. The occurring of Pseudo-regurgitation-B was confirmed, as illustrated in Fig. 8.

Volume of Pseudo-regurgitation

The volume of pseudo-regurgitation can be determined from the distance h_r through which the

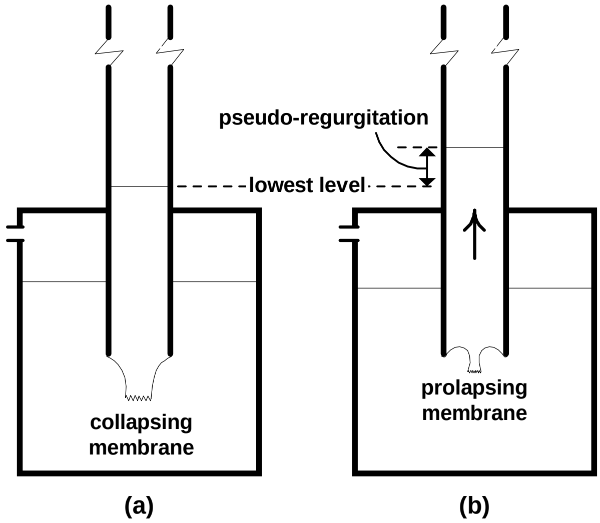

Fig. 8 Origination of Pseudo-regurgitation-B

column reverses until the collapsed membrane starts prolapsing into the tube (or a closed valve stops). With a tube diameter d_t the volume of pseudo-regurgitation V_{psr} is given by

$$V_{psr} = \pi d_t^2 h_r/4. \quad (1)$$

Since pseudo-regurgitation should mainly depend on the valve size, a normalized pseudo-regurgitation (NPR) may be defined as

$$NPR = V_{psr} / d_v^3 \quad (2)$$

where d_v is a characteristic valve diameter. From Eqs. (1) and (2) follows

$$NPR = (\pi /4)(d_t^2/d_v^2)(h_r/d_v). \quad (3)$$

For the thread model, V_{psr} was estimated as $0.5d_t^3$ [Talukder and Komerath, 1995]. With d_t/d_v=0.9, the corresponding NPR would be approximately 0.36. For the membrane model, NPR was estimated as 0.25. A comparable value of NPR is obtained for the average aortic back flow volume of 0.5 cm^3 measured in mongrel dogs weighing 11-20 kg [Spencer and Greiss, 1962] (assuming an average d_v of 1.25 cm).

CONCLUSIONS

From the results of the earlier and recent experimental studies discussed above, it may be concluded that there is hardly any scope of regurgitation during heart valve closure and that pseudo-regurgitation is a characteristic of heart valves.

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