



Understanding the Anterior Leaflet of the Mitral Valve

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Editorial

Since the first prosthetic valve became commercially available in 1961, debate has continued over the best way to manage the dysfunctional native mitral valve. Transcatheter mitral valve insertions (TMVI) have introduced new controversies which can be better understood by reviewing the old.

In 1991, Waggoner et al. [1] published the outcomes of 7 patients who underwent bioprosthetic mitral valve insertion over a 5 year period, without resection or any modification of the anterior leaflet of the mitral valve (AML) and who had post valve insertion echocardiography. Six of the seven patients died within 2 months of surgery and a retrospective review of the post operative echocardiography showed left ventricular outflow tract obstruction in all 7 patients. Since then, there have been very few reports of full retention of the mitral valve at the time of open heart bioprosthetic valve insertion, but full retention is now a requirement for the catheter deployed mitral prostheses. Therefore, it is necessary to look at the rationale for performing the procedures reported by Waggoner et al and understand why the outcomes were so poor, particularly after Cooley et al. [2] had reported successful outcomes with mechanical valve insertion.

Anterior Mitral Leaflet Function

The rationale for retention of the native mitral valve at the time of prosthetic valve insertion is based on the physiological function of the AML. The anterior leaflet contributes to left ventricular (LV) function by reducing LV work and improving efficiency. It has long been known that the retention of the annulo-papillary connection through the posterior leaflet [3,4] improves outcomes for patients undergoing mitral valve replacement for mitral incompetence and that the clinical extent of this benefit is inversely related to pre-operative left ventricular function. However, AML support of LV function is greater than that of the posterior leaflet [5] for the following reasons.

Intraventricular baffle

Magnetic Resonance Imaging (MRI) for assessment of blood flow velocities through the ventricle during diastole has shown that the AML acts as a mid ventricular baffle which provides laminar blood flow characteristics [6]. Following excision of the AML associated with mitral mechanical valve insertion, MRI shows turbulence and stasis of blood within the ventricle during diastole (Figure 1). With the AML baffle, blood flows through the inlet part of the ventricle, around the apex towards the outflow tract, and this flow is maintained throughout diastole such that systole provides acceleration of that diastolic flow. Without the AML baffle, greater LV work is required to re-initiate blood flow from a situation of turbulence and stasis.

AML/papillary muscle apparatus

The mitral valve closes immediately after the onset of systole. The mitral leaflets move from their diastolic intraventricular positions to the plane of the mitral annulus pulling the papillary muscles and left ventricular wall towards the mitral and aortic valves (Figure 2). This dynamic action “turbo-charges” the LV. A diastolic passive action of annulo-papillary continuity prevents excessive LV dilatation which can be achieved with the posterior leaflet connection alone. The height of the AML (distance from annulus to free edge chordal attachments) is considerably greater than that of the posterior leaflet. This has 2 important implications. (a) The excursion of the papillary muscle movement due to AML support is substantially greater and the turbo-charging effect is proportionally greater [5]. (b) Fixing the free edge of the AML to the annulus will impair the diastolic excursion of the papillary muscles away from the mitral annulus, with impaired diastolic filling of the LV.

Accordingly, the rationale for maintenance of the AML at the time of mitral prosthetic insertion

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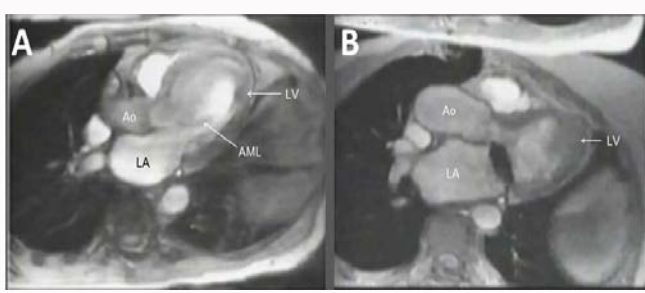


Figure 1: MRIs showing blood flow into the ventricle during early diastole. A: Normal flow in an adult, showing the AML directing flow posteriorly, away from the interventricular septum and around the apex. This is followed by continuing flow towards the outflow tract up to the onset of systole. B: Flow through a bileaflet mechanical prosthetic valve, showing flow into all parts of the ventricle towards the apex. This is followed by turbulence and stasis within the ventricle. (With permission from Professor Joachim Laas, Bad Bevensen, Germany).
 AML: Anterior Mitral Leaflet; Ao: Aorta; LA: Left Atrium; LV: Left Ventricle

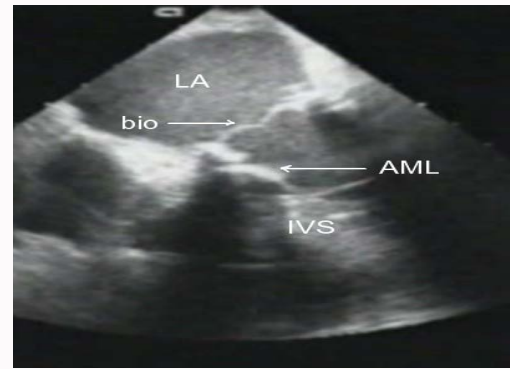


Figure 3: Left ventricular outflow tract obstruction due to systolic anterior motion of the anterior mitral leaflet (AML) following insertion of a mitral bioprosthesis (bio). The AML is seen abutting the interventricular septum (IVS) while the bioprosthesis valve is closed during systole.

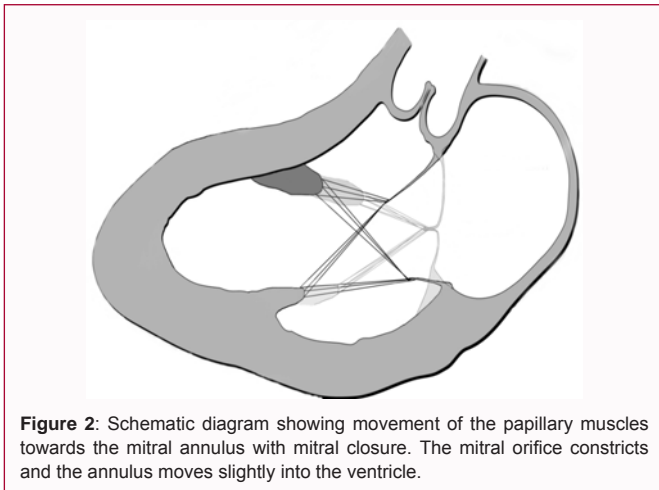


Figure 2: Schematic diagram showing movement of the papillary muscles towards the mitral annulus with mitral closure. The mitral orifice constricts and the annulus moves slightly into the ventricle.

is the improved ventricular function due to both turbo-charging and baffle effects. It is of note that the Washington University experience was with selected patients with poor LV function, on anti-failure therapy and most with ischaemic mitral incompetence. These patients are considered to be at high risk with conventional valve surgery and are the ones to derive greatest benefit from preservation of LV function by any means possible. It is inappropriate to attribute poor outcomes of conventional mitral valve replacement to an acute increase in after load when it is known that the method of mitral replacement impairs LV function.

The Causes of LV Outflow Tract Obstruction (LVOTO)

As the LVOTO is due to systolic anterior motion (SAM) of the AML (Figure 3), it would be intuitive to use a small mitral bioprosthesis rather than a large one that would likely position the base of the AML closer to the outflow tract. However, the use of a small bioprosthesis appeared to increase the risk of LVOTO in a sheep model reported in 2011 [7]. Although this study was underpowered to achieve significance for the various risk factors, it did demonstrate that reducing the antero-posterior (A-P) diameter of the prosthesis by cinching the prosthesis abolished LVOTO when present, thus confirming the intuitive desire to keep the base of the AML away from the outflow tract. However, small bioprosthesis have short distances between stent posts and this was the likely reason for

a trend towards a higher risk of LVOTO with smaller prostheses. The distance between the stent posts should exceed the width of the AML to allow the AML to move freely away from the outflow tract at the onset of systole. All the benefit of the smaller A-P diameter in a small valve is lost by having a stent posts under each side of the AML. Therefore, a bioprosthesis with a large anterior leaflet (wide anterior stent posts) would need to be inserted with the stent posts at the native commissures. The A-P diameter of the valve annulus should be reduced (elliptical shape) and the valve leaflets should be asymmetrical with a large leaflet anteriorly and 2 small equal sized leaflets posteriorly. This design was registered with the US patent office in 2011 by Alain Carpentier and Edwards Life Sciences (Patent no. US 8,034,104 B2). It has not been produced commercially due to the limited knowledge regarding the extent of benefit of AML retention. Accepting a documented hazard of LVOTO for the unproven benefit of AML retention would have little commercial appeal.

AML Management with Trans-catheter Mitral Valve Implantation (TMVI)

There are 2 principle methods of AML management at the time of TMVI, based on the design of the prostheses used.

1. AML entrapment by the prosthetic stent apparatus provides fixation of the device on the ventricular side and avoids the hazard of LVOTO. As stated above, reefing the AML at the level of the mitral annulus will inhibit diastolic filling of the LV. The first human implant of such a device was in an 86 year old man with severe LV dysfunction and severe mitral incompetence. The implantation was successful with minimal residual mitral incompetence but the patient died 3 days later from multi-organ failure. It is likely that the valve had an adverse effect on LV function and this may have simply been due to immobilization of the AML and restriction of diastolic filling. No devices that use AML entrapment have yet achieved desirable clinical outcomes in human trials [8]. Use of these devices in humans is usually based on compassionate grounds where no other form of therapy can be reasonably offered and the patients’ estimated survival without treatment is poor. These patients have poor LV function and are sensitive to interventions with any adverse effect on LV function. These valves are unlikely to be trialed in patients with good LV function, who might easily tolerate relatively minor disturbances in LV function. Indeed, many animal studies have been performed to confirm the function of these valves when implanted into normal animal hearts.

Valves that capture only the portions of the AML adjacent to the commissures, along with fixation to the posterior leaflet may achieve the benefit of retention of the baffle effect of the AML without diastolic restriction. The restriction of the para-commissural parts of the AML may be sufficient to prevent SAM.

2. LV apical tethering of the valve prosthesis effectively fixes the valve on the ventricular side. As with prostheses that use AML entrapment, fixation on the atrial side is achieved with a flange. Apical tethering does not involve attachment to the anterior leaflet but the nitinol stent supporting the valve extends into the LV cavity and restricts AML motion. The AML cannot reach the plane of the mitral annulus during systole and so the turbo-charge effect is largely lost but the baffle effect is maintained and diastolic LV filling is not restricted. There is a risk of LVOTO which makes accurate sizing of the valve very important. Undersizing the valve will likely result in a paravalvular leak and oversizing increases the risk of LVOTO by the AML due to SAM. If LVOTO is recognized before the apical tethering fixation is completed, the prosthesis can be easily retrieved or repositioned. There are options for management of LVOTO after completion of the implantation. Stenting of the LVOT can substantially reduce the gradient [9]. In the sheep model of LVOTO following open heart implantation of bioprosthesis, volume loading tended to increase the SAM of the AML which is the opposite of that seen following mitral valve repair. Where volume loading after repair increases the cross-sectional area of the LVOT and reduces SAM, it appears that the valve prosthesis causes a relatively fixed cross sectional area of the outflow tract so that volume loading simply increases the cardiac output and blood velocity through the outflow tract, thereby increasing the risk of SAM. Accordingly, it may be better to reduce cardiac output with ventricular pacing and or beta blocker therapy. Urgent alcohol septal ablation has been performed with immediate resolution of the SAM [10]. This was due to the immediate paralysis of the sub-aortic septum with onset of paradoxical motion. So far, the device that uses apical tethering has been the most successful in human use, presumably due to the preservation of LV function in patients who are poor candidates for any other form of therapy.

These important advances cannot be achieved without information from both human and animal experiences. Unfortunately, a lot of information exists in the industry sector, which is not available to the broader community. When it is considered that a company with a

single device not yet tested in humans can be worth US\$450M, the risks of industrial espionage are considerable. Even the information gained within various industry sectors is piecemeal and not shared. Many theories might be proven incorrect as more information is gained. There is still much to learn with plenty of room for new ideas.

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