Why does the interventricular septum "resist" the scalpel?

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Abstract

Abstract The posterior rupture of the left ventricle has been a complication recognized since the beginning of mitral valve surgery and, despite advances in cardiac surgery, the outcome of the rupture of the left ventricle has remained tragic. During mitral valve surgery, care must be taken not to traumatize the free wall of the left ventricle. On the other side, septal Myectomy is performed on hypertrophied septums to address the left ventricular outflow tract obstruction. In this article I have presented a theory that could give a part of the explanation of the resistance of the interventricular septum of surgical trauma unlike the ventricular wall.

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Introduction

Every time I perform the interventricular septum (In-S) Myectomy during a left ventricular outflow tract obstruction (LVOTO) correction operation, I always think: "Why is the In-S so resistant to the surgeon's scalpel compared to the rest of the left ventricle wall?"

Discussion

Two of the leading concepts of mural ventricular architecture are the unique myocardial band and the myocardial mesh model[(1)]. Torrent-Guasp suggested the presence of the ventricle walls act as a continuous myocardial bar, which runs from the root of the pulmonary trunk to the root of the aorta[(1)]. The sculpted muscle fascia of the ventricular mass was maintained to form two rings (basal and apical), which surround the two ventricular cavities. Presumably there are split planes or sliding surfaces between these segments, which allow parts of the band to move over each other[(1)]. As we know, aortic dissection occurs when an injury to the innermost layer of the aorta allows blood to flow between the layers of the aortic wall, forcing the layers apart. The point of entry and return of dissection are usually distant. If the blood-filled false lumen breaks through an outward weakness, it creates a spill point. This mechanism occurs due to the layered nature of the aorta. Thus, if the concept of the unique myocardial band were true, logically in cases of a ruptured left ventricle, the entry point and the exit point should be spaced apart. Instead the classification of a ventricular rupture between the mitral ring and papillary muscles during the replacement of the mitral valve shows that the rupture is close to the initial point of trauma. In one case report we previously presented[(8)], it can be verified that the ventricle rupture entry and exit points are positioned face to face (figura2). I think this observation could favour the myocardial mesh model concept in which the myocardium is represented as a continuous 3D meshwork that has no large-scale subdivision[(1)].

The shape of the left ventricle approximates to that of a cone with the right ventricle almost seeming to embrace it. Consequently, the septal component of the ventricular wall is curved. Normally, the left ventricular free wall is thicker at the cardiac base and it gradually becomes thinner moving towards the apex. At the very tip of the ventricle, the musculature is only 1–2 mm thick, even in hypertrophied ventricles[(2)]. On the other hand, if the concept of a unique band were realistic, then the exit point of the rupture of the left ventricle, following a lesion of the free wall under the mitral valve, should appear at the level of the ventricular apex where it is thinnest. In fact for many years the classification of left ventricular rupture

has been limited to levels above the papillary muscles[3]. It might be thought that the thickness of the left ventricle could be a protective factor from ventricular rupture, but in reality, the rupture of the left ventricle following MVR has been described at the thickened part of the free wall of the left ventricle above the papillary muscles.

However, whatever the composition of the wall of the ventricles, the complex architecture, that connects the ventricular wall with the fibrous skeleton of the heart through the papillary muscles, the cords and the leaflets of the valves, support the ventricle wall during the cardiac cycle. It has been demonstrated that the loss of annulo-ventricular continuity still leads to progressive left ventricular dilatation with eventual decline of left ventricular function in the long-term[4]. The role of the secondary (strut) chordae was determined to maintain the left ventricular geometry and function. Therefore, the secondary chordae are placed centrally within the subvalvular apparatus to ensure the force transmission and geometric stability of the left ventricle[5]. Hansen demonstrated that transection of chordae to the anterior mitral leaflet (AML) reduced the left ventricular function to a greater degree compared to the transection of chordae to the posterior mitral leaflet (PML)[6].

In fact, as we have learned from mitral surgery, the posterior left ventricular (LV) wall is exposed to iatrogenic trauma. Thus, during mitral valve repair or replacement, the surgeon pays meticulous attention to the possible occurrence of ventricular ruptures that mostly involve the posterior left ventricular wall. Particularly, the mechanical injury levied at the sites of papillary muscles (PM), especially during mitral valve replacement (MVR) performed under ischaemic arrest with a flaccid heart, can be responsible for left ventricular rupture[7].

Nevertheless, the myomectomy of the In-S, which is itself a myocardial trauma, has been used for 50 years with no, or few, descriptions of surgery-induced ruptures or inter-ventricular defects.

Left ventricular rupture after MVR is classified into three types, according to its variable location between the atrioventricular groove and the base of the PM[3]. We introduced the concept of a fourth type of rupture (type IV), that could be located at the site between the base of the PM and the left ventricular apex which can be the consequence of the ventricular access required to apply new procedures as we pointed out in one of our previous works[8]. Depending on the time of the tear appearance post surgery, left ventricular rupture has been also categorized as immediate, delayed, and chronic. Chronic tears occur days to years after MVR and share clinical and morphologic aspects with left ventricular pseudo-aneurysm[8], and this confirms that the rupture starts as an endocardial break. Thus, the iatrogenic trauma of the left ventricular endocardium may be the initial phase of the wall rupture process.

From a surgical point of view, Cobbs considered the mitral valve as a morphological and functional unit, called "the mitral-loop"[3]. It includes two arms: the inner arm, composed of the PMs, the cords and the leaflets, which is a longitudinally coursing loop connected at both ends to the mitral annulus; the outer arm of this loop, consisting of longitudinal muscle fibres and the wall of the left ventricle, superiorly fixed to the mitral annulus. Cobbs' "untethered ventricle" theory suggests that spontaneous rupture of the left ventricle could occur in some patients following excision of the posterior leaflet and the PM, creating an "untethered loop"[3]. The Cobbs theory is very remarkable and was supported by others, for example Ross' and Streeter's "Letter to the Editor"[3]. It is well known that preservation of the mitral valve posterior leaflet is the basic requirement to avoid serious complications. Respecting this pivot of mitral surgery has led to a dramatic reduction of left ventricular rupture cases reported in the literature. This supports the idea that iatrogenic trauma to the left ventricular endocardium and the "untethered ventricle" could both contribute to the rupture of the left ventricle free wall.

As an extension of this theory, observing the anatomy of the heart, two other loops divided by the septum could be envisaged (fig.1). First, the left loop: the outer arm formed by the ventricular apex, PMs, cords, anterior leaflet and anterior annulus between the trigons. The septal arm (central arm) of this loop consists of the mitro-aortic continuity, the aortic valve and the inter-ventricular septum. Secondly, the apex of the right ventricle, the septal portion of the tricuspid annulus and the tricuspid septal leaflet form the right loop of the outer arm. The septal arm (central arm) of this loop consists of the inter-ventricular septum.

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Therefore, it can be hypothesized that the In-S is supported longitudinally during diastole by these two loops on both sides, right and left, and that this support is missing in the free wall of the left ventricle.

On the other hand, dilation in the transverse plane of the left ventricle at the level of the In-S results from a combination of various factors. Septal motion originates from the blood pressure applied on the right and left sides of the In-S, as well as the active tension and the intrinsic stiffness of the tissue itself. Thus, during the cardiac cycle, the In-S is affected by the stress of the different pressures and volumes in the right and left ventricles. The changes in the diastolic curvature of the In-S, from a slight flattening to a complete inversion towards the left ventricle, determined by the right ventricle volume overload[(9)], demonstrate the presence of a direct cross-sectional support of the right ventricle to the In-S. A similar support to the LV free wall is lacking, particularly after opening the pericardium. However, in the presence of hypertrophic obstructive cardiomyopathy, the end-diastolic dimension is lower than the 'normal', even at full ventricular filling which may be particularly due to In-S and ventricular wall thickness .

Histological analysis of tissue from patients with hypertrophic obstructive cardiomyopathy (HOCM) show muscle fibre disarray that is absent in normal subjects. This histopathology typically involves more than 10% of the myocardium, is widely distributed and typically localized to the hypertrophied inter-ventricular septum[(10)]. The myomectomy procedure thins the In-S, increasing its elasticity compared to the rest of the ventricular wall. During end diastole, the dissected zone of the septum should undergo further tension with increased diastolic stress on the ventricle. It is possible that this stress exposes the thinner septum to the risk of a subsequent rupture, which could lead to a ventricle septal defect, a rare eventuality, as the majority of the reports in the field. This might support the hypothesis that the longitudinal support, described above, is pivotal in giving the resistance to the surgical trauma caused by myomectomy. In cases where this longitudinal support is impaired, right ventricular volumes become responsible for cross sectional support of the inter-ventricular septum.

Obviously, further studies focused on for example hemodynamics and exploiting new accurate imaging technologies are required to demonstrate this theory.

Legend figures

Figure 1

A and B: the right and left loops, separated by the interventricular septum, are indicated by dark blue and light blue circles respectively.

Figure 2

An urgent CT scan confirmed the presence of the perforation site beside the LV apex. In this image it can be verified that the positions of the entry and exit points of the perforation are face to face.

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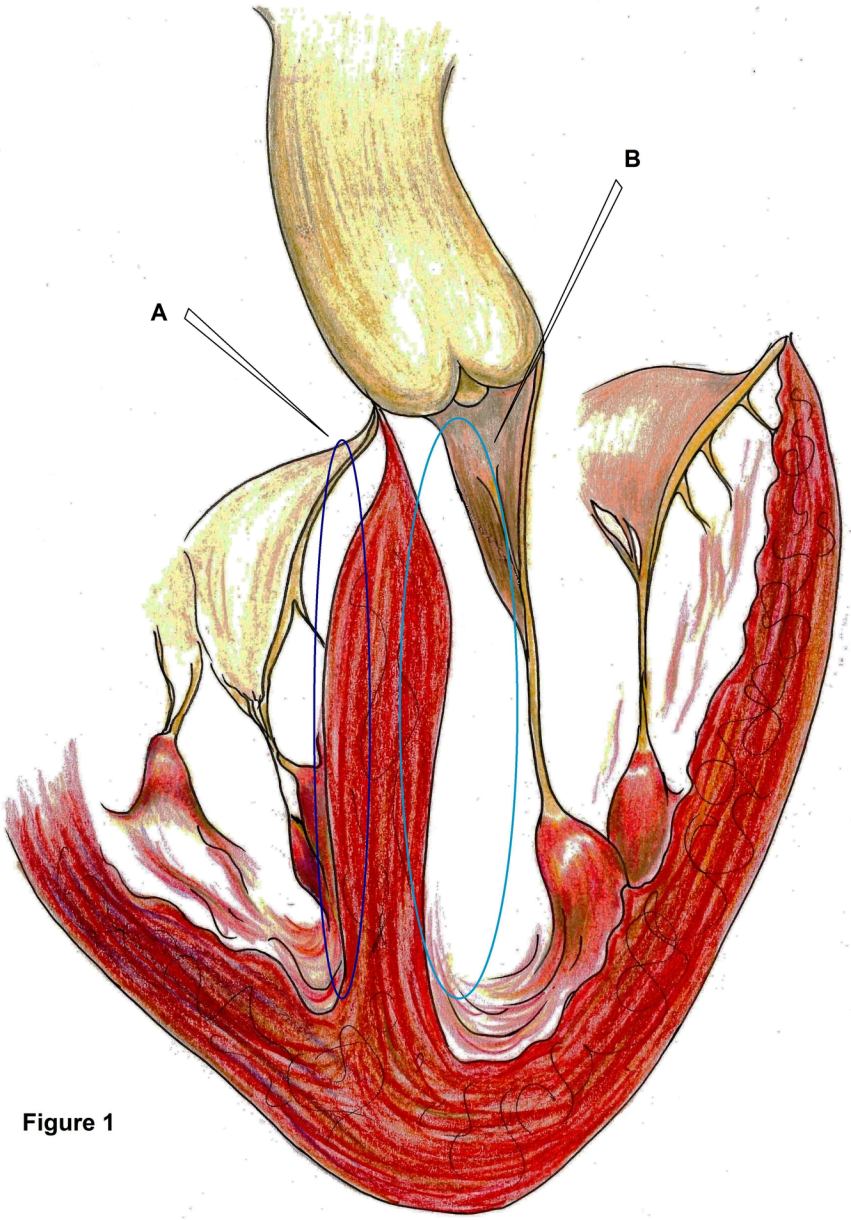

Figure 1

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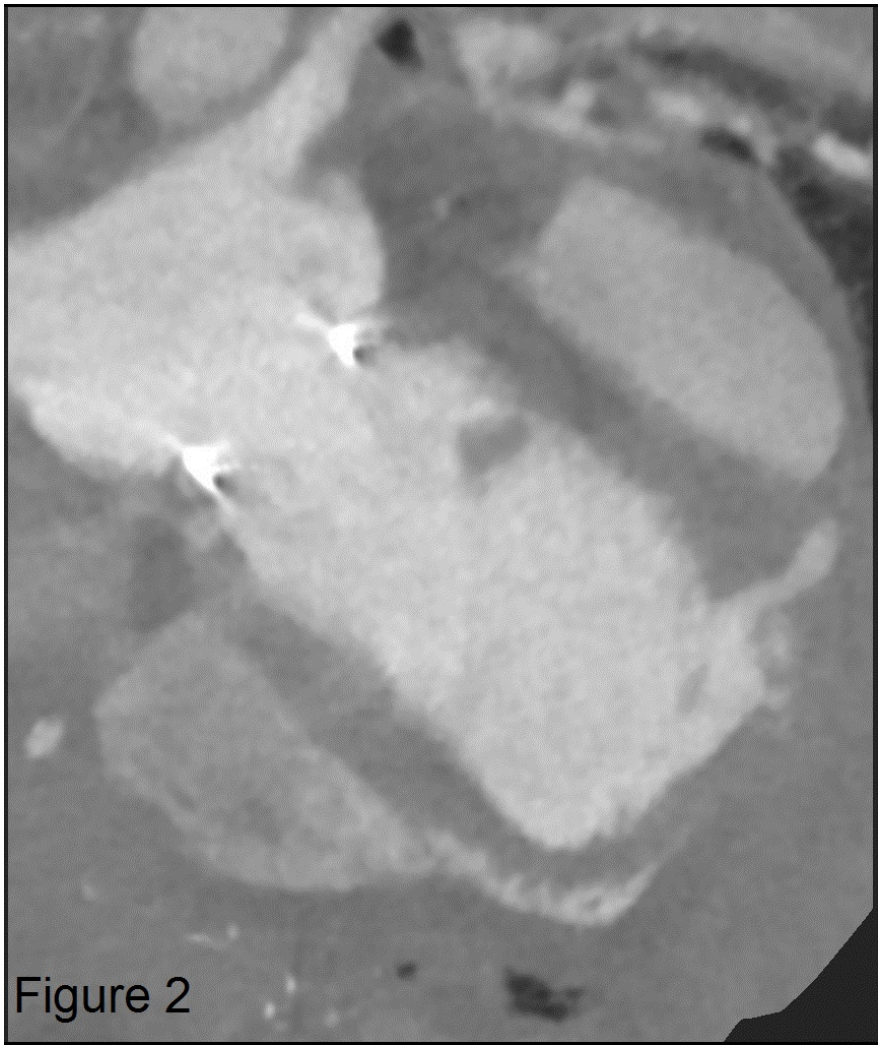

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