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Rewind the heart: A novel technique to reset heart fibers' orientation in surgery for ischemic cardiomyopathy

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Summary Ischemic cardiomyopathy is the most common cause of dilated cardiomyopathy and congestive heart failure. It affects approximately 1 out of 100 people, most often middle-aged to elderly men. Left ventricular restoration surgery is a challenging therapeutic approach to this pathology: it aims to rebuild a near-normal ventricular chamber in a heart damaged by a myocardial infarction, reducing its volume and improving the fraction of blood ejected by each systole. This is obtained by eliminating the akinetic/dyskinetic part of the cardiac muscle and closing the final defect with or without a synthetic patch. Optimization of surgical repair is mandatory as far as ischemic cardiomyopathy is a worldwide disease responsible for many cardiac deaths and because of its potential use as an alternative to heart transplantation in selected patients.

Until now, this surgery has been performed without caring for myocardial fibers' disposition but recent evidences clarified the key role of fibers' alignment in heart physiology. The myocardium of the left ventricle has a unique three-dimensional, multilayered structure: it constitutes the anatomical basis for the cardiac function and for left ventricular torsion, a key movement of normal heart. Myocardial infarction alters myocardial structure in the site of the necrosis and subsequent cardiomyopathy eliminates left ventricular torsion. On the other hand, histological evidences show that myofibers' orientation in the thickness of residual normal myocardium is not changed and that transmural courses of fiber orientation angles near infarct zones were similar to those of normal myocardium.

We hypothesize that, with a particular surgical technique, it could be possible to realign the anatomically normal fibers of the residual myocardium in order to rebuild a physiologic setting. We planned a novel surgical technique of left ventricular restoration using a very narrow, string-shaped patch and a particular suturing sequence and technique, whose aim is to near normally oriented residual myocardial fibers. The renewal of left ventricular torsion was evident at sight just at the end of this kind of ventricular restoration, still in the operating room, then confirmed by 2D speckle tracking echocardiography. These observations are indirect proofs of fibers' realignment, as the torsion movement of the left ventricle is due to the interlaced, oblique orientation of myocardial fibers. We herein propose a theoretical explanation of this outcome, drawing a geometrical modeling of the surgical procedure.

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Introduction

Ischemic cardiomyopathy is the most common cause of dilated cardiomyopathy and congestive heart failure. Mortality for heart failure is very high, at around 40% within the first year after diagnosis. Left ventricular restoration surgery is a challenging therapeutic approach to this pathology: it aims to rebuild a near-normal ventricular chamber in a heart damaged by a myocardial infarction, by reducing its volume and improving the fraction of blood ejected by each systole. This is obtained by eliminating the akinetic/dyskinetic part of the cardiac muscle and closing the final defect with or without a synthetic patch.

We herein hypothesize that a novel surgical technique can help in realigning myocardial fiber in a physiologic setting. It is a logical consequence of the state of the art of this surgical treatment and derives from studies about the pathological changes that intervene in the myocardium after an acute infarction. In particular, left ventricular torsion, a peculiar movement of the normal heart that optimizes ejection efficiency and energy expenditures, is lost due to the dilation of the apex and the loss of contractile myocardium [1].

In most of the patients in which this technique was applied, we observed the renewal of left ventricular torsion as a specific marker of restored fibers' orientation, both immediately after the surgical correction in the operating room and at late follow-up.

This evidence motivated us to hypothesize an explanation for its positive outcome.

The *restitutio* of ventricular torsion was never demonstrated before in any technique of ventricular restoration [2].

Heart structure

The myocardium of the left ventricle has a unique three-dimensional, multilayered structure: interlaced myocardial fibers depict a directional gradient in the thickness of the ventricular wall which guarantees the best hydrodynamic efficiency at the lowest energy expenditure and constitutes the anatomical basis for the cardiac function [3].

This structure is the result of a complex embryologic development of the heart, which undergoes a transition from a peristaltic tubular heart (something like a ''contracting vessel'') to a synchronously contracting four-chambered organ through a genetically programmed looping and growing process, determined by a delicate balance of proliferative and apoptotic events. Fibers' disposition is then strictly supported and interlaced with extracellular matrix of the heart, insulating tissue and anchoring fibrous structures, to constitute a self-regulating functional system, modeled and remodeled by hydrodynamic forces.

Serial sections across the LV wall demonstrate that fiber angle increases gradually in opposite directions in sections approaching the epicardial and endocardial surfaces. In particular, fibers at the apex of the heart have oblique, opposite directions [4].

This weave obtains a ''myocardial continuum'' which is the basis of the complex function of the heart.

Heart function and left ventricular torsion

Heart function is characterized by the unique ability to translate 15% linear sarcomere shortening into ejection fractions greater than 50% and wall thickening greater than 30%.

This is obtained thanks to the interlaced fibers' disposition that optimizes work per spatial unit and distributes stress to the adjacent extracellular tissue. The most important determinant and evidence of normal left ventricular function is out of doubt left ventricular torsion, that is the systolic counterclockwise movement of the apex with respect to the base which completely ''squeezes'' blood out of the ventricle and gains elastic energy for the diastolic recoil.

Since 1911 [5], torsion is recognized to be essential for ventricular ejection and many studies confirm its importance as a sensitive marker of normal ventricular function in physiologic and pathologic settings [6—8]. It is well known that the twisting motion of the left ventricle about its long axis results from the contraction of the opposite, obliquely oriented epicardial and endocardial fibers. The anisotropy due to the variation in muscle fiber angle across the wall induces this rotation. Helicoidal fibers' direction and chamber volume are the two major determinants of torsion. Normal value of torsion angle is about $11.2 \pm 1.3^{\circ}$ at apex, epicardial level.

Impact of myocardial infarction

Myocardial infarction completely alters the anatomical structure of the heart in the region tributary of the occluded vessel. The necrosis induced by myocardial infarction alters the myocardial continuum, forcing the whole ventricle to work at a lower efficiency level. Necrotic area spaces out the residual normal tissue, and negative