Myocardial Remodeling in Congenital Heart Disease

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Myocardial structure in congenital heart defects had not been extensively analyzed until recently. It was not until 15 or 20 years ago that the first studies appeared in the literature.

Back in the first half of the last century, the first challenge in the study of congenital heart defects was the understanding of the morphology and the approach to diagnosis. Morphology has now been studied for more than one century, and nowadays virtually all the anomalies have their anatomy well clarified.

With the advent of cardiac surgery in the 1950s, the next step was to correct or palliate the defects, and this, to a great extent, has also been achieved today.

In parallel to the improvement of diagnostic methods and surgical technique, clinical management also experienced an enormous development during the last five decades. Patients with heart defects, now surviving to adolescence and adulthood, required the specialization of clinicians and surgeons, in order to understand well the late outcome of their diseases and to treat their residual lesions.

The next goals to be achieved are the prevention of myocardial remodeling with an early approach to the defects and the understanding of the previous or intrinsic myocardial abnormalities.

The distorted cardiac morphology in congenital heart defects leads to variable hemodynamic consequences that promote myocardial adaptation and may, eventually, induce heart failure. Since these particular conditions of volume and pressure overload are present from cardiac morphogenesis, the remodeling process occurs in parallel to the growth process of the heart, before and after birth.

Although the term “myocardial remodeling” has been sometimes taken as a synonym to “cardiac dilation”, its concept implies a series of changes in size, shape and function of the heart, that histologically correspond to myocyte hypertrophy or loss (due to necrosis or apoptosis), changes in the amount of extracellular matrix (mainly fibrosis), and changes in the microvasculature, such as capillary proliferation. Particularly in small children, the mechanism of myocyte hyperplasia may also take part in the process.

Histological myocardial remodeling has been studied in different types of congenital defects, although not extensively.

Key words
Heart defects, congenital; myocardium.
in their number is believed to increase the potential for ischemic damage. Although hypoxia is considered to be an effective stimulus for endothelial cell growth, experimental data of capillary supply adaptation in response to hypoxia are controversial. Numbers of capillaries were considered inadequate in hearts of humans presenting hypoplastic left heart syndrome\(^\text{16}\) and in tricuspid atresia\(^\text{4}\). This may be interpreted as an inherent abnormality with implications for ventricular development or indicate a greater vulnerability of these hearts to ischemia.

In summary, when thinking about myocardial remodeling in congenital heart disease, we should consider that: it is an ongoing process occurring since intrauterine life, because the defects are present since morphogenesis; it develops in parallel with the normal growth process, which makes it unique and different from remodeling in mature hearts, and it continues even after surgery, because children continue to grow and may have residual defects.

References