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Modeling the impact of concomitant aortic stenosis and coarctation of the aorta on left ventricular workload

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ABSTRACT

Coarctation of the aorta (COA) is an obstruction of the aorta and is usually associated with bicuspid and tricuspid aortic valve stenosis (AS). When COA coexists with AS, the left ventricle (LV) is facing a double hemodynamic load: a valvular load plus a vascular load. The objective of this study was to develop a lumped parameter model, solely based on non-invasive data, allowing the description of the interaction between LV, COA, AS and the arterial system. First, a formulation describing the instantaneous net pressure gradient through the COA was introduced and the predictions were compared to in vitro results. The model was then used to determine LV work induced by coexisting AS and COA with different severities. The results show that LV stroke work varies from 0.98 J (no-AS; no-COA) up to 2.15 J (AS: 0.61 cm^2 + COA: 90%). Our results also show that the proportion of the total flow rate that will cross the COA is significantly reduced with the increasing COA severity (from 85% to 40%, for a variation of COA severity from 0% to 90%, respectively). Finally, we introduced simple formulations capable of, non-invasively, estimating both LV peak systolic pressure and workload. As a conclusion, this study allowed the development of a lumped parameter model, based on non-invasive measurements, capable of accurately investigating the impact of coexisting AS and COA on LV workload. This model can be used to optimize the management of patients with COA and AS in terms of the sequence of lesion repair.

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1. Introduction

Coarctation of the aorta (COA) is a congenital heart disease characterized by a narrowing of the isthmus zone, i.e., the section of the descending aorta distal to the left subclavian artery. COA is encountered in 0.1% of newborns (De Mey et al., 2001) and is the third most prevailing defect in infants and children (5 to 8% of all congenital heart disorders) (Rao, 1995). In severe cases COA can result in serious complications such as hypertension, left ventricular failure, rupture of the aorta and premature coronary artery disease. As a result, 60% of adults over 40 years old with uncorrected COA have symptoms of heart failure and 75% of these patients die by the age of 50 and 90% by the age of 60 (Brickner et al., 2000).

COA often occurs in combination with other congenital cardiovascular diseases. In the majority of cases COA coexists with aortic stenosis (AS) (between 30% to 50%) (Brickner et al., 2000; Braverman et al., 2005; Hamdan, 2006). The left ventricle (LV) then faces a double-pressure-overload: AS + COA. This leads to a significant increase in left ventricular work and systolic pressure, thus increasing the risk of heart failure. Under such conditions, it is particularly important to determine: (1) the total load supported by the LV and (2) the respective load induced by each pathology independently. This information will contribute to optimize the clinical procedure in terms of the sequence of lesion repair: valve replacement, COA repair or both (Chu et al., 2011). The total load supported by the LV could be better characterized by LV stroke work determination, which can be used to assess the inotropic state of the left ventricle (Burkhoff et al., 2005) and represents the work of the left ventricle during each heart beat. Thus, LV stroke work has been shown to effectively characterize patient's outcome (Garcia and Durand, 2006). However, this parameter requires invasive determination of the instantaneous ventricular pressure and volume, thus limiting its in vivo application. An alternative way to estimate LV stroke work and to investigate the impact of pathological conditions on LV performance is to model the cardiovascular system using lumped parameter models. This approach has been extensively used to model both healthy and pathological conditions (Segers et al., 2000, 2001, 2002, 2003; Garcia et al., 2005a; Tanné et al., 2008). Interestingly, only a limited number of models have been dedicated to simulate coarctation of the aorta (Engvall et al. 1991, 1994). More recently,

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