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ORIGINAL ARTICLE

Effect of left ventricular pacing mode and site on hemodynamic, torsional and strain indices

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Abstract *Introduction:* Several reports have indicated that left ventricular (LV) lead placement at an optimal pacing site is an important determinant of short- and long-term outcome. This study investigated the effect of pacing mode (atrioventricular [AV] or ventricular) and site (LV apical or lateral) outside the ischemic region on the LV hemodynamic, torsional and strain indices in the ischemic myocardium.

Methods: Experiments were conducted in anesthetized open-chest pigs ($n = 15$) 30 min after LAD ligation to investigate the hemodynamic effects of temporary epicardial AV and ventricular LV pacing at the LV apical (outside the ischemic region) or lateral wall. LV hemodynamic data were recorded (ejection fraction, stroke volume, dP/dt_{max}, systolic pressure, cardiac output and e/e' ratio) and torsional (twist, rotation), as well as deformation (radial and circumferential strain), indices of LV function were assessed using two-dimensional speckle tracking imaging.

Results: The LV function was highly dependent on the pacing mode and site. LV dP/dt_{max}, systolic pressure and twist decreased significantly during LV pacing in comparison to sinus rhythm ($p = 0.004$, $p<0.001$, $p = 0.002$, respectively). Torsion in sinus rhythm decreased significantly during AV-pacing at the lateral wall ($0.11 \pm 0.04^{\circ}$/mm vs. $0.06 \pm 0.02^{\circ}$/mm, $p = 0.005$) but did not change significantly during AV-pacing at the apex ($0.07 \pm 0.05^{\circ}$/mm).

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Conclusions: LV pacing at the apical or lateral wall, in the ischemic myocardium, leads to a suboptimal response in comparison to sinus rhythm. LV pacing at the apex outside the ischemic area exhibits a better response than pacing at the lateral wall, possibly because pacing from this site leads to a more physiological propagation of electrical conduction.

1. Introduction

There is growing interest in seeking methods that use different pacing modes and/or pacing sites to maximize the benefits and minimize the harmful effects of artificial cardiac stimulation on left ventricular (LV) function. Biventricular or LV pacing has been proposed as an appropriate choice for patients with preexisting LV dysfunction who require ventricular pacing because they are more likely to develop further deterioration of their LV function after right ventricular apical pacing.[1–3] Previous studies have shown that cardiac resynchronization therapy (CRT) is less effective in ischemic than in non-ischemic cardiomyopathy.[4–6] The explanation for these observations is that pacing within the necrotic area is a negative predictor for CRT response. The effects on LV function of pacing sites outside the necrotic region are matters that have not been studied explicitly. The identification of the optimal LV lead position remains controversial.[8] LV lead position at the lateral wall is recommended because this is the site of greatest contractile delay.[9,10] Conversely, LV apical pacing maintains a closer to normal electric activation pattern as well as mechanic synchrony and coordination.[11,12]

Two-dimensional speckle tracking echocardiography (STE) allows detailed evaluation of LV mechanics, including LV mechanical dyssynchrony, LV strain and LV torsion.[13,14] This technique provides important additional information for the selection of the optimal pacing site. The role of STE in the assessment of the effects of RV apical pacing on LV function and the upgrade from RV to biventricular pacing have been evaluated in few studies.[15,16] Data based on STE comparing the effects of different LV pacing modes (dual chamber vs. single chamber pacing) and sites on the LV mechanics, LV strain and LV torsion are still lacking.

The aim of this experimental study was to investigate the effect of pacing mode (atrioventricular or ventricular) and site (LV apical or lateral) outside the ischemic region on LV hemodynamic, torsional and strain indices in the ischemic myocardium (MI).

2. Methods

The protocol complied with the "Principles for the Care of Experimental Animals" and the "Guidelines for the Care and Use of Experimental Animals" issued by the US National Academy of Sciences and National Institutes of Health (version 85-23, revision 1996) and was approved by the Scientific Committee of the Alexandra University Hospital.

2.1. Surgical preparation

The experiments were performed on 15 pigs weighing 37 ± 2 kg. The animals were sedated with an intramuscular administration of midazolam 5 mg/kg and ketamine hydrochloride 5 mg/kg, anesthetized with intravenous (IV) sodium thiopental 5 mg/kg, intubated and controlled by mechanical ventilation (Sulla 808V, Drager Medizintechnik GmbH, Germany). Anesthesia was maintained with IV propofol 0.1—0.2 mg/kg. During the experiment, analgesia was maintained with an intravenous infusion of fentanyl 3 μg/kg/h. Additional anesthetic was administered during the experiment as needed. A 7F sheath was inserted into the right internal jugular vein for the delivery of drugs and fluids. Through the left external carotid artery, a 6F pigtail catheter was placed into the LV cavity and used for LV pressure monitoring. Lead II of the standard electrocardiogram (ECG), LV pressure, and hemoglobin oxygen saturation were monitored throughout the experiment as previously described in detail.[17] A regular median sternotomy was performed after thymic resection, followed by a longitudinal pericardiotomy. Two 3—0 Prolene (Ethicon, Johnson & Johnson Co., European Logistics Centre, Sint-Stevens-Woluwe, Belgium) sutures were placed after the origin of the first diagonal branch of the left anterior descending coronary artery (LAD) to be used for future ligation.

Temporary myocardial pacing leads (Medtronic, type 6500, Minneapolis, Minnesota) were attached to the surface of the right atrium and to the epicardium of the LV apex in the territory of the LAD and of the LV lateral wall (approximately 2 cm below the base). The apical pacing lead was placed outside the region where epicardial ischemia was observed. The leads were connected to an external pacemaker (Medtronic AV Pacing System Analyzer Model 5311B). The two LV electrodes were connected to a two-channel external pulse stimulator (Medtronic model 2883), allowing setting of thresholds for each electrode separately and pacing through each of the electrodes separately. Pacing was unipolar with an indifferent electrode positioned in between the intercostal muscles. During atrioventricular (AV) pacing, the AV delays were short enough to produce an activation wave originating from the ventricular pacing lead and were not modified between the different pacing configurations. Under all conditions, pacing was performed at about twice the stimulation threshold. The pacing rate was set at 10 beats/min above the intrinsic heart rate in each case.