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Review Article

Physiological cardiac pacing: Current status

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ABSTRACT

Adverse hemodynamics of right ventricular (RV) pacing is a well-known fact. It was believed to be the result of atrio-ventricular (AV) dyssynchrony and sequential pacing of the atrium and ventricle may solve these problems. However, despite maintenance of AV synchrony, the dual chamber pacemakers in different trials have failed to show its superiority over single chamber RV apical pacing in terms of death, progression of heart failure, and atrial fibrillation (AF). As a consequence, investigators searched for alternate pacing sites with a more physiological activation pattern and better hemodynamics. Direct His bundle pacing and Para-Hisian pacing are the most physiological ventricular pacing sites. But, this is technically difficult. Ventricular septal pacing compared to apical pacing results in a shorter electrical activation delay and consequently less mechanical dyssynchrony. But, the study results are heterogeneous. Selective site atria pacing (atrial septal) is useful for patients with atrial conduction disorders in prevention of AF.

1. Introduction

From its first human implantation (October 8th, 1958 by Swedish Surgeon Ake Senning), the right ventricular (RV) apical pacing has saved millions of lives. But, within one decade, it was proved to be non-physiological as it causes several adverse hemodynamic effects. Contemporary thinking led to the assumption that pacing the atrium and ventricle sequentially may solve the problem of unsynchronized contraction. So, dual chamber cardiac pacing (DDD/R) was introduced as the "physiologic" pacing mode.

The term "physiological" was first used in Canadian Trial of Physiological Pacing (CTOPP) to reflect the terminology at the time of development of the trial.[1] However, despite maintenance of atrio-ventricular (AV) synchrony, the dual chamber pacemakers (DDD/R) in different randomized controlled trials (RCTs) have failed to show its superiority over single chamber RV apical pacing in terms of death, progression of heart failure (HF), and atrial fibrillation (AF).[1–3] This mysterious inability to show an advantage of physiologic DDD/R versus non-physiologic ventricular pacing may be explained by a factor common to all modes of ventricular pacing and also influencing short- and long-term cardiac pump function: ventricular asynchrony. Retrospective analysis of the Mode Selection Trial (MOST) suggests that the risks of HF hospitalization and AF can be directly linked to right ventricular apical (RVA) pacing burden (cumulative percent ventricular pacing) regardless of pacing mode.[2] This probably can be partially managed by manipulation of pacing modes

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and timing cycle operation among patients with reliable AV conduction to minimize unnecessary ventricular pacing and preserve normal ventricular conduction. But, in patients with high presumed ventricular pacing burden (when ventricular pacing cannot be avoided and/or abnormal ventricular conduction is already present) pacing at alternate ventricular site(s) to attenuate the adverse effects imposed by ventricular desynchronization should be employed.

2. Ventricular pacing for AV conduction disorder

Most common indication for ventricular pacing is AV conduction disease. This AV disease can be at the level of AV node (nodal), at the His bundle (intra-Hisian) or below the level of His bundle (infra-Hisian). Determining the level of block is clinically important. A narrow QRS complex is most compatible with an AV nodal or intra-His problem. A wide QRS complex is most compatible with an infra-His problem. However, a wide QRS complex certainly may occur with AV nodal or intra-Hisian disease in presence of co-existent bundle branch block.[4] Normal PR interval (≤160 ms) of a conducted P wave indicates disease in the His bundle or His-Purkinje system and a PR of >300 ms indicates block in the AV node. His bundle escape rhythm typically has a rate of 45–60 beats per minute. So, in presence of third degree block, if the ventricular rate is greater than 50 bpm, the escape pacemaker is likely to be located high in the AV junction, and the site of block is likely to be in the AV node. In a patient with two-to-one AV block, improvement of conduction by atropine, beta agonists, or exercise suggests an AV nodal site of block. Carotid sinus pressure worsens the block in case of AV nodal block. Failure of conduction to improve with isoprenaline or atropine and paradoxical improvement with carotid sinus pressure suggest intra-His or infra-His block. In case of AV nodal block, there is no VA conduction. But, in case of intra- and infra-Hisian block, there may be presence of retrograde conduction. So, when a pacemaker is to be implanted for assumed or proven intra- or infra-His block, the operator has to consider the possibility of pacemaker syndrome or pacemaker-mediated tachycardia and choose the appropriate pacemaker and programming to prevent their occurrence. Multiple levels of AV block may coexist in the same patient, and they can produce a confusing ECG picture that is extraordinarily difficult to interpret without an intracardiac electrophysiology study.[4]

3. Adverse effects of RV apical pacing

RV apical pacing induces a slower myocyte-to-myocyte propagation of the electrical activation wave front throughout both the RV and left ventricle (LV), rather than rapid propagation through the His-Purkinje network. As a result, surface electrocardiograms exhibit a wide QRS complex and left bundle branch block pattern, characteristic of electrical dyssynchrony. This asynchronous electrical activation leads to asynchronous mechanical contraction which induces a spectrum of systolic and diastolic hemodynamic abnormalities. The MOST study showed that the patients with baseline lower ejection fraction (EF), history of myocardial infarction, and a worse New York Heart Association (NYHA) functional class are more likely to have these adverse events.[2] Batista and his colleagues have shown that even in patients with normal baseline ventricular function, conventional RV apical pacing leads to change in functional class, worsening in walk test, increased B type natriuretic peptide levels at the end of 2 years.[5] Over time, the sequelae of chronic pacing from the RV apex are a higher risk of development of left ventricular dysfunction, heart failure, AF, and death.[6–9] Interestingly, despite these provocative observations, clinical experience indicates that the majority of pacemaker patients tolerate chronic RVA pacing reasonably well. In the MOST study, only about 10% of patients had HF during follow-up.[2]

4. Physiological ventricular pacing

4.1. *Direct His bundle pacing*

Direct His bundle pacing (HBP) utilizes the native His-Purkinje conduction system and is supposed to be the most physiological ventricular pacing site for patients with AV nodal or intra-Hisian block. Direct HBP does not induce interventricular or intraventricular asynchrony or trigger the myocardial perfusion disorders described with RVA pacing as it produces ventricular contraction via the specific conduction system.[10] Direct HBP is accomplished with a steerable catheter (Selectsite), inserted into the right atrium via the subclavian vein, through which a dedicated bipolar, lumen less screw in, steroid-eluting, 4.1-Fr lead (Select Sure) is advanced into the area of triangle of Koch and mapping of the triangle of Koch is performed until the best near-field His bundle signal is recorded. This electrophysiologic mapping is guided by the quadripolar catheter previously positioned with the distal bipole on the His. Once the His signal has been recorded by means of the pacing lead, a clockwise turn is applied in order to fix the lead to the heart. Alternatively, it can be done with conventional active fixation leads. The safety and feasibility of HBP with conventional pacing leads have been shown in several studies.[11]

There are certain problems unique for the HBP with conventional active fixation pacing leads: (1) higher pacing threshold owing to the fibrous structure (less myocardium), (2) the close proximity of the tricuspid valve and its movements contribute to the greater instability of the lead, (3) low success rate.[10] Because the His-region block can become enlarged and encompass the lead site, an additional safety lead should be considered at the apex or right outflow tract to prevent asystole, especially in patients with pure His-bundle capture. This lengthens the surgical procedure time and results in a higher cost. These problems can be overcome by Para-Hisian pacing (PHP) rather than direct HBP. PHP involves simultaneous activation of the His bundle and ventricular septal myocardium. PHP is simpler and more reliable, seems to guarantee physiological ventricular activation of the high muscular part of the intraventricular septum, and also early invasion of the His-Purkinje conduction system, very similar to the activation that can be achieved by direct HBP.[11]