Hypertrophy of the heart muscle is one expression of many genetic, metabolic, or haemodynamic disorders. The process that leads to asymmetric septal hypertrophy may obstruct the subaortic outflow tract of the left ventricle. Narrowing of the outflow tract is caused by anterior movement of the mitral valve during systole, called systolic anterior movement (SAM), and by bulging of the septum into the outflow tract. As these two structures approach one another the flow velocity increases and a Venturi effect causes a pressure drop that draws the mitral valve even closer to the septum. This aspiration of the anterior mitral valve leaflet can lead to incomplete closure of the valve and in consequence to mitral regurgitation. The outflow tract obstruction is therefore dependent on left ventricular cavity size, systolic septal thickening, stroke volume and the related flow velocity, contractility, and the anatomical arrangement of the mitral valve.

So in patients with obstructive hypertrophic cardiomyopathy patterns of mitral valve systolic anterior movement are diverse and determined largely by the interrelation of left ventricular outflow tract geometry, the size and mobility of the mitral leaflets, and the presence of other anatomical or structural abnormalities.1

Several groups have reported short-term and long-term improvement in haemodynamic and clinic variables after pacemaker treatment of hypertrophic cardiomyopathy.2-6 Pacemakers were generally implanted in patients with refractory hypertrophic obstructive cardiomyopathy who were candidates for surgery (myotomy, myectomy, and mitral valve replacement) in whom pacing reduced the pressure gradient by more than 30%. The success of the procedure depended on full ventricular capture at the apex. To achieve this the electrode had to be placed carefully at the right ventricular apex in order to activate the intraventricular septum at the apex. Such apical pre-excitation is best achieved by a short atrioventricular delay during VDD or DDD pacing. In my hospital we found that long atrioventricular intervals often resulted in fusion between activation by the His bundle and the pacemaker of the septum, and did not reduce the gradient to a minimum. On the other hand atrioventricular delays that were too short reduced ventricular filling. Because diastolic function is abnormal in hypertrophic cardiomyopathy this delay is crucial. The atrioventricular delay must be long enough to achieve atrial filling of the ventricle but short enough to capture fully the left ventricle from the apex. Because in many patients with hypertrophic obstructive cardiomyopathy the intrinsic atrioventricular delay is usually short, drug treatment must be continued. In addition ablation of the atrioventricular node has been proposed for refractory cases in which the intrinsic atrioventricular delay is too short for ventricular pacing to be effective.

How do pacemakers influence obstruction in hypertrophic cardiomyopathy? Though pacemaker activation of a heart beat was known to alter left ventricular function the mechanism responsible for this effect was not investigated until two groups reported that pacing reduced the pressure gradient in hypertrophic obstructive cardiomyopathy and that this had long-term clinical benefits.14 Jeanrenaud et al reported that right ventricular apical stimulation inverted the activation sequence of the left ventricle and that as a consequence most of the systolic ejection was accomplished before the septum could move towards the mitral valve. Other factors, as yet unknown, may also contribute to the therapeutic effect of pacing in patients with hypertrophic cardiomyopathy. The fact that the left ventricular ejection fraction is slightly lower with ventricular pacing than with normal His-Purkinje activation has been recognised for many years but never explained. When activation maps were used in animal studies to compare His-Purkinje system activation with pacemaker induced activation of the left ventricle they showed that impulse propagation, and in consequence mechanical activation, was slower with pacing.6 Perhaps pacing "normalises" the hypercontractility of hypertrophied hearts?

The finding that pacing had a beneficial effect on short-term and long-term haemodynamic function in patients with hypertrophic cardiomyopathy was fortuitous and led to a major advance in treatment. It also showed that we still do not fully understand what pacing does to the heart.

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