

Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION



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Circulation 2003;108:e64-e66

DOI: 10.1161/01.CIR.0000085657.09097.38

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75214

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Cardiac Resynchronization Therapy

A Patient's Guide

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Cardiac resynchronization therapy (CRT) is an important new treatment for symptoms associated with congestive heart failure (CHF) caused by weakening of the heart muscle (cardiomyopathy). Cardiomyopathy is most commonly caused by irreversible damage from coronary artery disease (such as by a heart attack), but may also be the result of genetic factors, viral infections, or toxins (such as alcohol). The symptoms of CHF typically include shortness of breath, swelling of the feet and legs, fatigue, exercise intolerance, diminished appetite, and depression.

It is estimated that over 22 million people are living with CHF worldwide and an additional 2 million people are newly diagnosed each year. Physicians group CHF patients into 4 different categories or functional classifications on the basis of the individual's physical limitations due to CHF symptoms. These are referred to as New York Heart Association (NYHA) functional classes I, II, III, and IV. Class I and II patients are minimally or mildly symptomatic, whereas class III and IV patients are moderately or severely symptomatic.

Medications are the mainstay of therapy for patients with cardiomyopathy and CHF. Medications help rid the body of extra fluid, strengthen the heart's contraction, and ease the heart's workload by relaxing the blood vessels and reducing the resistance to pumping blood. Lifestyle adjustments are an equally important part of living with CHF and include regular exercise and dietary restrictions, such as minimizing salt and fluid intake, which help to prevent fluid build-up.

Many patients with cardiomyopathy and CHF also have an abnormality of the heart's electrical system resulting in an uncoordinated (asynchronous) contraction pattern of the heart muscle. This further reduces the pumping ability of a weakened heart muscle.

How Does the Heart's Electrical System Work?

The heart is a muscular pump composed of 4 chambers. The 2 upper chambers are referred to as the atria, and 2 lower, or main pumping chambers, are known as the ventricles. The coordinated pumping action of these 4 chambers is controlled by an electrical system that is contained in the heart muscle. The normal heartbeat is stimulated by a tiny

electrical signal that originates in a region of the right atrium known as the sinoatrial or SA node. The electrical signal then spreads through both atria, causing them to contract and squeeze blood into the ventricles. The electrical signal then passes through an electrical control station known as the atrioventricular or AV node. After a split-second delay, the signal spreads to the ventricles by way of specialized routes called the left and right bundle branches. The bundle branches fan out in the ventricles, thereby enabling the electrical signal to stimulate both ventricles to contract simultaneously. This simultaneous, coordinated contraction of the ventricles is necessary for optimal pumping of blood to the body and lungs.

Some patients with cardiomyopathy and CHF have an abnormality of the electrical system. The most common abnormality is a delay in electrical conduction through the left bundle branch. This is known as left bundle branch block (LBBB). Because the electrical signal to the left ventricle is delayed by left bundle branch block, the right ventricle begins to contract a fraction of a second before the left ventricle instead of simultaneously. The result is an asynchronous contrac-

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 (Circulation. 2003;108:e64-e66.)
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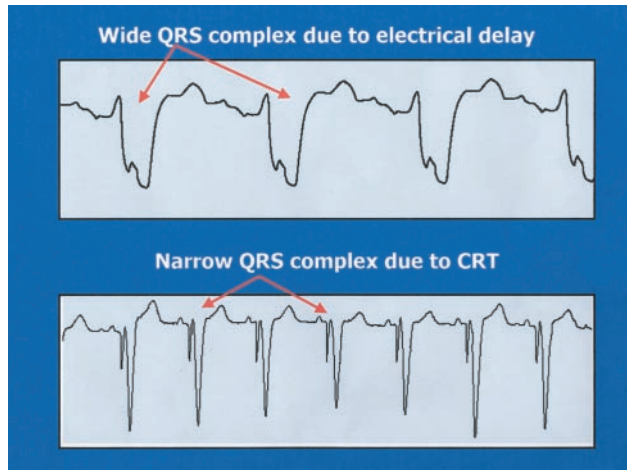


Figure 1. Top, ECG showing electrical delay (left bundle branch block) associated with cardiomyopathy. Bottom, ECG showing improvement in electrical delay with CRT.

tion of the ventricles. This uncoordinated ventricular contraction further reduces the pumping efficiency of an already weakened heart muscle in CHF patients. It is estimated that up to 40% of patients with cardiomyopathy and CHF have an uncoordinated ventricular contraction caused by electrical delay, most often LBBB. This electrical delay is visible on an electrocardiogram (ECG) as widening of the QRS complex and helps to identify patients who might benefit from CRT (Figure 1, top).

What Is CRT?

The idea behind CRT is simple: Restoration of the normal coordinated pumping action of the ventricles by overcoming the delay in electrical conduction caused by bundle branch block (also called resynchronization). This is accomplished by means of a unique type of cardiac pacemaker. Common pacemakers are typically used to prevent symptoms associated with excessively slow heartbeats (for a detailed explanation of how pacemakers work, please see the Cardiology Patient Page by Wood and Ellenbogen [Cardiac pacemakers from the patient’s perspective. *Circulation*. 2002;105:2136–2138]). The pacemaker continuously monitors the patient’s heartbeat and delivers a tiny, imperceptible electri-

cal charge to stimulate the heartbeat when necessary. Most pacemakers typically have 2 electrodes (or leads),

one in the right atrium and one in the right ventricle, which permit the pacemaker to maintain the normal coordinated pumping relationship between top and bottom of the heart. These leads are connected to a battery pack (pulse generator) placed under the skin in the upper chest. In addition to the 2 leads (right atrium and right ventricle) used by a common pacemaker, CRT pacemakers have a third lead that is positioned in a vein on the outer surface of the left ventricle (Figure 2). This allows the CRT pacemaker to simultaneously stimulate the left and right ventricles and restore a coordinated, or synchronous, pumping action. This is sometimes referred to as biventricular pacing because both ventricles are electrically stimulated (paced) at the same time. This reduces the electri-

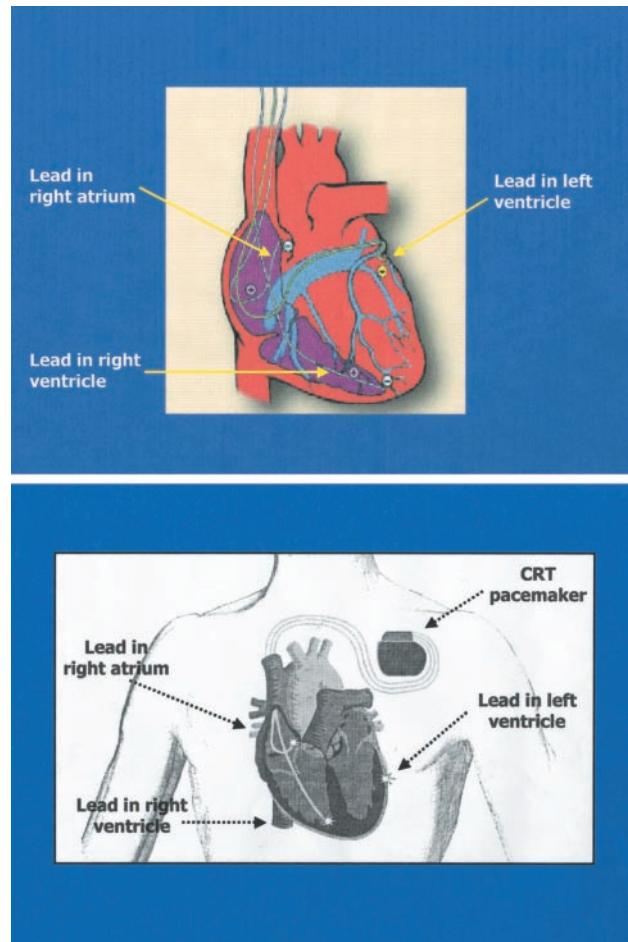


Figure 2. Schematic representation of CRT pacemaker showing 3 leads in the heart.

cal delay and results in a narrower and more normal QRS complex on the ECG (Figure 1, bottom).

Who Is a Candidate for CRT?

The ideal candidate for a CRT device is the individual with:

- CHF, moderately to severely symptomatic despite medical therapy (NYHA functional class III–IV)
- Cardiomyopathy (weakened and enlarged heart muscle)
- Significant electrical delay across the lower pumping chambers (bundle branch block)

Some CHF patients who are candidates for CRT are at also high risk of dying suddenly from rapid heartbeats arising from the ventricles. For these patients, a special CRT device is available that can stop potentially life-

threatening rapid heartbeats by delivering an electrical shock known as defibrillation. This device incorporates a standard implantable cardioverter-defibrillator (ICD) with a CRT pacemaker (for a detailed explanation of how ICDs work, please see the Cardiology Patient Page by Reiffel and Dizon [The implantable cardioverter-defibrillator: patient perspective. *Circulation*. 2002;105:1022–1024]). This device is referred to as a CRTD device (the D refers to defibrillation). Whether you need a CRT or CRTD device is based on many clinical factors and should be discussed in detail with your physician.

What to Expect From CRT

The response to CRT may vary greatly between patients. Scientific studies involving more than 2000 patients worldwide have consistently demonstrated modest improvements in exer-

cise tolerance, CHF class, and quality of life in most patients. Though these improvements may be noticed almost immediately in many patients, they may not be fully realized for weeks or months in others. Unfortunately, there are a small number of CHF patients who do not benefit from CRT therapy. Further research is needed to identify those patients who are most likely to benefit.

Additional Resources

HeartCenterOnline home page. Available at: <http://www.heartcenteronline.com>. Accessed August 1, 2003.

Guidant Corporation home page. Available at: <http://www.guidant.com>. Accessed August 1, 2003.

Medtronic, Inc. Heart failure. Available at: <http://www.Medtronic.com/heartfailure/>. Accessed August 1, 2003.

North American Society of Pacing and Electrophysiology–Heart Rhythm Society home page. Available at: <http://www.NASPE.org>. Accessed August 1, 2003.