

# Indications for Cardiac Pacing\*

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The indications for implantation of a long-term cardiac pacemaker consist of two major categories—the prevention of Stokes-Adams attacks and an inadequate cardiac output caused by a slow ventricular rate.

Stokes-Adams attacks are defined as episodes of cerebral ischemia due to cardiac arrest in patients who have atrioventricular block of any degree, not only during the episode of arrest but at least intermittently at other times as well. One Stokes-Adams episode presents an unquestionable, urgent indication for pacemaker implantation. Questions may arise, however, about the extension of this indication to patients who have high-degree A-V block but have not had an attack. After all, Stokes-Adams attacks are notoriously unpredictable in frequency, severity, and mechanism, and the first attack may be a fatal one. When Stokes-Adams attacks may be considered to have a significant probability, the risk of an initial attack should perhaps be obviated by the implantation of a cardiac pacemaker. A decision of this nature is a matter of legitimate discussion at the present time. The risks, disadvantages, and complications of the procedure must be balanced against the risk of a Stokes-Adams seizure. Many patients with high-degree A-V block never have a Stokes-Adams seizure. Furthermore, in the presence of transient or reversible factors (such as temporary anoxia, cardiac depressing drugs, anesthetic agents, and acute rheumatic fever or diphtheria) even a documented seizure may not be a sufficient indication for a pacemaker.

The indication may also be considered in patients with multifascicular block, i.e. intermittent bilateral bundle branch block, right bundle branch block plus left anterior hemiblock. These patients may go on to develop complete block and then perhaps suffer Stokes-Adams attacks. The likelihood of this development is not entirely clear, however. Although many patients with high-degree A-V block and Stokes-Adams attacks are known to have had multifascicular block earlier, it is not known how many of all the patients with multifascicular block go through this sequence.

The indication for temporary pacemaker application to prevent Stokes-Adams attacks is even more difficult to assess than for long-term use. It has been proposed in patients with acute myocardial infarction when first-degree A-V block or bundle branch block appears, and even in uncomplicated inferior wall myocardial infarction. Early enthusiasm for pacemaker application in acute myocardial infarction has subsided considerably with the demonstration that this procedure does not improve mortality statistics. The placement of a catheter electrode often constitutes a considerable, undesirable stress for the seriously ill patient with acute myocardial infarction. Furthermore, presence of the electrode in the chamber may provoke dangerous ectopic ventricular activity. Finally, electric stimulation of an ischemic area is especially hazardous since the threshold for repetitive response and ventricular fibrillation is much lower in such an area than in normal myocardium.

At times there may be great difficulty in making a diagnosis of A-V block and even of cardiac syncope in patients with intermittent seizures. A-V block may be incomplete and intermittent, so as to escape detection. Seizures that were thought to be neurological were suffered two or three times a week for 35 years by one patient in our series. His electrocardiograms repeatedly showed normal sinus rhythm with normal intraventricular conduction. Finally, a tracing was obtained from a cardiac monitor equipped with a "memory loop" of recording tape and alarm system that showed transient A-V block and ventricular standstill. His disease was at last cured by the appropriate therapy of implantation of a long-term cardiac pacemaker. His electrocardiograms now almost always show competition of his normally conducted intrinsic ventricular beats with the electrically stimulated beats of his fixed-rate pacemaker, a phenomenon that produces no untoward clinical symptoms. Rarely, A-V block is present and only the pacemaker rhythm is seen.

A final extension of this primary indication for pacemaker application is concerned with the management of cardiac arrhythmias in the absence of A-V block—so that the diagnosis of Stokes-Adams disease is technically improper. The provision of a reliable electrically paced rhythm of appropriate rate will prevent syncopal episodes due to depression of rhythmicity at any site

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regardless of whether A-V block is involved. Furthermore, many tachycardias and even fibrillation may be suppressed by overdriving with electric pacemakers at a sufficiently rapid rate.

The second major indication for pacemaker implantation is an inadequate cardiac output due to a slow ventricular rate. The slow rate may result from a slow sinus rhythm with normal A-V conduction or from varying degrees of A-V block. The inadequate cardiac output may be manifested by congestive heart failure, by diminished tolerance for exercise and consequent limitation of activity, by the presence of angina pectoris, and by reduced renal and cerebral function, i.e. azotemia and confusion or coma. The

reduction in renal and cerebral function may at times be insidious and occult, and become apparent only in retrospect after correction by a normal pacemaker rate. In some patients with slow ventricular rates, therefore, a temporary pacemaker at a fast rate should be applied on a trial basis for several days to uncover possible manifestations of inadequate cardiac output. If no significant effect of a slow rate is found, it may be a reasonable clinical judgment not to implant a cardiac pacemaker, but to tolerate a harmless bradycardia. The only regular consequence of a slow ventricular rate is the appearance within a few months of left ventricular hypertrophy.