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EDITORIALS

Changing Trends in Pacemaker Therapy

The first totally implantable pacemaker for the correction of complete heart block in man was reported in 1960.¹ Technologic advances, improved components, more durable electrodes and rigid quality control testing have since resulted in improved pacemaker performance and reliability. Despite the successes, failsafe function has been limited to one and one-half to three years in the majority of subjects. In the main, this has been due to unsatisfactory duration of energy source derived from mercury cell batteries. The availability of several successful pacing techniques requires the physician to be aware of physiologic advantages of each to best treat his patient. One must consider the factors of age, stability of heart block and existence of arrhythmia in selection.

Pacemakers perform as fixed rate, standby or demand, and synchronous. They are electively implanted on the myocardium via thoracotomy, or endocardium by the pervenous route. It is the consensus that the intravenous implant will suffice for the majority of elderly, infirm subjects because it is simple to insert, relatively atraumatic and can be achieved under local anesthesia.

In older individuals with fixed rate pacers, maximum cardiac output is not important to sustain their limited physical requirements; therefore, such patients perform their daily tasks quite adequately. Recently, however, sudden deaths due to ventricular fibrillation have been attributed to this type pacer. This catastrophic event has been ascribed to the electrical stimulus firing in the vulnerable period of the cardiac cycle. Such an event is likely to occur when normal atrioventricular conduction is present—a phenomenon well known in the natural history of patients in complete heart block. In

some instances, return to normal conduction may be the result of improved coronary flow owing to pacemaker correction itself. It has been stated that the stimulus of the fixed rate pacer is miniscule in relation to ventricular tachycardia or fibrillation threshold. This is unquestionably true during normal circumstances, where the set-rate stimulus fires countlessly in the vulnerable period without incident. Experimentally, however, lowered threshold levels have been produced in myocardial infarction, anoxia, electrolyte imbalance, and drug toxicity;² all common clinical states. In these settings, pacemaker stimuli may then trigger ventricular tachycardia or fibrillation.

Recognition of pacemaker-induced ventricular fibrillation has led to the development of standby or demand pacemaking.³ This is a fixed rate, pre-set system of cardiac stimulation at ventricular rates below 70. Above 70, the pacer stimulus fuses instantaneously with the R-wave avoiding the vulnerable period of the cardiac cycle. It is for this reason, as well as simplicity of application that standby has largely replaced fixed-rate pacing in many centers. A physiologic disadvantage of the standby mode is the continuation of a dissociated atrioventricular relationship, and inability to augment required increments of heart rate as contrasted to synchronous pacing.

In younger, more vigorous persons, greater cardiac output requirements are served by synchronous pacing and thoracotomy placement of more reliably sutured myocardial electrodes. The advantage gained by atrioventricular synchronization and autonomically controlled rate change⁴ has been shown to provide maximal augmentation of cardiac output being especially beneficial in heart failure.