2–5 Characteristics and Hemodynamic Advantages of a New Ventricular Synchronized Pacemaker

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Numerous patients with pacemakers may exhibit periods of spontaneous sinus rhythm or, after some time of artificial pacing, may show a return to a normal A – V conduction. In these instances, pacing by fixed-rate pacemakers may give rise to competition with the spontaneous rhythm and prevent the heart from exploiting the physiological mechanisms of adaptation to increased effort. To overcome these difficulties a new pacemaker (1) has been designed which stimulates the heart at a fixed rate when the ventricular rate is low and synchronizes on it when the spontaneous ventricular rate is higher than an established value.

Technique. One of the main features of the pacemaker is the utilization of only one electrode for stimulation and picking up the synchronising ventricular signal. This simplifies the implantation procedure which normally consists in the introduction of an intracavitary electrode (Lagergren type) through an external jugular vein to the apex of the right ventricle.

The pacemaker consists of two sections. The input section amplifies and normalizes the ventricular signals of depolarization, when present. The output section generates 6 mA - 2 msec pacing pulses, by means of a multivibrator synchronized from the output of the first section. When A — V conduction or spontaneous ventricular activity falls below a pre-established level, pacing takes place automatically at a fixed rate. The pacemaker consumes about 250 μW. With 5 standard pacemaker mercury cells with a total volume of about 17 cm^3, the theoretical life-time is 30,000 hours.

The pacemaker has been designed for external use and subcutaneous implantation. Its external dimensions are 60 x 60 x 22 mm and it weights about 150 g.

Results. The hemodynamic behaviour of patients with this synchronized pacemaker and that of others with fixed rate pacemakers was investigated during exercise on a bicycle ergometer. A cardiac catheter was introduced into the right atrium for measuring the pressure and injecting radio-iodinated serum albumin (RISA) to ascertain the cardiac output and the right ventricular and pulmonary blood volumes. These parameters were determined by the precordial counting technique. All the patients were submitted to a 10 minute period of exercise at a rate of 300 kgm/min. Measurements were performed at rest and repeated after the fifth minute of exercise.

Of the 9 patients studied, 4 showed no increase in heart rate (about 70 beats/min) even during exercise, 4 developed a sinus rhythm of about 140 beats/min with exercise, and 1 presented an extrasystolic rhythm with an increase of 55 – 92 beats/min. The cardiac output rose remarkably in all the patients, and was only weakly related to changes of heart rate. In all the patients the pulmonary blood volume (PBV), expressed as a percentage of the total blood volume (TBV) during exercise was considerably augmented with respect to the resting value, but whereas the increment was striking in the patients with a fixed heart rate, it was considerably smaller where this rate was increased. There appears to be a negative correlation between PBV/TBV and the increase in heart rate. The right ventricular end-diastolic blood volume (EDV_{RV}) showed a strong negative correlation with the heart rate. In patients with a fixed heart rate during exercise the ratio EDV_{RV}/TBV increased enormously, while those with an augmented heart rate showed either no elevation or even a slight fall. The mean right atrial pressure displayed a similar behavior, it went up considerably during exercise when the heart rate dit not rise, but otherwise remained around the control value.

Discussion. The hemodynamic data show that the comparable elevation of cardiac output observed in the patients with a fixed and increased heart rate is associated with quite different distributions of blood in the pulmonary circulation and heart. During exercise at a fixed heart rate there is a profound redistribution of blood from the systemic to the central circulation: the lungs are congested and the right ventricle, and probably also the left, are greatly dilated. These observations suggest that during exercise at a fixed heart rate the increase in cardiac output is attained through a mechanism which calls for a much greater cardiac effort than is needed during exercise with an increased heart rate. Atrial synchronized pacemakers with endocavitary leads would appear provide a simple way for patients with stable A – V block to overcome the observed hemodynamic derangements even during effort.