"Optimal Pacing Rate"

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The optimal rate for asynchronous cardiac pacing is very important in the treatment of patients with atrioventricular block. It has been well known that most patients do well at the fixed pacing rate about 70 per minute. But in some cases, various complaints appear at this rate. This paper deals with experimental and clinical studies on the rate-dependency in failed heart, the optimal rate in coronary sclerosis and an indicator for optimal pacing rate.

Subjects and Methods

Experimental Study

The first group of experiment was designed to explore the relationship between pacing rate and other hemodynamic parameters in the failed heart. In the experimental dogs anesthetized with α-chlorarose, atrioventricular block was made by infiltration of one per cent solution of procaine to the atrioventricular node through the right atrial wall. The dogs were paced with external cardiac pacemaker with electrodes placed on the anterior wall of the right ventricle. Hemodynamic measurements were performed while pacing rate was changed from 40 to 280/min. Cardiac output was measured with electromagnetic flowmeter fixed around the aortic root. Arterial pressure and central venous pressure were measured by electromanometer through cannulae in the femoral artery and vein respectively. Cardiac dysfunction was produced by ligation of the anterior descending branch of the left coronary artery of those dogs whose hemodynamic control data were determined as described above, thereafter hemodynamics in heart failure state were again measured with the same methods.

The second group of experiment was carried out to clear the relationship between pacing rate and hemodynamics in the canine heart perfused with fixed low coronary flow as a model of coronary sclerosis. Arterial blood was drawn from the left subclavian artery, and pumped into the left coronary artery by means of an occlusive

Fig.1. Hemodynamic differences between in control and in the failed heart with ligated coronary artery.

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coronary perfusion pump through catheter, which was secured in the origin of the left main coronary artery with a Rumell tourniquet. The coronary pump was adjusted to deliver low coronary flow, ranging from two third to one half of expected normal coronary flow. The heart was paced at the ventricular rate of 60, 90, 120, 150 and 180 per minute. Aortic pressure (AP), left ventricular end-diastolic pressure (LVEDP), cardiac output (CO) and left ventricular oxygen consumption (MVO₂) were measured at each paced rate. Left ventricular work (LVW) and cardiac efficiency (LVEf.) were determined according to following formulas.

\[
LVW = \frac{(AP_{\text{mean}} - LVEDP) \times CO}{100} \times 13.6 \text{ (Kg-M)}
\]

\[
LVEf. = \frac{LVW}{MVO_2 \times 2.06} \times 100\%
\]

Control measurements were made in the dogs with intact coronary circulation.

The third group of experiment was performed in dogs to observe the relationship between ventricular pacing rate and atrial rate as an indicator for optimal pacing rate. Ventricular pacing rate was altered by 10 beats/min. step up from 60 to 250/min. for three minutes interval. During this period, atrial rate, cardiac output, arterial pressure and right atrial pressure were measured. The changes in atrial rate at the different pacing rate were also observed after blocking the stellate ganglion and bilateral vagi.

**Clinical Study**

Clinical observations were made in patients with atroventricular block who were admitted to the Department of Thoracic Surgery in the University of Tokyo Hospital between 1963 and 1971. Coronary sclerosis and non-sclerosis were differentiated by histological study of the cardiac muscle or by coronary angiography and other factors such as age or the clinical course of heart block.

The cardiac output was determined by an indicator-dilution method with coomassie blue dye and a Cambridge Mark II recorder with an
earpiece oximeter or, in some cases, by direct measurement of aortic blood flow with the square-wave electromagnetic flowmeter and probe. The femoral arterial pressure and right atrial pressure were measured by catheterization and electromanometers. The atrial rate was observed by P-wave monitoring on the electrocardiogram.

**Results**

**Experimental Study**

Figure 1 clearly shows the results of the first group of experiment, the typical hemodynamic differences between in control and in the failed heart with ligated coronary artery.

Cardiac output: In control, maximal cardiac output was obtained in the range of 120 to 250/min. of pacing rate; namely plateau formation of cardiac output was observed in this range. In the failed heart, this range was limited from 190 to 220/min., and cardiac output clearly decreased at the rate of cardiac beat below or above it.

Arterial pressure: Arterial pressure increased until ventricular beats reached up to 190/min. in control, whereas it stopped increasing at the
ventricular rate of 140/min. in the failed heart.

Central venous pressure: minimal value was obtained in the range of 120 to 220/min. of ventricular rate in control, whereas it increased gradually after increasing pacing rate in the failed heart.

Figure 2 shows the result of the second group of experiment; the fall of left ventricular cardiac efficiency at higher pacing rate in the dogs with fixed low coronary flow. This values remained relatively unchanged both at slow and rapid rates in the dogs with intact coronary circulation.

The results of the third group of experiment are shown in figure 3 and 4. The relationship among pacing rate, atrial rate and hemodynamics are demonstrated in fig. 3. From top to bottom,
the changes in atrial rate, cardiac output, arterial pressure and right atrial pressure are plotted against paced ventricular rate. The lowest graph shows the circulatory function curve which indicates the relation between cardiac output and right atrial pressure when the pacing rate is changed from 60 to 250/min. During increase in the pacing rate, atrial rate gradually decreased to reach the minimal value at the ventricular rate of 140 to 220/min., thereafter it started to increase again as shown in fig. 3. The range of the minimal atrial rate is indicated by the shaded area. The range in which higher cardiac output and lower right atrial pressure are obtained is indicated by the heavy solid line. This experiment revealed that those two ranges were approximately the same. Minimal atrial rate was obtained at the pacing rate which produced the best circulatory condition. In other words, minimal atrial rate indicated the optimal pacing rate.

These alteration in atrial rate could be entirely eliminated after blocking the stellate ganglion. Figure 4 shows the changes in atrial rate when the autonomic nerves are blocked. The ordinate indicates the changes in atrial rate and the abscissa the ventricular pacing rate. The upper curves show the relation between atrial rate and pacing rate when the bilateral vagi were divided at the cervical region. The lower curves indicate the same relation when the bilateral vagi were interrupted and the right stellate ganglion was blocked with one per cent procaine solution. The atrial rate changed in response to the change of the pacing rate when the vagal nerve reflex was abolished, but not when the sympathetic nerve routes were blocked.

Clinical Study

Figure 5 shows the “circulatory function curves” in 6 paced patients, 3 with coronary sclerosis and the rest without sclerosis. Each curve indicates hemodynamic conditions, cardiac output and right atrial pressure, at the various paced ventricular rate. By graphical analysis, the optimal circulatory range was estimated and shown by the thickened area of the curve in each case. The range of optimal pacing rate was determined as the range that gave the optimal circulatory function, higher cardiac output and lower right atrial pressure. The difference between the ranges of optimal pacing rate in the two underlying disease groups was as follows. The optimal pacing rate in coronary sclerosis was from 59 to 80 per minute, and from 77 to 87 in non-sclerosis. The width of the range was from 2 to 8 beats per minute in sclerosis, and from 25 to 35 in non-sclerosis.

Figure 6 shows the distribution of optimal pacing rate determined by monitoring the atrial rate in 32 paced patients; 19 with coronary sclerosis and 13 without sclerosis. The ordinate indicates the number of patients and abscissa shows the optimal pacing rate. The black area is for non-sclerosis and the dotted area for sclerosis. The optimal pacing rate in coronary sclerosis tends to be less than 70 per minute and in non-sclerosis it tends to be more than 70. The average optimal pacing rate was 64 in sclerosis and 76 in non-sclerosis.

Discussion

Since the cardiac pacemaker was introduced to the clinical practice, the problems relating to the optimal pacing rate and rate-dependency of diseased cardiac function have become the prime of importance. McGregor and Klassen investigated patients in heart failure and found that under these circumstances the cardiac output was
highly rate-dependent until the heart failure was successfully treated. Sowton examined one patient after one year of continuous artificial pacing and found that the optimal pacing rate had not altered but the cardiac output was considerably less dependent upon pacemaker rate than initially observed. These findings suggested that improved myocardial function reduced the rate-dependency of cardiac function.

In the first group of experiments as in fig. 1, the anterior descending branch of the left coronary artery was ligated to produce cardiac dysfunction. The complete A-V block was induced by infiltration of procaine solution around the region of A-V node to minimize the surgical stress to the myocardium while making this arrhythmia. The range of pacing rate which produced maximal cardiac output was narrower in the failed heart than in the intact canine heart. In other words, the high rate-dependency of cardiac output was demonstrated in heart failure. Regarding the arterial pressure, the pacing rate which produced maximal blood pressure was lower in heart failure than in control. In addition, the minimal central venous pressure was obtained at the range of pacing rate from 120 to 220/min. in control heart without any remarkable undulation, whereas it did not remain stable, but gradually increased along with the augmentation of pacing rate in failed heart. This finding indicates the high rate-dependency of central venous pressure in heart failure. As far as the circulatory function is concerned, central venous pressure is very important as an indicator for venous return function. Increase in central venous pressure indicates reduced function of venous return. The range of pacing rate which produced higher cardiac output, high arterial pressure and low central venous pressure is considered to be very much limited in heart failure.

In the second group of experiments as in fig. 2, fall of cardiac efficiency at higher rates in the dogs with low coronary flow was observed. These hemodynamical deteriorations were considered to be related to the restricted coronary flow at the higher pacing rates. This result suggests that the optimal pacing rate in coronary sclerotic patient is relatively low.

In the patient with complete A-V block, the atrial rate during ventricular pacing often provides a simple index of cardiac function. Our third group of experiment, as shown in fig. 3, revealed that the best overall hemodynamic status which produced higher cardiac output with lower right atrial pressure seemed to be obtained at the pacing rate when atrial rate reached its minimal value. We had demonstrated that the best circulatory function was obtained at the ventricular pacing rate that produced the minimal atrial rate in patients with complete A-V block. These observations suggest that atrial rate is an appropriate indicator for optimal pacing rate. These changes in atrial rate are mainly controlled through the autonomic nerve reflex. The sympathetic nerve reflex, as shown in fig. 4, seems to take part in the feed-back system between peripheral circulatory demand and the atrial rate. On the basis of these experimental results, it is suggested that the ventricular pacing rate which produces minimal atrial rate indicates the optimal pacing rate which neither call for the peripheral circulatory demand nor excite the sympathetic nerve reflex. In some cases, high atrial rate is ascribable to fever, pain, respiratory insufficiency, low cardiac output or various mental conditions. It is to be investigated how to determine the proper pacing rate in such conditions.

The optimal asynchronous pacing rate varies from patient to patient. We investigated the optimal rate in the patients from the standpoint of coronary sclerosis or non-sclerosis by measuring hemodynamics as in fig. 5 and by monitoring atrial rate as in fig. 6. The results revealed that the optimal pacing rate in coronary sclerosis tended to be less than 70 per minute. As the greater part of coronary flow occurs during diastolic phase, high rate which shortens diastole results in reduction of coronary blood flow. As shown in fig. 2, coronary flow in normal circulation increases at the higher rates to fill up the myocardial oxygen demand, whereas it is restricted in sclerosis. As a result of this, cardiac efficiency decreases at the higher rates in low coronary flow state. Our clinical studies suggest that many patients could do well by pacing at the rate of 70/min., but in some cases the optimal pacing rate differs from 70 and its range is narrower than in others.

**Summary**

1) The range of optimal pacing rate is narrow in heart failure.
2) The optimal pacing rate under fixed low coronary flow state is relatively low.
3) Atrial rate is a reliable indicator for optimal pacing rate.

4) The optimal pacing rate in the patients with coronary sclerosis is lower and narrower in range.

REFERENCES


