CHANGES IN SINUS NODE RATE DURING HIGH-FREQUENCY ATRIAL STIMULATION

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UDC 616.125.4-008.3-02:615.84.6

KEY WORDS: pacemaker; atrium; frequency; stimulation; retrograde conduction.

High-frequency atrial stimulation (AS) is widely used nowadays as a test of sinus node function [1, 4, 5]. The recovery time of sinus node function after high-frequency AS is used as the corresponding parameter. However, the behavior of the sinus node during AS is also interesting on its own account. Too little attention has been paid to this question.

The aim of the present investigation was to study changes in sinus node function during high-frequency AS and the mechanism participating in alterations of sinus node function.

EXPERIMENTAL METHOD

Experiments were carried out on the frog (Rana temporaria) heart. The sinus venosus (SV) was isolated together with the atrium, unfolded into a flat slab of tissue, and immersed in Ringer's solution at pH 7.2. Potentials were derived by suction electrodes with a tip not more than 10 μ in diameter. The potentials were recorded on an ÉLKAR electrocardiograph and the signal led to its dc input. The atrium was stimulated extracellularly by above-threshold electrical pulses. The stimulating electrodes were made of silver wire 0.5 mm in diameter and the distance between them was 2 mm. One electrode was grounded, the other received square pulses 5 msec in duration from a "Physiovar" stimulator. The frequency of stimulation in all experiments was higher than the frequency of natural excitation of the preparation. Tests were carried out at the following frequencies of stimulation: 37.5, 43, 50, 63, and 77 ± 4 beats/min. The duration of stimulation at each frequency was 30-40 sec. After stimulation at each frequency the preparation was excited at its natural rhythm for 20-30 sec.

For reversible blocking of conduction of excitation between the pacemaker tissue and atrium, a strip of necrotic tissue 1 mm wide and 2 cm long was applied along the whole of the sinoatrial boundary [2, 6].

Electrical potentials for analysis were recorded on an SDR-41 tape recorder (Japan) and processed on a specialized ATAC-350 physiological signal analyzer (Japan), which automatically plotted an intervalogram of the periods of excitation of SV. The accuracy of measurement of the periods was 5 msec. The experimental results were analyzed by Student's t test.

EXPERIMENTAL RESULTS

In the experiments of series I the effect of high-frequency AS on the frequency of excitation of SV was studied. Nine preparations were used for this purpose. The mean frequency of natural excitation of all the preparations was 33.4 ± 1.7 beats/min. AS was carried out, as stated above, in gradually increasing frequency. In all cases of stimulation rhythm binding by the atrium was complete.

During AS with a frequency exceeding that of the natural frequency of the preparation by not more than 20%, SV of all preparations was observed to shift to the frequency of stimulation. For instance, stimulation with a frequency of 37.5 beats/min (T = 1600 msec) and with a frequency of 43 beats/min (T = 1390 msec) led to a switch by SV to these frequencies.

I. M. Sechenov First Moscow Medical Institute. M. V. Lomonosov Moscow University. (Presented by Academician of the Academy of Medical Sciences of the USSR V. V. Kupriyanov.) Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 94, No. 10, pp. 7-10, October, 1982. Original article submitted April 7, 1982.

0007-4888/82/9410-1317$07.50

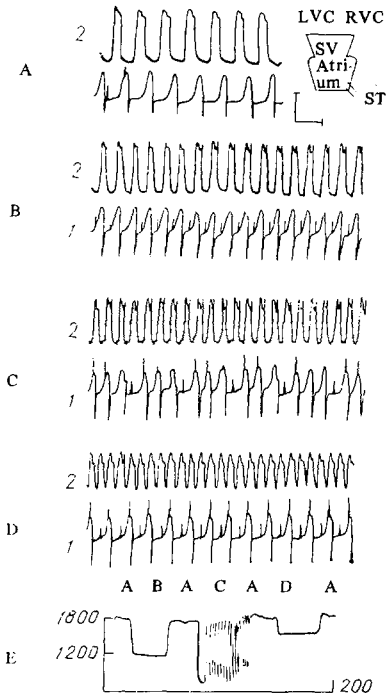

Fig. 1. Changes in frequency of SV during AS with different frequencies. Top right – scheme of preparation. LVC) Left vena cava, RVC) right vena cava. ST) Stimulating electrodes. A) Extracellular action potentials recorded without stimulation, B-D) the same, during AS with different frequencies. Recordings from: 1) SV, 2) atrium. E) Intervalogram of periods of excitation of SV. Letters above portions of intervalogram correspond to letters alongside places of extracellular potentials. Calibration: horizontal, 2 sec; vertical 10 mV.

When the frequency of stimulation was 40-50% higher than the natural frequency of the preparation, SV of some preparations was excited irregularly. For instance, in the case of stimulation with a frequency of 50 beats/min (T = 1190 msec) SV of four of the nine preparations changed to an irregular rhythm. All periods of excitation of SV under these circumstances differed from the period of stimulation. SV of the other five preparations were excited with a frequency of stimulation of 50 beats/min.

If the frequency of stimulation exceeded the natural frequency of excitation by more than 100%, SV switched to a frequency which was a multiple of the stimulation frequency. This frequency was half the stimulation frequency. For instance, when the atrium was stimulated with a frequency of 63 beats/min (T = 950 msec), four SV with natural frequencies of excitation of under 30 beats/min switched to a frequency of 31.6 beats/min. The remaining five SV were excited with an irregular frequency.

However, when the atrium bound a frequency of 77 beats/min (T = 780 msec), all nine SV were excited at half that frequency – 38.5 beats/min (T = 1560 msec); for every two excitations of the atrium there was one excitation of SV. The switch by SV to the frequency of stimulation, to an irregular rhythm, or to half the frequency of stimulation took place during one or two excitations of the atrium (1-2 sec).

An example of one experiment is shown in Fig. 1. The natural frequency of excitation of the preparation in this experiment was 33.6 beats/min (T = 1785 msec; Fig. 1A, 1, 2). During AS with a frequency of 50 beats/min (T = 1190 msec) SV was excited with the same frequency (Fig. 1B, 1, 2). When the atrium bound a frequency of 63 beats/min (T = 950 msec), SV switched to an irregular rhythm (Fig. 1C, 1, 2). AS with a frequency of 77 beats/min (T = 780 msec) caused SV to switch to a frequency of 38.5 beats/min (Fig. 1D, 1, 2). A change in the period of excitation of SV is reflected in the intervalogram (Fig. 1E).

The results of these experiments thus showed that a change in the frequency of AS leads to a change in the frequency of SV. Consequently, besides the direct influence of SV on the atrium, the atrium also has a reciprocal influence on SV.

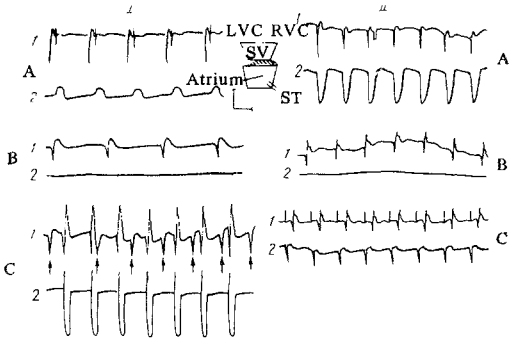

Fig. 2. Changes in frequency of excitation of SV during AS when retrograde conduction was blocked. Top center – scheme of preparation. A (I, II) traces of extracellular action potentials before conduction block, without stimulation; B (I, II) the same after conduction block as indicated on scheme; C (I, II) during AS. Arrows in Fig. 2C (I) correspond to time of excitation of SV. Calibration: horizontal 1 sec, vertical 10 mV. Remainder of legend as to Fig. 1.

It was accordingly considered to be important to study the mechanism of this influence. Some workers consider that the atrium can influence SV through retrograde conduction of excitation [3].

In the experiments of series II the aim was to discover if such a mechanism can in fact completely explain the phenomena observed. In 19 experiments of this series conduction was blocked by applying necrotic tissue along the whole of the sinoatrial border. A conduction block developed a few seconds after application of the tissue and the atrium ceased beating. The frequency of excitation of SV under these circumstances decreased by 5-30%.

Stimulating electrodes were arranged in the same way as previously (see schemes in Figs. 1 and 2). Since the frequency of excitation of SV fell, there was no point in stimulating the atrium within the same frequency range as previously. Accordingly, AS was used with a frequency 10-50% higher than that of excitation of SV after the blocking of conduction. The amplitude of stimulation was the same as in the previous series. In all experiments rhythm binding by the atrium was complete.

When retrograde conduction was blocked AS caused an increase in the frequency of SV. The characteristic time for establishing the new frequency of SV varied from 10 sec to 1 min. In the absence of a block, however, the time was 1-2 sec.

The frequency thus established in 11 of 19 experiments was below the frequency of stimulation, but 10-30% higher, on average, than its initial value ($P < 0.05$).

An example of one such experiment is given in Fig. 2, I. The natural frequency of excitation of the preparation was 26 beats/min (T = 2300 msec; see Fig. 2A, 1, 2). After the conduction of excitation was blocked the frequency of SV fell by 30% to 18.2 beats/min (T = 3300 msec), and the atrium ceased to beat (Fig. 2B, 1, 2). AS with a frequency of 39 beats/min caused SV to switch to a frequency of 34 beats/min (T = 1770 msec; see Fig. 2C, 1, 2); the trace C 1 in Fig. 2, moreover, clearly shows a progressive fall in the periods of excitation of SV – the end of establishment of the new frequency.

A frequency of excitation of SV in the remaining eight experiments was established equal to the stimulation frequency. This frequency could be as much as 50% higher than the initial value.

After the end of stimulation SV reverted to its initial frequency. An example of one such experiment is given in Fig. 2, II. The natural frequency of the preparation was 40 beats/min (T = 1500 msec; see Fig. 2A, 1, 2). After conduction had been blocked SV operated with a frequency of 34 beats/min (T = 1765 msec), i.e., the frequency was 15% lower, and the atrium stopped beating (Fig. 2B, 1, 2). AS with a frequency of 39 beats/min (T = 1538 msec) caused a shift of SV to a frequency of 39 beats/min (Fig. 2C, 2, 1, 2).

These experiments showed that besides retrograde conduction, another mechanism also is involved in the change of frequency of SV.

It can be tentatively suggested that this is an influence either of electrical pulses of the stimulator or of bioelectrical currents generated by the atrium during excitation, or the mechanical activity of the atrium on the formation of the general pacemaker rhythm.

In series III (eight experiments) the effect of electrical pulses of the stimulator on the change in frequency of SV was investigated. To rule out any possible influence of the atrium, the atrium was not stimulated. Excitation of the atrium took place at the frequency assigned by SV. Stimulating electrodes were lowered into the Ringer's solution at the same place as during AS (the atria were shifted to one side so that the electrodes did not touch them). During stimulation the frequency was gradually increased, just as in the experiments of series I. Pulses of the same amplitude as in the previous series were applied to the electrode, and changes in the frequency of SV were recorded. The action of the stimulating pulses caused no change in the frequency of SV. The results of this series of experiments showed that electrical pulses of the stimulator itself do not affect the frequency of SV. The frequency of SV changes only when the atria are excited.

During high-frequency AS the sinus node thus alters its own frequency. The change in frequency of the sinus node depends on the frequency of AS. Activity of the atrium has some influence on the formation of the rhythm of SV and this influence, moreover, is exerted by some means other than retrograde conduction of excitation. Evidently under the conditions of the present experiments some influence of mechanical activity of the atrium on frequency formation in SV likewise cannot be ruled out.

Changes in rhythmic activity and the possibility of onset of arrhythmias in the sinus node of the heart must be taken into consideration in clinical practice when electrical stimulation of the atrium is carried out.

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POSITIVE INOTROPIC ACTION OF BLOOD PLASMA ON RABBIT HEART PAPILLARY MUSCLE

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UDC 612.172.014.46:615:38]-085.23

KEY WORDS: blood plasma; positive inotropic action; rabbit heart papillary muscle.

When studying the action of blood plasma from rabbits in a state of burn shock on electrical and contractile activity of the papillary muscles, we used blood plasma from healthy animals in control experiments [1]. When so doing we found that the addition of control plasma in Tyrode solution (in the ratio of 1:1) causes a marked increase in amplitude of the contractions of the papillary muscles. This served as the starting point for the present investigation.

Its aim was to study the effect of normal blood plasma on ionotropism–rhythm relations in the rabbit myocardium and on the resting potential (RP) and action potential (AP) of myocardial cells. To study the nature of the cardiostimulating factors in blood plasma, propranolol, a β-adrenoblocker, was used.

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(Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Ékskperimental'noi Biologii i Meditsiny, Vol. 94, No. 10, pp. 10-13, October, 1982. Original article submitted February 28, 1982.

0007-4888/82/9410-1320$07.50