Repolarization of Ventricular Myocardium in Atrioventricular Electrical Stimulation of the Heart in Dogs

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The duration of ventricular myocardium excitation increases during atrioventricular stimulation of dog heart and the sequence of depolarization of the right and left ventricles is desynchronized. Significant shortening of the activation—recovery interval in the intramural and subendocardial layers of the left ventricular base leads to modification of the repolarization sequence in this area and to an increase of total dispersion of activation—recovery intervals in the cardiac ventricles, as a result of which repolarization sequence starts partially repeating the depolarization sequence.

Key Words: *activation—recovery interval; electrical stimulation; myocardium*

Processes leading to disorganization of cardiac activity develop in the heart with implanted pacemaker [6]. Transmural gradients of action potentials duration are inverted in patients with implanted pacemakers, which leads to arrhythmias [3]. It is however unknown how local duration of repolarization in the apical and basal regions of the right (RV) and left ventricles (LV) is changing in electrical stimulation of the heart.

We studied local lengths and sequence of repolarization of ventricular myocardium under conditions of atrioventricular stimulation (AVS) of dog heart.

MATERIALS AND METHODS

Experiments were carried out on mongrel adult dogs (n=15) of both sexes (15-35 kg). The animals were narcotized by zoletil (15 mg/kg intramuscularly) and transferred to artificial ventilation of the lungs. Median sternal thoracotomy was carried out and the pericardial sac was opened. After opening of the chest, the body temperature was maintained at the level of 37-38°C.

Electrical stimulation of the right atrium (control, 150 str/min) and regular AVS (3.5 mV, 1.0 msec, 150 str/min) were carried out. Mapping of the cardiac electric potentials was realized by means of multipolar intramural needle electrodes using an electrocardiotopographic device. The following parameters were determined in each of 64 myocardial leads: activation moment (by the dV/dt_{min} value during *QRS*), repolarization moment (by dV/dt_{max} value during *ST-T* period), duration of activation—recovery interval (ARI; by the interval between activation and reporalization moments). Activation, repolarization, and ARI dispersions were calculated as the difference between the maximum and minimum values of each parameter. The differences in the time parameters were evaluated using Wilcoxon, Friedman and Newman—Keuls tests. The data are presented as arithmetic mean±standard deviation ($M \pm \sigma$).

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RESULTS

Atrioventricular stimulation did not change the transmural sequence of RV and LV activation in any of the areas, except the LV apex, which was characterized by the absence of transmural differences in the time of activation ($p<0.05$). The earliest activation of the myocardium was observed at the site of stimulus application (RV apex), the latest in the LV base. Heart stimulation did not appreciably change the dispersion of RV and LV activation time (Table 1), but prolonged ($p<0.05$) the time of ventricular myocardium activation [2]. Atrioventricular stimulation led to desynchronization of cardiac ventricles activation, as a result of which LV was stimulated only when RV was completely depolarized (Table 1).

Transmural ARI gradient in RV did not change during AVS in comparison with the supraventricular rhythm (control). The LV base was characterized by shorter ARI in the intramural and subendocardial layers (Fig. 1). Changes in ARI in the intramural and subendocardial layers during AVS were presumably caused by an increase in the current

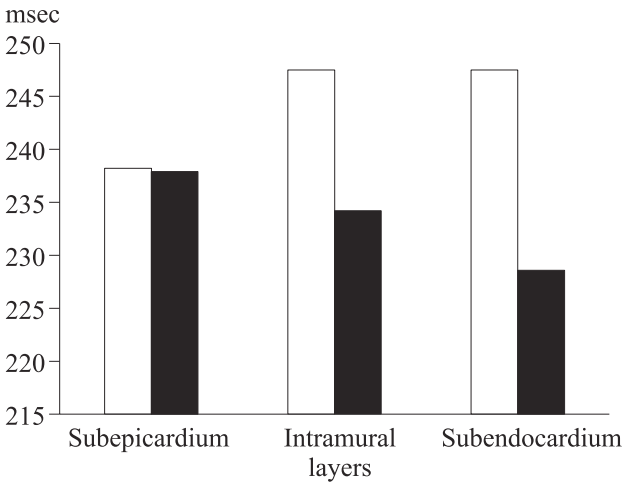

Fig. 1. Transmural ARI gradient in the LV base myocardium in supraventricular rhythm (light bars) and AVS (dark bars).

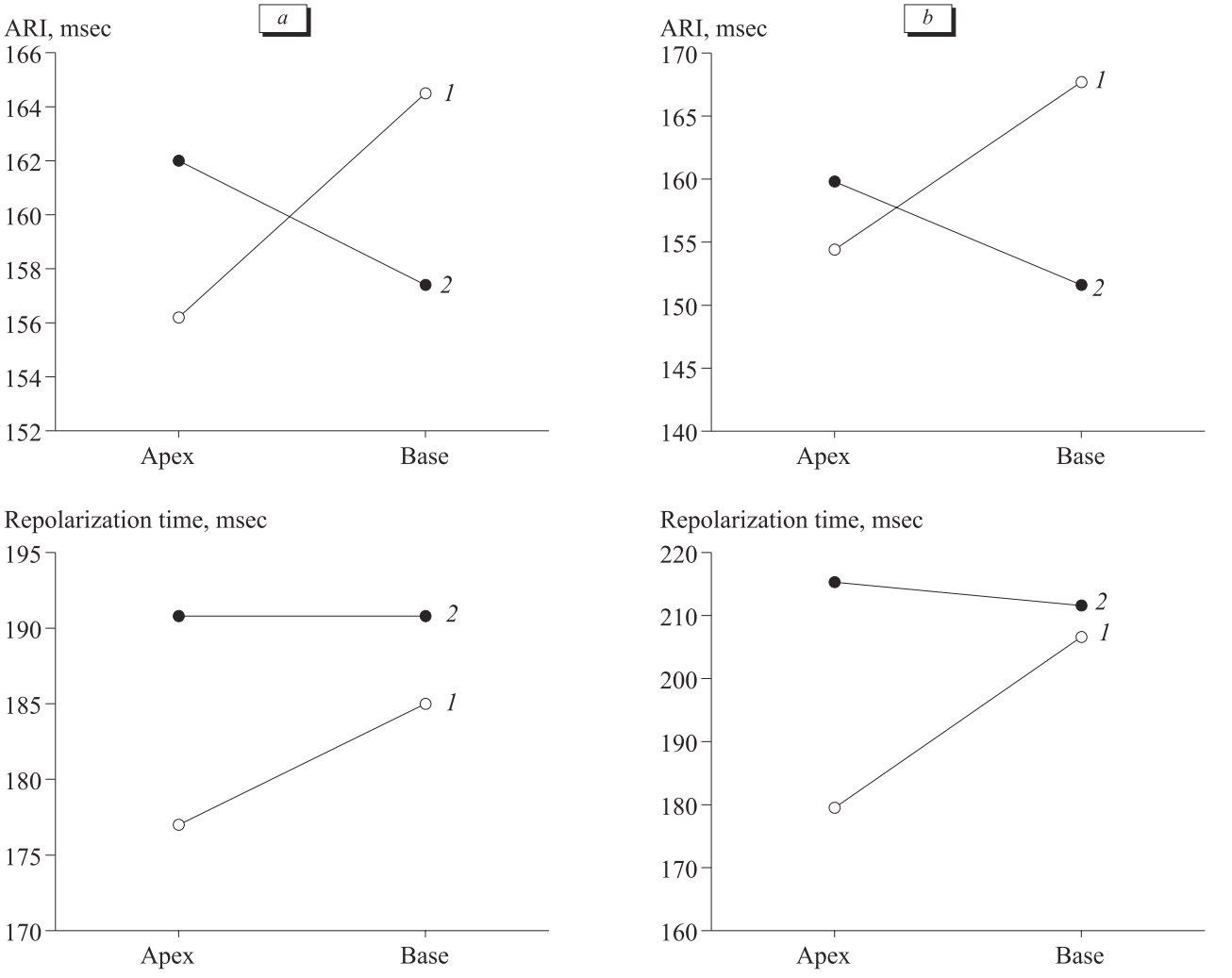

Fig. 2. Apicobasal gradients of ARI distribution and repolarization time in RV (*1*) and LV (*2*) in supraventricular rhythm (*a*) and AVS (*b*).

TABLE 1. Electrophysiological Parameters of Dog Ventricular Myocardium in Supraventricular Rhythm and AVS (msec; *M*±*s*)

Parameter		Supraventricular rhythm	AVS	*p*
Total dispersion of activation		29.9±7.3	57.1±19.4	$p<0.05$
Myocardial activation dispersion	LV	25.9±6.9	35.5±6.0	—
	RV	21.4±6.0	29.5±14.3	—
Maximum time of myocardial activation	LV	34.1±6.6	70.0±15.5	$p<0.0002$
	RV	30.1±5.2	40.5±12.0	$p<0.02$
Minimum time of myocardial activation	LV	8.2±3.5	31.5±15.6	$p<0.0004$
	RV	8.7±2.7	11.0±9.0	—
Total dispersion of ARI distribution		75.1±12.1	88.2±31.3	$p<0.05$
Dispersion of ARI distribution in myocardium	LV	64.8±11.8	70.1±31.7	—
	RV	49.0±17.5	52.7±29.5	—

density (I_{Ks}) [4] and inversion of the current gradient (I_{Kr}), respectively [5]. Left ventricular apical myocardium possesses no transmural heterogeneity of ARI in supraventricular rhythm and in AVS.

The RV myocardium was characterized by an increase in ARI duration from the apex to the base ($p<0.005$) due to the transmural gradient of the current (I_{to}) [7,8]. The ARI in the LV myocardium increased from the base to apex ($p<0.05$) due to the transmural current gradient [8]. The apicobasal gradient of ARI distribution in the RV and LV in AVS did not change in comparison with the supraventri-

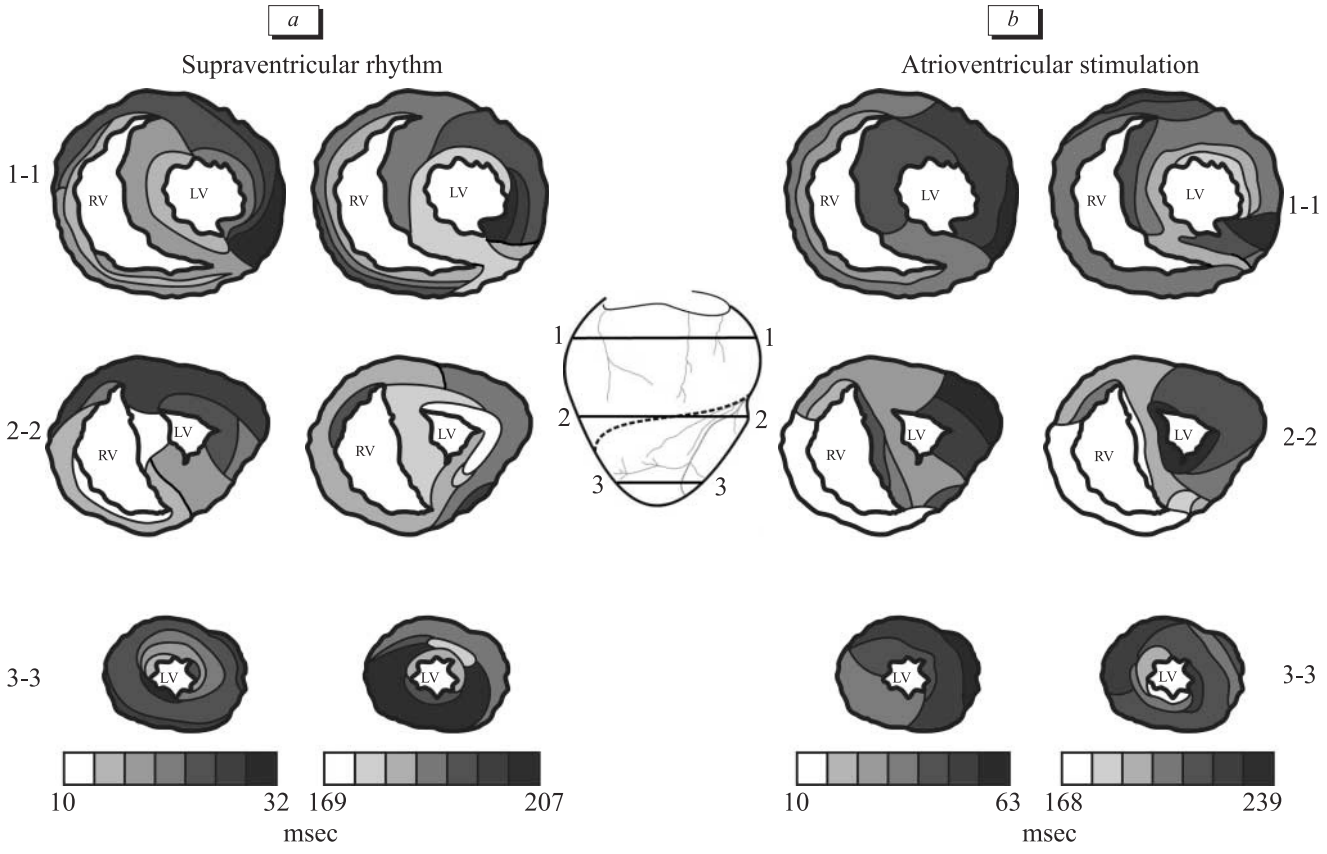

Fig. 3. Sequences of excitation (*a*) and repolarization (*b*) of dog ventricular myocardium in supraventricular rhythm and AVS. Time starting from the beginning of *QRS* complex in 2nd standard lead. 1-1, 2-2, 3-3: planes in which intramural multipolar needles were injected into ventricular myocardium.

cular rhythm (Fig. 2). Atrioventricular stimulation increased ($p<0.05$) total ventricular ARI dispersion, but did not change the individual RV and LV myocardial ARI dispersion (Table 1).

No appreciable transmural differences in the repolarization time in the RV myocardium and LV apex during AVS were detected. The subendocardial layers of the myocardium were the first to be repolarized in the LV basal myocardium, while the subepicardial layers repolarized the last ($p<0.05$). Similarly as in supraventricular rhythm, the transmural sequence of repolarization in the myocardial areas without transmural gradient of ARI distribution repeated the activation sequence.

No apicobasal gradient of repolarization was detected in LV myocardium; in RV repolarization started in the apex and passed to the base (Fig. 2). Atrioventricular stimulation led to an increase in ventricular myocardial activation and repolarization time in comparison with the supraventricular rhythm (Fig. 3; $r=0.18$ and $r=0.37$, respectively), but the relationship between repolarization time and ARI remained significant ($r=0.73$ and $r=0.56$, respectively). The main cause of the appearance of relationship between repolarization and activation is alteration of ARI duration, while inhibition of stimulation conduction in ventricular myocardium due to myogenic depolarization of cardiac ventricles can be the second cause [1].

Hence, AVS is associated with desynchronization of excitation of cardiac ventricles: excitation of LV begins only when RV is completely depolarized. The duration of ARI intervals in the intramural and subendocardial layers of LV base decreased. As a result, the transmural gradient of repolarization lengths in this area is altered and the heterogeneity of ARI distribution increases. Overall sequence of repolarization starts partially repeating the activation sequence. These changes can serve as an early marker of disorganization of the ventricular pumping function in electrocardiostimulation.

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