Repolarization of Epicardial Ventricular Surface of Rabbit Heart in Acute Stenosis of the Aortic Arch

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 146, No. 8, pp. 144-146, August, 2008
Original article submitted December 24, 2007

Overload of the cardiac left ventricle causes opposite local changes in repolarization duration (activation-recovery intervals) on the right- and left-ventricular epicardium, which depend on the duration of overload. The activation—recovery intervals on the right-ventricular lateral surface decrease after 1-min overload, but increase after 10-min overload. The length of activation—recovery intervals on the lateral surface of the left-ventricular apex increases after 10-min aortic stenosis more markedly in comparison with that after 1-min overload. Decrease/increase of local lengths of activation—recovery intervals results in modification of the general sequence of cardiac ventricular surface repolarization.

Key Words: *cardiac ventricles; aortic stenosis; repolarization; rabbit*

Mechanical load changes the duration of cardiomyocyte repolarization [4]. Pressure and/or volume overload of the heart can lead to electrophysiological changes, causing arrhythmias [3]. The degree and direction of mechanoelectrical effects in the heart are determined by the duration of exposure [6] and myocardial region [2]. The majority of studies of the effects of mechanical load on electrical characteristics of the heart were carried out on isolated heart [7] and myocardial strips [1].

We studied local duration of activated status in various ventricular sites and evaluated the sequence of ventricular epicardial surface repolarization in 1- and 10-min acute stenosis of rabbit aorta.

MATERIALS AND METHODS

The study was carried out on 15 adult male and female Chinchilla rabbits. The animals were intraperitoneally narcotized with urethane (1.5 g/kg), after which were intubated, transferred to artificial respiration, and the chest was opened. Electrical potentials were recorded through 64 electrodes in unipolar epicardial leads placed on rabbit cardiac ventricles. The electric field of the heart was studied under conditions of spontaneous sinoatrial rhythm and the temperature was maintained at the level of 38°C. Ventricular overload was induced by stenosing (ligation) of the aortic arch, the systolic blood pressure (SBP) in the aorta being elevated to 150 mm Hg for 1 and 10 min. The initial status of animals (before aortic arch stenosis) served as the control.

The time of epicardial activation was evaluated by the dV/dt_{min} value during *QRS*, repolarization moment by dV/dt_{max} value during *ST-T*, and duration of activation—recovery interval (ARI) by the interval between activation and repolarization moments [5]. Corrected ARI (ARIc) was estimated by the formula: $ARIc=ARI/RR^{1/2}$. Differences between the time parameters were evaluated using Student's *t* test with Bonferroni correction. The differences were considered significant at $p<0.05$.

RESULTS

Depolarization of the epicardium before overload starts in the middle part of the right ventricle (RV)

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0007-4888/08/1462-0180

and apical part of the left ventricle (LV); activation wave then propagates along ventricular surfaces and the last place it reaches is LV base.

The shortest ARI are located on the apex of the heart ($p<0.01$) and the longest in RV base and lateral surface (Table 1). ARI on LV base and free wall is longer than on its apex ($p<0.01$) and shorter than on RV base and lateral surface ($p<0.01$). On the whole, local ARI on LV are much shorter than on RV ($p<0.01$). Repolarization sequence correlates with distribution of ARI values. Foci of the earliest repolarization are located on LV apex. The lateral surface of LV and its base are repolarized later ($p<0.01$). The last in the sequence is repolarization of RV middle part of free wall and base. Hence, the total sequence of ventricular epicardial repolarization is directed from LV to RV and from the heart apex to base.

After stenosing of the aortic ostium for 1 and 10 min (SBP=150-160 mm Hg) heart rate decreases from 236±24 to 209±29 and 214±18 bpm, respectively ($p<0.03$; $p<0.004$). The sequence and depolarization virtually do not change under these conditions. Epicardial activation starts in the median part of RV free wall and/or LV apical part and propagates over the entire ventricular surface. Cardiac ventricular bases are depolarized the last.

The lengths of ARI on LV and RV epicardium change significantly during repolarization (Fig. 1). The direction of changes in ARIc on RV lateral surface depends on the duration of overload. ARI decreases by 8±1 msec ($p<0.01$) after 1-min aortic stenosis, while after 10-min overload it increases by 16±6 msec in comparison with the control ($p<0.05$). After 1-min stenosis, ARIc on the heart apex and LV free wall adjacent to it virtually do not change, while after 10-min stenosis they are 28±18 msec longer ($p<0.01$).

The main regularities in repolarization sequence are retained after 1-min overload: the apical areas are repolarized before the base ($p<0.001$) and LV repolarized before RV ($p<0.01$). Ten-min overload leads to the appearance of a delayed repolarization zone on the apex; on the whole, the heart apex is repolarized 17±10 msec later compared to the control ($p<0.01$).

Hence, acute stenosis of rabbit aortic ostium involves local changes in ARIc duration on ventricular epicardium, depending on length of overload. Activation-recovery intervals on RV decrease after 1-min LV overload, while on LV they remain unchanged. Longer stenosis (10 min) leads to an increase in ARIc lengths on the epicardium of both ventricles. As a result, changes in local lengths of ARI involve modification of the entire sequence of repolarization of ventricular epicardial surface.

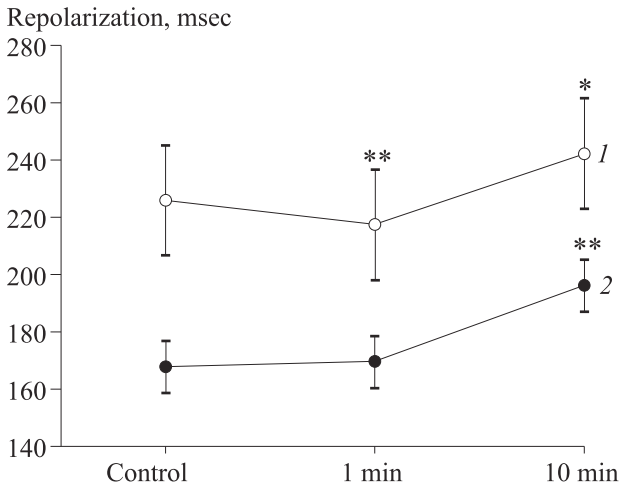

Fig. 1. Local changes in the epicardial activation-recovery intervals during LV load for 1 and 10 min. *1*) RV lateral zone; *2*) LV apex. *$p<0.05$, **$p<0.01$ compared to values before overload.

TABLE 1. Duration of Activation—Recovery Intervals (ARIc) in Different Epicardial Zones of Rabbit Cardiac Ventricles in Health and LV Overload

Area	ARIc, msec	Before overload	
		1 min	10 min
RV	230±13	222±4	235±24
LV	188±12	176±11	197±15
RV base	225±20	221±10	241±20
LV base	192±10	182±12	205±20
RV lateral surface	231±18	227±5	245±24
LV lateral surface	189±13	179±11	186±12
Heart apex	171±12	170±19	207±24

The study was supported by the Integration Project of Basic Studies of the Ural and Far Eastern Divisions of Russian Academy of Sciences.

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