Time Correlation Between Initial Activation of Ventricular Myocardium and Cardiac Electric Potentials on Body Surface in Dogs

V. A. Vityazev, S. N. Kharin, Ya. E. Azarov, K. K. Mostivenko, N. A. Antonova, I. M Roshchevskaia, D. N. Shmakov, and M. P. Roshchevskii

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 131, No. 4, pp. 388-391, April, 2001
Original article submitted December 13, 2000

Parameters of cardioelectric field on body surface and propagation of excitation in ventricular myocardium during initial activation were studied using multichannel synchronous electrocardiotopography. It was shown that inversion of areas of negative and positive potentials of cardioelectric field on body surface at the moment of excitation propagation to the epicardium reflected changes in the main direction of excitation wavefront in ventricles.

Key words: *electrocardiotopography*; *cardiac electric field*; *activation of the myocardium*; *intramural layers of the myocardium*; *potential distribution*

Foci of initial excitation, from which excitation and contraction waves spread through the ventricular myocardium (VM), are located near the Purkinje-myocardium contacts. These areas play a crucial role in normal cardiac function.

The initial ventricular depolarization corresponds to the *Q* wave on ECG. High diagnostic value of *Q* wave in cardiac pathologies associated with abnormal excitation propagation during initial ventricular activation was reported [5,7]. Multichannel synchronous recording of electric cardiac potentials is widely used in clinical investigations of the heart along with conventional ECG. High diagnostic value of body surface potential mapping was now established for a variety of cardiac pathologies [1,6,9,10]. The aim of the present study was to correlate the pattern of ventricular depolarization with parameters of cardiac electric field at the body surface at the period corresponding to the *Q* wave on ECG. The study was performed on dogs, because the pattern of activation in these animals is similar to that in humans [4].

MATERIALS AND METHODS

Cardiac electric potentials (CEP) at the body surface and extracellular potentials in the intramural layers of the ventricular myocardium were recorded in the supine position in 11 adult male and female mongrel dogs weighing 6 to 40 kg. The animals were anesthetized with sodium thiopental (50 mg/kg, intraperitoneally).

CEP were synchronously recorded from 64 leads placed uniformly on the body surface (Fig. 1, *a*), plotted as instantaneous equipotential maps, and analyzed.

The pattern of VM activation was studied using modified intramural multipolar needle electrodes [3]. Intramural needles were introduced into VM in five planes parallel to the heart base (Fig. 1, *b*) Unipolar electrograms were recorded synchronously from 64 electrodes positioned in a selected ventricle section plane. The moment when excitation wavefront reached a recording point corresponded to the maximum rate of voltage decrease [8].

Laboratory of Physiological Information of Biophysical fields, Institute of Physiology of the Komi Science Center, Ural Division of the Russian Academy of Sciences, Syktyvkar

0007-4888/04/1314-0327$25.00

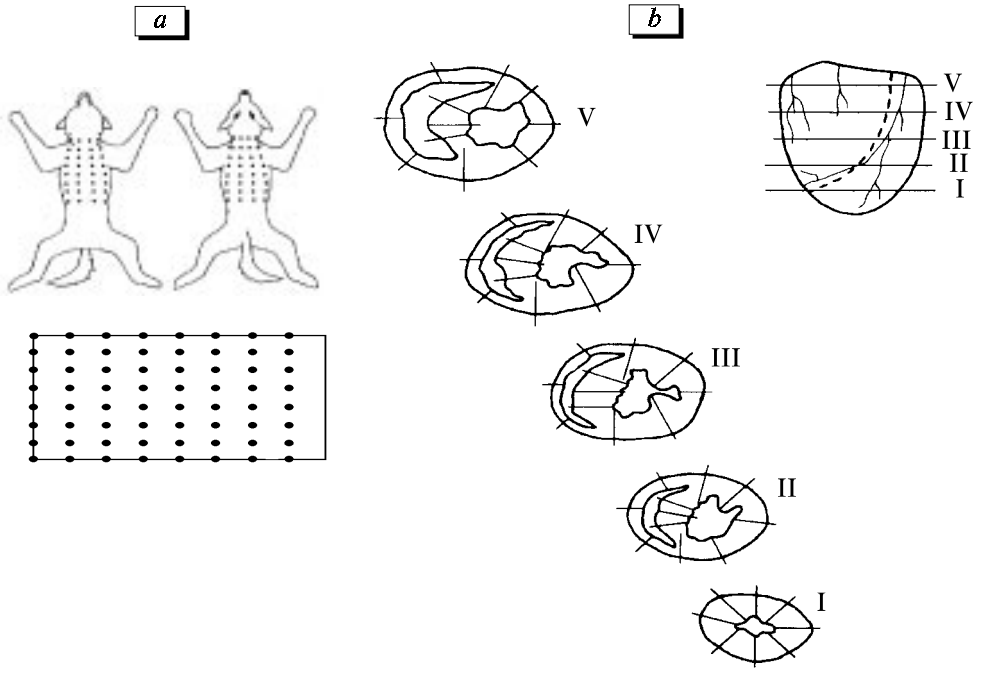

Fig. 1. Position of recording electrodes on body surface and its rectangular planar scheme corresponding to the format of isopotential maps (*a*) and position of multipolar intramural needle electrodes in ventricles (*b*). Here and on Fig. 2 and 3: I, II, III, IV, V denote ventricular sections from apex to base.

In order to correlate cardiac potentials at the body surface with the pattern of VM activation *QRS* complex in lead I was used as a reference potential.

RESULTS

The earliest foci of ventricular activation in dogs were small areas in the intramural layers (close to the endocardium) of the lower (apical) part of the interventricular septum adjacent to the left ventricle (Fig. 2, *a*). The area of positive potential spread over the cranial portion of the ventral and a part of dorsal body surface with a maximum located on the ventral body surface cranially to heart base projection (Fig. 2, *b*). The rest thorax surface was negative with the maximum on the left lateral body surface within projection of the heart ventricles. The time interval corresponded to baseline just before the *Q* wave in lead I (or before *R* wave if *Q* wave is absent). In our experiments *Q* wave in lead I was recorded in 35% animals, which is normal for ECG recorded in a supine dog under narcosis [2].

Ten-twelve milliseconds after the start of activation, the lower (apical) third of the interventricular septum from the left ventricular to right ventricular endocardium was involved in depolarization spreading in intramural layers of VM (Fig. 3, *a*). At this term, all myocardial layers in the apical portion of the right ventricle are involved into excitation, and the front of depolarization attained the epicardium surface. The time interval from the start of activation to excitation of the epicardium is characterized by inversion of positive and negatives areas on thorax surface: the positive area moved caudally relatively to the negative area (Fig. 3, *b*); ventral thoracic surface was primarily occupied by positive, while dorsal surface by negative areas with positive and negative maxima located on the ventral and dorsal surfaces, respectively. This moment corresponded to the peak of *Q* wave, or the very beginning of the ascending *R* wave(in the absence of the *Q* wave). Inversion of positive and negative areas

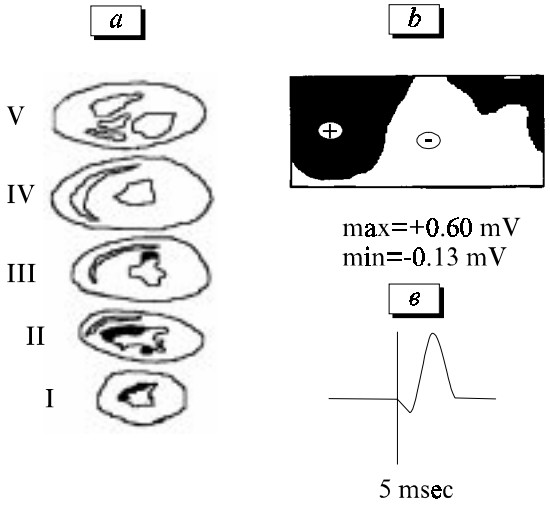

Fig. 2. Excitation of ventricular myocardium (*a*) and distribution of potential on body surface (*b*) at the beginning of ventricular depolarization. Here and on Fig. 3: black areas correspond to the regions of excitation in the myocardium (*a*) and areas of positive potential on the map (*b*). Symbols "+" and "-" indicate maxima of positive and negative potential (the corresponding values are given under the map). The mark on ECG lead I indicates the time passed from the beginning of ventricular excitation (*c*).

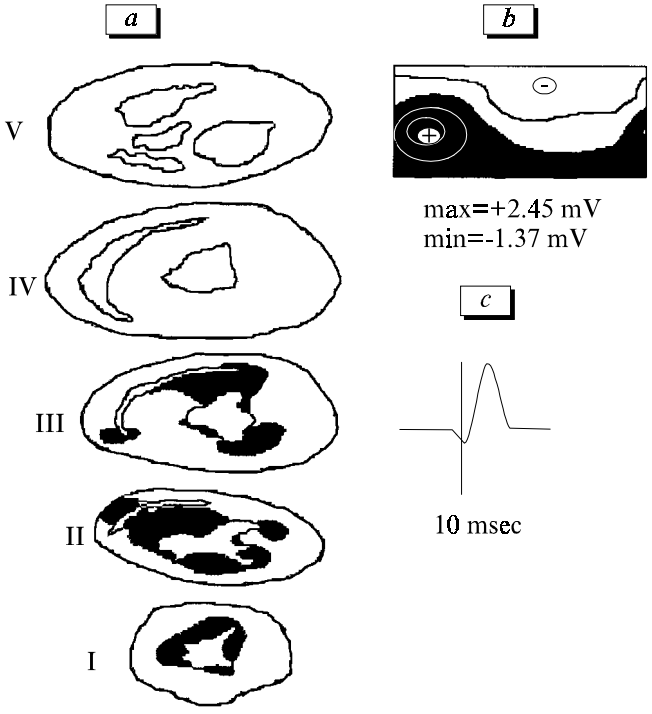

Fig. 3. Ventricular excitation (*a*) and distribution of potential on body surface (*b*) 10 msec after appearance of the primary excitation foci in ventricles.

on the thorax surface was completed 10-12 msec after the beginning of activation. The resultant CEP distribution is characteristic of the ascending phase or the peak of *R* wave recorded in lead I. At the early stages of activation, depolarization moved from endocardial primary excitation foci to the epicardium, *i. e*. from the internal to external myocardial layers. When excitation involved the epicardium, depolarization wavefront changed its direction and moved towards the heart base.

Our findings indicate that mapping of body surface potential is a more sensitive way to study electric activity associated with local myocardial depolarization at initial moments of activation. Conventional ECG recorded from extremities does not reflect bioelectric activity generated in primary excitation foci located in the intramural layers of the myocardium, but this activity can be clearly seen on instantaneous isopotential maps of CEP distribution on the body surface (Fig. 2, *a* and *b*). Inversion of negative and positive potential areas on body surface at the moment corresponding to wave *Q* (or beginning of the wave *R* when *Q* is absent) occurs when the excitation wave reaches the epicardium, and the wavefront of ventricular depolarization changes its main direction.

REFERENCES

1. V. P. Nuzhnyi. *Cardiac Electric Field at Thoracic Surface in Humans and Dogs with Implanted Cardiac Stimulator System*, Abstract of Cand. Biol. Sci. Dissertation, Syktyvkar (1997).
2. A. M. Khil'kin and V. A. Svetlov, *Experimental Model of Cardiac and Vascular Lesions* [in Russian], Moscow (1979).
3. D. N. Shmakov, Method of Needle Electrode Preparation, *Byull. Izobret*, No. 28, (1993).
4. D. Durrer, R. van Dam, G. E. Freud, *et al*., *Circulation*, **41**, No. 6, 899-912 (1970).
5. C. Fisch, *ACC Cur J. Rev*, **6**, No. 3, 71-75 (1997).
6. L. S. Green and J. A. Abildskov, *Clin Cardiol*., **18**, 245-249 (1995).
7. T. C. Mathew, Sh. Lakshmiprasad, and D. H. Spodick, *Am. J. Cardiol.*, **82**, 809-811 (1998).
8. M. S. Spach, R. C. Barr, G. S. Serwer, *et al*., *Circ. Res.*, **30**, 505-519 (1972).
9. B. Taccardi, *J. Electrocardiol.*, **23**, Suppl. 150-154 (1990).
10. T. Yung-Zu, T. Chuen-Den, L. Huey-Ming, *et al*., *J. Electrocardiol.*, **23**, No. 2, 127-136 (1990).