SIMULATION OF VENTRICULAR TACHYCARDIA WITH A LENGTHENED Q–T INTERVAL

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Raised sympathetic tone plays an essential role in the genesis of ventricular arrhythmias and sudden death. Enhanced functional activity of the left stellate sympathetic ganglion in patients with an elongated Q–T interval syndrome contributes to the development of ventricular arrhythmias which lead to fibrillation of the heart [6]. The treatment of such patients is aimed at blocking sympathetic influences on the myocardium either by means of drugs or by surgical methods through left-sided stellectomy [2, 5, 7]. Practical experience shows, however, that the suggested methods of treatment are not always effective, and patients with an elongated Q–T interval syndrome often develop attacks of ventricular tachycardia during or after treatment [3].

The aim of the investigation was to create a model of vetricular tachycardia against the background of an elongated Q–T interval, taking account of modern views on the pathogenesis of this pathological form.

EXPERIMENTAL METHOD

Experiments were carried out on 12 dogs weighing 10-15 kg and anesthetized with hexobarbital (0.7 mg/kg). The aorta was catheterized through the left carotid artery, the coronary sinus through the right jugular vein, and the superior vena cava through the left jugular vein. After intubation of the animal, sternotomy was performed. The left and right stellate ganglia were isolated. To stimulate the stellate ganglia electrically, a Soviet SUNS-01n stimulator was used, with bipolar electrodes, voltage 8 V, pulse frequence 50 Hz, and time constant 0.3 msec. The duration of stimulation was 60 sec and of the pause 30 sec. The arterial and venous pressure and the ECG in three standard leads were recorded on a "Galileo" polygraph (Italy). Plasma levels of potassium, sodium and calcium were determined in arterial blood and blood from the coronary sinus on a No. 614 potassium–sodium electrode analyzer ("Corning," England). To reproduce a model of an elongated Q–T interval, cesium

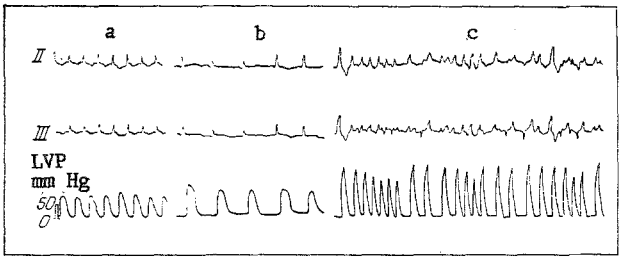

Fig. 1. Polymorphic ventricular tachycardia. a) Initial state; b) 1 min after cesium chloride infusion; c) stimulation of left stellate ganglion against the background of cesium chloride. II, III) ECG leads. LVP) Left ventricular pressure. Here and in Figs. 2 and 3, paper winding speed 25 mm/sec.

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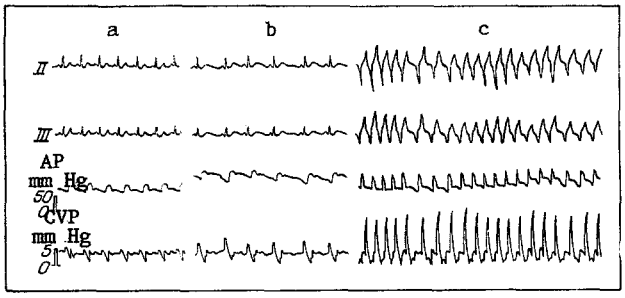

Fig. 2. Paroxysmal ventricular tachycardia. AP) Pressure in aorta, CVP) pressure in superior vena cava. Remainder of legend as to Fig. 1.

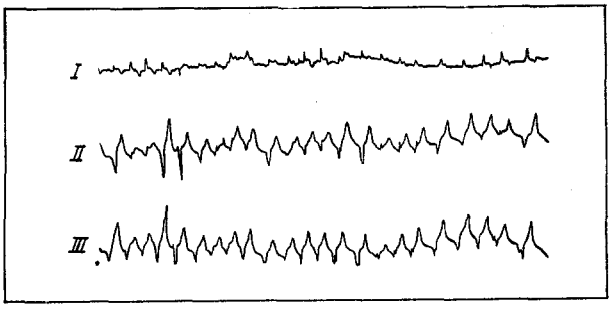

Fig. 3. Ventricular tachycardia of "pirouette" type in response to stimulation of left stellate ganglion and against the background of cesium chloride. I, II, III) ECG leads.

chloride was injected through the catheter in the left jugular vein in a dose of 1 mmole/kg [4].

EXPERIMENTAL RESULTS

Intravenous infusion of cesium chloride into the dogs was followed by changes in the hemodynamics, the cardiac rhythm, and the electrolyte balance of the heart (Figs. 1 and 2). From the first minute after injection of the compound sinus arrhythmia developed, with a heart rate of 70-126 beats/min. In five experiments ventricular extrasystoles of the bigeminy type were observed. A considerable rise of arterial pressure, both systolic and diastolic, was observed (on average of 60%). The central venous pressure was doubled compared with initially. Considerable changes were observed during the study of myocardial electrical activity. A characteristic feature of the action of cesium chloride was the lengthening of electrical systole of the myocardium: the Q–T interval. During ECG analysis the corrected Q–T interval also was used, for this eliminates any dependence of the duration of electrical systole on changes in heart rate [1]. On average the corrected Q–T increased by 32%. The ST segment was displaced below the isoelectric line and passed into a flattened and widened positive or biphasic T wave, which often was merged with a U wave when it appeared. The plasma potassium concentration in arterial blood and blood from the coronary sinus rose injection of cesium chloride from 3.82 to 8.08 mmoles/liter and from 3.36 to 8.71 mmoles/liter, respectively. The plasma calcium and sodium levels were unchanged. Comparison of the ECG data with the plasma electrolyte concentrations showed the presence of myocardial hypocaliemia, one cause of which could be depression of the potassium flows in response to cesium chloride injection [4]. Marked changes in the hemodynamics and electrical activity of the heart were observed during the first 5-7 min after cesium chloride infusion, but these changes disappeared 15 min after its infusion.

Stimulation of the left stellate ganglion in the first 5 min after cesium chloride infusion not only increased the force and frequency of the cardiac contractions, but also led to the development of ventricular tachyarrhythmia in 10 of the 12 dogs, appearing 8-10 sec after the beginning of stimulation and continuing for 27 min. In three experiments the ventricular arrhythmia was more stable and lasted 40 sec. During analysis of the rhythm dis-

turbances, polymorphic ventricular tachycardia was observed most frequently (Fig. 1c). Various ventricular complexes, differing in shape and duration, were observed on the ECG and the heart rate was 141-226 beats/min. The arterial pressure varied from 180/127 to 150/110 mm Hg. Paroxysmal ventricular tachycardia with a more or less regular rhythm of 200-220 beats/min was observed in two animals (Fig. 2c). In some experiments, ventricular flutter developed, described by the name of "pirouette" (Fig. 3). After stopping the stimulation of the left stellate ganglions, ventricular tachyarrhythmia continued for a further 15 sec, after which sinus rhythm was restored. In two of the 12 experiments stimulation of the left stellate ganglion was accompanied only by a positive ino-chronotropic effect without any disturbances of rhythm. In these experiments cesium chloride caused virtually no increase in the Q–T interval. Stimulation of the right stellate ganglion in dogs with an elongated Q–T interval caused ventricular tachycardia in only two cases. The different effects of the left and right sympathetic ganglia on the rhythmic activity of the heart may depend on the topography of their nerve fibers in the myocardium. The right sympathetic nerves are connected mainly with the sinus node and they control the heart rate, whereas the left are connected mainly with the atrioventricular node and with the ventricles. Stimulation of the right stellate ganglion in the present experiments was accompanied by an increase in the heart rate of more than 50%. The fast rhythm of the sinus node, however, can inhibit ventricular ectopic beats. Increased activity of the right stellate ganglion is also known to raise the threshold of ventricular fibrillation. High tone of the left stellate ganglion lowers the threshold of fibrillation [6]. The blood plasma potassium concentration in these experiments remained high during stimulation of the stellate ganglia, namely 7.15-8.2 mmoles/liter.

Thus, electrical stimulation of the left stellate ganglion against the background of electrical instability of the myocardium led in 83% of cases to the appearance of stable, polytopic ventricular tachycardia. The model created provides evidence of the role of intracardiac pathology (electrolyte imbalance), combined with increased activity of the left stellate ganglion, in the mechanism of ventricular arrhythmias associated with an elongated Q–T interval.

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