

The aim of the study was to evaluate the complex determination of predictors of “arrhythmogenic cardiomyopathy” in patients with ventricular extrasystoles (VE) without structural heart changes to predict the development of cardiovascular diseases in a prospective study.

**Methods.** Experimental study. Rats were used to model VE by the mechanism of early postdepolarization (aconitine arrhythmia), rabbits — for delayed postdepolarization (barium chloride-induced arrhythmia), and dogs — for re-entry peroxide arrhythmia. In the electrocardiogram (ECG), in addition to the conventional parameters, the pre-excitation interval, its variability and the index of intrinsic deviation of VE (IDVEi) were analyzed. **Clinical study.** We observed 412 patients without structural changes of the heart aged from 16 to 43 years (mean  $28.4 \pm 0.8$  years), and the number of VEs per day of observation ranged from 6157 to 37254 (mean  $19706 \pm 656$  VEs). The same parameters were determined by the ECG as in experimental arrhythmias: they were calculated separately for mono- and polymorphic left- and right ventricular extrasystoles (LVE and RVE). The duration of patient follow-up was up to 10 years. Results. In the modeling of ventricular arrhythmias by the mechanism of delayed postdepolarization, polymorphic VE, early postdepolarization — early monomorphic VE, re-entry — early and late monomorphic VE were registered. In the animals with the modeling of arrhythmia by the mechanism of re-entry IDVEi was significantly higher in comparison with VE caused by the mechanisms of early and delayed postdepolarization. The main predictors of “arrhythmogenic cardiomyopathy” in patients without structural changes of the heart with VE, which determine the development of organic heart pathology, such as coronary heart disease (CHD) and mitral valve prolapse (MVP), are IDVEi and QRSVE complex duration. Increased values of these parameters ( $>0.42$  units and 148 m/s, respectively), characterize the risk group of cardiovascular pathology formation. The development of CHD in patients without structural heart changes with VE highly correlated with  $IDVEi \geq 0.56$  units, duration of QRSVE complex  $\geq 157$  m/s in monomorphic LVE, use of class III drugs. The development of MVP in these patients highly correlated with duration of QRSVE complex  $\geq 159$  m/s in polymorphic VE, efficacy of class I drugs and to a lesser extent of the class III drugs.

**Conclusion.** In patients without structural heart changes with VE, the increase in IDVEi values and QRSVE complex duration  $>0.48$  units and 149 m/s, respectively, determine the risk group of cardiovascular pathology formation. In patients without structural heart changes with VE, the development of CHD highly correlated with  $IDVEi \geq 0.56$  units, QRSVE complex duration  $\geq 157$  m/s in monomorphic LVE, and MVP — with QRSVE complex duration  $\geq 159$  m/s in polymorphic VE.