

# THE EFFECTS OF VARIOUS SWIMMING TRAINING PROTOCOLS ON CARDIAC CAPACITY AND VENTRICULAR FIBRILLATION THRESHOLD IN RATS

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**Abstract.** Irregular heartbeats and different forms of ventricular ectopic activity are a common occurrence among elite athletes with high contractile cardiac capacity. At the same time, experiments demonstrated that the electrical stimulation threshold, causing ventricular fibrillation, increases during adaptation to physical exercise, without the increase in the contractile cardiac capacity. The research purpose is to examine the dependence of ventricular fibrillation threshold and contractile cardiac capacity on intensity and duration of swimming sessions, as well as duration of the training period. Female Wistar rats were assigned to five groups: sedentary (S), training 1 (T1, low intensity), training 2 (T2, moderate intensity), training 3 (T3, long-term), training 4 (T4, exhaustive). At the end of the experimental period, the rats were anesthetized and their ventricles irritated with rectangular pulses of 10 ms duration, to determine the minimum current causing ventricular fibrillation. The cardiac capacity was assessed by the maximum pressure in the left ventricle, at full aortic-cross clamping. The ventricular fibrillation threshold was increased by 60% in T1, 57.5% in T2 and 74% in T3, but no difference in T4 was observed, compared with S. The pick pressure in the left ventricle after aortic cross-clamping in T1 and T2 was not enhanced, compared with S; in T3 and T4, however, it was significantly increased. Physical exercise training changed the ventricular fibrillation threshold and cardiac contractile capacity, independently of the intensity of exercise. The rise of the ventricular fibrillation threshold and its contractile capacity can be demonstrated during a long adaptation to moderate-term sessions of aerobic exercises.

**Key words:** swimming training, ventricles, cardiac capacity, fibrillation

## Introduction

Elite athletes with clearly increased contractile cardiac capacity have irregular heartbeats (Ector et al. 1984), complex forms of ventricular ectopic activity and some ECG alterations, typical for ventricles repolarization abnormalities (Crawford et al. 1979). Athletes' sudden deaths during competitions (Maron et al. 2009; Marijon et al. 2011) can also be caused by various types of cardiac arrhythmia, in particular, ventricular fibrillation (Green et al. 1976). At the same time, experiments with isolated animal hearts demonstrated that electrical stimulation threshold,

causing ventricular fibrillation, increases during adaptation to physical exercise (Noakes et al. 1983). Whole-body experiments also demonstrated increasing resistance to fibrillation, accompanied by ischemic stroke (Hull et al. 1994) and myocardial infarction (Meerson et al. 1987). According to the views of recent authors, such effect appears only during moderate-intensity physical load with unaltered left ventricular systolic pressure after aortic cross-clamping, compared to controls. By contrast, fibrillation threshold caused by electric stimulation decreases with cardiac contractile capacity, enhanced as a result of excessive physical loads. Consequently, alterations in the contractile capacity and cardiac electrical stability caused by physical activity may have a different direction. It is not clear which training programs enhance improvement of both indicators, and what is the effect of the intensity and duration of exercise sessions and training period. The aim of the present study is to examine the dependence of ventricular fibrillation threshold and cardiac contractile capacity on exercise sessions intensity, and the duration of the training period.

## Materials and methods

### Animals care

All of the protocols and surgical procedures used were in accordance with the Association for Assessment and Accreditation of Laboratory Animal Care. Female Wistar rats (220 to 250 g,  $n = 50$ ) were used. The animals were weighed weekly and housed in five-per-cage units at a controlled room temperature (22°C) with a 12-h dark–light cycle, and fed standard rat chow *ad libitum*. The rats were randomly assigned to five experimental groups: sedentary control (S,  $n = 10$ ), swimming trained following protocol 1 (T1,  $n = 10$ ), swimming trained following protocol 2 (T2,  $n = 10$ ), swimming trained following protocol 3 (T3,  $n = 10$ ), and swimming trained following protocol 4 (T4,  $n = 10$ ), as described below.

### Exercise Training Protocols

Protocol 1 (T1, low intensity training) consisted of 60 min a day of swimming, five times per week for 8 weeks without overload. Protocol 2 (T2, moderate intensity training): animals performed the same training protocol as in T1, but swam with 2% to 7% body weight (BW) overload (1% increase per week until the 7th week). Protocol 3 (T3, long-term training) consisted of the same rules of conduct as T2 until the 8th week, from the 9th to 13th week rats continued swimming with 7% BW overload. Protocol 4 (T4, exhaustive training) was the same as T3 until the 10th week; in the 11th, 12th and 13th weeks rats swam 120 min a day, 180 min a day and 240 min a day respectively, with 7% BW overload. The density of water in the pool consisted of 0.45 m overload. Clothespins were attached to the animal's back closer to the head.

### Markers of aerobic training

#### Heart rate measurements

HR was determined from the ECG, recorded at the end of the experimental period in the drowsy state of the caged animals.

#### Cardiac hypertrophy measurements

Cardiac hypertrophy was assessed by measuring the ratio of the heart weight to body weight (HW/BW mg/g).

### Ventricular fibrillation threshold

After opening the chest, artificial lung ventilation ventricles irritated rectangular pulses of 10 ms duration at the minimum current (with Nihon Kohden – SEN -1101 stimulator), causing ventricular fibrillation.

### Cardiac capacity

Cardiac contractility was evaluated by the maximum pressure of the left cardiac ventricular with aortic cross-clamping, using “Mingograph-34” (Siemens-Elema). Catheter entered the ventricle through the top of the heart. Results were registered before and 30 seconds after the aortic cross-clamping.

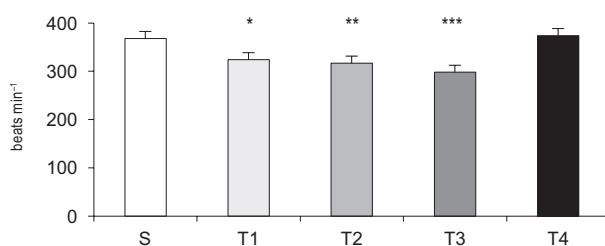
### Statistical analysis

The results are represented as mean  $\pm$ SEM for 10 rats per group. Data were initially analyzed for normality (Shapiro-Wilk test) and homoscedasticity. All analyzed variables presented normal distribution and homoscedasticity. Differences between groups were tested by independent Students t test. P values  $<0.05$  were accepted as statistically significant with a confidence level of 95%.

## Results

### Aerobic training markers

Figure 1 shows a significant HR decrease after the swimming protocol T1 ( $342 \pm 8$  beats  $\text{min}^{-1}$ ), T2 ( $317 \pm 11$  beats  $\text{min}^{-1}$ ), and T3 ( $298 \pm 7$  beats  $\text{min}^{-1}$ ) respectively, compared with group S ( $368 \pm 9$  beats  $\text{min}^{-1}$ ). There was no difference in group S in T4 ( $374 \pm 7$  beats  $\text{min}^{-1}$ ). The table shows that at the end of the experimental period HW/BW were the same in T1 ( $3.04 \pm 0.13$  mg/g) and in T2 ( $3.16 \pm 0.15$  mg/g), compared with group S ( $2.82 \pm 0.11$  mg/g). HW/BW increased significantly in T3 ( $3.38 \pm 0.12$  mg/g) and in T4 ( $3.42 \pm 0.15$  mg/g). An increase of the HW/BW in T4 was partly caused by the ongoing 3-week underweight of 8.8%, compared to T3.



**Figure 1.** Effect of aerobic training on rest HR. Groups: S – sedentary control; T1 – swimming training protocol 1; T2 – swimming training protocol 2; T3 – swimming training protocol 3; and T4 – swimming training protocol 4. Data are reported as means  $\pm$ SEM \* P  $< 0.05$  versus S; \*\* P  $< 0.01$  versus S; \*\*\* P  $< 0.001$  versus S.

### Ventricular fibrillation threshold

Table 1 shows significant differences between the four groups in the ventricular fibrillation threshold; T1, T2 and T3 had an increase of 61% ( $9.8 \pm 1.0$  mA), 57% ( $9.6 \pm 1.2$  mA) and 74% ( $10.6 \pm 0.8$  mA) respectively, compared with group S ( $6.1 \pm 0.6$  mA); in T4 ( $5.7 \pm 0.7$  mA) there was no difference in comparison with group S.

### Cardiac capacity

The pressure in LV before the aortic clamping was similar in all groups (92–97 mm Hg). After the complete aortic clamping (Table 1) it increased more than 2-fold and T1 ( $201 \pm 6$  mm Hg)/ T2 ( $202 \pm 9$  mm Hg) groups were not different from S ( $199 \pm 8$  mm Hg). In T3 ( $242 \pm 7$  mm Hg) and T4 ( $233 \pm 7$  mm Hg) groups, the maximum pressure in LV was 21.6% and 17.1% higher than that in S, respectively.

**Table 1.** Ventricular fibrillation threshold, left ventricular pressure and cardiac weight in different swimming training protocols (mean  $\pm$  SEM)

Animals groups (n)	Ventricular fibrillation threshold (mA)	Left ventricular pressure (mmHg)		Heart weight (mg)	Heart weight/ body weight (mg/g)
		before aorta clamping	after aorta clamping		
S (10)	$6.1 \pm 0.6$	$93 \pm 6$	$199 \pm 8$	$1050 \pm 40$	$2.82 \pm 0.11$
T1 (10)	$9.8 \pm 1.0^{**}$	$93 \pm 7$	$201 \pm 6$	$1110 \pm 38$	$3.04 \pm 0.13$
T2 (10)	$9.6 \pm 1.2^*$	$97 \pm 9$	$202 \pm 9$	$1141 \pm 41$	$3.16 \pm 0.15$
T3 (10)	$10.6 \pm 0.8^{**}$	$95 \pm 6$	$242 \pm 7^{***}$	$1184 \pm 36^*$	$3.38 \pm 0.12^{**}$
T4 (10)	$5.7 \pm 0.7$	$92 \pm 6$	$233 \pm 7^{***}$	$1121 \pm 45$	$3.42 \pm 0.15^{**}$

\*  $P < 0.05$ ; \*\*  $P < 0.01$ ; \*\*\*  $P < 0.001$  opposed to S.

### Discussion

As demonstrated above, moderate- and low-intensity training protocols did not elevate maximum systolic left ventricular pressure with aortic clamping. The results of other researchers (Kapelko et al. 1977) were the same during moderate physical exercise. When interpreting them, authors admitted that the expected increase of myocardial contractile capacity had not fully occurred during uncommon cardiac isometric load, caused by aortic clamping. However, it does not seem to be the only reason. First of all, the pressure on myocardial structure caused by aortic cross-clamping is not fully isometric, as coronary blood flow does not stop. Secondly, training including long-term and exhaustive physical exercise elevates left ventricular pressure to significantly higher level compared to controls. These data mean that workloads intensity and/or their total duration is the main increasing factor for the left ventricular contractility.

Indeed, according to the data (Krames et al. 1984), ventricular myocardium generates higher isometric tension during intensive physical exercises. At the same time, HB/BW was increased by 20%; it was partly caused by the underweight of BW. The dependence of the maximum left ventricular pressure with aortic clamping on heart mass is demonstrated in other studies (Kammereit et al. 1975). Increasing maximum pressure in our research was also demonstrated during long-term and exhaustive swimming exercise, causing HW/BW elevation by 19,9% and 21,3% respectively, compared to controls.

Furthermore, it is important to note that the aerobic swimming exercise of moderate duration and low to moderate intensity caused similar increasing level of the ventricular fibrillation threshold. Moreover, that effect was demonstrated after 8 training weeks i.e. within the time when maximum left ventricular pressure with aortic clamping had not yet been altered. When swimming training duration reached 13 weeks, further ventricular fibrillation threshold was not significantly elevated, but cardiac contractile capacity did increase. However, these two indicators can reach high level only in terms of relatively narrow range of physical activities. As demonstrated above, when swimming, exercise duration increases up to 120–240 min. At the same time, ventricular fibrillation threshold decreases and reaches control level in the short term, although myocardial contractile capacity stays high.

When evaluating the increase of ventricular fibrillation threshold during swimming, training of low and moderate intensity did not cause any significant myocardial hypertrophy, but it contributed to the increase of selective structure mass of sarcoplasmic reticulum, responsible for calcium transfer (Guski et al. 1981). During such physical exercise, total phospholipid number in myocardial cell membrane increases by a quarter and phosphatidylserine, responsible for sarcolemmal calcium binding, increases by 50% (Tibbits et al. 1984). During long-term physical exercise these alterations are aimed to increase, according to the level of ventricular fibrillation threshold. Cardiac contractile capacity grows correspondingly, owing to moderate myocardial hypertrophy (12.8%). This could be facilitated by such factors as increased capillary density in myocardium (Natan et al. 2012), increased calcium sensitivity of cardiomyocytes (Wisshoff et al. 2001), and inotropic effect mediated by beta- and alpha1-adrenergic cardiac receptors (Chinkin et al. 1987). When such positive phenomenon do not occur during exhaustive physical exercise, cardiac contractile capacity becomes higher, mainly through increased mobilization of catecholamines from adrenal glands, and activation of sympathetic nervous system (Sitdikov et al. 1987).

Thus, the development of alterations in ventricular fibrillation threshold does not depend on cardiac capacity alterations. Evidently, myocardial cell membrane is its most vulnerable structure, which changes properties more rapidly than contractile apparatus, experiencing both positive and negative impact. The rise of both ventricular fibrillation thresholds and its contractile capacity, can be demonstrated only during long adaptation to moderate-term sessions of aerobic exercises.

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