

# Effect of Exercise on Heart Rate: Curiosity Turned into an Experiment

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## ABSTRACT

The heart pumps and circulates the blood throughout the system, contracting and relaxing in a rhythm with each beat. The autonomic nervous system (ANS) controls involuntary body function (heart rate, BP, breathing, and more), and the sympathetic nervous system (SNS), a branch of ANS, is activated in response to exercise and stress. SNS activation releases hormones like epinephrine and norepinephrine, which cause an increase in heart rate involving specific receptors on heart cells. It is very well known how the nervous system controls heart function; this review article summarizes the findings on sympathetic regulation of heart rate. Of note, some pilot experiments were conducted in random groups of high school students to elucidate the effect of exercise on heart rate. A statistically significant increase in heart rate was observed after exercise.

## Introduction

### The Regulation of the Heart via the Sympathetic Nervous System

The sympathetic nervous system (SNS) is responsible for flight or fight response via the release of neurohormones (epi or norepinephrine). The parasympathetic nervous system (PNS) is responsible for rest and digestion via the release of acetylcholine. When the SNS is activated, it increases heart rate and contractions. It is to be noted that the sustained activation of SNS leads to pathological heart failure (Gordan R et al., 2015). The nervous system can cause an increase or decrease in the heart rate based on the hormones it releases. The sinoatrial (SA) node, a specialized group of cells in the heart, sets the rhythm of the heart rate and generates action potentials, which are electrical impulses that travel through the heart and cause it to contract. The rate and rhythm of these action potentials are influenced by the nervous system.

### Receptor-Mediated Regulation of Heart Rate

Neurohormones like norepinephrine and epinephrine, once released by SNS, bind to adrenergic receptors (ARs) specifically beta-1 and beta-2 on heart cells causing the heart muscle to contract with force (Gorelik J et al., 2013). Adrenergic receptors are members of the family of huge G protein-coupled receptors (GPCR (Motaiejunaite J et al. 2020). Heart cell's  $\beta_1$  adrenergic receptor ( $\beta_1$ AR) and  $\beta_2$  adrenergic receptor ( $\beta_2$ AR) are coupled to  $G_s$  to produce second messenger cyclic adenosine monophosphate (cAMP) which then leads to activation of protein kinase A (PKA). The activated PKA molecule in turn instigates various signals thereby regulating the heart rate (Fu Q et. al., 2015)

### Chronic Sympathetic Stimulation of the Heart

In normal physiological conditions, the SNS plays an important role in the maintenance of heart function via activation of specific ARs. Acutely, and if the heart functions properly, the activation of the ANS will promptly restore cardiac function. It is noted that uncontrolled activation of AR has bad long-term effects leading to pathological conditions such as hypertrophy and heart failure wherein hyperactive ANS will continue to push the heart to work at a level much higher than the heart muscle can handle eventually causing the heart to malfunction (Joca HC et. al. 2020; Lymperopoulos et. al., 2013). In heart failure, SNS is overactive throughout the body and can affect skeletal muscle function and reduce exercise capacity (Borovac JA et. al. 2020).

## **Pilot Study**

### **Aim**

To determine the heart rate of 44 high school students in resting and exercise mode.

## **Design & Methodology**

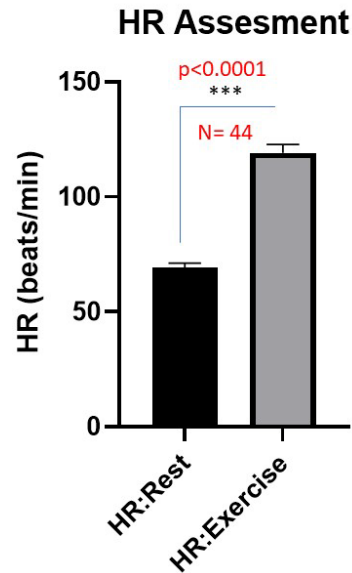
Forty-four random school students (N= 44, including males and females in the age group of 14-15 yrs.) were selected for the study, irrespective of their pre-existing heart conditions. Subjects were asked to measure their heart rate before and after exercise. The heart rate was measured as the number of contractions the heart makes per unit of time.

### **Data Analysis**

All data was analyzed using GraphPad Prism 9.5.1. Data are displayed as mean  $\pm$  SD (beats per minute). A paired t-test was used to compare between heart rate at rest and after exercise ( $p < 0.05$  considered significant).

## **Results**

We observed that the mean heart rate of students when resting was 69.05 beats per minute with a standard deviation of 13.97 beats per minute [69.05  $\pm$  13.97]. Interestingly, the mean heart rate of the students when exercising was 119.0 beats per minute with a standard deviation of 24.59 beats per minute [119.0  $\pm$  24.59]. We compared the above groups and observed a statistically significant increase in heart rate after exercise ( $p < 0.0001$ ).



**Figure 1.** Comparison of heart rate at rest and after exercise. Forty-four high school students' data on heart rate (beats per minute) was collected and analyzed. We observed a statistically significant increase in heart rate in the exercise group when compared with the rest group. Mean heart rate for the rest group  $69.05 \pm 13.97$  beats per minute, while  $119.0 \pm 24.59$  beats per minute for the exercise group.  $p < 0.0001$

### Supplemental Information

Excel file for raw data on heart rate for forty-four high school students during rest and after exercise is attached.

## Discussion

Exercise triggers an increase in norepinephrine, leading to increased calcium levels within cardiac myocytes via AR activation and second messenger cAMP production and augments the rate of contraction in the heart. Calcium plays a crucial role in the contraction of cardiac muscle. When calcium levels rise, it initiates a cascade of events that allow the cardiac myocytes to contract more forcefully and quickly. This increased contractility leads to a higher heart rate, which is reflected in the histograms showing a significant increase in heart rate after exercise (Figure 1). The sympathetic stimulation of the heart serves as a protective mechanism to ensure an adequate blood supply and oxygen delivery to cardiac muscles during exercise. By increasing the heart rate and cardiac output, the body can meet the increased metabolic demands of the exercising muscles. Of note, while sympathetic stimulation is beneficial during acute mode of exercise, its chronic or prolonged activation can have detrimental effects on heart function. Chronic SNS stimulation puts excess pressure on the heart, resulting in pathological conditions like hypertrophy (enlargement of heart cells), impaired relaxation between contractions, and problems in electrical conduction in the heart. Over time these changes can contribute to the development of heart failure and arrhythmias, a condition where the heart becomes unable to pump blood effectively and loses its electrical activity. In summary, the increase in heart rate after exercise is a result of sympathetic stimulation, which leads to elevated norepinephrine levels, increased calcium levels in cardiac myocytes, and subsequent enhanced cardiac contractions. Chronic stimulation can have detrimental effects on the heart and potentially lead to heart failure and arrhythmias.  $\beta$ -adrenergic receptor blockers are the main pharmacological approach for patients with heart failure with reduced ejection fraction (Paolillo et. al., 2020).  $\beta$ -blockers, which

specifically block  $\beta$ -adrenergic receptors, work by decreasing the activity of the heart via blocking the action of neurohormones.

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