How Cardiac Functions Can Be Affected in Toad Heart: The Relationship between Adrenaline and the Heart Rate

Since cardiovascular disease (CVD) has become the biggest burden of disease in some countries and the major cause of morbidity and mortality in the world, how cardiac functions can be controlled has become more and more considerable. The purpose of the study is to advance our knowledge on the relationship between the electrical and mechanical activity of the heart. This scientific report is about to investigate the consequences on drugs on cardiac physiology. The drug applied in the experiment is adrenaline whereas the animal model used is the heart of the cane toad (Rhinella marina). According to the previous background research, the research question can be stated that there is a positive relationship between adrenaline (IV) and the heart rate (DV) of the toad. The Electrocardiogram (ECG) test was employed in this experiment to assess activities of the heart. An unpaired, two tailed two-tailed, t-test with Welch’s correction was used to run the statistic analysis which also demonstrated that there was a significant difference between the group of treatment and control. Thus, it is found that the heart rate can be increased by adrenaline.

Key Words
Cardiac / Adrenaline / Electrical Activity / Mechanical Activity / Cane Toad / ECG / Heart Rate / Electrocardiogram
Introduction

Adrenaline is the main hormone of the Adrenal medulla (ADM) (Krzeminski, Cybulski & Nazar, 2009). Adrenal medulla would secrete adrenaline when there is a danger or stress. Since the adrenal medulla secretes adrenaline directly into the bloodstream, plasma adrenaline levels usually reflect neural outflow to the adrenal medulla (Ito, Sato, Shimamura & Swenson, 1984). This hormone has profound effects on the cardiovascular system which increases the heart rate and the stroke volume of the heart beat (Campbell, Reece & Meyers, 2006). This is because that adrenaline increases pacemaker current in the nodal cells, in that way raising the rate of depolarisation and thus requires less time to reach threshold. Moreover, adrenaline increases Ca2+ current in all myocardial cells which also makes the threshold more negative (Brown, Difrancesco & Noble, 1979). Nevertheless, adrenaline creates shorter action potentials accordingly of the actions it has on some specific currents (Boron & Boulpaep, 2009). Thus, adrenaline plays an important role to our key research objective that what effects adrenaline would have on the heart rate of the toad.

Adrenaline usually exerts an inotropic action which enhances myocardial contractility. The process linking depolarisation to contraction is called excitation contraction coupling which the contraction is determined and initiated by the rises of intracellular Ca2+ (Ward & Linden, 2008). Additionally, contraction and relaxation times are significantly affected by adrenaline concentration where ventricular tissues respond to adrenaline as well (Skov, Bushnell, Tirsgaard & Steffensen, 2009). Therefore, adrenaline can make the heart to pump faster, raise the speed of conduction and increase cardiac output (Griffiths, Irving & Dixon, 1976). On the use of medicine, adrenaline is frequently used to stimulate the heart when heart failure. As a result, adrenaline can be observed to increase the heart rate of the toad in this experiment.

Hypothesis

Adrenaline will increase the heart rate of the toad heart.

Methods

A toad heart was employed as the model for this experiment due to the special characteristics of amphibians. Besides, Electrocardiogram (ECG), a useful test, was applied to this experiment to measure the electrical activity of the heart.

The protocol of this experiment is going to be described as following. Basic toad heart was set up previously. By using ECG, sufficient baseline data was recorded where the essential heart rate of the toad could be determined from this baseline data. A comment was added on the ECG with adrenaline. Then, four drops of adrenaline
solution (1mM) was applied to the heart by using a plastic pipette and at that time sufficient data was recorded. After that, Ringer’s solution was used to rinsing the heart for several times and then thoracic cavity was filling with Ringer’s solution. Paper towel was used to blot the Ringer’s solution away. Finally, this rinse procedure was repeated for several times which allowed the heart to recover back to baseline.

The method of data analysis is going to be demonstrated as following. The heart rate was calculated base on several continuous cardiac cycles from both baseline data and adrenaline added. This calculation was repeated for three times for each data. Next, Prism was used to create a chart by putting in the data of the heart rate. Finally, an unpaired, two-tailed, t-test with Welch’s correction was used to run the statistic analysis which was in order to determine whether there’s a significant different between control and treatment.

Results

There was a significant increase in heart rate during adrenaline treatment compared with the baseline condition (p<0.05, see Figure 1).

Figure 1. Comparison of Heartbeat rate with control and adrenaline added. There was a significant (p=0.002) increase in heart rate during adrenaline treatment (70.20 ± 0.025) compared with the baseline data (35.64 ± 1.534). Columns and bars are means ± SEM, *P <0.05 compared with control was considered to be statistically significant.
The unpaired, two tailed, t-test was used to analyse the data difference of baseline condition and adrenaline added. Since the F test aimed to compare variances was resulted in significant differences, Welch’s correction was applied in this test analysis where equal variances were not assumed. From the chart above (Figure 1), we can clearly perceive that heartbeat rate of the toad was increased after adrenaline added which confirms with our hypothesis. Moreover, the data analysis also demonstrates that there was a significant difference between control and treatment, say here, without adrenaline and with adrenaline. Therefore, according to these results, we can determine that this hypothesis is supported.

Discussion

Adrenaline is believed to have a considerable effect on the increase of the heart rate. Our findings highlight that when adrenaline was applied to the toad heart, the heart rate would become faster than the original heart rate. The sinoatrial node is the normal pacemaker of the heart and produces the electrical impulse for the regular contraction of the heart (Solaro, Rosevear & Kobayashi, 2008). The pacemaker activity of sinoatrial node cells is based on the incidence of the diastolic depolarisation phase of the action potential where cells depolarise spontaneously in the direction of the action potential threshold, and hence determined the heart rate (Bucchi, Baruscotti, Robinson & Do Francesco, 2007). The pacemaker potential decays because of a slowly reducing outward K+ current set against inward currents (Ward & Linden, 2008). Factors that affect these currents alter the rate of decay and the time to reach threshold, and as well as the heart rate (Verkerk, Ginneken & Wilders, 2009). In this experiment, adrenaline can increase the rate of decay which means that adrenaline decreases the permeability of K+ and therefore, accelerates inactive of K+ channels. For that reason, there is a rapid drift to threshold where the depolarisation rate increases. Thus, increases the heart rate.

As a result, the positive chronotropic outcome of adrenaline is mediated by the increased rate of pacemaker depolarisation. Conversely, an increase of K+ would tend to decelerate the pacemaker depolarisation. Moreover, according to the research, Brown, Difrancesco & Noble found that there is an additional current, lr (Na+ and K+), is stimulated within the range of voltage occurs. This could show the importance for both in normal pacemaking and in adrenaline-induced acceleration.

These results correlate with previous findings that cardiac functions can be affected by several variables (Ershow & Costello, 2006). For instance, some drugs might activate or block autonomic receptors on pacemakers and cardiomyocytes, thus, produce an impact on the heart rate or contractility (Satish, 2005). For this experiment,
the hypothesis that adrenaline increases the heart rate of the toad is verified. The findings shed further insights on the use of drugs on the cardiac cells and tissues which also implied the physiology and mechanism of molecular circulating and responding. This study could be significant for further development of the use of adrenaline on medicine for the treatment of cardiac disease or heart failure.
References


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