Cardiovascular physiology assignment

Psychology

Introduction The cardiac muscles involved in a heartbeat are the specialized cells in conducting system and the contractile cells. The natural pacemaker is the sinoatrial (SA) node, which is comprised of heart cells that are the most active in generating electrical impulses. This impulse propagates throughtout the heart via the conducting system. The electrical signals can then be tracked down by an electrocardiogram (ECG or EKG). The conducting system includes the SA node, atrioventricular (AV) node, and conducting cells.

The conducting cells form the AV bundle, bundle branches, and Purkinje fibers, which distribute the stimulus from the AV node to the ventricular myocardium. The internodal pathway distributes the stimulus from the SA node to the AV node. The contractile cells, about 99% of the the muscle cells in the heart, shorten and contracts when they receive the electrical signals [1]. The SA node is affected by both sympathetic and parasympathetic stimulations. The vagas nerve brings parasympathetic stimulus and the sympathetic stimulus from the medulla oblongata.

When the heart is overly stimulated, the result is tachycardia, which is the increase of heart rate. The opposite is a situation known as bradycardia, where the heart rate is slower than usual. Experimental Direct Heart Stimulation The heart will be stimulated with direct contact. This is done by placing a conducting wire on the heart to send an electrical signal. This signal is controlled to stimulate right at the beginning of ventricular contraction, near the peak of ventricular contraction, and done in two quick successions.

The signal could also be sent as multiple stimulus, where it administer repeated stimuli at a rate of 20 per second to the heart. Vagus Nerve Stimulation This uses the electrode to stimulate the vagus nerve which is then connected to the heart. The vagus nerve stimulation can only be applied with multiple stimuli. Effect of Chemicals Certain hormones will be applied to the heart at a specified temperature, such as 23 ?? C. Epinephrine, pilocarpine, atropine, and digitalis are the chemicals experimented with. A few drops will be applied to the heart and then the heart rate will be observed.

Data When the direct heart stimulation was applied during the beginning of ventricular contraction, no changes were made. However, when the stimulus was applied during the peak of ventricular, a second ventricular systole occurred right after the first. During the multiple stimulus simulation, the heart rate was changed dramatically. Sometimes it would flat line after a few good heart beats or have a double systole right after the atrial contraction. When the double systole occurred, some had a diastole close to normal range, while in other cases, the ventricular diastole was cut short.

Table 1 ??? The effects of chemical substance added to the heart on the heart rate. | Chemical Added | Heart Rate | | Control | 59 | | Epinephrine | 78 | | Pilocarpine | 44 | | Atropine | 69 | | Digitalis | 41 | The control was at 23 ?? C without any chemical added. It was the value that all other chemicals were compared with. Analysis

During the direct heart stimulation, the stimulus applied right at the beginning of the ventricular contraction, no change was observed because at

that moment, the heart muscles were under absolute refractory period. In this period, no matter how many or how strong the stimulus applied is, the cardiac muscle cannot contract again. However, during the peak of the ventricular contraction, it is the relative refractory period. This meant any stimulus applied here would cause another contraction of systole. Refractory period on the multiple stimulus section was shown during the normal cycle and the double ventricular systole.

This is because if there was shown no change, the heart could have either beat between the two stimulus or because the stimulus occurred during the absolute (or also known as the effective) refractory period. The double systole that showed only a very small amount ventricular diastole showed that the heart was stimulated during the relative refractory period. Both cases showed that summation does not occur. This is important because if summation had occurred the strength of the heart pumps would increase and enter a moment of tetanus where the stimulation rate increase until the relaxation phase is eliminated.

This would cause distress onto the heart and the circulatory system if summation occurred as the increase stroke volume would result in deteoritation of the blood vessels and consistent contraction of the heart would not provide a steady flow of blood throughout the body. Epinephrine, a hormone secreted by the adrenal glands, stimulates the heart via the bloodstream. When this occurs, it increases the metabolic rate, which then the heart rate would increase [2, 3]. It is a sympathetic stimulus.

The effect of Atropine shows similar but not as dramatic results, as the heart rate also increase. It is a cholinergic drug that showed a different effect than pilocarpine, and acts as a sympathetic stimulus by inhibiting acetylcholine by binding to the receptors and opposes the vagus nerve. Pilocarpine and digitalis are both drugs that mimic the parasympathetic stimuli, slowing down the heart rate. Since pilocrapine is a drug that acts like acetylcholine in the body [4]. Digitalis is the drug that interferes with the conduction pathway in the heart by having a positive intropic effect.

It prevents the normal conduction by blocking the internodal pathway between the atrial impulses to the ventricles by affecting the sodium ion pumps. From the EKG, the P wave represents atria depolarization, T wave represents the ventricles repolarize, and the QRS complex represents the ventricle depolarization. In the cardiac cycle, it starts with ventricular diastole and atrial systole for 100 msec. From 100 msec until 370 msec is the ventricular systole and atrial diastole. The atrial diastole lasts until the rest of the cardiac cycle (800 msec on average).

The cardiac muscle is different than the typical muscle in that there is a longer refractory period in both absolute and relative; there is also accumulation in the summation of stimulus. The depolarization occurs when sodium ions enter the chanel and ends when the voltage regulated sodium channel closes. Then muscle enters the plateau within the absolute refractory period. Calcium ions enters and after about 175 msec, the calcium channels closes. The repolarization of the muscle includes the exit of potassium ions, which occurs within the relative refractory period [1].

Although the SA node generates 80 to 100 action potential per minute but the isolated cells of the AV node depolizes only 40-60 times per minute, but due to the parasympathetic effects, the heart rate is slower than 80 to 100 beats per minute Conclusion The heart will not contract again during absolute refractory period regardless of the amount and strength of the stimulus. If the stimulus occurs during the relative refractory period, the heart can undergo another ventricular contraction. However, if too much stimulation occurs at once, neurotransmitters can become depleted, resulting in a temporal flat-line.

Stimulation of the vagus nerve would slow down the heart rate. Epinephrine and atropine are sympathetic stimuli, increasing the heart rate. These drugs can be used if a patient’s heart rate is slow and desires to increase the heart rate. If these drugs were to be given to a normal human, they would have a case of tachycardia. Digitalis and pilocarpine are drugs that are parasympathetic stimuli and slow down the heart rate. These drugs would be used to slow down the heart rate. When a healthy person absorbs these drugs, they will develop a case of bradycardia plus other parasympathetic nervous system symptoms such as salivating.

Reference [4]http://www. mercksource. com/pp/us/cns/cns_hl_dorlands_split. jsp? pg=/ppdocs/us/common/dorlands/dorland/seven/000091035. htm [2] Collart F. 1985. Acta Cardiologica. Relationships between Blood-Pressure, Heart-Rate and Plasma Epinephrine, Norepinephrine, Angiotensin-II Concentrations, Plasma-Renin Activity During Chronic Guanfacine Therapy in Patients with Essential Arterial-Hypertension. [3] Holmberg. 1984. American Journal of Physiology. V. 247 . Intracoronary Epinephrine Attentuates

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