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The effect of training on heart rate

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Abstract

The heart rate is a useful parameter for monitoring the reaction of the athlete's body to training and the HRM provides a convenient method for measuring and recording heart rate during exercise. Heart rate, on its own, does not allow for an accurate assessment of the training effectiveness over time and cannot tell the coach or athlete which aspects of the training program are having a positive or negative influence on training adaptation.

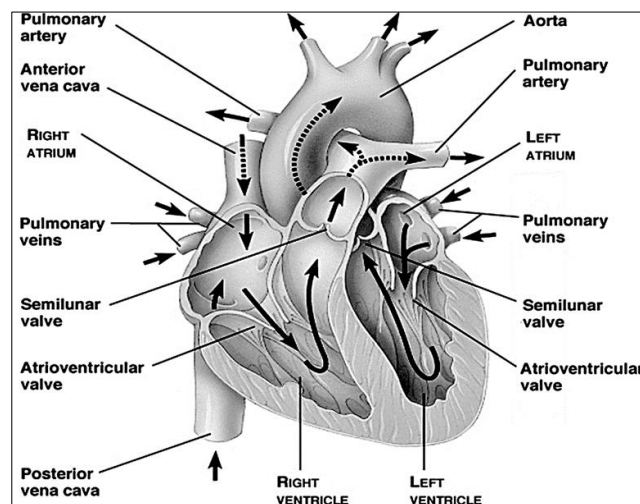
Keywords: the effect, heart rate, text books physical education, health awareness

Introduction

The heart is one of the most important organs in the entire human body. It is really nothing more than a pump, composed of muscle which pumps blood throughout the body, beating approximately 72 times per minute of our lives. The heart pumps the blood, which carries all the vital materials which help our bodies function and removes the waste products that we do not need. For example, the brain requires oxygen and glucose, which, if not received continuously, will cause it to lose consciousness. Muscles need oxygen, glucose and amino acids, as well as the proper ratio of sodium, calcium and potassium salts in order to contract normally. The glands need sufficient supplies of raw materials from which to manufacture the specific secretions. If the heart ever ceases to pump blood the body begins to shut down and after a very short period of time will die.

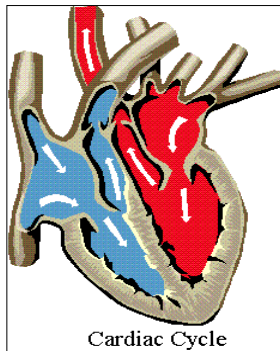
The heart is essentially a muscle (a little larger than the fist). Like any other muscle in the human body, it contracts and expands. Unlike skeletal muscles, however, the heart works on the "All-or-Nothing Law". That is, each time the heart contracts it does so with all its force. In skeletal muscles, the principle of "gradation" is present. The pumping of the heart is called the Cardiac Cycle, which occurs about 72 times per minute. This means that each cycle lasts about eight-tenths of a second. During this cycle the entire heart actually rests for about four-tenths of a second.

Make-up of the heart



The walls of the heart are made up of three layers, while the cavity is divided into four parts. There are two upper chambers, called the right and left atria, and two lower chambers, called the right and left ventricles. The Right Atrium, as it is called, receives blood from the upper and lower body through the superior vena cava and the inferior vena cava, respectively, and from the heart muscle itself through the coronary sinus. The right atrium is the larger of the two atria, having very thin walls. The right atrium opens into the right ventricle through the right atrioventricular valve (tricuspid), which only allows the blood to flow from the atria into the ventricle, but not in the reverse direction. The right ventricle pumps the blood to the lungs to be reoxygenated. The left atrium receives blood from the lungs via the four pulmonary veins. It is smaller than the right atrium, but has thicker walls. The valve between the left atrium and the left ventricle, the left atrioventricular valve (bicuspid), is smaller than the tricuspid. It opens into the left ventricle and again is a one way valve. The left ventricle pumps the blood throughout the body. It is the Aorta, the largest artery in the body, which originates from the left ventricle.

The circulatory system



The Heart works as a pump moving blood around in our bodies to nourish every cell. Used blood, that is blood that has already been to the cells and has given up its nutrients to them, is drawn from the body by the right half of the heart, and then sent to the lungs to be reoxygenated. Blood that has been reoxygenated by the lungs is drawn into the left side of the heart and then pumped into the blood stream. It is the atria that draw the blood from the lungs and body, and the ventricles that pump it to the lungs and body. The output of each ventricle per beat is about 70 ml, or about 2 tablespoons. In a trained athlete this amount is about double. With the average heart rate of 72 beats per minute the heart will pump about 5 liters per ventricle, or about 10 liters total per minute. This is called the cardiac output. In a trained athlete the total cardiac output is about 20 liters. If we multiply the normal, non-athlete output by the average age of 70 years, we see that the cardiac output of the average human heart over a life time would be about 1 million liters, or about 250,000 gallons.

Normal heart sounds

With the aid of a stethoscope you can hear the characteristic sounds of the normal heartbeat, typically described as a "lub-dub." These sounds are produced by the closure of the heart valves. The first heart sound or "lub" results from closure of the tricuspid and mitral valves. It is a rather low-

pitched and a relatively long sound which, as indicated in, represents the beginning of ventricular systole.

The second heart sound, or "dub," marks the beginning of ventricular diastole. It is produced by closure of the aortic and pulmonary (pulmonic) semilunar vanes when the intraventricular pressure begins to fall. This "dub" sound is typically heard as a sharp snap because the semilunar valves tend to close much more rapidly than the AV valves. Because diastole occupies more time than systole, a brief pause occurs after the second heart sound when the heart is beating at a normal rate. Therefore, the pattern that one hears is one of: "lub-dub" pause, "lub-dub" pause, and so on.

Sometimes, especially in young normal individuals, a third heart sound can be heard. This sound is produced by the very rapid influx of blood into the partially filled ventricle. It is typically very faint and as such difficult to hear.

Cardiac output as a function of stroke volume and heart rate

Let us concentrate now on the work that is performed by the beating heart. The volume of blood pumped by one ventricle during one beat is called the stroke volume.

Because cardiac work performance is typically related to a fixed time interval (i.e., one minute), it is possible to calculate the cardiac output by simply multiplying the stroke volume by the number of times the ventricles beat per minute. Thus, the cardiac output represents that volume of blood pumped by one ventricle in one minute. For example, in a resting adult the heart might be at 72 times per minute and pump about 70 ml of blood with each ventricular contraction. This being the case,

$$\begin{aligned}\text{Cardiac output} &= \text{stroke volume} \times \text{heart rate (number of} \\ &\text{ventricular contractions/min)} \\ &= 70 \text{ ml/stroke} \times 72 \text{ strokes/min} \\ &= 5040 \text{ ml/min (5.04 liters/min)}\end{aligned}$$

Although the cardiac output for the resting adult heart is ordinarily on the order of 5 to 6 litres per minute, during strenuous exercise the heart may increase its output four or five times. Simple multiplication demonstrates that under such conditions the heart may pump as much as 20 to 30 litres of blood per minute. Even more amazingly, the heart of a well-trained athlete can increase its output up to seven times.

The maximum percentage that the cardiac output can be increased above normal is defined as the cardiac reserve. Therefore, if during exercise the output can increase to a maximum of five times normal, the cardiac reserve is 400%. From the previous equation, it should be clear that cardiac output varies as a function of either stroke volume or heart rate. Let us examine just how these two important variables are regulated.

Stroke volume

Stroke volume, or the volume of blood pumped by one ventricle during one contraction, has a direct effect upon cardiac output. Although the ventricles do not eject all of their blood when they contract, the more forcefully they contract, the greater the volume of blood ejected. Moreover, the volume of blood returned to the heart via the great veins varies from time to time. Thus, stroke volume is regulated mainly by venous return and by sympathetic stimulation.

The volume of blood delivered to the heart by the great veins, the venous return, together with subsequent stretching of the cardiac muscle, is perhaps the most important determinant of cardiac output. The greater the volume of blood returned to the heart via the veins, the greater the volume of blood pumped by the heart. This relationship, known as Starling's law of the heart, permits the heart to pump all of the blood returned to it within physiological limits. As additional quantities of blood fill the chambers of the heart, cardiac muscle cells are stretched to a greater extent and subsequently contract with a greater force. Thus, increased quantities of blood are pumped into the arteries. Increasing the stroke volume in this manner can raise the normal output of 5 liters per min to a maximum output of approximately 14 liters per minute.

When the venous return is even greater, the heart is able to keep pace with the excessive volume only through sympathetic stimulation, leading to an increased heart rate and force of contraction. The release of noradrenaline by sympathetic nerve fibres not only increases heart rate but also increases the force of cardiac muscle contraction. This increased force of contraction is distinct from that which is brought about by an increased blood volume, as mentioned previously. Adrenaline, a hormone released by the adrenal medulla, has a similar effect on cardiac muscle. Thus, when the force of contraction increases, the stroke volume increases, and this in turn leads to an increase in cardiac output.

Regulation of heart rate is influenced by several factors

We know that the heart is capable of beating independently of its bodily control systems. However, in order to adapt its rate to the changing needs of the body, it is subject to the most careful regulation by the nervous system. Additional factors such as hormones, fluctuations in body temperature, and concentrations of various ions also influence heart rate.

Autonomic nervous system

The heart is innervated by both components of the autonomic nervous system. Parasympathetic fibres decrease heart rate, whereas sympathetic fibres increase heart rate. Parasympathetic innervation originates in the cardiac inhibitory centre in the medulla oblongata of the brain stem and is conveyed to the heart by way of the vagus nerve (Cranial Nerve X).

Both the SA and AV nodes are richly supplied with vagal fibres. There is a minor distribution of vagal fibres to muscle of the atria and ventricles. When these parasympathetic fibres are stimulated they release acetylcholine, which slows the heart rate.

Normally, the parasympathetic innervation represents the dominant neural influence on the heart.

Maximal stimulation of vagal fibres can actually lead to a complete cessation of ventricular contraction. This can result from either a block in impulse transmission through the AV junctional fibres or complete inhibition of rhythmic signal generation by the SA node. Even with continued parasympathetic stimulation, the ventricles will begin to beat (10 to 40 beats per minute) after a short interval (typically 5 to 10 seconds). This phenomenon is called ventricular escape and is the result of new rhythmic impulses being generated in an abnormal site, for example, the AV bundle.

The heart receives its sympathetic innervation from nerves originating in the medulla (cardiac accelerating centre) and upper thoracic spinal cord. These reach the myocardium via several nerves sometimes referred to as the accelerator nerves. Sympathetic fibres innervate SA and AV nodal tissue as well as cardiac muscle cells themselves. When stimulated, the sympathetic fibres release noradrenaline, which leads not only to an increase in heart rate, but to an increase in the strength of ventricular and atrial contraction as well. The heart rate may nearly triple, and the strength of contraction may nearly double, under the influence of maximal sympathetic stimulation.

Various parts of the circulatory system relay messages (e.g., regarding blood pressure) to the cardiac centres, which respond by sending messages to the heart via the vagus nerves. In this manner the cardiac centres are responsible for maintaining a balance between the inhibitory effects of the parasympathetic nerves and the stimulatory effects of the sympathetic nerves. When the parasympathetic messages decrease, the sympathetic nerves are able to function in an unopposed manner and thereby increase the heart rate. For example, severance of vagal nerve fibres results in an increased heart rate.

Hormonal influence

Under conditions of stress, adrenaline and noradrenaline are released from the tissues of the adrenal medulla into the general circulation. Each of these hormones produces an increase in heart rate.

Thyroid hormones, thyroxine (T₄) and triiodothyronine (T₃), also accelerate the heart rate and this is most likely due to a direct effect of these substances on the heart. The strength of heart contraction is also modulated by thyroid hormones. In slight excess they increase the strength of contraction, whereas in marked excess they actually reduce the strength of contraction.

Temperature

Elevation of the body temperature markedly increases the heart rate. This most probably results from an increased permeability of cardiac muscle-cell plasma membranes to the passage of various ions, thereby causing an accelerated generation of rhythmic action potentials. During fever, for example, it is not uncommon for the individual to experience a heart rate in excess of 100 beats per minute. Lowering of the body temperature, or hypothermia, is accompanied by a reduction in heart rate. This latter observation is taken advantage of clinically, for example, when the patient's temperature is deliberately lowered during heart surgery.

The heartbeat

During rest, the heart beats about 70 times a minute in the adult male, while pumping about 5 liters of blood. The stimulus that maintains this rhythm is self-contained. Embedded in the wall of the right atrium is a mass of specialized heart tissue called the sino-atrial (S-A) node. The S-A node is also called the pacemaker because it establishes the basic frequency at which the heart beats. The interior of the fibers of heart muscle, like all cells, is negatively charged with respect to the exterior. In the cells of the pacemaker, this charge breaks down spontaneously about 70 times each minute. This, in turn, initiates a similar discharge of the nearby muscle fibers of the atrium. A tiny

wave of current sweeps over the atria, causing them to contract.

When this current reaches the region of insulating connective tissue between the atria and the ventricles, it is picked up by the A-V node (atrio-ventricular node). This leads to a system of branching fibers that carries the current to all parts of the ventricles. The contraction of the heart in response to this electrical activity creates systole. A period of recovery follows called diastole. The heart muscle and S-A node become recharged. The heart muscle relaxes. The atria refill.

Method of assessment of heart rate

During ventricular contraction blood is ejected into the arteries with a pressure and this pressure wave is transmitted throughout the arterial system which can be felt easily in superficial artery. The number of pressure waves per minute felt at the arteries is called pulse rate. Normally it is about 70 to 80 per minute. In trained person it is found to be as low as 40 to 50 beats per minute. Measurement of heart rate can be done by the following way: (a) Stethoscope, (b) by pulse, (c) through electrocardiogram and (d), radio-telemetric devise.

Age	Normal heart rate (beats per minute)
newborn	130
3 months	150
6 months	135
1 year	125
2 years	115
3 years	100
4 years	100
6 years	100
8 years	90
9 years	95
12 years	85
Adult	60-100

Miguel Indurain, a cyclist and five times Tour de France winner, had a resting heart rate of 28 beats per minute, one of the lowest ever recorded in a healthy human.

Functioning

Starting in the right atrium, the blood flows through the tricuspid valve to the right ventricle. Here it is pumped out the pulmonary semilunar valve and travels through the pulmonary artery to the lungs. From there, blood flows back through the pulmonary vein to the left atrium. It then travels through the mitral valve to the left ventricle, from where it is pumped through the aortic semilunar valve to the aorta. The aorta forks and the blood is divided between major arteries which supply the upper and lower body. The blood travels in the arteries to the smaller arterioles, then finally to the tiny capillaries which feed each cell. The (relatively) deoxygenated blood then travels to the venules, which coalesce into veins, then to the inferior and superior venae cavae and finally back to the right atrium where the process began.

The heart is effectively a syncytium, a meshwork of cardiac muscle cells interconnected by contiguous cytoplasmic bridges. This relates to electrical stimulation of one cell spreading to neighboring cells.

Some cardiac cells are self-excitabile, contracting without any signal from the nervous system, even if removed from

the heart and placed in culture. Each of these cells has its own intrinsic contraction rhythm. A region of the human heart called the sinoatrial node SA node, or pacemaker, sets the rate and timing at which all cardiac muscle cells contract. The SA node generates electrical impulses, much like those produced by nerve cells. Because cardiac muscle cells are electrically coupled by intercalated disks between adjacent cells, impulses from the SA node spread rapidly through the walls of the atria, causing both atria to contract in unison. The impulses also pass to another region of specialized cardiac muscle tissue, a relay point called the atrioventricular (AV) node, located in the wall between the right atrium and the right ventricle. Here, the impulses are delayed for about 0.1s before spreading to the walls of the ventricle. The delay ensures that the atria empty completely before the ventricles contract. Specialized muscle fibers called Purkinje fibers then conduct the signals to the apex of the heart along and throughout the ventricular walls. The Purkinje fibres from conducting pathways called bundle branches. The impulses generated during the heart cycle produce electrical currents, which are conducted through body fluids to the skin, where they can be detected by electrodes and recorded as an electrocardiogram (ECG or EKG).

A. Statement of the problem

The purpose of the study is to compare the Resting, Normal, and Exercise heart rate before and after training of the athletes.

B. Hypothesis

1. It is hypothesized that normal heart rate in the athletes will be less after training.
2. It is hypothesized that resting heart rate in the athletes will be less after training.
3. It is hypothesized that Exercise heart rate in the athletes will be more after training.

C. Limitations

This study is limited only to the 15 M.P.Ed. Students.

D. Delimitations

1. This study was delimited on 15 M.P.Ed final year students of the year 2008-09, of KUD.
2. Further the study was delimited on 21-26 years age group students.

E. Definitions of the term

Heart rate is a term used to describe the frequency of the cardiac cycle. It is considered three of the four vital signs. Usually it is calculated as the number of contractions (heart beats) of the heart in one minute and expressed as "beats per minute" (bpm).

Maximum heart rate (HR_{max}) is the highest number of times your heart can contract in one minute, or the heart rate that a person could achieve during maximal physical exertion. It is not the maximum one should obtain often during exercise. MHR is used as a base number to calculate target heart rate for exercise.

Heart rate drift is the increase in heart rate seen over time while exercising at a constant workload. Some studies have found that your heart rate can increase by as much as 5 to 20

bpm during exercise lasting 20 to 60 minutes even when the work rate does not change.

Heart rate variability describes the variations in the intervals between consecutive heartbeats. Even when the heart rate is stable, the time between consecutive beats can vary considerably. At rest, heart rate variability is larger in aerobically trained individuals than in untrained individuals.

F. Significance of the study

- This study will help the individual to know their fitness level.
- This study will guide the coaches and physical education teachers to pickup physically fit players.
- This study will help the athletes to find out if they're over trained.
- This will help the coaches to give training.

Methodology

The purpose of the study was to compare the resting, normal and exercise heart rate of athletes. A selected 15 males from M.P.Ed Final year students of P.G. Dept of Studies in Physical Education, Karnatak University, Dharwad for this study in between the age group of 21 to 25 years. Resting heart rate was taken for 1 minute of this subject for 7 days early in the morning before they would get-up from the bed and average was taken. Average resting, normal and exercise heart rate was taken before and after training. Once in a week exercise heart rate was taken for 5 weeks on week ends. The below following methods are used to check the exercise heart rate. i.e. - fast continuous run for 45 minutes, agility exercise, uphill running, plyometrics, weight training, passive resistance training etc with the intensity of 90% to 100%.

Measuring heart rate



Measuring the pulse at the neck and wrist

The pulse rate (which in most people is identical to the heart rate) can be measured at any point on the body where an artery's pulsation is transmitted to the surface - often as it is compressed against an underlying structure like bone. Some commonly palpated sites are as listed.

1. The inside of the wrist on the side of the thumb (radial artery),
2. The neck (carotid artery),
3. The inside of the elbow, or under the biceps muscle (brachial artery)
4. The groin,
5. Behind the medial malleolus on the feet (posterior tibial artery)
6. Middle of dorsum of the foot (dorsalis pedis).
7. Behind the knee (popliteal artery)
8. Over the abdomen (abdominal aorta)

9. The chest. (aorta) This can be felt with one's hands or fingers but it is possible to auscultate the heart by utilizing a stethoscope.

Producing an electrocardiogram, or ECG is one of the most precise methods of heart rate measurement. Continuous electrocardiographic monitoring of the heart is routinely done in many clinical settings, especially in critical care medicine. Commercial heart rate monitors are also available, consisting of a chest strap with electrodes. The signal is transmitted to a wrist receiver for display. Heart rate monitors allow accurate measurements to be taken continuously and can be used during exercise when manual measurement would be difficult or impossible (such as when the hands are being used).

Your resting heart rate is best measured when you first wake up in the morning, before your feet leave the sheets.

Grab a stopwatch or a clock or watch with a second hand, then find your pulse. You can locate your pulse either in your radial artery on your wrist or at your carotid artery in your neck. Choose the spot that works best for you.

The only trick to measuring your heart rate is that you must use the correct fingers to do the measuring. Your thumb has a light pulse and can create some confusion when you are counting your beats. It's best to use your index finger and middle finger together.

After you find the beat, you need to count how many beats occur within 60 seconds. The shortcut to this method is to count the number of beats in 10 seconds, and then to multiply that number by 6. This method gives you a 60-second count.

Example: You count 7 beats in 10 seconds: $7 \times 6 = 42$ beats per minute.

If you have trouble finding your pulse or separating the beats in your body from the ticks of your watch, ask a friend for help. Have your friend count your pulse beats while you watch the clock or vice versa.

Calculation of maximum heart rate

The easiest and best known method to calculate your maximum heart rate (MHR) is to use the formula

- $MHR = 220 - \text{Age}$.

Calculation of a zone value

The calculation of a zone value, X%, is performed in the following way:

- Subtract your RHR from your MHR giving us your working heart rate (WHR)
- Calculate the required X% on the WHR giving us "Z"
- Add "Z" and your RHR together to give us the final value

Example: The athlete's MHR is 180 and their RHR is 60 - determine the 70% value

- $MHR - RHR = 180 - 60 = 120$
- $70\% \text{ of } 120 = 84$
- $84 + RHR = 84 + 60 = 144 \text{ bpm}$

What is my heart rate reserve or working heart rate? What is the Karvonen formula?

Your heart rate reserve is the gap between your resting heart rate and your maximum heart rate. For example, my

maximum heart rate is 190bpm (four beats a minute higher than the rule of Thumb based on my age predicts); and my resting heart rate is 48bpm. This means my heart rate reserve is:

$$HRR = 190 - 48 = 142 \text{ bpm.}$$

To get the heart rate corresponding to the aerobic threshold,

which is at 85% of my heart rate reserve, I have to add 85% of my heart rate reserve rate to my resting heart rate. So the heart rate corresponding to my aerobic threshold is estimated to be: $AT = 48 + (0.85 \times 142) = 169$.

This way of calculating a heart rate zone is known as the Karvonen formula.

What are the heart rate zones?

Zone	What it does	% of Heart Rate Reserve (Karvonen)
Long, slow runs, easy or recovery runs	Training in this zone improves the ability of your heart to pump blood and improve the muscles' ability to utilize oxygen. The body becomes more efficient at feeding the working muscles, and learns to metabolize fat as a source of fuel.	60-70%
Aerobic zone or "target heart rate zone"	Most effective for overall cardiovascular fitness. Increases your cardio-repitory capacity: that is, your ability to transport oxygenated blood to the muscle cells and carbon dioxide away from the cells. Also effective for increasing overall muscle strength.	70-80%
Anaerobic zone	The point at which the body cannot remove lactic acid as quickly as it is produced is called the lactate threshold or anaerobic threshold. It generally occurs at about 80-88% of the Heart Rate Reserve. Training in this zone helps to increase the lactate threshold, which improves performance. Training in this zone is hard: your muscles are tired, your breathing is heavy.	80-90%
VO2 max "Red line zone"	You should only train in this zone if you re very fit, and only for very short periods of time. Lactic acid develops quickly as you are operating in oxygen debt to the muscles The value of training in this zone is you can increase your fast twitch muscle fibers which increase speed.	90-100%

Does it matter where in the heart rate zone I train?

Yes. Training at particular heart rates in the zone will be more beneficial for you in terms of the impact on your body. Have a look at the heart rate training paces.

Training zones based on heart rates

Zone	Why you should do it	Frequency	Heart rate % of reserve	Heart rate beats per minute
Recovery runs	Gives you time to recover from harder workouts.	Use recovery runs the day after hard workouts.	< 70%	< 152
Long, slow runs.	Builds endurance, and develop the strength of your muscles, bones and joints. Helps develop the metabolic system to enable you to burn more fat. Burn more calories, and so reduce weight.	At least one long, slow run a week. 80-90% of your training mileage should be at recovery run pace or long slow run pace.	67% - 77%	148 - 162
Lactate (or anaerobic) threshold pace	Increases the ability of the running muscles to use available oxygen to convert carbohydrate and fat fuel into output.	No more than once a week. No more than 10 to 15 percent of total training mileage. About 3-8 miles a week.	Beginner: 77% - 83% Experienced: 82% - 88%	162 - 170 168 - 176
VO2 max pace	Improves the body's ability to transport blood and oxygen. Improves running economy.	No more than once a week. No more than 4 to 8 percent of total training mileage.	95% -98%	185 - 189

Analysis of data

However valid, reliable and adequate the data may be, it does not serve any useful purpose unless it is carefully processed, systematically classified and tabulated, scientifically analyzed, intelligently interpreted and rationally concluded.

After the data had been collected, it was processed and tabulated using Microsoft Excel - 2007 Software. The data collected on normal, resting and exercises heart rate of 15 athletes. The purpose of the study is to compare the resting, normal, and exercise heart rate before and after training of the athletes. Then the data were analyzed with reference to the objectives and hypotheses by using independent t-test to find out the effect of training on heart rate by using SPSS 14.0 statistical software and the results obtained there by have been interpreted. The level of significance set at

0.05% level of significance was considered to reject or accept the null hypothesis. On the basis of objectives the following hypotheses were formed.

Resting health rate

Hypothesis: It is hypothesized that resting heart rate in the athletes will be less after training.

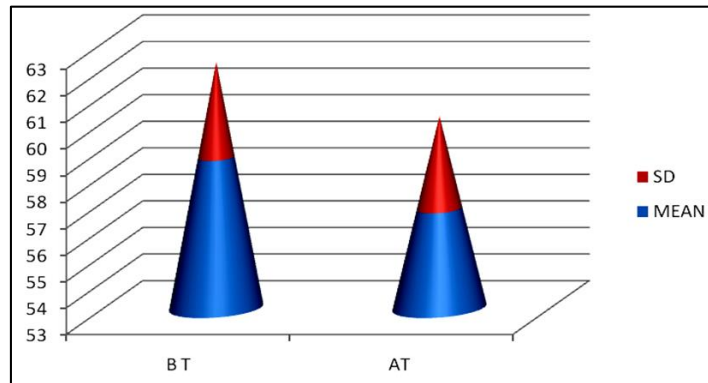
To achieve this hypothesis, the t-test was applied and the results are presented in the following table.

Group statistics

	A.T	N	Mean	Std. Deviation	Std.Error Mean
RHR	1.00	15	58.6667	3.57904	.92410
	2.00	15	56.7333	3.47371	.89691

Independent Samples Test

		t-test for Equality of Means						
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper	
RHR	Equal variances assumed	1.501	28	.144	1.93333	1.28779	-.70459	4.57126
	Equal variances Not assumed	1.501	27.975	.144	1.93333	1.28779	-.70470	4.57137



Conclusion

There is no significance difference between that before and after training (bpm) resting heart rate in the Athletes. Therefore a null hypothesis is accepted.

Normal heart rate

Hypothesis: It is hypothesized that normal heart rate in the athletes will be less after training.

To achieve this hypothesis, the t-test was applied and the results are presented in the following table.

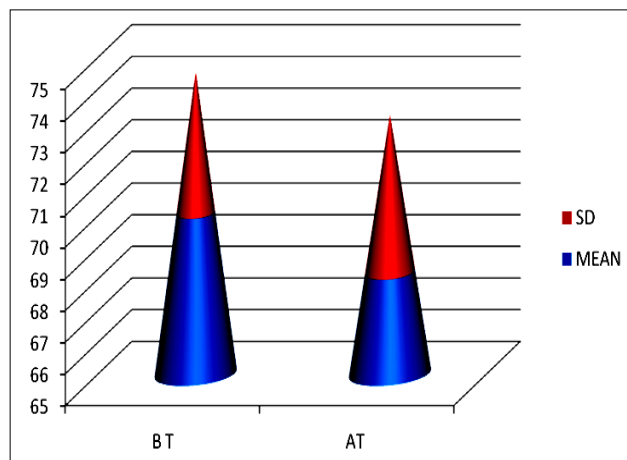
Group statistics

	A.T	N	Mean	Std. Deviation	Std. Error Mean
NHR	1.00	15	70.0667	4.46361	1.15250
	2.00	15	68.2000	4.97422	1.28434

Independent Samples Test

		t-test for Equality of Means						
		t	df	Sig. (2tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper	
NHR	Equal variances assumed	1.082	28	.289	1.86667	1.72562	-1.66811	5.40145
	Equal variances not assumed	1.082	27.678	.289	1.86667	1.72562	-1.66997	5.40330

Graph



Conclusion

In general, the higher your resting heart rate, the less physically fit and the lower your heart rate, the greater physically fit you are. (Some athletes have resting heart

rates in the 40s.) One way to see if your new workout is succeeding is to check your resting heart rate over a few months. See if it has increased, decreased, or remained the same. If your workouts are effective, your resting heart rate will slowly decrease, or at least remain constant. Your body has many ways of telling you when enough is enough, and if your resting heart rate has increased, you should start listening to your body by decreasing your workout frequency or intensity.

As the M.P.Ed students are already been training for past 2 to 3 years (not elite training) there is no significance difference between that before and after training in resting and normal heart rate where as in exercise heart rate there is a significance difference(at 0.05 level) between that before and after training. The reason behind it is that the M.P.Ed course is a teaching course not training. Daily the students run for 3 to 4 rounds jog then go for rotation for joints and some conditionings for about 30 minutes there pulse will be around 120-140/min. This type of training was given for 5 weeks. After limited training they go for teaching the skills or learning the technique which will be part by part method.

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