Heart rate

Heart rate is the speed of the heartbeat measured by the number of contractions (beats) of the heart per minute (bpm). The heart rate can vary according to the body's physical needs, including the need to absorb oxygen and excrete carbon dioxide. It is usually equal or close to the pulse measured at any peripheral point. Activities that can provoke change include physical exercise, sleep, anxiety, stress, illness, and ingestion of drugs.

The American Heart Association states the normal resting adult human heart rate is 60–100 bpm.[1] Tachycardia is a fast heart rate, defined as above 100 bpm at rest.[2] Bradycardia is a slow heart rate, defined as below 60 bpm at rest. During sleep a slow heartbeat with rates around 40–50 bpm is common and is considered normal. When the heart is not beating in a regular pattern, this is referred to as an arrhythmia. Abnormalities of heart rate sometimes indicate disease.[3]

While heart rhythm is regulated entirely by the sinoatrial node under normal conditions, heart rate is regulated by sympathetic and parasympathetic input to the sinoatrial node. The accelerans nerve provides sympathetic input to the heart by releasing norepinephrine onto the cells of the sinoatrial node (SA node), and the vagus nerve provides parasympathetic input to the heart by releasing acetylcholine onto sinoatrial node cells. Therefore, stimulation of the accelerans nerve increases heart rate, while stimulation of the vagus nerve decreases it.[4]

Due to individuals having a constant blood volume,[*dubious – discuss*] one of the physiological ways to deliver more oxygen to an organ is to increase heart rate to permit blood to pass by the organ more often.[3] Normal resting heart rates range from 60-100 bpm.[5][6][7][8] Bradycardia is defined as a resting heart rate below 60 bpm. However, heart rates from 50 to 60 bpm are common among healthy people and do not necessarily require special attention.[1] Tachycardia is defined as a resting heart rate above 100 bpm, though persistent rest rates between 80–100 bpm, mainly if they are present during sleep, may be signs of hyperthyroidism or anemia (see below).[3]

- Central nervous system stimulants such as substituted amphetamines increase heart rate.
- Central nervous system depressants or sedatives decrease the heart rate (apart from some particularly strange ones with equally strange effects, such as ketamine which can cause – amongst many other things – stimulant-like effects such as tachycardia).

There are many ways in which the heart rate speeds up or slows down. Most involve stimulant-like endorphins and hormones being released in the brain, many of which are those that are 'forced'/'enticed' out by the ingestion and processing of drugs.

This section discusses target heart rates for healthy persons and are inappropriately high for most persons with coronary artery disease.[9]

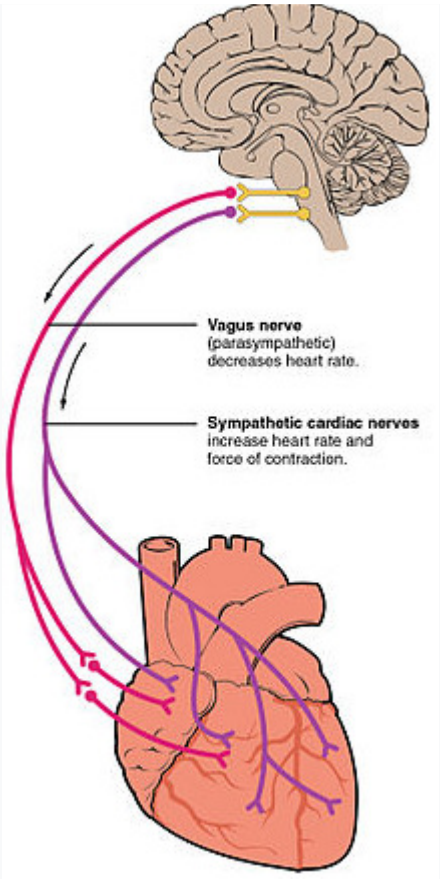
Influences from the central nervous system[edit]

Cardiovascular centres[edit]

The heart rate is rhythmically generated by the sinoatrial node. It is also influenced by central factors through sympathetic and parasympathetic nerves.[10] Nervous influence over the heart rate is centralized within the two paired cardiovascular centres of the medulla oblongata. The cardioaccelerator regions stimulate activity via sympathetic stimulation of the cardioaccelerator nerves, and the cardioinhibitory centers decrease heart activity via parasympathetic stimulation as one component of the vagus nerve. During rest, both centers provide slight stimulation to the heart, contributing to autonomic tone. This is a similar concept to tone in skeletal muscles. Normally, vagal stimulation predominates as, left unregulated, the SA node would initiate a sinus rhythm of approximately 100 bpm.[11]

Both sympathetic and parasympathetic stimuli flow through the paired cardiac plexus near the base of the heart. The cardioaccelerator center also sends additional fibers, forming the cardiac nerves via sympathetic ganglia (the cervical ganglia plus superior thoracic ganglia T1–T4) to both the SA and AV nodes, plus additional fibers to the atria and ventricles. The ventricles are more richly innervated by sympathetic fibers than parasympathetic fibers. Sympathetic stimulation causes the release of the neurotransmitter norepinephrine (also known as noradrenaline) at the neuromuscular junction of the cardiac nerves. This shortens the repolarization period, thus speeding the rate of depolarization and contraction, which results in an increased heartrate. It opens chemical or ligand-gated sodium and calcium ion channels, allowing an influx of positively charged ions.[11]

Norepinephrine binds to the beta–1 receptor. High blood pressure medications are used to block these receptors and so reduce the heart rate.[11]



Autonomic Innervation of the Heart - Cardioaccelerator and cardioinhibitory areas are components of the paired cardiac centers located in the medulla oblongata of the brain. They innervate the heart via sympathetic cardiac nerves that increase cardiac activity and vagus (parasympathetic) nerves that slow cardiac activity.[11]

Parasympathetic stimulation originates from the cardioinhibitory region with impulses traveling via the vagus nerve (cranial nerve X). The vagus nerve sends branches to both the SA and AV nodes, and to portions of both the atria and ventricles. Parasympathetic stimulation releases the neurotransmitter acetylcholine (ACh) at the neuromuscular junction. ACh slows HR by opening chemical- or ligand-gated potassium ion channels to slow the rate of spontaneous depolarization, which extends repolarization and increases the time before the next spontaneous depolarization occurs. Without any nervous stimulation, the SA node would establish a sinus rhythm of approximately 100 bpm. Since resting rates are considerably less than this, it becomes evident that

parasympathetic stimulation normally slows HR. This is similar to an individual driving a car with one foot on the brake pedal. To speed up, one need merely remove one's foot from the brake and let the engine increase speed. In the case of the heart, decreasing parasympathetic stimulation decreases the release of ACh, which allows HR to increase up to approximately 100 bpm. Any increases beyond this rate would require sympathetic stimulation.[11]

Effects of Parasympathetic and Sympathetic Stimulation on Normal Sinus Rhythm - The wave of depolarization in a normal sinus rhythm shows a stable resting HR. Following parasympathetic stimulation, HR slows. Following sympathetic stimulation, HR increases.[11]

Input to the cardiovascular centres[edit]

The cardiovascular centres receive input from a series of visceral receptors with impulses traveling through visceral sensory fibers within the vagus and sympathetic nerves via the cardiac plexus. Among these receptors are various proprioreceptors, baroreceptors, and chemoreceptors, plus stimuli from the limbic system which normally enable the precise regulation of heart function, via cardiac reflexes. Increased physical activity results in increased rates of firing by various proprioreceptors located in muscles, joint capsules, and tendons. The cardiovascular centres monitor these increased rates of firing, suppressing parasympathetic stimulation or increasing sympathetic stimulation as needed in order to increase blood flow.[11]

Similarly, baroreceptors are stretch receptors located in the aortic sinus, carotid bodies, the venae cavae, and other locations, including pulmonary vessels and the right side of the heart itself. Rates of firing from the baroreceptors represent blood pressure, level of physical activity, and the relative distribution of blood. The cardiac centers monitor baroreceptor firing to maintain cardiac homeostasis, a mechanism called the baroreceptor reflex. With increased pressure and stretch, the rate of baroreceptor firing increases, and the cardiac centers decrease sympathetic stimulation and increase parasympathetic stimulation. As pressure and stretch decrease, the rate of baroreceptor firing decreases, and the cardiac centers increase sympathetic stimulation and decrease parasympathetic stimulation.[11]

There is a similar reflex, called the atrial reflex or Bainbridge reflex, associated with varying rates of blood flow to the atria. Increased venous return stretches the walls of the atria where specialized baroreceptors are located. However, as the atrial baroreceptors increase their rate of firing and as they stretch due to the increased blood pressure, the cardiac center responds by increasing sympathetic stimulation and inhibiting parasympathetic stimulation to increase HR. The opposite is also true.[11]

Increased metabolic byproducts associated with increased activity, such as carbon dioxide, hydrogen ions, and lactic acid, plus falling oxygen levels, are detected by a suite of chemoreceptors innervated by the glossopharyngeal and vagus nerves. These chemoreceptors provide feedback to the cardiovascular centers about the need for increased or decreased blood flow, based on the relative levels of these substances.[11]

The limbic system can also significantly impact HR related to emotional state. During periods of stress, it is not unusual to identify higher than normal HRs, often accompanied by a surge in the stress hormone cortisol. Individuals experiencing extreme anxiety may manifest panic attacks with symptoms that resemble those of heart attacks. These events are typically transient and treatable. Meditation techniques have been developed to ease anxiety and have been shown to lower HR effectively. Doing simple deep and slow breathing exercises with one's eyes closed can also significantly reduce this anxiety and HR.[11]

Factors influencing heart rate[edit]

Table 1: Major factors increasing heart rate and force of contraction[11]

Factor	Effect
Cardioaccelerator nerves	Release of norepinephrine
Proprioreceptors	Increased rates of firing during exercise
Chemoreceptors	Decreased levels of O_2; increased levels of H^+, CO_2, and lactic acid
Baroreceptors	Decreased rates of firing, indicating falling blood volume/pressure
Limbic system	Anticipation of physical exercise or strong emotions
Catecholamines	Increased epinephrine and norepinephrine
Thyroid hormones	Increased T3 and T4
Calcium	Increased Ca^{2+}
Potassium	Decreased K^+
Sodium	Decreased Na^+
Body temperature	Increased body temperature
Nicotine and caffeine	Stimulants, increasing heart rate

Table 2: Factors decreasing heart rate and force of contraction[11]

Factor	Effect
Cardioinhibitor nerves (vagus)	Release of acetylcholine
Proprioreceptors	Decreased rates of firing following exercise
Chemoreceptors	Increased levels of O_2; decreased levels of H^+ and CO_2
Baroreceptors	Increased rates of firing, indicating higher blood volume/pressure
Limbic system	Anticipation of relaxation
Catecholamines	Decreased epinephrine and norepinephrine
Thyroid hormones	Decreased T3 and T4
Calcium	Decreased Ca^{2+}
Potassium	Increased K^+
Sodium	Increased Na^+
Body temperature	Decrease in body temperature

Using a combination of autorhythmicity and innervation, the cardiovascular center is able to provide relatively precise control over the heart rate, but other factors can impact on this. These include hormones, notably epinephrine, norepinephrine, and thyroid hormones; levels of various ions including calcium, potassium, and sodium; body temperature; hypoxia; and pH balance.[11]

Epinephrine and norepinephrine[edit]

The catecholamines, epinephrine and norepinephrine, secreted by the adrenal medulla form one component of the extended fight-or-flight mechanism. The other component is sympathetic stimulation. Epinephrine and norepinephrine have similar effects: binding to the beta-1 adrenergic receptors, and opening sodium and calcium ion chemical- or ligand-gated channels. The rate of depolarization is increased by this additional influx of positively charged ions, so the threshold is reached more quickly and the period of repolarization is shortened. However, massive releases of these hormones coupled with sympathetic stimulation may actually lead to arrhythmias. There is no parasympathetic stimulation to the adrenal medulla.[11]

Thyroid hormones[edit]

In general, increased levels of the thyroid hormones (thyroxine(T4) and triiodothyronine (T3)), increase the heart rate; excessive levels can trigger tachycardia. The impact of thyroid hormones is typically of a much longer duration than that of the catecholamines. The physiologically active form of triiodothyronine, has been shown to directly enter cardiomyocytes and alter activity at the level of the genome.[*clarification needed*] It also impacts the beta adrenergic response similar to epinephrine and norepinephrine.[11]

Calcium[edit]

Calcium ion levels have a great impact on heart rate and contractility: increased calcium levels cause an increase in both. High levels of calcium ions result in hypercalcemia and excessive levels can induce cardiac arrest. Drugs known as calcium channel blockers slow HR by binding to these channels and blocking or slowing the inward movement of calcium ions.[11]

Caffeine and nicotine[edit]

> This section **needs expansion**. You can help by adding to it. *(February 2015)*

Caffeine and nicotine are both stimulants of the nervous system and of the cardiac centres causing an increased heart rate. Caffeine works by increasing the rates of depolarization at the SA node, whereas nicotine stimulates the activity of the sympathetic neurons that deliver impulses to the heart.[11] Both stimulants are legal and unregulated, and nicotine is very addictive.[11]

Effects of stress[edit]

Both surprise and stress induce physiological response: elevate heart rate substantially.[12] In a study conducted on 8 female and male student actors ages 18 to 25, their reaction to an unforeseen occurrence (the cause of stress) during a performance was observed in terms of heart rate. In the data collected, there was a noticeable trend between the location of actors (onstage and offstage) and their elevation in heart rate in response to stress; the actors present offstage reacted to the stressor immediately, demonstrated by their immediate elevation in heart rate the minute the unexpected event occurred, but the actors present onstage at the time of the stressor reacted in the following 5 minute period (demonstrated by their increasingly elevated heart rate). This trend regarding stress and heart rate is supported by previous studies; negative emotion/stimulus has a prolonged effect on heart rate in individuals who are directly impacted.[13] In regard to the characters present onstage, a reduced startle response has been associated with a passive defense, and the diminished initial heart rate response has been predicted to have a greater tendency to dissociation.[14] Further, note that heart rate is an accurate measure of stress and the startle response which can be easily observed to determine the effects of certain stressors.

Factors decreasing heart rate[edit]

The heart rate can be slowed by altered sodium and potassium levels, hypoxia, acidosis, alkalosis, and hypothermia. The relationship between electrolytes and HR is complex, but maintaining electrolyte balance is critical to the normal wave of depolarization. Of the two ions, potassium has the greater clinical significance. Initially, both hyponatremia (low sodium levels) and hypernatremia (high sodium levels) may lead to tachycardia. Severely high hypernatremia may lead to fibrillation, which may cause CO to cease. Severe hyponatremia leads to both bradycardia and other arrhythmias. Hypokalemia (low potassium levels) also leads to arrhythmias, whereas hyperkalemia (high potassium levels) causes the heart to become weak and flaccid, and ultimately to fail.[11]

Heart muscle relies exclusively on aerobic metabolism for energy. Severe hypoxia (an insufficient supply of oxygen) leads to decreasing HRs, since metabolic reactions fueling heart contraction are restricted.[11]

Acidosis is a condition in which excess hydrogen ions are present, and the patient's blood expresses a low pH value. Alkalosis is a condition in which there are too few hydrogen ions, and the patient's blood has an elevated pH. Normal blood pH falls in the range of 7.35–7.45, so a number lower than this range represents acidosis and a higher number represents alkalosis. Enzymes, being the regulators or catalysts of virtually all biochemical reactions - are sensitive to pH and will change shape slightly with values outside their normal range. These variations in pH and accompanying slight physical changes to the active site on the enzyme decrease the rate of formation of the enzyme-substrate complex, subsequently decreasing the rate of many enzymatic reactions, which can have complex effects on HR. Severe changes in pH will lead to denaturation of the enzyme.[11]

The last variable is body temperature. Elevated body temperature is called hyperthermia, and suppressed body temperature is called hypothermia. Slight hyperthermia results in increasing HR and strength of contraction. Hypothermia slows the rate and strength of heart contractions. This distinct slowing of the heart is one component of the larger diving reflex that diverts blood to essential organs while submerged. If sufficiently chilled, the heart will stop beating, a technique that may be employed during open heart surgery. In this case, the patient's blood is normally diverted to an artificial heart-lung machine to maintain the body's blood supply and gas exchange until the surgery is complete, and sinus rhythm can be restored. Excessive hyperthermia and hypothermia will both result in death, as enzymes drive the body systems to cease normal function, beginning with the central nervous system.[11]

In different circumstances[edit]

Heart rate (HR) (top trace) and tidal volume (Vt) (lung volume, second trace) plotted on the same chart, showing how heart rate increases with inspiration and decreases with expiration.

Heart rate is not a stable value and it increases or decreases in response to the body's need in a way to maintain an equilibrium (basal metabolic rate) between requirement and delivery of oxygen and nutrients. The normal SA node firing rate is affected by autonomic nervous

system activity: sympathetic stimulation increases and parasympathetic stimulation decreases the firing rate.[15] A number of different metrics are used to describe heart rate.

Resting heart rate[edit]

newborn (0–3 months old)	infants (3 – 6 months)	infants (6 – 12 months)	children (1 – 10 years)	children over 10 years & adults, including seniors	well-trained adult athletes
99-149	89–119	79-119	69–129	59–99	39–59

Electrical Shock Hazards & Its Effects on Human Body

Electrical Technology

0 2 minutes read

Electrical Shock Hazards & Effects

What is Electrical Hazard?

Electrical hazard or ***Electric Shock*** may be defined as "*Dangerous event or condition due to direct or indirect electrical contact with energized conductor or equipment and from which a person may sustain electrical injury from shock, damage to workplace environment, damage to property or both.*

When working with or nearby electrical installations an **electrical shock**, **arc flash** or **arc blast** can occur and a current can go through the body, due to the following situations:

- Direct contact with live parts of the installation (exposure to live parts)
- Contact with parts that normally are not live, but as a consequence of a fault become live accidentally (indirect contact)
- Existence of potential difference between different points in the ground

Related Post: Protective Actions to Avoid & to Reduce Electric Hazardous

The effects of electrical current through the human body vary according to:

- The voltage
- The time the current flows
- The value of the current

- The frequency of the network
- The pathway of the current
- The ability of the person to react

Electrical installations with voltages up to 50 V, in dry places, and up to 25V, in wet or humid places (AC) and up to 120 V in DC are considered safe in what concerns direct and indirect contacts.

The duration of the fault must be limited by the action of protection relays and devices. Below is a given table which shows the different values of current and its effect on human body. Keep in mind that it is the average value and not meant to be same for everyone as it depends on different factors.

Increase of frequency decreases the danger of current through the human body. The called "industrial frequencies" (50 Hz or 60 Hz) are the more dangerous.

Related post: Emergency Planning for Safety & Protection in Industries & Installations

AC or DC, Which one is dangerous ?

Keep in mind that **both the AC and DC voltages and Currents are dangerous & hazardous**. But AC with 50 or 60Hz frequency is more dangerous than the same voltage of level for DC. We will discuss in next post which one is more dangerous and why?

Table 2 – AC and DC Shock Comparison & its effect on human body

The value of the current through the body depends of the resistance of the skin. This resistance depends of several factors such as:

- Humid or wet skin
- Thickness of the skin at the contact point
- Psychological condition
- Weight
- Sex
- Age

The pathway of the current through the human body is unpredictable, and pathways through the heart are the most dangerous. Figure 1 shows possible pathways.

Figure 1 – Pathways of current

When an electrical current flows through the physiological tissues there is an interference with the electrical processes of the human body, namely with nerves, muscles and the heart.

The tissues are also subjected to heating and electrochemical reactions that provoke the deterioration of the tissues.

Related post: All About Electrical Protection Systems, Devices And Units

The hazardous of electrical shock are the following:

- Loose of motion control
- Respiratory arrest
- Pain
- Physical fatigue
- Ventricular fibrillation
- Cardiac arrest
- Burns

Fig 2 – Electrical Shock Hazards & Effects on Human Body – Current & Resistance Chart

Some of these injuries can cause death, namely respiratory arrest, ventricular fibrillation, cardiac arrest and burns.

Loose of motion control can cause pain and secondary injuries due to falls.

Chart: American Burn Association Study of Body burns due to Electric Shock

Ventricular fibrillation is due to the uncontrolled contractions of the heart fibers, that can provoke a cardiac arrest and the lack of brain irrigation, and it is one of the main causes of death due to electrical shock.

Electrical burns are the consequences of Joule effect, and taking into account the injuries they provoke, they are classified in 3 degrees – 1, 2 and 3, being this one the most dangerous and that can cause dead.

Note: Its must to use PPE when working on live and energized equipments for safety and protection