

## Key Medical Terms Associated with the Cardiovascular System

**Aneurysm:** A thin, weakened section of the wall of an artery or vein that bulges outward, forming a balloon-like sac. Common causes are atherosclerosis, syphilis, congenital blood vessel defects, and trauma.

**Angiography:** A diagnostic procedure in which a radiopaque dye is injected through a catheter that has been introduced into a blood vessel and guided to the blood vessel to be examined. The dye flows into the appropriate blood vessel, making abnormalities such as blockages visible on x-rays. Angiography is used to study the blood vessels of the heart (coronary angiography), aorta and its branches (aortography), and blood vessels in the legs (femoral angiography).

**Deep vein thrombosis:** The presence of a thrombus (blood clot) in a deep vein of the lower limbs. It may lead to pulmonary embolism, if the thrombus dislodges becoming an embolus and then lodges within the pulmonary arterial blood flow.

**Arteriosclerosis:** A general term that refers to any thickening and hardening (and loss of elasticity) of the arteries. Atherosclerosis is a specific type of arteriosclerosis, but the terms are sometimes used interchangeably. **Atherosclerosis** refers to the buildup of fatty materials such as cholesterol in and on your artery walls (plaques), which can restrict blood flow.

### Blood Pressure

Arterial pressure is most commonly measured via a **sphygmomanometer**, which historically used the height of a column of mercury to reflect the circulating pressure. Blood pressure (BP) values are generally reported in millimeters of mercury (mmHg), though electronic devices do not use mercury.

For each heartbeat, BP varies between systolic and diastolic pressures. A long standing example of normal measured values for a resting, healthy adult human is 120 mmHg systolic and 80 mmHg diastolic (written as 120/80 mmHg). Although new guidelines from the American Heart Association indicate that a blood pressure of the 120/80 mmHg is actually the low range for prehypertension (see chart below) and a normal resting blood pressure should be less than 120/80 mmHg.

**Systolic pressure** is peak pressure in the arteries, which occurs near the end of the cardiac cycle when the ventricles are contracting. Systolic blood pressure is a function of **ventricular contraction** and is a direct reflection of **cardiac output (stroke volume)**. The systolic pressure is determined by the amount of blood being forced into the aorta and arteries with each ventricular contraction, i.e., stroke volume, and also by the force of contraction. Similarly, if the arterial wall becomes stiffer, as happens in **arteriosclerosis**, the vessels are not able to distend with the increased blood volume and so the systolic pressure is increased

**Diastolic pressure** is minimum pressure in the arteries, which occurs near the beginning of the cardiac cycle when the ventricles are filled with blood. Diastolic blood pressure is the **direct reflection of the vasculature and degree of peripheral resistance**. It can be **high due to increased arteriolar vasoconstriction by the sympathetic nervous system**. This will impede blood flowing out of the arterial system to the capillaries and diastolic pressure will rise. Conversely, if the **peripheral resistance is reduced due to vasodilation**, more blood will flow out of the arterial system and thus diastolic pressure will fall. Drugs that modify the degree of **arterial vasoconstriction** and alter the **peripheral resistance** will affect the diastolic pressure. Vasodilator

drugs such as hydralazine hydrochloride (Apresoline) and minoxidil (Loniten) can be used in the treatment of severe hypertension.

**Which number is more important, top (systolic) or bottom (diastolic)?** This has been a topic of debate and while both are important, studies have shown that systolic blood pressure is a better predictor of events such as coronary heart disease, cardiovascular disease, heart failure, stroke, and end-stage renal disease than diastolic blood pressure DBP. In most people, systolic blood pressure rises steadily with age due to increasing stiffness of large arteries, long-term build-up of plaque, and increased incidence of cardiac and vascular disease.

**This chart reflects blood pressure categories defined by the American Heart Association.**

| Blood Pressure Category                                       | Systolic mm Hg (upper #) |     | Diastolic mm Hg (lower #) |
|---|--------------------------|-----|---------------------------|
| <b>Normal</b>   | less than <b>120</b>     | and | less than <b>80</b>       |
| <b>Prehypertension</b>  | <b>120 – 139</b>         | or  | <b>80 – 89</b>            |
| <b>High Blood Pressure (Hypertension) Stage 1</b>             | <b>140 – 159</b>         | or  | <b>90 – 99</b>            |
| <b>High Blood Pressure (Hypertension) Stage 2</b>             | <b>160 or higher</b>     | or  | <b>100 or higher</b>      |
| <b><u>Hypertensive Crisis</u><br/>(Emergency care needed)</b> | Higher than <b>180</b>   | or  | Higher than <b>110</b>    |

**Hypotension:** Low blood pressure; most commonly used to describe an acute drop in blood pressure as occurs during excessive blood loss.

**Orthostatic hypotension:** An excessive lowering of systemic blood pressure when a person assumes an erect or semi erect posture.

**White coat (office) hypertension:** A stress-induced syndrome found in patients who have elevated blood pressure when being examined by health-care personnel, but otherwise has normal blood pressure.

**Syncope (SIN-ko-pe):** Fainting. A sudden, temporary loss of consciousness followed by spontaneous recovery. It is usually due to decreased blood flow to the brain.

**Vasovagal syncope** (vaso = vessel; vagal = vagus nerve): is the most common (80%) type of syncope that occurs when your body overreacts to triggers, such as emotional stress, trauma, pain, the sight of blood, prolonged standing and straining (such as to have a bowel movement) and being

scared. The nervous system malfunctions causing your heart rate to slow and the blood vessels to vasodilate. This allows blood to pool in your legs, which lowers your blood pressure resulting in diminished blood flow to your brain, and you faint. The medulla oblongata of the brainstem malfunctions resulting in the simultaneous activation of **parasympathetic nervous system (vagal tone)** and deactivation of **sympathetic nervous system tone**.

This results in the following hemodynamic responses:

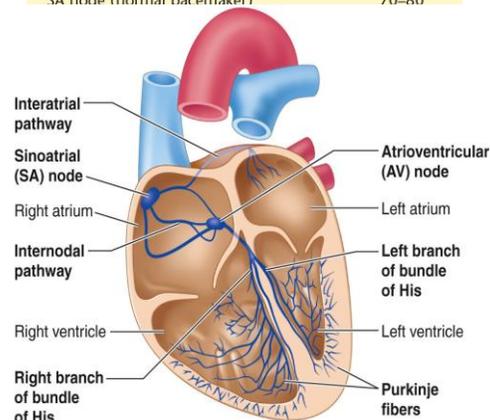
1. **Activation of parasympathetic nervous system** causes a **cardioinhibitory** response, characterized by a drop in heart rate and in contractility leading to a decrease in cardiac output that is significant enough to result in a loss of consciousness.
2. **Deactivation of the sympathetic nervous system** causes **vasodilation** due to a withdrawal of sympathetic nervous system on vascular tone. Characterized by a drop in blood pressure (to as low as 80/20) without much change in heart rate.

Prior to losing consciousness, the individual frequently experiences symptoms such as lightheadedness, nausea, the feeling of being extremely hot (accompanied by sweating), ringing in the ears (tinnitus), uncomfortable feeling in the heart, tunnel vision, and sometimes a feeling of nervousness.

**Normal Pacemaker Activity:** The various autorhythmic cells have different rates of slow depolarization to threshold thus they have different rates of generating action potentials. The heart cells with the fastest rate of action potential initiation are localized in the **SA node**. The following analogy shows how the SA node drives the rest of the heart at its own pace. Suppose a train has 100 cars, 3 of which are engines capable of moving on their own; the other 97 cars must be pulled. One engine (the SA node) can travel at 70 miles/hour (mph) on its own, another engine (the AV node) at 50 mph, and the last engine (the Purkinje fibers) at 30 mph. If all these cars are joined, the engine that can travel at 70 mph will pull the rest of the cars at that speed. The engines that can travel at lower speeds on their own will be pulled at a faster speed by the fastest engine and therefore cannot assume their own slower rates long as they are being driven by a faster engine. The other 97 cars (nonautorhythmic, contractile cells), being unable to move on their own, will likewise travel at whatever speed the fastest engine pulls.

**Abnormal Pacemaker Activity:** If for some reason the fastest engine breaks down (**SA node damage**), the next-fastest engine (**AV node**) takes over and the entire train travels at 50 mph; that is if the SA node becomes nonfunctional, the **AV node assumes pacemaker activity** (Figure b). The non-SA nodal autorhythmic tissues are latent pacemakers that can take over, although at a lower rate, if the normal pacemaker fails. If impulse conduction becomes blocked between the atria and the ventricles, the atria continue at the typical rate of 70 beats per minute, and the ventricular tissue, not being driven by the faster SA nodal rate, assumes its own much slower autorhythmic rate of about 30 beats per minute, initiated by the ventricular autorhythmic cells (**Purkinje fibers**). This situation is like a breakdown of the second engine (AV node) so that the lead engine (SA node) becomes disconnected from the slow third engine (Purkinje fibers) and the

| TABLE 9-1 Normal Rate of Action Potential Discharge in Autorhythmic Tissue of the Heart |                              |
|---|------------------------------|
| Tissue  | Action Potential per Minute* |
| SA node (normal pacemaker)  | 70–80                        |



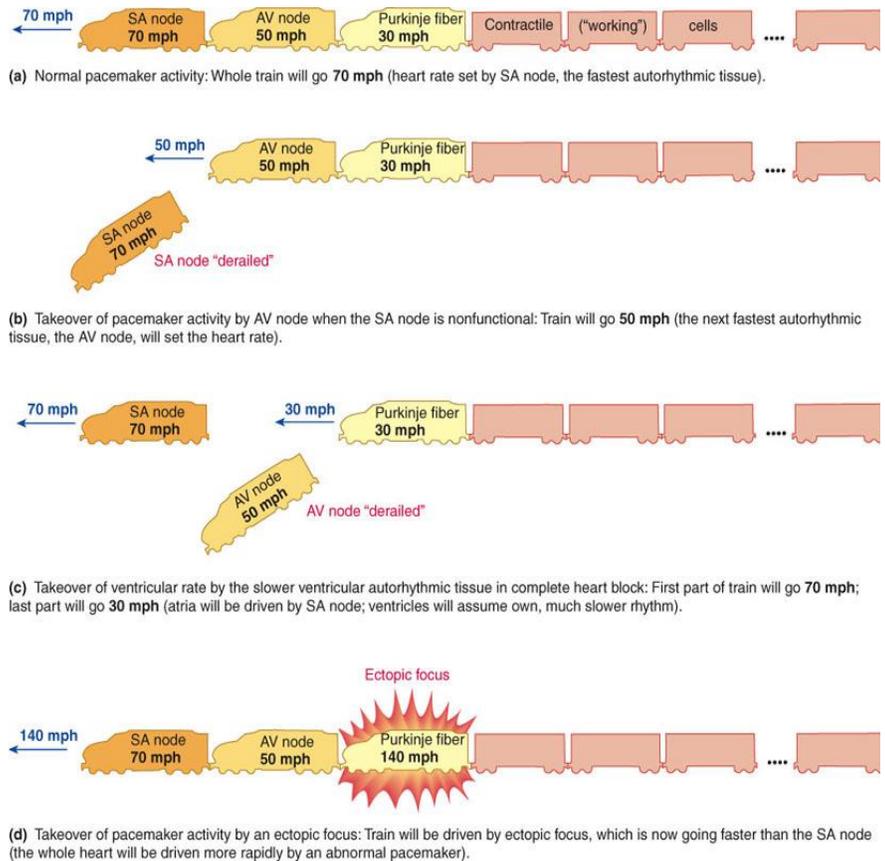
(a) Specialized conduction system of the heart

rest of the cars (Fig c). The lead engine (and cars connected directly to it; that is, the arterial cells) continues at 70 mph while the rest of the train proceeds at 30 mph. This **complete heart block** occurs when the conducting tissue between the atria and ventricles is damaged, as for example during a heart attack, and becomes nonfunctional. A ventricular rate of 30 beats per minute will support only a very sedentary existence and the person could become comatose.

When a person has an abnormally low heart rate, as in SA node failure or heart block, an **artificial pacemaker** can be used. Such an implanted device rhythmically generates impulses that spread throughout the heart to drive both the atria and ventricles at the typical rate of 70 beats per minute.

**Premature ventricle contractions (PVC):** Occasionally an area of the heart, such as a Purkinje fiber, becomes overly excitable and depolarizes more rapidly than the SA node. (The engine suddenly goes faster than the lead engine – Fig d). This abnormally excitable area, an **ectopic focus** or **ectopic pacemaker**, initiates a premature action potential that spreads throughout the rest of the heart before the SA node can initiate a normal action potential (**ectopic** mean “out of place”). An occasional abnormal impulse from a ventricular ectopic pacemaker or focus produces a **premature ventricular contraction (PVC)**. Single PVCs are common and not dangerous. If the ectopic focus continues to discharge at its more rapid rate, pacemaker activity shifts from the SA node to the ectopic focus. The heart rate abruptly becomes greatly accelerated and continues this rapid rate for a variable time period until the ectopic pacemaker returns to normal. These **rapid PVCs** are called **ventricular tachycardia** (defined as four or more PVCs without intervening normal beats), also known as **VT** or **V-tach**. Such overly irritable areas may be associated with heart disease, but more frequently they occur in response to **anxiety, lack of sleep, or excess caffeine, nicotine, or alcohol consumption**. Medications can be used to slow down rapid heart rates, or the abnormal portions of the conducting system can be destroyed.

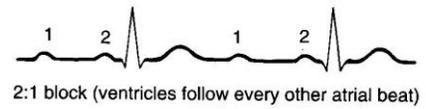
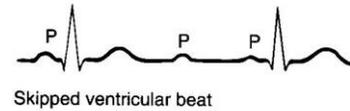
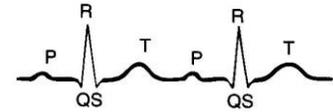
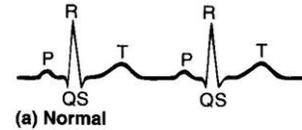
**Ventricular fibrillation** can follow in situations of Multiple PVCs or V-tach. The resulting condition is also known as **cardiac arrest**, is rapidly fatal, because the heart quivers and stops pumping blood.



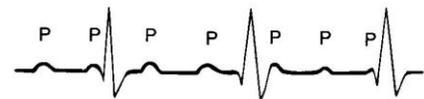
**Diagnosing Abnormal Heartbeats due to Atrioventricular Block:** The normal way by which impulses can pass from the atria into the ventricles is through the AV node and A-V bundle (bundle of His). Damage to this conduction pathway can slow or block the rate of impulse conduction. Conditions that can cause this include:

1. Compression (mechanical distortion) of the AV bundle by scar tissue or by calcified portions of the heart can depress or block conduction.
2. Ischemia of the AV node or bundle fibers can decrease their conduction
3. Infection and inflammation from conditions such as myocarditis due to rheumatic fever or diphtheria.

The resulting slowing or blocking of the impulses at this region results in a condition that called a heart block. Figure (a) shows a normal ECG and (b) through (d) are examples of the types of heart blocks.



(c) Second-degree blocks



(atrial beats occur regularly, ventricular beats occur at slower, unrelated pace)

**First-degree heart block:** Figure (b), The AV node and the AV bundle slow the passage of impulses that are heading for the ventricular myocardium. A first-degree block is a delay of conduction from the atria to the ventricles but not an actual blockage of conduction. Although a delay exists, the regular rhythm of the heart continues, and each atrial beat is followed by a ventricular contraction. First-degree heart block rarely causes any symptoms, and it usually doesn't require treatment.

**Second-degree heart block:** Figure (c), Occurs when the delay at the AV node last long enough that it may not complete the signal to the ventricles. The result is that the ventricles will not be stimulated, and the normal “atria-ventricles, atria-ventricles” pattern will disappear. In this instance, there will be an atrial P wave but not a QRS wave or T wave, and it is said that there are “dropped beats” of the ventricles. A mild second-degree block may produce only an occasional skipped beat, but more substantial delays, the ventricles will follow every second atrial beat. The resulting pattern of “atria, atria-ventricles, atria, atria-ventricles” is known as a two-to-one (2:1) block because you have two P waves before the QRS and T wave. Three-to-one or even four-to-one blocks are also encountered. Depending on often the 2<sup>nd</sup> degree block occurs the person can have symptoms of dizziness and may require a pace maker as treatment.

**Third-degree heart block or complete heart block:** Figure (d), The conducting pathway stops functioning. The atria and ventricles continue to beat, but their activities are no longer synchronized. The atria follow the pace set by the SA node, beating 70-80 times per minute, and the ventricles follow the commands of the AV node, beating at a rate of 40-60 beats per minute. Complete heart block can result in sudden cardiac arrest and death. Pace maker implantation will be required for treatment.