4

# The Electrics

## Quick Look



The previous two chapters explored the mechanics of the heart, looking first at cardiac anatomy, then at the factors that affect cardiac output. Attention now turns to the heart's control centre, the electrical conduction system.

Without the innervation by the heart's electrical pathways, the heart muscle will simply not pump. An absence of cardiac electrical activity - the "flat line" seen on a monitor - is definitely not a good sign!

Starting with the SA node, each of the major electrical components of the heart are explored in this chapter. Attention is focused on pacemaker and non-pacemaker sites. The processes of depolarization and repolarization are briefly addressed. Finally, the autonomic control of heart rate is discussed.

While this may sound akin to discussing particle physics, the heart's electrics are much simpler and at least as interesting. There is much to be digested over the next dozen pages or so. Let's dig in.

*There must be a seat...from which heat and life are dispensed to all parts...the heart is this place... I trust no one will deny.*

William Harvey (1628)

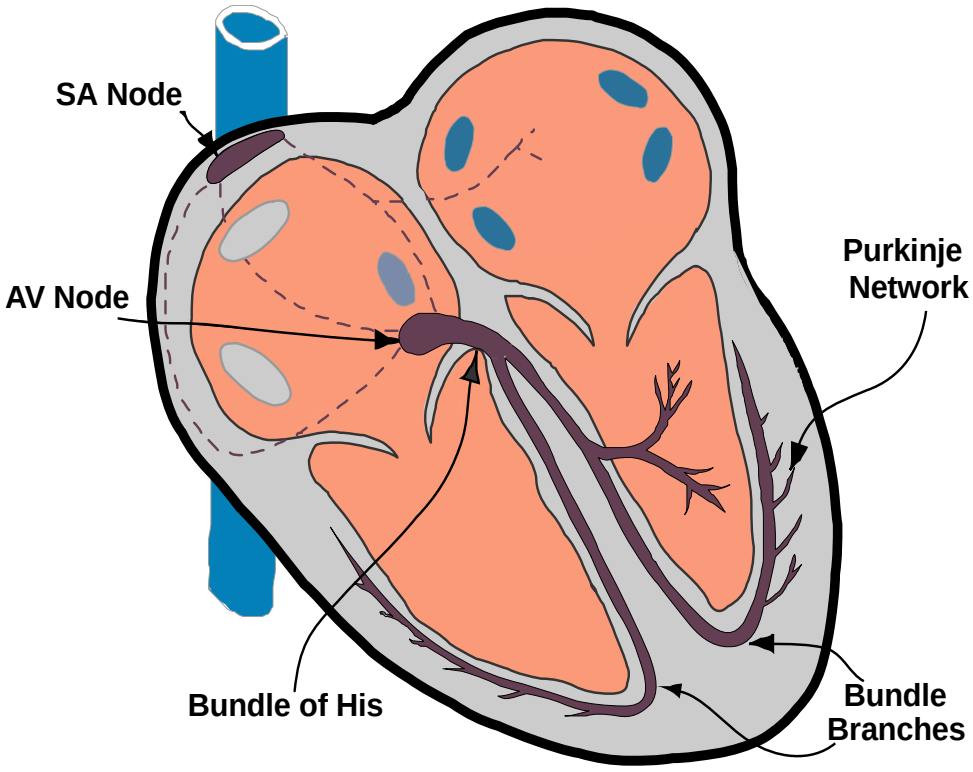
# Electrical Overview

Late in the 1700s, physiologists realized that an electric stimulus causes muscles to contract. In the past 200 years, electrophysiologists have continued to uncover many secrets of cardiac electrophysiology. With these discoveries, an in depth understanding of cardiac electrophysiological events has led to insights into dysrhythmia interpretation and treatment.

New findings continue at a brisk pace, revealing the many evolutionary features of the heart.

The heart's electrical system is composed of five significant components: the SA node, the AV node, the Bundle of His, the Bundle Branches, and the Purkinje network. These electrical structures work as a cohesive interdependent team, conducting electrical impulses rapidly throughout the heart. Their location within the heart is depicted in the figure 3.1 below.

**Figure 4.1 The Heart's Electrical Pathway**




*A wave of depolarization normally begins with the SA node. This electrical wave from the SA node passes quickly across the atria, through the AV junction (the AV node and the Bundle of His) then across the ventricles via the bundle branches and the Purkinje network.*

As an electrical impulse is initiated and then conducted over the heart, affected cardiac cells undergo an ionic shift, called **depolarization**. The interior of cardiac cells at rest have a negative charge relative to the outside of the cell. During depolarization, positive ions enter the cell, changing the cell's polarity. Soon after, the cells experience a series of ionic shifts that return the cell to its resting state. This subsequent process is called **repolarization**. Contraction of cardiac cells is initiated during repolarization.

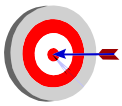
The cells that initiate and then conduct this impulse can be split into two groups, pacemaker cells and non-pacemaker cells. **Pacemaker cells** have the ability to self-initiate an electrical impulse. This relative independence from the body enables the heart to continue beating even if removed from the body (up to 20 minutes). The SA and the AV nodes contain groups of pacemaker cells. **Non-pacemaker cells** conduct the impulse to neighboring cells but usually do not initiate an impulse (i.e. atrium).

The electrical activity of the heart, then, is dependent on pacemaker cells to initiate each wave of depolarization. Normally, this depolarizing wave passes across the cells of the atria, through the AV node, and the Bundle of His. The bundle branches carry this wave through to the Purkinje network where the wave enters the ventricular endocardium, the myocardium and the epicardium in that order. The atria and the ventricles depolarize then contract *from the inside out*.

A wave of repolarization follows. Normally, the repolarization of the endocardium is delayed, so the wave of repolarization begins in the epicardium, proceeds through to the myocardium and finishes at the endocardium - opposite the direction of depolarization. For a more detailed explanation of depolarization and repolarization, see Chapter 4.

# SA Node

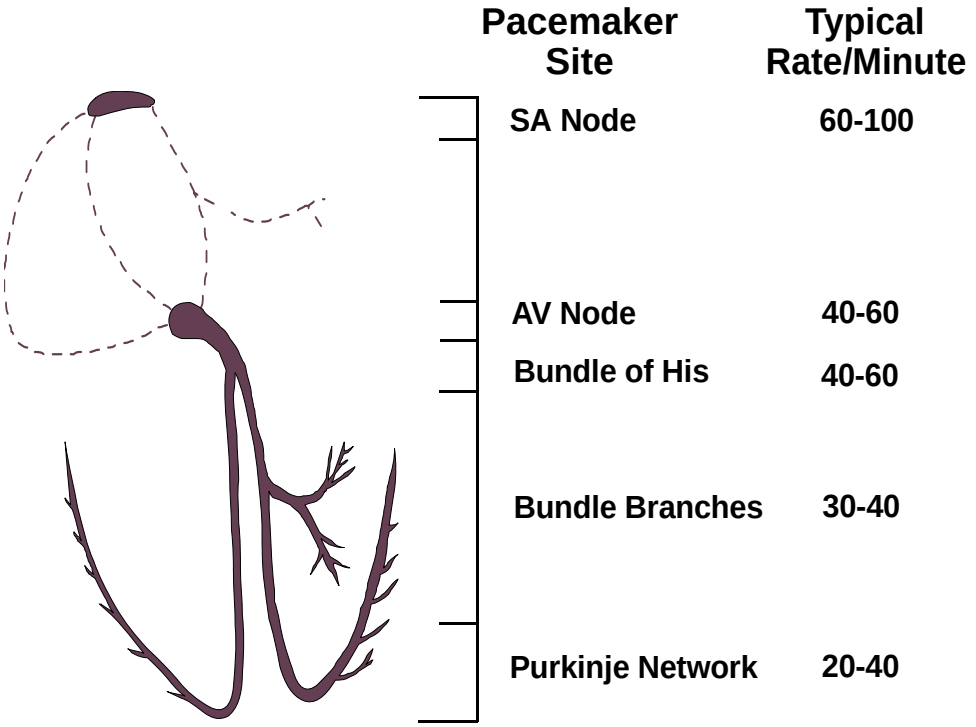
The **SA node**, usually the dominant pacemaker, is located in the right atrium at the opening of the superior vena cava. The SA (sinoatrial) node is a clump of hundreds of specialized cardiac cells that have the ability to self-initiate an electrical impulse. This pacemaking ability, called **automaticity**, makes the SA node a pacemaker site in the heart.



As a general rule, the site in the heart that is able to self-generate the quickest rate, **RULES** the heart. This site is almost always the sinoatrial node (SA node). Thus, the SA node is often called the dominant pacemaker. If an ectopic site (site other than the SA node) begins to fire faster than the SA node, the ectopic site tends to drive the heart.

The SA node initiates an electrical impulse at a rate faster than other pacemaker sites (see Figure 3.2). In the heart, the pacemaker which fires at the quickest pace takes control of heart rate. This is why the SA node is the "dominant pacemaker". A cardiac rhythm that originates from the SA node is called a sinus rhythm.

**Figure 4.2 Pacemaker Sites and Normal Rates**




*These are typical heart rates from various pacemaker sites. Heart rates can vary, though, for each site above and below the range specified. Note that the typical pacemaker rate decreases as the distance from the SA node increases. Lower pacemakers serve as"back-up" in case higher pacemakers fail. The bundle branches and the Purkinje network (both from the ventricles) typically provide an exceptional slow heart rate that is often associated with poor cardiac output. Note also that the absence of atrial activity results in a loss of atrial kick, impacting an already low cardiac output further.*

The SA node normally generates electrical impulses at 60-100 /minute. This rate tends to increase with sympathetic stimulation (norepinephrine and epinephrine) and slows with parasympathetic stimulation (acetylcholine and the Vagus nerve). Therefore, Vagal stimulation can slow the SA node to rates below 60/minute causing a sinus **bradycardia**. Sympathetic stimulation can cause rapid sinus rhythms called sinus **tachycardias**. The control of heart rate is addressed in more detail later in this chapter.

# Atrial Conduction and the AV Junction

Once the SA node initiates an electrical impulse, the resulting electrical wave moves across the right and left atria. The atrial septum serves as an electrical insulator. Bachman's Bundle tunnels through the atrial septum to continue the electrical wave across the left atrium. The wave takes approximately 3/100 of a second to cross the atria and arrive at the AV node.

The atrioventricular (AV) node is a rounded bulbar structure of specialized cells similar to the SA node. The AV node also has intrinsic automaticity, with the ability to serve as a pacemaker in case of SA nodal failure. The AV node usually does not initiate impulses, though, as its intrinsic firing rate is normally 40-60 /minute, slower than the SA node. Remember, the fastest pacemaker site rules.

**Figure 4.3 Connective Tissue Structures and Electrical Conduction**

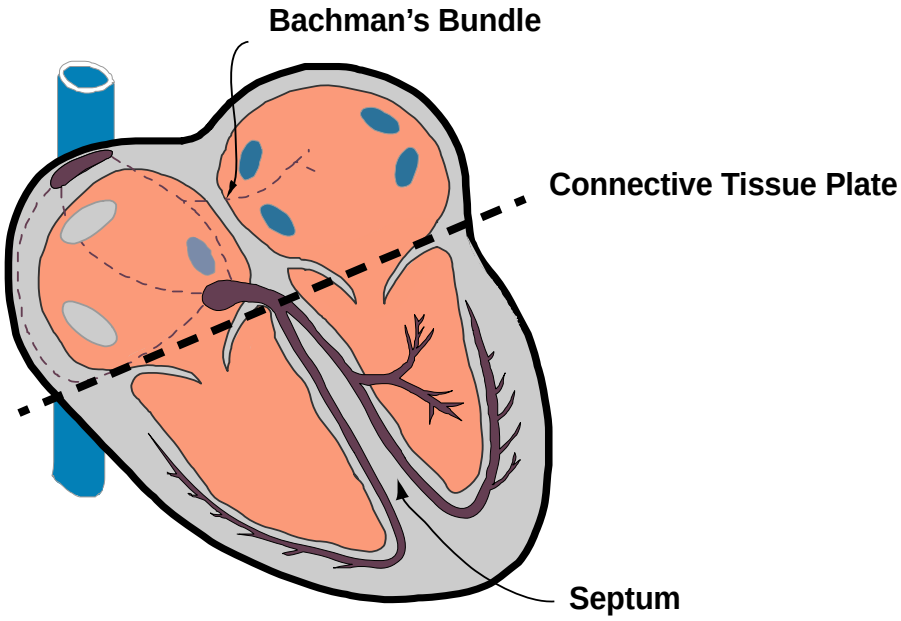



*Figure 3.3 depicts the structures that form the cardiac skeleton, the septum and the plate of connective tissue that separates the atria from the ventricles. Connective tissue does not conduct electrical impulses, serving rather as an electrical insulator or barrier. To connect the left and right atria electrically,* ***Bachman's bundle*** *burrows through the atrial septum. The* ***Bundle of His*** *performs a similar function, connecting the atria electrically with the ventricles. Note that without the Bundle of His, supraventricular impulses would not be transmitted through to the ventricles. The ventricles would then be dependent on their own slow intrinsic pacemakers.*

The AV node has a second important role. The AV node and the bundle of His slows impulse conduction to allow the atria time to contract prior to ventricular contraction. In other words, the AV junction provides the time delay for an atrial kick. The time taken to cross the small AV junction is 10-12/100 of a second (a significantly lengthy period for such a small structure).

The **Bundle of His** serves as an electrical connection between the atria and the ventricles, traversing the fibrous plate that separates the atrial and ventricular electrical systems. The AV node and bundle of His form the AV junction (sometimes just called the junction).

Note that the AV junction, atria and SA node are the three main supraventricular (located above the ventricles) electrical sites. This is an exceptionally simple and important distinction. As you will soon discover, in order for an impulse to be transmitted down the bundle branches, the impulse must be supraventricular in origin.

# The Ventricular Conduction System

The ventricles' electrical system is exceptionally efficient. To produce a forceful, coordinated contraction, the electrical wave must travel quickly through the large ventricles. Knowing that the atria depolarize over 3/100 of a second, how long would depolarization take to crest across the ventricles (three times the size of the atria)?

Venture a guess? The electrical wave crosses the ventricles in a mere 1/100 of a second. The wave moves like lightning! The question is: How is this rapid conduction accomplished?

**Table 4.1 .Duration of Depolarizing Waves**

| Structure | Duration of Depolarizing Wave |
|---|---|
| Atria | 0.03 seconds (3/100) |
| AV Junction | 0.10-0.12 seconds (10-12/100) |
| Ventricles | 0.01 seconds (only 1/100) |

*The time taken for depolarization is somewhat counterintuitive. The depolarizing wave takes the longest duration moving through the small AV junction (and thus allowing for atrial kick) and the shortest time covering the large ventricles.*

First, the specialized bundle branches and Purkinje network facilitate this rapid conductivity (refer to Figure 3.1 "The Heart's Electrical Pathway" on page 54). With the atria, only one wave is propagated. Via the bundle branches, this impulse is split into at least **three simultaneous waves**, thus reducing the distance each wave must travel. Less distance equates to less time. As a result, the time taken to depolarize the ventricles is reduced considerably.

Second, the bundle branches and Purkinje network are composed of Purkinje fibers, specialized cardiac cells that are tailored for fast conductivity. These rapidly conducting cells carry the impulses through connective tissue, reaching contractile cardiac tissue at the distal ends of the Purkinje network. This encapsulated electrical network is extremely efficient, rapidly carrying a depolarizing wave throughout the ventricles.

Note that the speed of contraction translates directly into the force of contraction. The faster that the ventricles can depolarize and subsequently contract, the greater the force of contraction. A greater force of contraction increases both stroke volume and cardiac output. Force of contraction is referred to as **contractility**.

Why is the speed of ventricular depolarization important? As mentioned in the box above, the faster the depolarization, the greater the force of contraction. But there is another good reason to take the time to fully grasp what causes the ventricles to depolarize with varied speeds.

We established that an electrical wave envelops the ventricles very quickly **IF** the bundle branches and the Purkinje network are utilized. This is comparable to getting off the back country roads and racing down the freeway. The rapidly conducting bundle branches could be called the Autobahn* of the heart.

Where is the only location to ramp onto the Autobahn of the heart? Remember that the bundle branches are largely encapsulated in connective tissue. The impulse must have travelled through the bundle of His to arrive at the bundle branches (the Autobahn). This is the only entry point to the Autobahn.

Where are we going with this? If the impulse travels through the bundle of His, then it originated in either the bundle of His or above the bundle of His (i.e. the AV node, the atria or the SA node). Simply stated, for a *rapid* wave of depolarization to envelop the ventricles, the impulse must originate above the ventricles.

Let's repeat this for effect. **In order for rapid depolarization of the ventricles to occur, the impulse must originate from a supraventricular site.**

***The Autobahn is an ultra-fast freeway that connects several countries in western Europe.**

Here's the crunch. On an electrocardiogram - an ECG - the QRS complex is often narrow. We are jumping ahead a bit here, but a QRS complex represents ventricular depolarization. An ECG is the graphical representation of the electrical activity of the heart, with the horizontal axis (width) of the ECG being a measurement of time. A narrow QRS then equates to rapid ventricular depolarization, taking very little time. A **narrow QRS, then, occurs when the impulse originates above the ventricles**.

**Figure 4.4 Ventricular Conduction Speed and the QRS Complex**

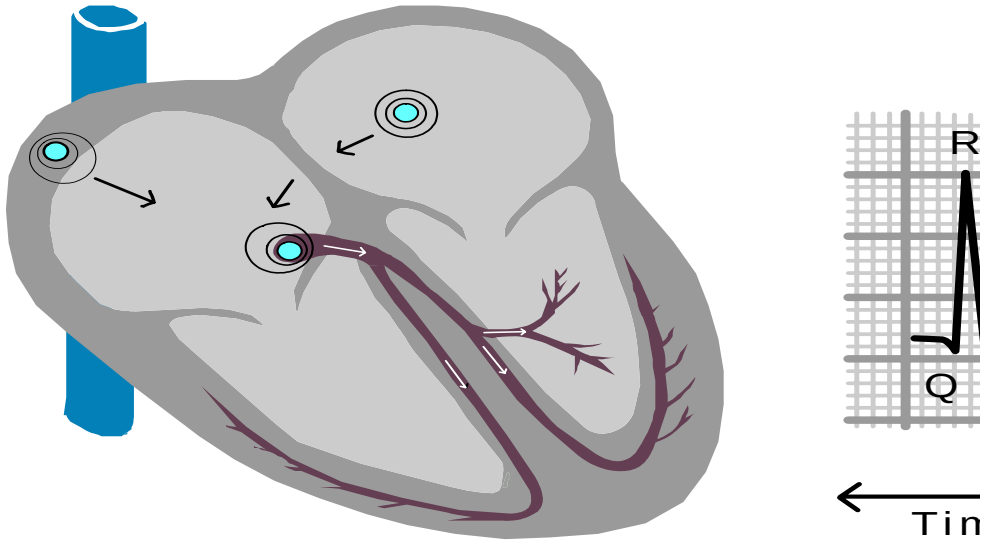



*Figure 3.4 illustrates an impulse originating from the SA node, the atria or the junction. These supraventricular impulses are transmitted to the ventricles via the bundle branches, metaphorically referred to as the Autobahn. Since the Autobahn or bundle branches are used, the ventricles will depolarize rapidly, resulting in a narrow QRS complex.*

Consider for a moment an ectopic impulse originating in the right ventricle. Instead of three simultaneous depolarizing waves, one wave depolarizes the right and then the left ventricle. Of course, more distance is covered by the one wave, taking more time. Instead of the Autobahn, the wave travels the back country roads. With more time taken for ventricular depolarization, a wide QRS results.

When interpreting an ECG, the location of the originating impulse for each beat is quite important. In fact, the naming of most cardiac rhythms begin with the site that the impulse originated. For example, a rhythm that consistently originates from the sinoatrial node is called a sinus rhythm. If a beat originates from the AV junction, it is called a junctional beat.

Therefore, based on what we know about the QRS and the speed of depolarization, a narrow QRS occurs with supraventricular rhythms. A wide QRS complex is commonly associated with ventricular rhythms. This is perhaps the most important step in

identifying cardiac rhythms. Is the QRS wide or narrow? The width and shape of the QRS is addressed in more detail in the next two chapters. As it stands, though, you are already equipped to differentiate between supraventricular and ventricular rhythms.

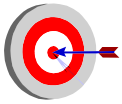
Asking the question, "Is the QRS wide or narrow" is an important step in ECG interpretation. A narrow QRS occurs when an impulse that originates from above the ventricles travels down the rapidly conducting bundle branches to depolarize the ventricles.

We sneaked ahead a little to look at one aspect of the ECG. It is important, though, to integrate your knowledge of the heart's electrical structures with the skill of ECG interpretation. Understanding why a QRS is narrow is much better than memorizing the particulars of every cardiac rhythm.

# Controlling Heart Rate

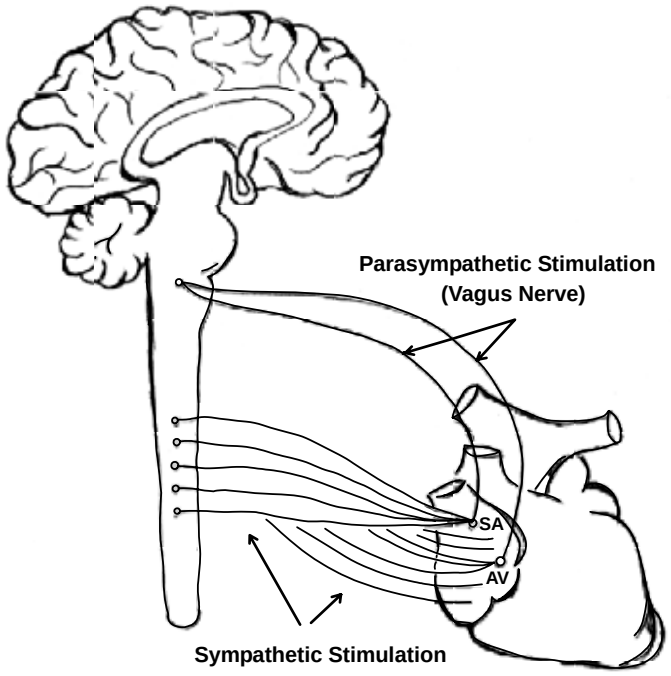
Heart rate, the numbers of beats or cardiac cycle per minute, is the result of three factors: intrinsic control by the heart's pacemakers, sympathetic stimulation and parasympathetic stimulation. The heart's pacemakers have their own intrinsic rate of impulse formation. For heart transplant recipients, without the benefit of cardiac innervation, this is a welcome phenomenon. Physiologists have determined that the SA node would beat at a rate of about 100/minute without any other influences.

Typical heart rates, though, range across a much wider continuum due primarily to the influence of the autonomic nervous system. Influence from the sympathetic nervous system increases heart rate and the speed of conductivity. Catecholamines released from the adrenal glands (i.e. epinephrine) can also produce a similar effect.



**Terminology**
The autonomic nervous system has dramatic effects on the cardiovascular system. When regards to the heart, sympathetic stimulation can yield a positive **chronotropic** (rate) effect, increasing heart rate. In contrast, the Vagus nerve produces a negative chronotropic effect slowing the heart rate. The Vagus nerve also slows the conductivity across the AV node. This is called a negative **dromotropic** (speed of conduction) effect. Sympathetic stimulation - particularly beta 1 stimulation - causes a positive **inotropic** (force of contraction) response, meaning that the force of contraction has increased. The medication Dopamine is known as a positive inotrope because it has the effect of increasing cardiac contractility.

**Figure 4.5 The Autonomic Nervous System and the Heart**




*While sympathetic stimulation innervates most of the heart, parasympathetic stimulation via the Vagus nerve is more specific, innervating the SA and AV nodes.*

The main effect of the catecholamines epinephrine and norepinephrine is an increased cardiac output. The alpha effect, vasoconstriction, shunts blood away from the periphery and improves preload and afterload (see Chapter 2). Coronary and cerebral perfusion are also enhanced. The beta 1 effects of epinephrine increase cardiac output by increasing both heart rate and stroke volume (contractility).

Unfortunately, beta 1 stimulation also increases myocardial oxygen demand. Beta 1 stimulation is also responsible for dysrhythmia generation. It is not surprising that beta blockers, which tend to slow heart rate, reduce cardiac contractility, decrease myocardial workload and decrease dysrhythmias are one of the most important medical treatments for myocardial infarctions (AMI). The morbidity and mortality of an AMI is reduced by as much as 40% with early beta blocker administration.

Parasympathetic innervation produces the opposite effect, slowing the rate of impulse formation by the SA node and slows conductivity through the AV nodes. Parasympathetic influence occurs via the Vagus nerve (acetylcholine). Vagal stimulation is also known to decrease the contractility of the atria as well as cause peripheral vascular dilation.

**Table 4.2 Cardiovascular Receptor Sites and Responses to the Autonomic Nervous System**

| Autonomic Nervous System | Principal Chemicals | Receptors (Effects) | Sites Affected | Site Response |
|---|---|---|---|---|
| Sympathetic | catecholamines (norepinephrine, epinephrine) | **Alpha** (constriction) | abdominal, peripheral, coronary blood vessels | arterial and venous constriction |
| | | **Beta 1** (increases heart rate, enhances contractility, increases cardiac irritability) | cardiac muscle | increased heart rate and strengthened force of contraction |
| | | **Beta 2** (dilation of bronchioles) | bronchioles | bronchioles dilates |
| Parasympathetic | acetylcholine | cholinergic receptors | SA node, AV node, atria, coronary vessels | slows rate, conductivity and weakens atrial contraction; dilates coronary vessels |

*Table 3.2 presents a summary of the cardiac effects of the autonomic nervous system. Parasympathetic and sympathetic innervation tend to produce opposite effects. Sites that experience both parasympathetic and sympathetic innervation, such as the SA and AV nodes, are constantly being pulled in different directions. For example, with significant Vagal stimulation (parasympathetic nervous system), the heart rate tends to slow. If Vagal stimulation is blocked by the administration of a vagolytic medication such as Atropine, the sympathetic nervous system exerts its effect, driving the heart rate up once again.*

Note that whereas sympathetic stimulation tends to blanket the heart, parasympathetic stimulation is limited primarily to the SA node, the atria and the AV node. During episodes of rapid heart rates that originate in above the ventricles (supraventricular tachycardias), Vagal stimulation can often slow or even terminate the fast rhythm by slowing the conductivity through the AV node.

Several techniques can cause **Vagal stimulation**: the Valsalva maneuver or "bearing down", carotid sinus massage, immersing a person's head in cold water, and even a change of position. Perhaps not as popular is the digital anal sweep. Note that all forms of Vagal stimulation can slow heart rates significantly.

Vagal stimulation can also produce very slow heart rates called **bradycardias**. For athletes, regular exercise tends to increase vagal tone with resting heart rates often being in the forties or fifties. Excess Vagal stimulation for people with structural heart disease can produce disastrous consequences, causing bradycardias that may be associated with hemodynamic compromise. Even periods of asystole - an absence of heart rate - can be caused by Vagal innervation.

Table 3.2 outlines the various effects of the sympathetic and parasympathetic nervous systems. Heart rate is the product of several competing drives. Behind the parasympathetic and sympathetic innervation is the body's need to quickly respond to internal and external stressors to keep its steady state. The ability of the heart to react quickly with changes in heart rate is a major factor in holding this state of homeostasis.

# Summary

In this chapter we completed what we began in Chapter 2: review the anatomy and physiology of the heart. Understanding the inner workings of the heart is vital to ECG interpretation and to responding effectively to acute cardiac events. Of course, this review has been a simple, high level review. Hurst's The Heart presents an *overview of the heart* in a short 2600 pages! Nevertheless, we have covered the essentials necessary to our work at hand.

This chapter is an abbreviated description of the heart's electrical system. The mechanical aspects of the heart are intimately connected to the heart's electrical system. A dysfunctional electrical system often negatively impacts the heart's effectiveness as a pump. For example, atrial fibrillation results in the loss of atrial kick.

The SA (sinoatrial) node has the ability to self-initiate an electrical impulse. This ability, called automaticity, makes the SA node a pacemaker site for the heart. The fact that the SA node normally fires at rates greater than other pacemakers (60-100/minute) makes the SA node most often the dominant pacemaker.

The AV node also has the ability to initiate impulses, serving as a back-up in the case of SA nodal failure. The AV node significantly slows down the transmission of the electrical wave of depolarization, providing time for atrial kick prior to ventricular contraction.

The bundle of His carries the impulse from the AV node in the atria to the bundle branches in the ventricle. The bundle of His and the AV node, called the AV junction, can serve as a pacemaker at 40-60 beats/minute.

The bundle branches and the Purkinje network facilitate rapid depolarization throughout the ventricles. These electrical structures also can self-initiate impulses if necessary with typical rates of 20-40/minute. We referred to the bundle branches/Purkinje fibers as the Autobahn of the heart. Impulses that originate above the ventricles are associated with a narrow QRS complex.

Lastly, heart rate control was explored. Heart rate is the product of three factors: intrinsic impulse formation, sympathetic and parasympathetic stimulation. Sympathetic stimulation increases heart rate and contractility whereas parasympathetic stimulation slows heart rate and reduces contractility. Measuring heart rate is a useful sign when assessing a person's state of homeostasis.

# Chapter Quiz

1. The Vagus nerve stimulates (circle all that apply):

a) the SA node
b) the AV node
c) the ventricles
d) the atria

2. Normally, the dominant pacemaker of the heart is the SA node.

True or False

3. Number the following structures in the expected order of electrical transmission beginning with the normal dominant pacemaker.

___ AV Node
___ SA Node
___ Bundle of His
___ Purkinje Network
___ Bundle Branches

4. The primary role of the bundle of His is to electrically connect the atria with the ventricle.

True or False

***Answers:*** ***1.*** *a),b),d);* ***2.*** *True;* ***3.*** *SA node, AV node, Bundle of His, Bundle branches, Purkinje network;* ***4.*** *True*

5. The (sympathetic, parasympathetic) nervous system uses the chemical norepinephrine. The action of norepinephrine on the SA node is to (increase, decrease) its rate of firing. (Automaticity, Synchronicity) is the property of cells to self-initiate an electrical impulse.

6. The layers of the heart depolarize in what order? (Number the layers in order)

___ Epicardium
___ Endocardium
___ Myocardium

7. Ventricular depolarization occurs most rapidly if the impulse has a (ventricular, supraventricular) origin.

8. A QRS complex represents:

a) atrial depolarization
b) atrial repolarization
c) ventricular depolarization
d) ventricular repolarization

9. Because hearts that are transplanted are not innervated by either the parasympathetic or sympathetic nervous systems, an electronic pacemaker is required to keep the heart beating.

True or False

10. The (epicardium, endocardium) experiences a delay in repolarization. This causes the (epicardium, endocardium) to begin the process of repolarization.

11. The following structures are supraventricular (circle all that apply):

a) bundle branches
b) SA node
c) bundle of His
d) AV node
e) Purkinje network

12. The SA node is located near the juncture of the (right, left) atrium and the _____.

***Answers:*** ***5.*** *sympathetic, increase, automaticity;* ***6.*** *endocardium, myocardium, epicardium;* ***7.*** *supraventricular;* ***8.*** *ventricular depolarization;* ***9.*** *False;* ***10.*** *endocardium, epicardium;* ***11.*** *b),c),d);* ***12.*** *right, superior vena cava;*

13. Depolarization is the same as contraction.

True or False

14. Pacemaker cells are normally located in the:

a) SA node
b) AV node
c) atrial myocardium
d) bundle branches
e) Purkinje network
f) bundle of His
g) ventricular myocardium

15. At rest, the interior of cardiac cells have a (positive, negative) polarity as compared with the cell exterior.

16. The atrioventricular (AV) junction serves the following functions:

a) protects the ventricles from overly rapid atrial rates
b) back up pacemaker during periods of SA nodal failure
c) slows conduction to allow time for atrial kick
d) the pathway between the atria and the ventricles
e) all of the above

17. The SA node is a cluster of hundreds of specialized cells that possess the ability to initiate impulses. An area of the heart that shares very similar cells is the:

a) the atria
b) the AV node
c) bundle branches
d) the ventricles

18. The skeleton of the heart is composed of connective tissue. This connective tissue occupies much of the septum as well as a plate that separates the atria from the ventricles. Because this connective tissue does not conduct electrical impulses, conducting cells burrow through to connect each of the heart's chambers. The structure that connects the atria electrically to the ventricles is called the:

a) SA node
b) Bachman's bundle
c) AV node
d) bundle of His

***Answers:*** ***13.*** *False;* ***14.*** *a),b),d),e),f);* ***15.*** *negative;* ***16.*** *e);* ***17.*** *b);* ***18.*** *d);*

19. Pacemaker sites furthest from the SA node tend to produce the slowest rates.

True or False

20. The depolarization of the ventricles progress very rapidly due to (circle all that apply):

a) specialized Purkinje fibers
b) simultaneous depolarization of several waves across the ventricles
c) the bundle branches (Autobahn of the heart)
d) the muscle cells are able to contract much faster than skeletal fibers

21. Normally, more time is taken to depolarize the AV junction than the rest of the heart combined.

True or False

22. An impulse that originates in the myocardium tends to depolarize the ventricles (slower, faster) than if the impulse originated from a supraventricular focus.

23. Heart rate is influenced by (circle all that apply):

a) intrinsic ability of the pacemaker cells
b) automaticity
c) sympathetic innervation
d) parasympathetic innervation
e) epinephrine released from the adrenals

24. A cardiac rhythm that originates from the SA node is called a (circle all that apply):

a) sinus rhythm
b) atrial rhythm
c) supraventricular rhythm
d) ventricular rhythm

25. The cardiac effects of the sympathetic nervous system include (circle all that apply):

a) positive chronotropic effect
b) negative dromotropic effect
c) positive inotropic
d) proarrhythmic
e) positive dromotropic
f) negative chronotropic

***Answers:*** ***19*** *True;* ***20.*** *a),b),c);* ***21.*** *True;* ***22.*** *slower;* ***23.*** *all of the above;* ***24.*** *a),c);* ***25*** *a),c),d),e)*

26. Vagal stimulation can help slow rapid ventricular dysrhythmias.

True or False

27. Examples of Vagal stimulation include (circle all that apply):

a) anal stimulation
b) sudden change of body position
c) face immersed in ice-cold water
d) carotid sinus massage
e) deep pain
f) vomiting

28. Supraventricular tachycardias can be slowed or terminated with _____ _____.

29. Sympathetic stimulation may be advantageous during periods of (low cardiac output, ischemia) and increase morbidity during episodes of (low cardiac output, ischemia).

30. Beta blockers reduce morbidity and mortality for those experiencing an acute myocardial infarction by as much as (10%, 20%, 30%, 40%).

# Suggested Readings and Resources

Alexander, W. et al. (2001). Hurst's the Heart. 10th ed. New York: McGraw-Hill

Katz, A.M. (2001). Physiology of the Heart. 3rd ed. London: Lippincott

HeartScape: The Anatomy of the Heart. SkillStat Learning Inc.
Web: http://www.skillstat.com/heartscapeDemo.html

The Heart: An Online Exploration. Web: http://sln.fi.edu/biosci/heart.html

Linappa, V. & Farey, K. (2000). Physiological Medicine. New York: McGraw-Hill

***Answers:*** ***26*** *False;* ***27.*** *all of the above;* ***28.*** *Vagal stimulation;* ***29.*** *low cardiac output, ischemia;* ***30.*** *40%*

# What's Next?

It's time to apply your knowledge of normal cardiac anatomy and physiology to the skills of ECG interpretation. The next chapter is a primer on the components of an ECG. Learn about the cardiac monitoring system, ECG waveforms, segments and complexes. Also become skilled at quickly determining heart rate. Proficiency in rapid ECG interpretation is definitely within reach.