AUTONONIC REGULATION OF THE HEART: AN OSTEOPATHIC PERSPECTIVE

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"The ANS comprises an elegant and complex series of interactions that directly impacts cardiovascular responses to physiologic stimuli and can play a large part in the pathogenesis of a variety of cardiac diseases."
S. Kapa

The autonomic influence on heart function is substantial. On the one hand, it can be summarized fairly simply: the sympathetic nervous system increases heart rate and force of contraction, and the parasympathetic nervous system decreases heart rate and force of contraction. On the other hand, the exact function and mechanism of each of these systems and the interplay between the two systems is complex.

This article assumes an understanding of cardiac anatomy and electrophysiology.

THE BEATING HEART

"When blood and lymphatics flow freely, the tissues can perform their physiologic functions without impedance…the rule of the artery is absolute, universal, and it must not be obstructed."
AT Still

Our osteopathic work supports circulatory freedom to all tissues. The heart is an essential piece of this circulatory health.

The lifelong task of our hearts is to assist in blood circulation: to help arterial supply to tissues, providing oxygen, nutrients, immune cells, chemokines, hormones, etc.; to receive oxygen-poor, waste-rich blood and lymphatic fluid; to move that blood to the lungs for replenishment; to receive that oxygenated blood, only to immediately send it on its way - to the digestive tract to receive nutrients, to the liver and kidneys for waste detoxification & removal; and once again to supply our cells.

The rate and strength of heart contraction is precise and responsive to our needs. This responsiveness is primarily mediated by the autonomic nervous system. ANS modulation of heart function is essential for optimal circulation & health, and our osteopathic work can influence that ANS modulation.

ANATOMY OF CARDIAC ANS

Before we examine ANS physiology, let's review the anatomy. (Figures 1, 2, 3)

Sympathetic:

Preganglionic fibers originate in the T1-5 intermediolateral column of the spinal cord, exiting via the ventral horn to the lateral chain. These synapse primarily in the stellate ganglia but also in the upper thoracic and lower cervical ganglia.

Postganglionic nerves exit the lateral chain as "cardiac nerves" and travel through cardiac ganglia to their destinations in the cardiac conduction system, cardiac muscle, great vessels, and coronary blood vessels.

- The ***superior cardiac nerve*** comes from the superior cervical ganglia in front of C2 & C3, travelling in front of the common carotid artery and the large muscles of the neck to the superficial and deep cardiac plexuses.
- The ***middle cardiac nerve*** exits the middle cervical ganglia in front of C6; the right goes behind the common carotid to the dorsal part of the cardiac plexus, while the left runs between the common carotid artery and the subclavian vein to the deep cardiac plexus.
- The ***inferior cardiac nerve*** exits the inferior cervical ganglion (between the transverse process of C7 and rib 1), or the stellate ganglion, traveling behind the subclavian artery and the anterior surface of the trachea to the deep part of the cardiac plexus.
- ***Thoracic cardiac nerves*** from T2-5 ganglia to the deep cardiac plexus
- There is variation in presence and distribution of these nerves.

Parasympathetic:

Cardiac vagal fibers originate primarily in the nucleus ambiguous, with some input from the dorsal motor nucleus. The vagus exits the skull via the jugular foramen. Left and right vagal fibers descend in the carotid sheaths lateral to the carotid artery and then branch:

- The superior cervical cardiac branch leaves the carotid sheath to meet the deep cardiac plexus.
- The inferior cervical cardiac branch exits in the thorax to join both deep and superficial cardiac plexuses.

The right vagus usually innervates the SA node, the left vagus the AV node; there is also vagal innervation to ventricles and atria.

Vagal afferent cell bodies are in the inferior (nodose) ganglion in the jugular foramen; the afferents synapse in the nucleus tractus solitarius (NTS).

Cardiac Plexuses:

At the base of the heart are the two cardiac plexuses, formed by SNS and PSNS cardiac nerves.

The superficial cardiac plexus is located below the arch of the aorta in front of the right pulmonary artery. It includes the left sympathetic superior cervical cardiac branch, and the lower superior cervical cardiac branch of the left vagus.

The deep plexus is in front of the tracheal bifurcation, behind the aortic arch. It is formed by both right and left sympathetic cardiac nerves and all cardiac branches of the vagus.

Cardiac Ganglia:

The cardiac ganglia are the synapse spots for preganglionic vagal fibers, as well as having a network of local intrinsic neurons. The ganglia are mostly in the atrial epicardium walls, but also in the adventitia of the aorta and pulmonary trunk, and in the interatrial septum. Postganglionic vagal fibers and efferent intrinsic neurons begin in the ganglia and supply the conduction system and the myocardium.

The Conduction System:

From an osteopathic perspective, two areas of interest are the SA node (high in the right atrium, near the entry of the superior vena cava) and the AV node (lower in the right atrium, near the tricuspid valve)

AUTONOMIC OVERVIEW:

Autonomic influence is powerful. Sympathetic stimulation can double the heart rate; parasympathetic stimulation can stop the heart. As early as the 1970's it was known that SNS and PSNS function are important in post-MI risk; current clinical research focuses on how to influence autonomic activity to help with heart conditions.

The autonomic nervous system modulates cardiac function in three different ways:

1. through the electrical conduction system, which determines the rate of the heart beat;

2. through direct action on the myocardium (muscles of the heart tissue);
3. through vasomotor influence on the coronary circulation (i.e., the blood vessels that supply/drain the walls of the heart).

The parasympathetic system is dominant at rest – the primary neurotransmitter, acetylcholine, acts to lower the intrinsic (i.e., 100 bpm) heart rate. The sympathetic nervous system balances the parasympathetic and is there for stress, emergencies, and energy expenditure (e.g., exercise), but also for normal life activities like postural and breathing changes. Norepinephrine, the primary transmitter, increases heart rate and contractility.

Let's briefly review the conduction system before looking at the ANS in more detail.

The Heart Beat – The Electrical Conduction System

The electrical conduction system – beginning in the fibrous skeleton of the heart, and ending in the myocardium – produces what is known as the "heart beat." (Figures 4, 5)

There are two main phases of the heart beat: **diastole** (relaxation of the ventricles) and **systole** (contraction of the ventricle wall); the atria also have their own diastole/systole rhythm, overlapping but not coinciding with the ventricular beat.

The electrical conduction of the heart beat begins with an action potential in the **sinoatrial node**. This action potential travels first to both atria (via anterior, middle and posterior tracts). Both atria contract and blood empties into the ventricles. Then, after a $1/10^{th}$ second delay, there is depolarization of the **atrio-ventricular node**, then of the av bundle **(Bundle of His**) and then of the **Purkinje fibers**, which finish in the myocardium of the ventricles. The ventricles contract simultaneously, blood from the right ventricle exiting into the pulmonary trunk, from the left into the aorta.

The volume of blood that is pumped per minute out of the ventricles is the **cardiac output**, which is a product of the heart rate (beats per minute) and stroke volume (volume of blood leaving the left ventricle per beat): CO = HR x SV. CO is determined by external influence (primarily from the ANS, but also from hormonal and chemical factors) on the intrinsic contraction of the heart.

Intrinsic Contraction:

The heart will contract without any outside neurological influence. The embryonic heart begins beating at 3 weeks, while the neural crest cells (future ANS) don't begin migrating to the heart and lateral chain until week 5.

The sinoatrial node determines the ***heart rate*** – it is the intrinsic pacemaker of the heart, beating, without outside influence, at 100 beats per minute,

The heart ***conduction strength*** is governed by the **Frank-Starling law**, which basically states that all the blood that enters the heart (i.e., venous return) will be pumped out (i.e., stroke volume) due to stronger ventricular contraction.

This intrinsic heart beat (rate set by the SA node, strength set by the Frank-Starling law) is modified and regulated by the ANS. The metabolic needs of the tissues, as well as external information e.g., from stress, exercise, limbic system messages, inflammation, and hormones in turn inform this autonomic regulation.

CARDIAC AUTONOMIC NEUROPHYSIOLOGY

Remember, the ANS influences the heart in three ways:

1. The electrical conduction system
2. The myocardium (heart muscle tissue)
3. Coronary vasculature

Sympathetic Nervous System:

The sympathetic nervous system is adrenergic; post-ganglionic neurotransmitters are primarily epinephrine and norepinephrine. Beta receptors (mostly Beta 1, but some Beta 2) are in the conduction system, the muscle walls of the heart and some coronary arteries; alpha receptors are also present, primarily in the coronary vasculature, but also in cardiomyocytes.

Electrical and Myocardium Effects: Beta activation changes both the conduction system and myocardium, resulting in *increased heart rate* (via SA node*), increased velocity* of AV conduction, and *increased cardiomyocyte* contraction.

Coronary Vessels: In systemic arteries, the action of the SNS/adrenal medulla is vasoconstriction – hence the osteopathic model of working with the SNS to change vasomotion. In coronary vessels adrenergic action is complicated and the research is contradictory. The ANS has perhaps less effects on coronary arteries than other local factors like NO release from vascular endothelium. However, it seems that the bigger arteries (in the epicardium, the outer layer of

heart tissue) have alpha receptors that respond to postganglionic sympathetic and circulating norepinephrine and epinephrine by vasoconstriction. However, smaller vessels in the myocardium and subendocardium have beta receptors – the response is smooth muscle relaxation and vasodilation!

In summary, post-ganglionic adrenergic norepinephrine (and epinephrine from the adrenal medulla) stimulation of Beta receptors result in:

1. Conduction: Positive chronotropy (heart rate) & dromotropy (conduction speed)
2. Myocardium: Positive inotropy (contractility) of atria & ventricles
3. Coronary vasculature: vasoconstriction of larger arteries, vasodilation of smaller vessels

Parasympathetic Nervous System:

The parasympathetic nervous system is cholinergic; pre and post ganglionic fibers release acetylcholine (and some other important neurotransmitters).

The parasympathetic supply to the heart is from cranial nerve X, the vagus. The nucleus ambiguous (NA) supplies 80% of efferent fibers, synapsing in the cardiac ganglia. The conduction system is densely innervated by postganglionic parasympathetic nerves. Activation of muscarine M2 receptors in the conduction system slows depolarization, resulting in *decreased heart rate* via the SA node, *and slower conduction* via the AV node. M2 receptor activation in the myocardium of both atria *reduces contractility* of cardiomyocytes.

It has been thought that only the atria received parasympathetic innervation. Recent research has definitively shown rich parasympathetic innervation in the ventricles as well, resulting in decreased ventricular contraction.

The NA vagal cardiac activity is modulated by respiratory rhythms, baroreceptor and chemoreceptor information, as well as by information from the NTS and other CNS centers.

Less understood are the unmyelinated fibers from the dorsal motor nucleus (DMNX), which compromise 20% of vagal cardiac efferents. They also slow heart rate and contractility; their action is slower and less responsive to changes such as baroreceptor stretch or respiratory rates.

Other parasympathetic neurotransmitters are VIP (vasoactive intestinal peptide) and NO (nitric oxide) – the former causes coronary artery vasodilation (n.b.:

acetylcholine does not influence coronary arteries), the latter protects against ventricular fibrillation.

In summary: the PSNS is dominant at rest, and serves to slow the intrinsic heart rate. PSNS cholinergic activity from acetylcholine and other neurotransmitters results in:

1. Conduction: negative chonotropy & dromotropy
2. Myocardium: negative inotropy
3. Coronary vasculature: limited influence – some vasodilation

The SNS and PSNS are often talked of as antagonistic, but in fact they work together to provide a nuanced response to demands, whether they be from a simple postural change or stress/exercise needs. Although, in general, the influence is on the whole heart, there can be specificity – e.g., the vagus can decrease heart rate even as the sympathetics activate the ventricles.

Neurotransmitter	Receptor	Heart Function			Coronary V Function
		Inotropy	Chronotropy	Dromotropy	
Norepinephrine, epinephrine	α1	+	+	+	vasoconstr
	β1	+	+	+	vasoconstr
	β2	+	+	+	vasodilatic
Acetylcholine, NO	Muscarine 2 (M2)	-	-	-	vasodilatic

Heart Rate Variability (HRV)

HRV is a good indicator of the health of ANS balance. "Heart-beat" is taken as an average – beats per minute – but those beats are not equally spaced. Small differences (milliseconds in the intervals between beats) is healthy and indicates a flexible and responsive system. Respiratory Sinus Arrhythmia (RSA) is the slight increase in heart rate on inspiration, decrease in expiration – RSA and HRV, although slightly different, are often used interchangeably.

High HRV, measured on the ECG, is a marker for better cardiac health. RR interval (time between R peaks) variability is indicative of vagal activity. Variability of the QT intervals indicates sympathetic responsiveness.

Higher HRV – shown by these small RR and QT interval changes - indicate SNS & vagal flexibility to modulate to immediate needs. Sympathetic overactivity and decreased parasympathetic activity are linked with reduced HRV which in turn is correlated with heart failure and poor post-MI outcomes. Reduced HRV correlates with other diseases such as diabetic neuropathy and cancer outcomes; higher HRV links with better attention, decision-making and emotional regulation.

The ANS is modulated by both the CNS and by a localized cardiac neuronal network.

Intrinsic Cardiac Autonomic Nervous System (ICANS)

This is a network of intrinsic neurons (sensory, interconnecting, and efferent) that work in and between cardiac ganglia to make adjustments to cardiac activity. Sometimes referred to as "the little brain," this network integrates local information from the heart with external information from the ANS. It then influences the conduction system at the SA and AV nodes as well as atrial and ventricular contraction.

CNS influence (Figure 6)

The CNS modulation of the ANS is a huge discussion in its own right. Vagal motor centers (NA & DMNX) are informed by medullary centers (e.g., respiratory & cardiovascular centers) as well as heart sensory information via the NTS. Preganglionic sympathetic neurons in the spinal cord are modulated by many brain areas including the NTS, the RVLM (rostral ventrolateral medulla), the medullary vasomotor center, the reticular formation and the hypothalamus. The limbic system also influences cardiac autonomic function in complex and vital ways. Some examples: the paraventricular nucleus (PVN) of the hypothalamus, with its oxytocin-rich neurons, supports vagal activity; the insula influences both SNS and PSNS cardiac activity; the pre-frontal cortex and the hypothalamus are linked to HRV.

Sensory/afferent ANS

So often we speak only of efferent neurology, when in fact the receiving and processing of sensory information is essential for cardiac regulation - and indeed all healthy physiology. 80% of vagal fibers are afferent!

Cardiac sensory information is processed locally by the ICANS, which modulates contractility and conduction.

Vagal afferents carry baroreceptor (described below), chemoreceptor and other sensory information (including inflammatory cytokines); they travel to the medullary nucleus tractus solitarius (NTS); the NTS informs the hypothalamus, the vagal efferent centers, the sympathetic output, and other brain centers

New information is highlighting the importance of sympathetic afferents which travel with the sympathetic nerves and synapse in the spinal cord via the dorsal root ganglia. They receive information on mechanical and chemical changes, lowering the receptivity of the baroreceptor system (see below) and transmit pain information; communicate branches synapse with the cervical and brachial plexuses & intercostal nerves, explaining the pain distribution in cardiac pathology.

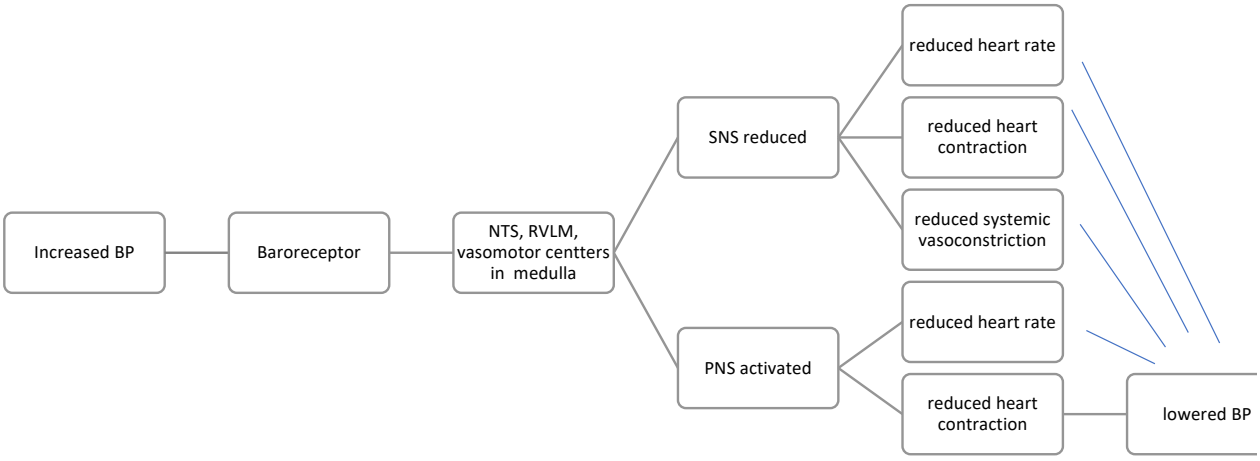
Baroreceptors & Chemoreceptors

The ANS is intricately linked with afferent baroreceptor and chemoreceptor input. See the appendix for details on chemoreceptor and baroreceptor function.

Chemoreceptor information (low O2/hypoxia, high H+/acidosis, high CO2/hypercapnia) is sent via the PSNS to the medulla; vagal activity is reduced, SNS activity is increased. Respiration, heart rate, and stroke volume are increased.

Baroreceptor activity is vital for healthy ANS regulation. So, increased blood pressure:

- stretches the baroreceptors
- information is relayed via the vagus to the NTS, medullary vasomotor center and rostral ventrolateral medulla (RVLM)
- RVLM reduces stimulation to SNS preganglionic spinal neurons, & noradrenergic effect on SA node & cardiac muscles is lowered
- NTS information results in vagal efferent (NA & DMNX) stimulation, cholinergic effects lower heart rate & ventricular contraction
- Blood pressure is lowered.



In health, this baroreceptor/ANS/cardiac circuitry is sensitive and rapid in response. However, if there is chronic hyper or hypotension, the baroreceptors reset to the new blood pressure and are less able to respond to blood pressure changes.

Baroreflex sensitivity (BRS)can be measured. As with HRV, healthy, higher BRS reflects the responsiveness and flexibility of the ANS. Reduced BRS is linked with poor outcomes/high risk in heart failure, ventricular fibrillation, hypertension and post-MI death.

ANS and Cardiac Pathophysiology:

"Cardiac dysfunction results from autonomic dysregulation of the contractile output of the heart."
H. Furstenwerth

Although our focus as osteopaths is on health and healthy physiology, we are indebted to current medical research on cardiac ANS function, stemming from the known influence of the ANS on cardiac pathology (e.g., atrial fibrillation, ventricular arrhythmias, myocardial infarction, heart failure, hypertension)

Simply stated, excessive SNS activation, increased catecholamines and decreased PSNS tone are linked with severity of heart failure, arrhythmias and increased mortality. Increased vagal/cholinergic activity is protective against ventricular fibrillation and other heart conditions.

When the normal homeostatic mechanisms of cardiac function are disrupted, there is negative feedback: abnormal ANS negatively influences cardiac function, resulting in pathology; cardiac pathology alters ANS function, which in turn leads to disease progression.

For example, after a myocardial infarction, many changes occur, including:

- Cardiac sensory nerve endings are damaged, altering afferent information. Dead muscle is replaced by scar tissue, further altering afferent input. The healthy responsive autonomic cycle: Heart afferent ⇒ CNS ⇒ANS ⇒Heart efferent is disrupted.
- SNS nerves are damaged (as far away as the stellate ganglia); there is abnormal repair, increasing ventricular arrhythmias.
- The MI elicits a powerful sympathetic response, resulting in post-MI sympathetic dominance, reduced HRV and increased arrhythmias

Medical interventions – pharmaceutical, surgical and some non-invasive techniques – are now being studied for their influence on HRV, BRS and ANS function. Osteopathic principles hold that treatment can support healthy autonomic function, which in turn will better equip the person to respond to internal and external challenges – medical research, it seems, is once again catching up to osteopathic thinking.

Some examples:

- **LCSD** – left cardiac sympathetic denervation. The left lower half of the stellate ganglion and the T1-4 lateral chain ganglia are surgically removed, reducing sympathetic nerve input. Used since the 1970's it is successful in preventing arrhythmias, tachycardia and reducing risk of death post-MI while still allowing the heart to respond to exercise.
- **Beta blockers** – these decrease hyperadrenergic drive by reducing activity at the Beta receptors
- **VNS** – vagal nerve stimulation. This is an invasive procedure with mixed results and many side effects
- **VTES or LLTS –** vagal transcutaneous electrical stimulation, or low level tragal stimulation. The concept here is to stimulate parasympathetic activity via a superficial afferent pathway – in this case, the auricular branch of the vagus. Stimulation is to the tragus or cymba conchae of the external ear. It produces a smaller acetylcholine release than VNS but is non-invasive and has no side effects. It has been shown to be useful for improving heart failure risks, lowering atrial fibrillation, and reducing post-MI ventricular arrhythmias.
- **Other non-invasive approaches**. HRV can be increased in a variety of ways, including , exercise, biofeedback, better breathing, improved sleep,

hydration, and, in a recent study, daily playing of the Native American Flute!

- **Osteopathy:** Our clinical experience is now being supported by some research that has documented quantitative changes in HRV from osteopathic treatment.

CARDIAC AUTONOMICS: A SUMMARY

Although a summary is simple (vagus: dominant at rest, slows heart rate and heart contractility; the sympathetic nervous system: dominant with activity and stress, increases heart rate and heart contractility), the reality is much more complex.

Texts and research often describe the two branches of the ANS as being antagonistic, with the parasympathetics regulated at rest and the SNS activating in emergency, stress and exercise. In reality, the two systems, although having very different physiological influence, work in concert – like a dance – to keep our bodies healthy. Normal function of the heart relies on a back-and-forth interplay of the sympathetic and vagal systems for the simplest of tasks like standing up from lying down. Indeed, an important indicator of health is RSA, where heart rate shifts with respiration due to tiny changes in vagal tone.

The autonomics work in concert to keep cardiac function responsive to our physiological needs – i.e. to make sure that our tissues have good nourishment and waste removal. This balance is continuously adjusted as our needs and environment changes. The ANS is modulated by internal & external information, e.g., baroreceptor stretch, activity level, stress, caffeine, information from our limbic system about perceived safety and danger, our emotional health, stress levels, health issues such as diabetes, and the balance of the neuroendocrine immune system (this last one being yet another topic in itself!)

It is when this delicate ANS balance is disrupted – often by persistent higher levels of the sympathetic nervous system – that this healthy function is disrupted, and cardiac pathology can occur.

OSTEOPATHY AND HEART AUTONOMICS

Osteopaths have been looking at and treating the autonomic nervous system since, well, *almost* the beginning of osteopathy. Still anecdotally came into a classroom and wrote, “No physiology” on the board, and certainly fought with Littlejohn when Littlejohn was dean at Kirskville. However, the contributions of Littlejohn, the early studies by Louisa Burns and the later work of Dr. Korr,

as well as so many osteopathic teachers, have made treatment of the autonomics an essential piece of osteopathic work.

Treatment of the autonomics is a discussion for an entire article in itself. However, I will say, in practice and in my teaching, I like to begin with a systemic view of the autonomic nervous system (e.g., SNS & PSNS individual function and balance) and then look at specifics (e.g., activity at the stellate ganglion).

Treating the cardiac vagus: Littlejohn accessed the vagus via inhibition at the occiput-atlas; other access areas are the medullary centers via the occiput and the 4th ventricle, the jugular foramen, the anterior neck/carotid sheath, and, from the anterior chest, the cardiac ganglia in the atria. It is also interesting to perceive the sensory information coming from the heart to the medulla (remember the vagus is 80% afferent) - I like to have a hold on the occiput (to perceive medullary function) and on the anterior chest to assess vagal afferent information.

With the sympathetic nervous system, we can look at T1-T5 (spinal cord preganglionic innervation), stellate ganglion (at the level of C6-T1), and the cervical lateral chain ganglia (origins of the cardiac nerves) in front of C2-7, and the course of the cardiac nerves in the anterior neck. Littljeohn used articulatory techniques to alter sympathetic function, but there are many ways of influencing sympathetic function at the thoracolumbar flow, the lateral chain and the course of the cardiac nerves including paraspinal inhibition, balanced ligamentous tension, myofascial release and simply observing/supporting change in activity.

The conduction system and cardiac plexuses & ganglia can also be explored via the anterior chest. (Figure 7)

The CV4 is vital in balancing fluids and normalizing medullary function. My experience is that this technique may not be helpful if the sympathetic tone is set too high.

The above techniques are suggestions, ideas to explore. For myself, I take a basic approach:

1. Are my hands quiet and perceptive?
2. Is the person in a neutral state – are they ready to begin treatment?
3. What can I observe/perceive? The more informed my anatomy and physiology is, the more patient I am, the more accurate my perception
    - How is the function of the SNS? The vagus?
    - The balance between them?

    - The specific ANS cardiac function?
    - Extent and cause of dysfunction? (From long-standing emotional stress? Acute health issues? How old?
4. Is there willingness of the system to change?
5. Treatment
    - Acknowledge, for example, sympathetic excitation to the heart
    - Provide safety and spaciousness
    - Ask "what can I do to assist healing?"
    - Treatment can be as simple as holding that spaciousness, or as specific as direct action (e.g., release of the pericardial fascia, or traction on the cervical and thoracic spine to create more space for the lateral chain).
6. Time for the treatment to settle and integrate.

Healthy heart function is essential for life; balanced autonomic regulation of the heart is necessary for healthy heart function. And osteopathy has an important role to play in balancing the autonomic nervous system.

Note: In the appendix, there are details on anatomy and physiology of the electrical conduction system, an explanation of how electrical conduction is seen on the electrocardiogram (ECG), more details of the diastolic and systolic phases and how they are seen on the ECG, and a review of how the heart valves open and close to allow coordinated filling of and flow out of the heart chambers.

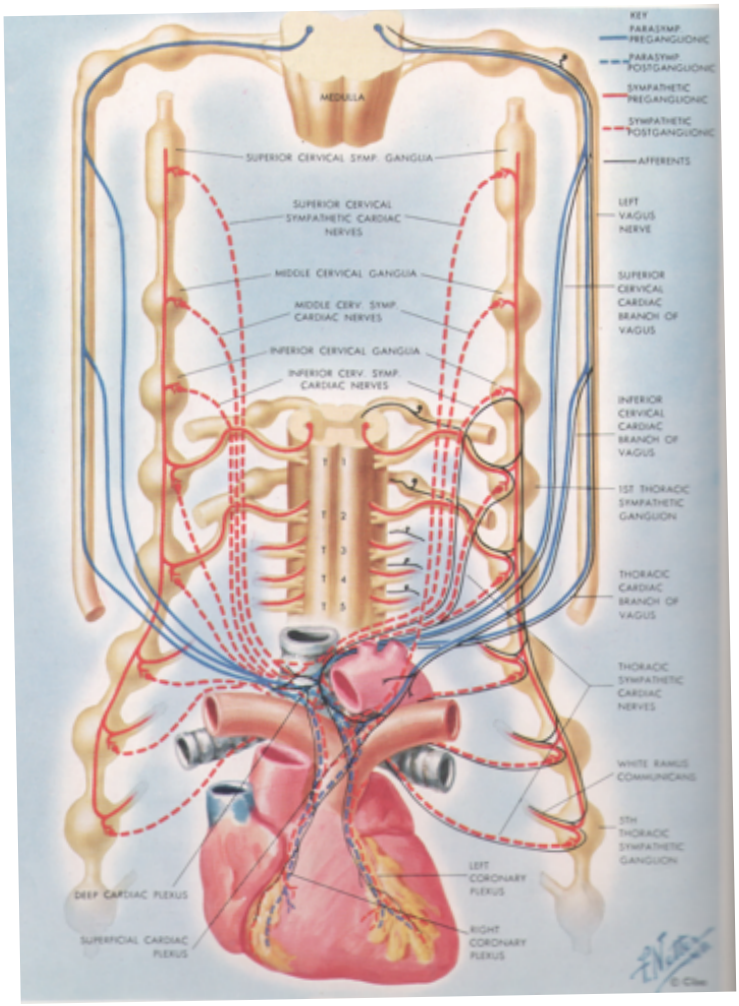


Figure 1 Cardiac Autonomics
from: Netter, F. Nervous System

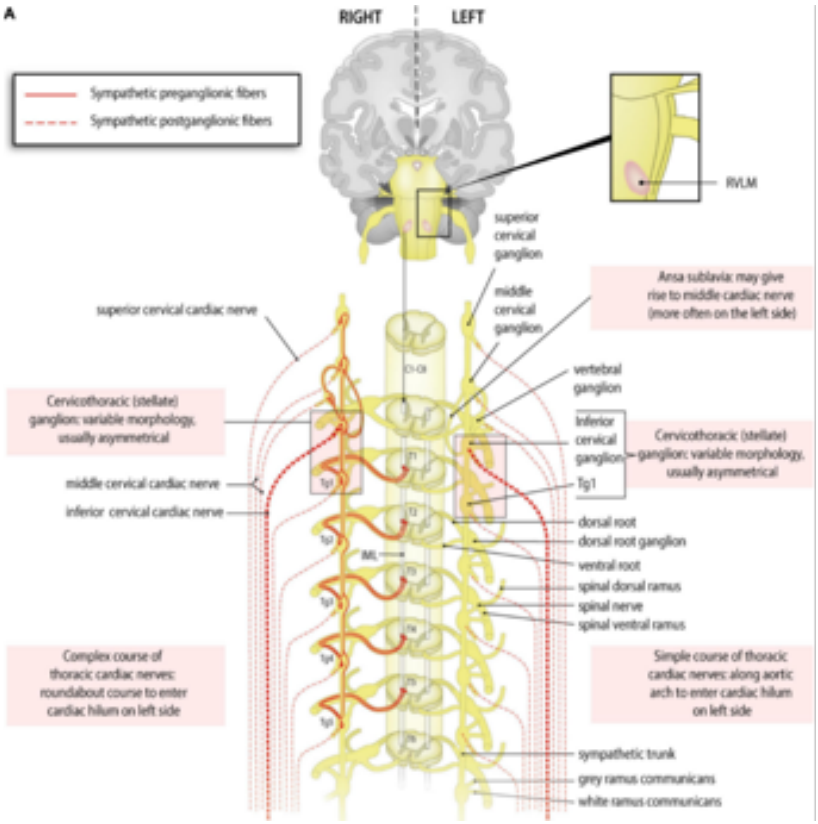


Figure 2: Sympathetic Innervation of the Heart
from: Zandstra, T.E. et al

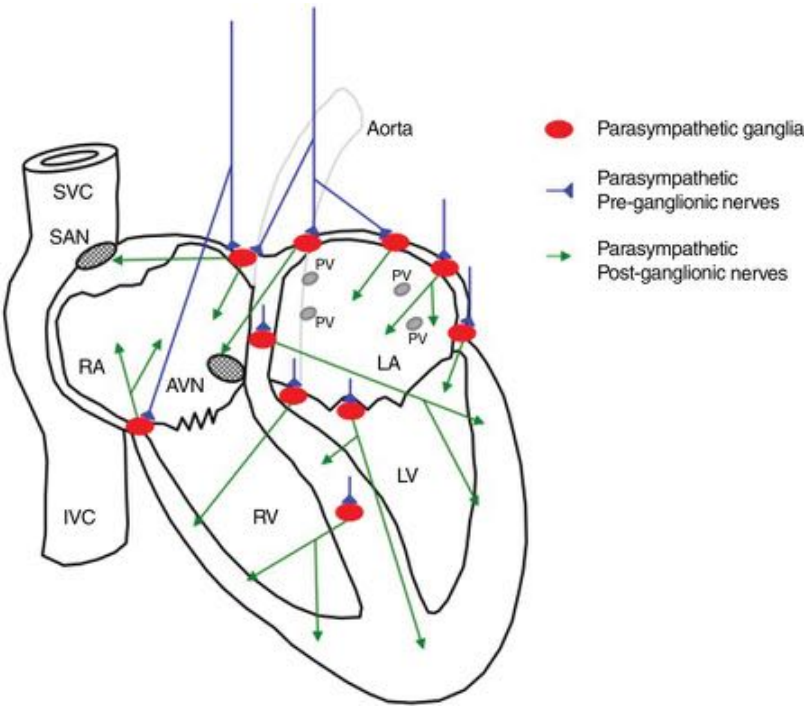


Figure 3: Parasympathetic Innervation of the Heart
from: Coote, J.H.

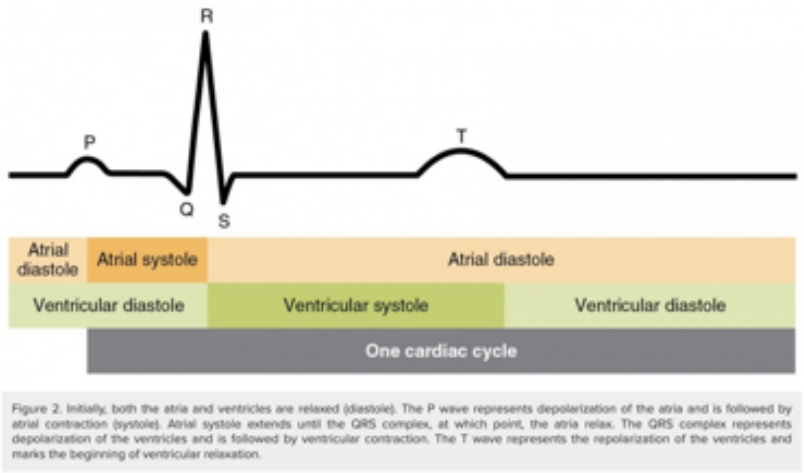


Figure 2. Initially, both the atria and ventricles are relaxed (diastole). The P wave represents depolarization of the atria and is followed by atrial contraction (systole). Atrial systole extends until the QRS complex, at which point, the atria relax. The QRS complex represents depolarization of the ventricles and is followed by ventricular contraction. The T wave represents the repolarization of the ventricles and marks the beginning of ventricular relaxation.

Figure 4: ECG of the Heartbeat

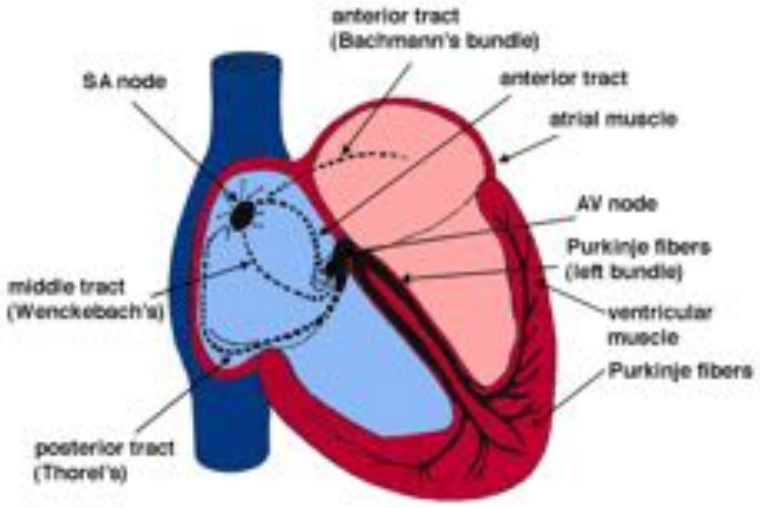


Figure 5: The Conduction System
from vhlab.umn.edu

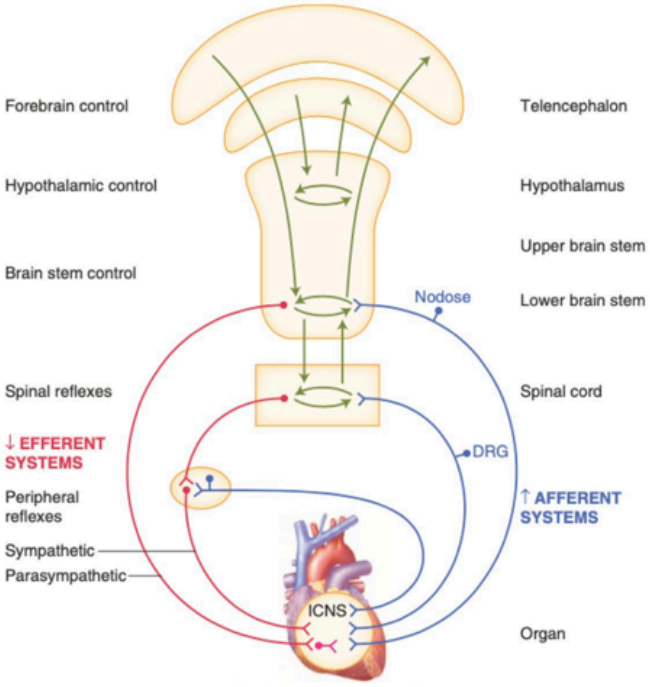
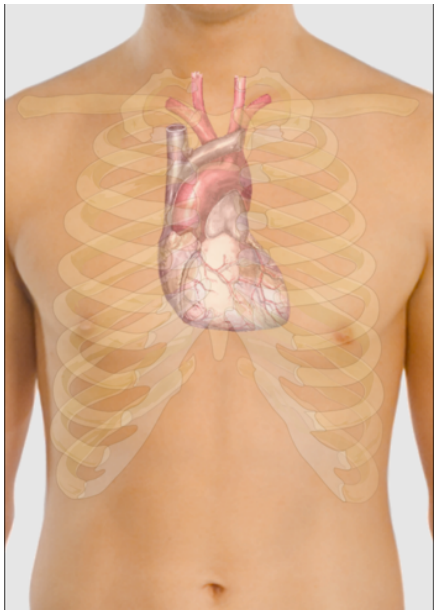


Figure 6: CNS Regulation of the ANS
from: Hadaya, J.



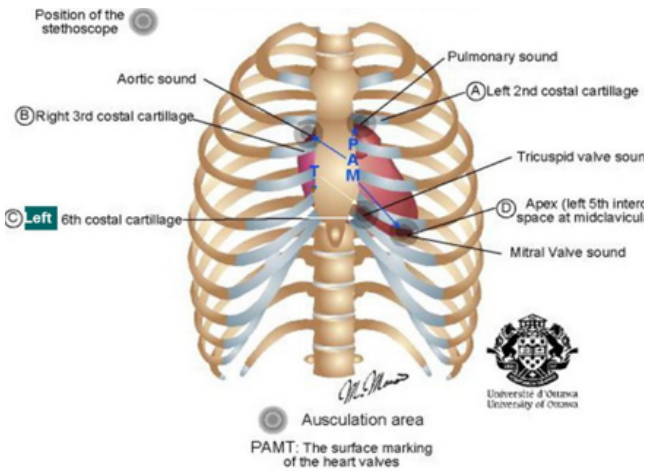


Figure 7: Surface Anatomy of the Heart