

EXERCISE-INDUCED TACHYCARDIA

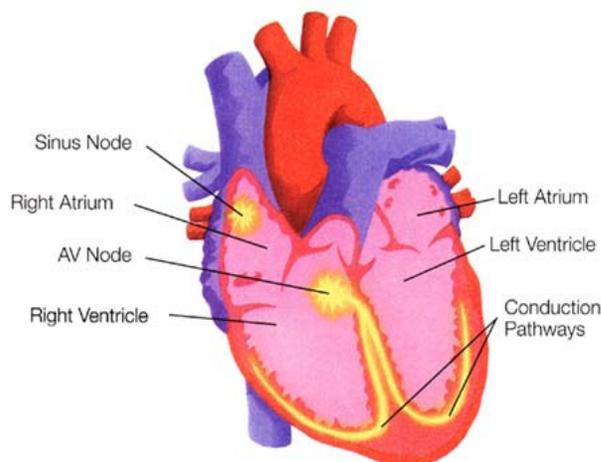
Mary Boudreau Conover

- **Inside the Heart**
- **Pacemaker of the Heart**
- **Exercise-Induced Sinus Tachycardia and its Benefits**
- **Observable Characteristics Of Sinus Tachycardia**
- **The “Atrial Kick”**
- **Summary**

INSIDE THE HEART

The sinus node. Your heart has kept you alive all this time with a nice normal rhythm at an appropriate rate. The structure responsible for such a dependable personal cardiac-history is called the **sinoatrial (SA) node** or **sinus node**. It can only be defeated if invaded or overpowered. This article has been written to acquaint you with the function of this little node during all of the activities of your life, including your workout, when it protects your organs by supplying you with an appropriate sinus tachycardia.

The discovery of the sinus node was published in 1907¹, describing a crescent-shaped area located within the uppermost wall of the right atrium. It was later demonstrated that there is an artery coursing through its length. The word “sinus” describes a curved, hollow, sac-like structure, a designation that probably reflected what Kieth and Flack could see with the equipment of the day in 1907. Modern technology and electromechanical mapping has revealed it to be a group of highly specialized cells tightly packed in connective tissue with an “irregular contour, variable borders, and extensions”⁴ into the surrounding myocardium.



The illustration rather dramatically represents the sinus node as a bright yellow spot against a pretty pink background--not even close to reality, but it does pinpoint its location fairly accurately. The sinus node is actually imbedded within the right atrial wall and not plastered against the inside wall as shown in the illustration. You will see it again in a more realistic animation below.

The atrioventricular (AV) node is a group of special cells in the floor of the right atrium where the sinus impulse is delayed slightly to allow for the atrial contraction to be completed.

The His bundle (pronounced “Hiss”) is the very important slender tract extending into the ventricles from the AV node. It is the only normal electrical connection between atria and ventricles. This link to an animated illustration of cardiac electrical and mechanical activation may help in the visualization of the cardiac conduction system and how the heart responds to it. This is a complicated link, but well worth seeing. Please click on the “forward” arrow at the bottom of the picture and you will see a perfectly stunning animation of the normal sinus rhythm, electrical and mechanical. The flash of light from SA node to the apex of the heart represents the electrical current that powers the heart. **NOTE:** It says “ventricular tachycardia” at the top of the illustration. It does not apply here, so ignore it, click on and enjoy.

<http://www.medmovie.com/mmdatabase/mediaplayer.aspx?Message=VG9waWNpZD03NDY7Q2xpZW50SUQ9NjY7VmVybmFjdWxhcklEPQ%3D%3D-sLVQQR4ka24%3D>

Papillary muscles and chordae. The strange looking finger-like protrusions that you see inside the two ventricles are diagrammatically illustrated below. They are strong muscles (papillary muscles) extending into tough string-like tendons (chordae) attached to mitral and tricuspid valve leaflets. Only one valve leaflet is shown. The papillary muscles and the chordae keep the valve leaflets from flipping up into the two atria during ventricular contraction and are also thought to be important in retaining the ventricular shape and function^{2,3}. Click on the animation again and watch the papillary muscle and chordae at work. What an incredible creation!

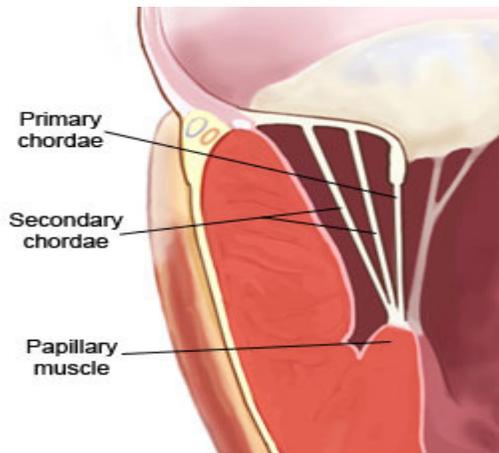


Illustration courtesy of: Department of Cardiothoracic Surgery, Mount Sinai Medical Center, New York City, NY

PACEMAKER OF THE HEART

THE pacemaker of the heart is the sinus node because it is automatic, needing no stimulation from elsewhere. There are other cells located geographically lower in the

heart (bundle of His) that are capable of firing automatically, but the sinus node does it best and dominates all other contenders. It also adjusts its firing rate under the control of the autonomic nervous system so that the speed at which the blood is pumped (heart rate) meets the needs of the body in all the varied activities of life—sleeping, eating, running, relaxing, splitting logs, climbing the face of El Capitan, skiing the steeps, surfing Mavericks, making love, fleeing danger, fighting tigers, or exercising CrossFit-style. Its rhythm normally changes to accommodate every situation you may throw at it and it does so smoothly—never abruptly—no sudden starts and stops. This last property distinguishes it from most abnormal tachycardias. Normally heart rate varies in tandem with respirations, increasing with inspiration and slowing with expiration. This is known as heart rate variability.

Muscle cells of the heart. The muscle cells of the heart, as opposed to the pacemaker cells, are not normally capable of automaticity. Their sole functions, other than that of a container for blood, are to respond to the electrical currents, pass it on, contract, and relax—restored and ready for the next cycle. They simply wait for the next impulse to arrive—and so it goes, each beat depending on a stimulus from the sinus node.

EXERCISE-INDUCED SINUS TACHYCARDIA

Sinus tachycardia is strictly defined as a rate of 100 beats/min or more and is called an “arrhythmia” by traditionalists and a “dysrhythmia” by purists. These designations for this particular rhythm are unfortunate because they fly in the face of appropriate exercise-related sinus tachycardia—certainly NOT an arrhythmia (without rhythm) or a dysrhythmia (disturbed rhythm). Every day that you do an intense, tough, CrossFit workout that tachy is yours! It is the normal response of the heart to signals from the brain that you need more oxygen.

Benefits. Studies have shown that aerobic interval training and the functional sinus tachycardia associated with it improve microcirculation, the health of the lining of our coronary arteries, and myocardial function at the cellular level.⁵ In fact, mixed-sports (aerobic and anaerobic) athletes survive longer than the general population; fewer cases of cardiovascular disease are thought to be the primary reason.⁶ Even in those athletes with cardiovascular disease who have sustained a heart attack, some of the negative effects following the event are reversed in hearts preconditioned with tachycardia.^{7,8}

Dangers. No athlete is unaware of the sudden death scenes of endurance marathon athletes and will not be surprised to learn from Dr. O’Keefe’s video (below) about a potential for heart damage when endurance exercise is excessive. This is especially true when the thinner right ventricle needs to be stretched beyond its capability in order to accommodate the need of skeletal muscles, heart and brain for more O₂. Of course stretching isn’t a bad thing except when it’s too much! Too much of anything is just that – **too much!** The excessive stretch of the myocardium over long periods can tear myocardial fibers. Such tears result in stiff scars, which reduce the effectiveness of the pump, both in relaxing and contracting, and create a milieu for arrhythmias. There are also the alarming possibilities for stiffening of the large arteries as well as calcification of

coronary arteries, the harbinger of heart attacks. Dr. O'Keefe introduces the subject and acquaints us with the data in this video and the article.

<http://vimeo.com/54864015>

http://www.msma.org/docs/communications/momed/Excessive_Endurance_Exercise_and_Heart_Disease_MOMED_JulyAug2012.pdf

OBSERVABLE CHARACTERISTICS OF SINUS TACHYCARDIA

All other forms of tachycardia are abnormal and not always well tolerated. So it behooves us to know this friend of ours so that we can recognize the unfriendlies, for ourselves and others. There are interesting physical clues that give the unfriendlies away. To understand these clues we will begin with an understanding of the characteristics of our friend—sinus tachycardia.

Acceleration. In healthy young adults during strenuous physical exercise the rate of the sinus node may be as much as 200 beats/min. The max rate during exercise may decrease with age to less than 140 beats/min. When the sinus rate accelerates in response to exercise it does so gradually and settles into a rapid, regular rhythm. Your respirations reflect your heart rate. You all know the feeling---open mouth, sucking air, spread eagle on the floor.

Deceleration. When the workout is completed and the physiological needs no longer exist, the heart rate decelerates along with respirations. The deceleration is easier to determine than the acceleration at the beginning of a workout—a busy time. However, at the end of a workout, it is fairly easy to put your fingers on a pulse and feel the heart rate become slower and slower. Try this after your own workout just to get the feel of normal deceleration of the sinus node rate in a healthy person. Acquaint yourself well with the normal and you will more easily recognize the abnormal.

THE “ATRIAL KICK”

One reason that sinus tachycardia doesn't land you flat on your back is because the electrical activation and contraction of the atria and ventricles are sequential. The ventricles, as with other muscles, perform best when stretched just prior to contraction. For this to happen, the atria contract before the ventricles, pushing their contents into the already passively filled ventricles and providing not only a stretch but also an important extra boost of blood known as the “*atrial kick*.” Hearts that are deprived of this atrial kick, such as those with no organized atrial contractions (atrial fibrillation), lose approximately 10% of their cardiac output – slows one down a bit.

REFERENCES

- 1) Keith A, Flack M: **The form and nature of the muscular connections between the primary divisions of the vertebrate heart.** J Anat Physiol 1907; 41:172.

- 2) Rodriguez F, Langer F, Harrington KB et al. **Importance of mitral valve second-order chordae for left ventricular geometry, wall thickening mechanics, and global systolic function.** *Circulation* 2004 September 14;110(11 Suppl 1):II115-II122.
- 3) Rodriguez F, Langer F, Harrington KB et al. **Effect of cutting second-order chordae on in-vivo anterior mitral leaflet compound curvature.** *J Heart Valve Dis* 2005 September;14(5):592-601.
- 4) D Sánchez-Quintana, JA Cabrera, et al: **Sinus node revisited in the era of electroanatomical mapping and catheter ablation.** *Heart* 2005 91(2):189-194.
- 5) Domenech RJ, Sanchez G, Donoso P, et al: **Effect of tachycardia on myocardial sarcoplasmic reticulum and Ca₂₊ dynamics: a mechanism for preconditioning?** *Journal of molecular and cellular cardiology*, 2003; 35: 1429-1437.
- 6) Teramoto M, Bungum TJ: **Mortality and longevity of elite athletes.** *J Sci Med Sport*. 2009 Jun 30; Department of Sports Education Leadership, University of Nevada, Las Vegas, USA.
- 7) Domenech RJ, Sanchez G, et al: **Effect of tachycardia on myocardial sarcoplasmic reticulum and Ca₂₊ dynamics: a mechanism for preconditioning?** *Journal of Molecular and Cellular Cardiology* 2003, vol. 35, n°12, pp. 1429-1437.
- 8) Kemi OJ, Ceci M, Condorelli G, et al: **Myocardial sarcoplasmic reticulum Ca₂₊ ATPase function is increased by aerobic interval training.** Institute of Biomedical and Life Sciences, University of Glasgow, UK. *Eur J Cardiovasc Prev Rehabil*. 2008; 15(2):145-148.

ACKNOWLEDGEMENTS

Coach Mark Rippitoe (Wichita Falls Athletic Club, Texas) reviewed the early formation of this article and suggested an expansion of the discussion on the sinus node. The reference from Sánchez-Quintana et al was particularly enlightening

Tara Muccilli, Michelle Mootz, and Eva Twardokens from CFSCC each added their own unique critique and helped immensely with their reviews.

mc 7/28/2009; revised 5/22/2013