

THE ATHLETE'S HEART

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Recently, Brian, my highly conditioned and athletic nephew mentioned to me that his physician was alarmed because of his slow heart rate. That prompted me to make the following list for him of possible ECG signs reflecting the athlete's heart. My purpose was to simply acquaint him with his athletic heart and the possibly alarming terms that might be used by his physician or that he might see spelled out on his electronically diagnosed 12 lead ECG.

Sinus Bradycardia is diagnosed when the heart rate is < 60 beats per minute (bpm). It's called "sinus" because of its origin in the sinus node at the top of the right atrium. It's called an arrhythmia or a dysrhythmia because of its rate. Most people know that athletes have nice slo-o-o-o-w heart rates! In highly conditioned athletes (swimming, rowing, bicycling, and long-distance running) sinus bradycardia is present even during normal daily activity. Joel, my 53 year old swimming coach had a resting heart rate of 36 bpm. In the days when I swam with the Masters he swam round trips to Alcatraz in the middle of that cold San Francisco bay every year; maybe he still makes that swim. His 36 bpm was recorded after I had dragged him away from setting up big board workout signs at the pool! I was trying to catch him and lay him out so I could record his "resting" ECG for my book. His heart rate would have been much lower had he truly been "resting". By the way, his post-exercise heart rate that day was 60 bpm, barely qualifying as "normal". The 60/minute was right after he had been competing (continuous lap swimming) with an Olympic triathlete and staying close in spite of the nearly 30 years difference in their ages! The "O" triathlete's resting heart rate was 42 bpm. Of course, Joel had that 30 years on the Olympian - more time to precondition his amazing heart. Sinus bradycardia in the athlete is the result of a predominance of vagal tone from aerobic physical training. The parasympathetic fibers of the vagus nerve serve the sinus node so that, when stimulated, the firing rate within that node decreases and the heart rate slows. When there is a predominance of vagal tone, the vagus nerve is dominating the nerves of the sympathetic nervous system which would otherwise increase the heart rate.

Sinus Arrhythmia (Respiratory Heart Rate Variability). A common type of bradycardia in athletes is the acceleration of the heart rate during inspiration and slowing during expiration. This is also a vagal effect and is very marked in children and athletes.

Notched P waves. The P wave is the little bump on the ECG prior to the spike. It reflects the depolarization (electrical activation) of the atria. The notched P wave can be seen in the normal ECG of conditioned athletes and in the abnormal ECG of persons with left atrial enlargement. One Physician found a slight notching in 18 out of 21 of his marathon athletes. The notch delineates the right atrium-left atrium depolarization in sequence.

First degree atrioventricular block (AV heart block). This is actually a misnomer because *there is no block*, but rather a lengthening of the PR interval to more than 200

msec., reflecting a slowing of conduction between the atria and the ventricles — obviously not an actual "block". It is another sign of vagal dominance in the trained athlete and perfectly normal, although it will be labeled on the ECG read-out as "abnormal". It typically normalizes after exercise or complete deconditioning. Although "first degree AV block" is common in the athlete, second or third degree AV block is not and, when present, the possibility of underlying heart disease is considered.

Q waves. Small Q waves in the athlete's ECG reflect normal electrical activation of the ventricular septum. They are compatible with, but not necessarily indicative of, cardiovascular disease, depending on their width and depth. Which leads they are normally seen in depends on the electrical orientation of the heart (horizontal, vertical, or somewhere in-between.)

High voltage QRS. In athletes, this one can easily be misinterpreted!

The QRS is the big spike on the ECG. It reflects ventricular depolarization. In well-conditioned athletes, especially the men, that QRS spike may be tall enough in some chest leads and deep enough in others to match the specifications for pathological thickening of the walls of the heart. However, in the heart of a well conditioned athlete, a perfectly normal condition evolves; the chambers of the ventricles enlarge to accommodate the increased volume of blood that needs to be pumped in support of elite athletic workouts and competition. So now, the Physician needs to distinguish between the two Ps, a huge distinction — pathological or physiological?

In a small percentage of athletes, mainly rowers, a thickening of the left ventricular wall occurs along with the enlarged left ventricular chamber. These changes cause the QRS on the ECG to match the voltage produced by an abnormal heart (hypertrophic cardiomyopathy). The hypertrophy in the athlete's heart is called "physiological". The hypertrophy in the diseased heart is called "pathological". Obviously, this is not to say that athletes never have pathological left ventricular hypertrophy.

Differentiating between the purely athletic heart and the pathologically enlarged heart may present a significant diagnostic challenge. When left ventricular thickness in the athlete's heart does not also have left ventricular chamber dilatation, the likelihood of pathology exists. It may help to know that the physiological hypertrophic signs seen in an athlete's ECG resolve with deconditioning. Therefore, the best way to differentiate the pathologic condition from the athlete's physiological condition is to check for the decrease in left ventricular cavity size and wall thickness after interruption of training for at least 3 months.. That decrease is not seen in the pathological condition of hypertrophic cardiomyopathy.

Incomplete Right Bundle Branch Block (RBBB). This is a common finding in trained athletes. The QRS will be 120 msec. or less. Complete RBBB (QRS > 120 msec.) is rare in athletes as is left bundle branch block and fascicular block.

This is a personal experience. RBBB and anterior fascicular block can also occur secondary to chest injury. I have both a complete RBBB and an anterior fascicular block resulting from a fractured sternum (ski injury). I got blasted right off the ground with a bull's-eye shot (skier's fist) to the base of the right ventricle just beneath the sternum where those two delicate branches begin.

Early repolarization syndrome (elevated ST segment). It won't be called by the syndrome name, but that is what it is. It is seen in highly conditioned athletes and manifests itself with a slightly elevated ST segment (the segment after the tall spike and before the curved little tent known as the T wave.) This one can be misdiagnosed as ischemia of the heart (a heart attack.)

Premature beats ("PVCs" or "VPBs"). The physician might identify these irksome things to a lay person as "extra beats". Atrial and ventricular premature beats are commonly seen in the athlete's ECG, possibly because the slow heart rate allows them "space" to emerge. In those without heart disease they are not clinically significant and are not treated. Often they feel like "skipped beats" followed by a "boomer" of a beat that you may actually feel against your chest wall. However, that big-beat is not the bad guy. In fact, it is the normal beat following the pause created by the premature ventricular beat. It is a stronger beat because the pause allows for more ventricular filling time. The ventricular muscles are therefore stretched against very full chambers and that good ole stretch-reflex kicks.

Note. All of these notable ECG patterns have been recorded in a study of 1,005 Italian Olympic Athletes by Pelliccia et al. However, make no mistake, there is a list of other conditions that can cause sudden death in athlete's, but that's another story!

References

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