Bradyarrhythmias and conduction disturbances in athletes

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The athlete’s heart: a historic perspective

- Osler 1892\(^1\): large heart - nature vs nurture
- Henschen 1898\(^2\): dilatation and hypertrophy and changes were normal and favourable
- Kirch 1935\(^3\): described hypertrophy in 35 athletes who experienced sudden death
- Kjellberg 1949\(^4\): chest radiograph. Relationship between cardiac volume and working capacity

\(^1\)Osler W. New York 1892; Appleton : pp635
\(^2\)Henschen S. Mitt med Klin Uppsala 1898.
\(^3\)Krch E. Verh Dtsch Ges Inn Med 1935; 47: 73-98
\(^4\)Kjellberg SR et al. Acta Radiol 1949; 31;113-22
Athletic heart syndrome, (AHS) also known as athlete's heart or athletic bradycardia, is a non-pathological condition commonly seen in sports medicine, in which the human heart is enlarged, and the resting heart rate is lower than normal. AHS is caused by significant amounts of aerobic exercise performed over a period of at least several months.
Parameters Affected by Training

- Heart size
- Stroke volume
- Heart rate
- Cardiac output
- Blood flow
- Blood pressure
- Blood volume
• Decreases with endurance training due to more blood returning to heart
• In sedentary individuals can decrease by 1 beat per min per week during initial training
• Highly trained athletes may have resting heart rates of 40 beats per min or less
Heart Rate Recovery Period

• It is the time after exercise that it takes your heart to return to its resting rate.
• With training, heart rate returns to resting level more quickly after exercise.
• Has been used as an index of cardiorespiratory fitness.
• Conditions such as altitude or heat can affect it.
• Should not be used to compare individuals to one another.
When exercise stops, heart rate drops as less blood is needed to be pumped around the body.

A trained individual will return to resting values quicker than a healthy sedentary person.
HEART RATE RESPONSE TO A SINGLE BOUT OF EXERCISE

Heart rate is controlled by two branches of the nervous system:

- The Sympathetic Nervous System is like an accelerator in a car and causes the heart rate to increase.
- The Parasympathetic Nervous System is like the breaks in a car and causes the HR to slow down the heart’s brake (slows down heart rate).
What is the Effect of Endurance Training on Resting Heart Rate?

- Heart rate is lower in endurance trained athletes than sedentary healthy individuals.

- Endurance training will cause the parasympathetic nervous system to fire more frequently.
How Does the Heart Rate Differ Between an Athlete and Untrained Person During Exercise?

A person who is endurance trained will have a lower heart rate than an untrained person when they are running at the same speed.
Sinus Arrhythmia

Inspiration

Expiration

SA nodal acceleration

SA nodal deceleration
Heart Beat Anatomy

Sinus Node (SA Node)

- The Heart’s ‘Natural Pacemaker’
- 60-100 BPM at rest

Sinus Node (SA Node)
**AV NODE**

- Receives impulse from SA Node
- Delivers impulse to the His-Purkinje System
- 40-60 BPM if SA Node fails to deliver an impulse
Heart Beat Anatomy

**Sinus Node** (SA Node)
- Begins conduction to the Ventricles
- AV Junctional Tissue: 40-60 BPM

**Atrioventricular Node (AV Node)**

**Bundle of His**
Heart Beat Anatomy

THE PURKINJE NETWORK

- Bundle Branches
- Purkinje Fibers
- Moves the impulse through the ventricles for contraction
- Provides ‘Escape Rhythm’: 20-40 BPM

Sinus Node (SA Node)

Atrioventricular Node (AV Node)

Bundle of His

Bundle Branches

Purkinje Fibers
Atrial Depolarization
Delay At AV Node
Conduction Through Bundle Branches
Conduction Through Purkinje Fibers
Ventricular Depolarization
Plateau Phase of Repolarization
Final Rapid (Phase 3) Repolarization
Normal Ranges in Milliseconds:

- **PR Interval**: 120 - 200 ms
- **QRS Complex**: 60 - 100 ms
- **QT Interval**: 360 - 440 ms
RHYTHM DISORDERS

Bradyarrhythmias
Common abnormalities of the heart rhythm seen in an athlete

- Sinus bradycardia (up to 91%)
  - reflects predominance of vagul tone
  - may exhibit junctional escape rhythm
- Sinus arrhythmia
- 1st and 2nd (type I) degree AV block (10% - 33%)
- Incomplete RBBB (up to 51%) - QRS width between 100 - 120 msec
- Early repolarization - mild J-point and ST segment elevation
  - differential diagnosis - Brugada Syndrome
  - elevated J-point swoops into a negative T-wave
- Premature atrial & ventricular contractions
Bradycardias

Bradycarrhythmia Classifications

Classification Based on Disorder

Impulse Formation Disorders

Impulse Conduction Disorders
Bradyarrhythmia Classifications
Classification Based on Disorder

Impulse Formation Disorders

- Sinus Bradycardia

Impulse Conduction Disorders
Sinus Bradycardia
Classification Based on Disorder

Bradyarrhythmia Classifications

Classification Based on Disorder

- Sinus Bradycardia
- Sinus Arrest

Impulse Formation Disorders

Impulse Conduction Disorders
• Failure of sinus node discharge
• Absence of atrial depolarization
• Periods of asystole
Impulse Formation Disorders
- Sinus Bradycardia
- Sinus Arrest
- Brady/Tachy Syndrome

Impulse Conduction Disorders
Brady/Tachy Syndrome

- Intermittent episodes of slow and fast rates from the SA node or atria
- Brady <60 BPM
- Tachy >100 BPM
Bradyarrhythmia Classifications

Classification Based on Disorder

Impulse Formation Disorders
- Sinus Arrest
- Sinus Bradycardia
- Brady/Tachy Syndrome

Impulse Conduction Disorders
- Sino-atrial Exit Block
Transient block of impulses from the SA node
Identified by P-P interval relationship
Lead II

Sino-Atrial Exit Block (type I)
Bradyarrhythmia Classifications

Classification Based on Disorder

- Sinus Arrest
- Sinus Bradycardia
- Brady/Tachy Syndrome

Impulse Formation Disorders

- Sino-atrial Exit Block
- AV block

Impulse Conduction Disorders
First-Degree AV Block

- PR interval > 200 ms
- Delayed conduction through the AV Node
  - Example shows PR Interval = 320 ms
Progressive prolongation of the PR interval until there is failure to conduct and a ventricular beat is dropped.

Known as Wenckebach Block.
• No impulse conduction from the atria to the ventricles
  - Ventricular rate = 37 BPM
  - Atrial rate = 130 BPM
  - PR interval = variable
Third degree AV block
Bradyarrhythmia Classifications

Classification Based on Disorder

Impulse Formation Disorders
- Sinus Arrest
- Sinus Bradycardia
- Brady/Tachy Syndrome

Impulse Conduction Disorders
- Exit Block
- 1\textsuperscript{st} Degree AV Block
- 2\textsuperscript{nd} Degree AV Block
- 3\textsuperscript{rd} Degree AV Block
Bradyarrhythmia Classifications

Classification Based on Disorder

**Impulse Formation Disorders**
- Sinus Arrest
- Sinus Bradycardia
- Brady/Tachy Syndrome

**Impulse Conduction Disorders**
- Exit Block
- 1st Degree AV Block
- 2nd Degree AV Block
- 3rd Degree AV Block
- Bundle Branch Block
Bundle Branch Block

Left bundle branch block
Causes of Bradyarrhythmias

**Congenital Heart Disease**
- Present at birth due to genetics, environment

**Acquired Heart Disease**
- Acute Myocardial Infarction, Ischemic Heart Disease, Dilated or Hypertrophic Cardiomyopathy, Hypertension, Valvular Heart Disease, Post-operative

**Drug-induced**

**Neurocardiogenic**
- Hypersensitive Carotid Sinus Syndrome
- Vasovagal Syncope
12-lead ECG in the athlete: physiological versus pathological abnormalities

D Corrado, A Biffi, C Basso, A Pelliccia, G Thiene

<table>
<thead>
<tr>
<th>Common and training-related ECG changes</th>
<th>Uncommon and training-unrelated ECG changes</th>
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<tbody>
<tr>
<td>Sinus bradycardia</td>
<td>T-wave inversion</td>
</tr>
<tr>
<td>First degree AV block</td>
<td>ST-segment depression</td>
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<tr>
<td>Incomplete RBBB</td>
<td>Pathological Q waves</td>
</tr>
<tr>
<td>Early repolarisation</td>
<td>Left atrial enlargement</td>
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<tr>
<td>Isolated QRS voltage criteria for left ventricular hypertrophy</td>
<td>Left axis deviation/left anterior hemiblock</td>
</tr>
<tr>
<td></td>
<td>Right axis deviation/left posterior hemiblock</td>
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<tr>
<td></td>
<td>Right ventricular hypertrophy</td>
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<tr>
<td></td>
<td>Ventricular pre-excitation</td>
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<tr>
<td></td>
<td>Complete LBBB or RBBB</td>
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<td></td>
<td>Long or short QT interval</td>
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<td></td>
<td>Brugada-like early repolarisation</td>
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AV, atrioventricular; LBBB, left bundle branch block; RBBB, right bundle branch block.

Trained athletes commonly (up to 80%) show ECG changes such as sinus bradycardia, first-degree atrioventricular (AV) block and early repolarisation, which result from physiological adaptation of the cardiac autonomic nervous system to athletic conditioning, such as increased vagal tone and/or withdrawal of sympathetic activity.
Sinus bradycardia/arrhythmia

Resting sinus bradycardia, as defined by a heart rate <60 beats/min, is almost universal in athletes, depending on the type of sport and the level of training/competition.

Escape junctional beats or rhythm may be recorded in athletes with more severe bradycardia and result in functional AV dissociation.

Sinus arrhythmia is also reported with widely varying frequency, from approximately 15% to 70%.

Sinus bradycardia/arrhythmia disappear during exercise, suggesting that high vagal tone causes slowing of the sinus atrial node.
Only profound sinus bradycardia and/or marked sinus arrhythmia (<30 beats/min) need to be distinguished from sick sinus syndrome. A sinus atrial node dysfunction can be reasonably excluded by demonstrating that:

1. The decrease in heart rate is appropriate for the level of training and type of sports;
2. Symptoms, such as dizziness or syncope, are absent;
3. Heart rate normalises during exercise, sympathetic manoeuvres or drugs, with preservation of maximal heart rate; and
4. Bradycardia reverses with training reduction or discontinuation.
First and second degree AV block may occur physiologically at an AV Nodal level:
- in response to premature atrial impulses or atrial tachyarrhythmias
- in settings of increased vagal tone (e.g., sleep, Valsalva maneuver, well-trained athletes)

BUT... persistent 3rd degree AV block is never physiologic
Reversibility and benign recurrence of complete heart block in athletes.
Cooper JP, Fraser AG, Penny WJ.
Department of Cardiology, University of Wales College of Medicine, Cardiff, U.K.

In two patients, symptoms associated with complete heart block and physical training resolved with deconditioning. In one patient, symptoms recurred after resumption of training but she has remained well for 4 years without a pacemaker. This suggests that complete heart block induced by fitness is benign, even when symptomatic.
Objective. To prove that long-distance running is safe for athletes with pacemaker devices, pacemaker function was evaluated in nine long-distance runners.

Method. Nine runners participated in a nine-month Training programme that involved running for 1000 or 2000 km in preparation for either a full or a half marathon. A professional coach, three cardiologists and a technician - all with running experience - conducted the training and medical checkups.
Results. All nine athletes completed the Amsterdam 2001 half or full marathon without any pacemaker dysfunction. A short survey after two years showed no pacemaker dysfunction.

<table>
<thead>
<tr>
<th>Table 1. Patient pacemaker indications and running experience.</th>
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</thead>
<tbody>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td><strong>Indication for pacemaker</strong></td>
</tr>
<tr>
<td>SSS + PAF</td>
</tr>
<tr>
<td>AVB</td>
</tr>
<tr>
<td>Asystole</td>
</tr>
<tr>
<td>AF + ablation</td>
</tr>
<tr>
<td><strong>Type of pacemaker</strong></td>
</tr>
<tr>
<td>VVIR</td>
</tr>
<tr>
<td>DDDR</td>
</tr>
<tr>
<td><strong>Sensors</strong></td>
</tr>
<tr>
<td>Activity (motion)</td>
</tr>
<tr>
<td>Activity + QT</td>
</tr>
<tr>
<td>No sensor (Medtronic Minix 8340)</td>
</tr>
<tr>
<td>Experience long-distance running</td>
</tr>
<tr>
<td>No experience in long-distance running</td>
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</tbody>
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SSS+PAF=sick sinus syndrome and paroxysmal atrial fibrillation, AVB=complete acquired or congenital atioventricular block, AF=atrial fibrillation, VVIR=single-chamber ventricular pacemaker, DDDR=dual-chamber pacemaker.
Conclusion
Long-distance running is safe for athletes with pacemaker implants. Overall fitness and sufficient endurance training remain the prerequisites for maintaining the condition necessary for successful completion of a marathon regardless of medical status. In our study, it became clear that for patients who had received a pacemaker because of complete heart block, the upper rate of the pacemaker programme needed to be adjusted to 170 to 180 ppm to insure 1:1 atrio-ventricular synchrony during high atrial rates. It is concluded that there is no a priori reason for cardiologists to advise against long-distance running in athletes with pacemakers.
Cardiologists do not need to discourage physically fit pacemaker patients from long-distance running activities. In our group of nine patient-athletes, none suffered pacemaker-associated symptoms that impaired their running performances. It is important to consider the underlying conduction problem, which resulted in pacemaker implantation in the particular patient.
Electrocardiographic findings in male veteran endurance athletes

- 20 male members of Scottish Veteran Harriers Club
- 20 age matched controls (golfers / bowlers)
- Non smokers and no CVS disease
- Resting, exercise & ambulatory ECG analysis
- Echocardiography

Echocardiographic findings in male veteran endurance athletes

STRUCTURE

<table>
<thead>
<tr>
<th></th>
<th>Athletes</th>
<th>Controls</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass (g)</td>
<td>265</td>
<td>221</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV mass (g/m²)</td>
<td>151</td>
<td>118</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVID diastole (mm)</td>
<td>47</td>
<td>47</td>
<td>ns</td>
</tr>
<tr>
<td>LVID systole (mm)</td>
<td>33</td>
<td>34</td>
<td>ns</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>57</td>
<td>58</td>
<td>ns</td>
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</tbody>
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This group of veteran endurance athletes developed concentric rather than eccentric LVH

## Bradycardia in Veteran Athletes

<table>
<thead>
<tr>
<th></th>
<th>Athletes</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean 24hr heart rate (bpm)</td>
<td>59</td>
<td>74</td>
</tr>
<tr>
<td>Asystole &gt;2s</td>
<td>7/20</td>
<td>2/20</td>
</tr>
<tr>
<td>2nd Degree Heart Block</td>
<td>4/20</td>
<td>0/20</td>
</tr>
<tr>
<td>3rd Degree Heart Block</td>
<td>3/20</td>
<td>0/20</td>
</tr>
</tbody>
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## Bradycardia in Veteran Athletes 12 Year Follow Up

<table>
<thead>
<tr>
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<th>1985</th>
<th>1997</th>
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<tbody>
<tr>
<td>Mean HR</td>
<td>59</td>
<td>55</td>
</tr>
<tr>
<td>Asystole &gt;2s</td>
<td>7/20</td>
<td>2/20</td>
</tr>
<tr>
<td>2nd Degree Heart Block</td>
<td>4/20</td>
<td>1/20</td>
</tr>
<tr>
<td>3rd Degree Heart Block</td>
<td>3/20</td>
<td>3/20 (2 paced)</td>
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The prevalence of incomplete RBBB (QRS duration <120 ms) has been estimated to range from 35% to 50% in athletes compared with 10% in young, healthy controls. The ECG pattern is more often noted in athletes engaged in endurance sports, with a striking male preponderance. It has been suggested that the right ventricular (RV) conduction delay is not within the His-Purkinje system, but is caused by the enlarged RV cavity size/increased cardiac muscle mass and the resultant conduction delay.
Incomplete RBBB does not require further tests in the presence of a negative family/personal history and physical examination. Because incomplete RBBB is a typical ECG finding in patients with an atrial septal defect of the “ostium secundum” type, particular attention should be paid to exclude related symptoms and a fixed split of the second tone by accurate cardiac auscultation.
Typical features of incomplete RBBB are uncommonly observed in patients with arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D). An underlying ARVC/D should be suspected when the pattern of incomplete RBBB is associated with disproportionate extent of T-wave inversion (beyond V2 to include midprecordial V3 and V4 leads) or in the presence of premature ventricular beats with a left bundle branch block (LBBB) morphology.
In some cases, incomplete RBBB should be differentiated from a Brugada ECG. The ECG pattern of the ion channel disorder, Brugada syndrome, is characterised by a slow, positive deflection at the R-ST junction ("J wave"), which is most evident in leads V1 and V2, with minimal or no reciprocal changes in other leads.
(A) Borderline Brugada ECG pattern mimicking incomplete right bundle branch block (RBBB).

Early repolarisation has traditionally been regarded as an idiopathic and benign ECG phenomenon, with an estimated prevalence in healthy young people ranging between 1% and 2%, and a clear male preponderance. The early repolarisation ECG pattern is the rule rather than the exception among highly trained athletes, in whom it is observed in 50-80% of resting ECGs.
Different patterns of precordial early repolarisation in two healthy athletes.

The magnitude of ST-segment elevation is characteristically modulated by autonomic influences, heart rate changes and drugs; this explains the dynamic nature of the ECG abnormalities and a waxing and waning of the ST-T segment over time.

Slowing of the heart rate exaggerates ST-segment elevation, whereas sinus tachycardia occurring during exercise or after isoproterenol administration reduces and often eliminates early repolarisation changes.
Early repolarisation is a physiological and benign ECG pattern in the general population of young people and athletes, and does not require further clinical evaluation. In trained athletes, right precordial ST-T changes due to early repolarisation show typical features that may allow differentiation from ARVC/D (fig 3) or Brugada syndrome (fig 4). In rare cases, athletes may require pharmacological testing with sodium channel-blocking agents, electrophysiological study or cardiac imaging study to achieve a conclusive diagnosis.
(A) Early repolarisation pattern in a healthy black athlete characterised by right precordial T-wave inversion (arrowhead) preceded by ST-segment elevation (arrow).

(B) Right precordial T-wave inversion in a patient with arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D).

Differential diagnosis between representative right precordial ECG patterns from (A) a patient with Brugada syndrome and (B,C) two trained athletes.

In athletes presenting with syncope or cardiac arrest, which remains unexplained after a detailed clinical work-up aimed to exclude cardiac causes and neuromediated mechanisms, an ECG pattern of early repolarisation in inferior and/or lateral leads, with a prominent terminal QRS slurring, should raise the suspicion of an underlying idiopathic ventricular fibrillation.
At the end:

Telling the difference between normal adaptations and disease is challenging.

An upper limit of exercise clearly exists.

The threshold level of training is important and when passed, results in harm.
What makes it difficult

- Different sports
- Different performance level
- Differences in race, sex and age
- Differentiating pathology
- Performance enhancing drugs
Thank you for the attention
Aerobic (endurance) training leads to
• improved blood flow, and
• increased capacity of muscle fibers to generate ATP.

Anaerobic training leads to
• increased muscular strength, and
• increased tolerance for acid-base imbalances during highly intense effort.
Second degree AV block
Electrocardiographic findings in male veteran endurance athletes

24 hour trend of heart rate in athletes and controls
Heart Rate During Exercise

Submaximal = 10-15% less from the maximal
- Decreases proportionately with the amount of training completed
- May decrease by 20 to 40 beats per min after 6 months of moderate training

Maximal = 220 - age
- Remains unchanged or decreases slightly
- Thought to decrease to allow for optimal stroke volume and maximize cardiac output
The athlete's heart was first described in 1899 by S. Henschen. He compared the heart size of cross-country skiers to those who lived sedentary lives. He noticed that those who participated in competitive sports displayed symptoms of athlete’s heart syndrome. Henschen believed the symptoms were a normal adjustment to exercise, and felt there was no need for concern.
Exercise and the Heart
Athlete’s Heart

Rhythm /ECG

- Sinus bradycardia /arrest
- Wandering pacemaker
- Junctional bradycardia
- First degree AV block
- 2nd Degree AV block (Wenckebach)
- Repolarisation “abnormalities” (ST & T wave changes)
This problem becomes more intriguing when Wenckebach A-V block is encountered in asymptomatic top-ranking athletes, because of medico-legal implications. We report 10 cases of highly-trained athletes, including three with mitral valve prolapse (MVP) features, with a spontaneous or induced Wenckebach second-degree A-V block.
Ambulatory ECG recordings in endurance athletes

- Sinus pauses >2s 37% (5.7%)
- 1st degree AV block 37% (14%)
- Wenckebach AV block 22.9% (5.7%)
- Mobitz type II AV block 8.6% (0%)
- Junctional rhythm 20% (0%)

1Vitassalo MT et al. Br Heart J 1982; 47: 213-20
Athlete's heart is common in athletes who routinely exercise more than an hour a day, and occurs primarily in endurance athletes, though it can occasionally arise in heavy weight trainers. The condition is generally believed to be a benign one, but may sometimes be hard to distinguish from other serious medical conditions. For example, on the results of an electrocardiogram (EKG) athletic heart syndrome may be mistakenly interpreted as evidence of serious heart disease. [3]