Agenda

Over two hours with a small break

- ECG Interpretation Tools
  - Atrial and Ventricular Hypertrophy
  - Wolff-Parkinson White
  - Hypertrophic Cardiomyopathy
  - Pulmonary Embolus
  - Prolonged QT

- Heart Failure Clinical Practice Guidelines
- 2 Lead ECG Practice
Objectives

1. Identifies abnormal findings associated with various pathologies.

2. Identifies 12 Lead ECG changes correlating to multiple pathologies and their management and therapies.

3. Selects appropriate clinical practice guidelines for the heart failure patient, including ACE-I/ARB and beta blocker therapies.
<table>
<thead>
<tr>
<th>Wall</th>
<th>Leads</th>
<th>Coronary Artery</th>
<th>Reciprocal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>V1, V2, V3, V4</td>
<td>LAD branch of LCA</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>RCA</td>
<td>I, aVL</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
<td>Circumflex branch of LCA</td>
<td>V1, V3</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1, V2</td>
<td>RCA, Circumflex</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(ST depression, tall R waves)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>V3, V4, V5, V6</td>
<td>LAD, RCA</td>
<td></td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, aVL, V1, V2, V3, V4, V5, V6</td>
<td>LAD, Circumflex</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Septal</td>
<td>V1, V2</td>
<td>LAD</td>
<td></td>
</tr>
<tr>
<td>LEAD</td>
<td>NORMAL WAVEFORM</td>
<td>USUAL DEFLECTION</td>
<td>AREA OF HEART IT VIEWS</td>
</tr>
<tr>
<td>------</td>
<td>-----------------</td>
<td>------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>I</td>
<td>![Waveform]</td>
<td>Positive</td>
<td>Lateral, atrial rhythms</td>
</tr>
<tr>
<td>II</td>
<td>![Waveform]</td>
<td>Positive</td>
<td>Inferior, SA node, atrial rhythms</td>
</tr>
<tr>
<td>III</td>
<td>![Waveform]</td>
<td>Usually positive, can be equiphasic</td>
<td>Inferior</td>
</tr>
<tr>
<td>AVR</td>
<td>![Waveform]</td>
<td>Negative</td>
<td>No specific</td>
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<tr>
<td>AVL</td>
<td>![Waveform]</td>
<td>Equiphasic</td>
<td>Lateral</td>
</tr>
<tr>
<td>AVF</td>
<td>![Waveform]</td>
<td>Positive</td>
<td>Inferior</td>
</tr>
<tr>
<td>V1</td>
<td>![Waveform]</td>
<td>Negative</td>
<td>Anterior septal, ST segment, BBB</td>
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<tr>
<td>V2</td>
<td>![Waveform]</td>
<td>Mainly negative</td>
<td>Anteroseptal</td>
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<tr>
<td>V3</td>
<td>![Waveform]</td>
<td>Equiphasic</td>
<td>Anterior</td>
</tr>
<tr>
<td>V4</td>
<td>![Waveform]</td>
<td>Equiphasic, becoming more positive</td>
<td>Anterior</td>
</tr>
<tr>
<td>V5</td>
<td>![Waveform]</td>
<td>Positive</td>
<td>Lateral</td>
</tr>
<tr>
<td>V6</td>
<td>![Waveform]</td>
<td>Positive</td>
<td>Lateral</td>
</tr>
<tr>
<td>Axis</td>
<td>Lead I</td>
<td>Lead II</td>
<td>Lead III</td>
</tr>
<tr>
<td>---------------------------</td>
<td>--------</td>
<td>---------</td>
<td>----------</td>
</tr>
<tr>
<td>Normal 0-90</td>
<td><img src="image1" alt="Diagram" /></td>
<td><img src="image2" alt="Diagram" /></td>
<td><img src="image3" alt="Diagram" /></td>
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<tr>
<td>Physiologic Left Axis 0--40</td>
<td><img src="image4" alt="Diagram" /></td>
<td><img src="image5" alt="Diagram" /></td>
<td><img src="image6" alt="Diagram" /></td>
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<tr>
<td>Pathological Left Axis -40 to -90</td>
<td><img src="image7" alt="Diagram" /></td>
<td><img src="image8" alt="Diagram" /></td>
<td><img src="image9" alt="Diagram" /></td>
</tr>
<tr>
<td>Right Axis 90-180</td>
<td><img src="image10" alt="Diagram" /></td>
<td><img src="image11" alt="Diagram" /></td>
<td><img src="image12" alt="Diagram" /></td>
</tr>
<tr>
<td>Extreme Right Axis No Man’s Land</td>
<td><img src="image13" alt="Diagram" /></td>
<td><img src="image14" alt="Diagram" /></td>
<td><img src="image15" alt="Diagram" /></td>
</tr>
</tbody>
</table>
General guidelines:
>1 mm in limb leads
> 2 mm in chest leads

Acute injury is occurring. Heart attack is happening now.
Q waves develop over 4 to 24 hours and remain for life.

Significant Q waves are 25–33% of the R wave. Q > 0.038 seconds
Ischemia  Inverted T waves

Supply and Demand problem.
Left Atrial Hypertrophy

Hypertension
Left atrial enlargement
  - Pulmonary edema

Broad or notched P–waves
Prolonged P wave (>0.12)
V1 broad trough
I, II, & V4-V6 notched
Right Atrial Hypertrophy

Lung disease
- COPD
Pulmonary Embolus
Pulmonary Hypertension

What will see
- Tall, peaked P-waves
  - II, III, aVF
Right Ventricular Hypertrophy

Increased right ventricular mass
- Pulmonary stenosis
- Primary pulmonary hypertension
- Pulmonary embolus
- Diastolic overload
- Atrial septal defect

Right axis deviation
- Tall “R” waves in right precordial leads
  - V1 most sensitive
- Deep “S” waves in left precordial leads
  - V6

Causes

What will see
Left Ventricular Hypertrophy

Increased LV muscle mass
- Hypertension
- Cardiomegaly
- Cardiomyopathy
- Valvular regurgitation
- Aortic stenosis

Left axis deviation

Measure
- V1 or V2 Deepest “S” wave
- V5 or V6 Tallest “R” wave

#mm add up > 35 mm
Heart Failure Epidemiology

- 1 month readmission rate of 25%
  - 50% at 6 months
- Over half of the total cost of HF care in the US is spent on hospitalizations.

<table>
<thead>
<tr>
<th>Lifetime Risk</th>
<th>Prevalence</th>
<th>Incidence</th>
<th>Mortality</th>
<th>Hospital Discharges</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>20% of Americans ≥ 40 years</td>
<td>~5.7 million</td>
<td>Over 650,000 annually</td>
<td>50% within 5 years 1 yr ~ 30%</td>
<td>&gt; 1,000,000 annually</td>
<td>&gt; $40 billion annually</td>
</tr>
</tbody>
</table>
### Definition of Heart Failure

<table>
<thead>
<tr>
<th>Classifications</th>
<th>Ejection Fraction</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Failure with Reduced Ejection Fraction (HFrEF)</td>
<td>≤ 40%</td>
<td>• Systolic HF&lt;br&gt;• Reduced Left Ventricle contractility&lt;br&gt;• Diminished ejection fraction</td>
</tr>
<tr>
<td>Heart Failure with Preserved Ejection Fraction (HFpEF)</td>
<td>≥ 50%</td>
<td>• Diastolic HF&lt;br&gt;• Stiffing of the ventricle&lt;br&gt;• Problem with ventricular filling or relaxation</td>
</tr>
<tr>
<td>HFpEF Borderline</td>
<td>41 to 49%</td>
<td>• Borderline or intermediate group</td>
</tr>
<tr>
<td>HFpEF Improved</td>
<td>≥ 40%</td>
<td>• Previously had HFrEF</td>
</tr>
</tbody>
</table>
Heart Failure Stages & Recommended Therapy

**STAGE A**
At high risk for HF but without structural heart disease or symptoms of HF

- Patients with:
  - HTN
  - Atherosclerotic disease
  - DM
  - Obesity
  - Metabolic syndrome
  - Using cardiotoxins
  - With family history of cardiomyopathy

**STAGE B**
Structural heart disease but without signs or symptoms of HF

- Patients with:
  - Previous MI
  - LV remodeling including LHV and low EF
  - Asymptomatic valvular disease

**STAGE C**
Structural heart disease with prior or current symptoms of HF

- Patients with:
  - Known structural heart disease and HF signs and symptoms

**STAGE D**
Refractory HF

- Patients with:
  - Marked HF symptoms at rest
  - Recurrent hospitalizations despite GDMT

**Therapy**
- Goals:
  - Heart healthy lifestyle
  - Prevent vascular, coronary disease
  - Prevent LV structural abnormalities

**Therapy Goals**
- Prevent HF symptoms
- Prevent further cardiac remodeling

- Drugs:
  - ACEI or ARB as appropriate
  - Beta blockers as appropriate
  - In selected patients:
    - ICD
    - Revascularization or valvular surgery as appropriate

**Therapy**
- Goals:
  - Control symptoms
  - Improve HRQOL
  - Prevent hospitalization
  - Prevent mortality

**Strategies**
- Identification of comorbidities

**Treatment**
- Diuresis to relieve symptoms of congestion
- Follow guideline driven indications for comorbidities, e.g., HTN, AF, CAD, DM

**Therapy**
- Goals:
  - Control symptoms
  - Patient education
  - Prevent hospitalization
  - Prevent mortality

**Drugs for routine use**
- Diuretics for fluid retention
  - ACEI or ARB
  - Beta blockers
  - Aldosterone antagonists

**Drugs for use in selected patients**
- Hydralazine/noradrenergic drinat
  - ACEI and ARB
  - Digitalis

**In selected patients**
- CRT
- ICD
- Revascularization or valvular surgery as appropriate

BNP – B type Natriuretic Peptide

Released by the cardiomyocytes with myocardial stretch.
Release modulated by calcium ions.

Poor prognosis if BNP stays chronically elevated.
Causes for elevated BNP levels

**Cardiac**
- Heart Failure, including right ventricle syndromes
- Acute coronary syndrome
- Heart muscle disease, including left ventricular hypertrophy
- Valvular heart disease
- Pericardial disease
- Atrial fibrillation
- Myocarditis
- Cardiac surgery
- Cardioversion

**Non-cardiac**
- Advancing age
- Anemia
- Renal failure
- Pulmonary causes; obstructive sleep apnea, severe pneumonia, pulmonary HTN
- Critical illness
- Bacterial sepsis
- Severe burns
- Toxic-metabolic insults
Biomarkers for Heart Failure

- Galectin - 3
  - Seen with fibrosis and remodeling
- ST 2
- Interleukin 6
- Procalcitonin
  - Also mentioned in sepsis campaign
  - Not one of 3 or 6 hour bundle elements
Pharmacologic Therapy for Heart Failure

- **ACE inhibitor or ARB**
  - Captopril, Enalapril, Fosinopril, Lisinopril, Ramipril
  - Candesartan, Losartan, Valsartan

- **Beta blocker**
  - Carvedilol, metoprolol succinate CR/XL

- **Diuresis**

- **Digoxin mixed reviews**

- **Aldosterone antagonist**
  - Spironolactone

- **Hydralazine/nitrate**
  - Hydralazine and isosorbide dinitrate

- **Chronic anticoagulation for permanent or persistent atrial fibrillation**

- **Omega 3 supplementation is reasonable**

- **Statins no benefit in solely HF**

- **Calcium Channel Blockers are not recommended in HFrEF**

- **Avoid NSAIDs**
Diuretics and NSAIDs

Don’t take together.

NSAIDs

- Inhibit renal prostaglandins – \( I_2 \) and \( E_2 \)
- Increase sodium and water retention
- Blunt the response to diuretics
- Lose nitric oxide vasodilation
Beta Blockers

- Reduce catecholamine levels
- Decrease myocardial ischemia and limit infarct size
- Reduce myocardial workload and oxygen demand
- Reduce heart rate and blood pressure
- Reduce supraventricular and malignant ventricular arrhythmias

Metoprolol – Lopressor, Toprol XL
Carvedilol – Coreg
Bisoprolol - Zebeta

Only three BBs have been shown in studies to help in heart failure.
Renin-Angiotensin-Aldosterone System

- Decrease in renal perfusion (juxtaglomerular apparatus)
- Angiotensinogen → Angiotensin I → Angiotensin II → Tubular Na⁺ Cl⁻ reabsorption and K⁺ excretion, H₂O retention
- Adrenal gland: cortex → Aldosterone secretion
- Arteriolar vasoconstriction, increase in blood pressure
- ADH secretion
- Collecting duct: H₂O absorption

Legend:
- Secretion from an organ
- Stimulatory signal
- Inhibitory signal
- Reaction
- Active transport
- Passive transport

Water and salt retention. Effective circulating volume increases. Perfusion of the juxtaglomerular apparatus increases.
ACE-I & ARBs

ACE-I
Lisinopril – Prinivil, Zerteril
Benazepril – Lotensin
Captopril – Capoten
Ramipril - Altace
Enalapril – Vasotec
Fosinopril – Monopril

Adverse effect – cough, angioedema, hyperkalemia
Watch renal function.

ARB
Losartan – Cozaar
Valsartan – Diovan
Irbesartan – Avapro

Tend not to have as many adverse effects. Cough not really seen.
Corlanor (ivabradine)

Funny current works on pacemaker (SA node) activity and modulations

- Patients did better with a decreased heart rate ~70.
- But do keep heart rate above 60 and cannot be 100% paced.

- 5 mg BID increasing to 7.5 mg BID with meals
  - Can start 2.5 BID per company.

- Adverse effects: Bradycardia, visual disturbances (enhanced brightness)
  - More about funny channel blockers @ [http://circres.ahajournals.org/content/106/3/434.full](http://circres.ahajournals.org/content/106/3/434.full)
Entresto (sacubitril / valsartan)

Neprilysin inhibitor results in an increased concentration of natriuretic peptides and inhibit RAAS.

- Promotes natriuretic and vasodilatory properties.

- Film-coated tablets (sacubitril/valsartan): 24/26 mg; 49/51 mg; 97/103 mg BID

- Adverse effects: Hypotension, angioedema, hyperkalemia, renal impairment
Nonpharmacological Interventions

- Nutritional supplements
  - For HFrEF patients
- Exercise training or regular physical activity
- Sodium restriction is reasonable
  - 2000-3000 mg daily, avoid potassium-based salt substitutes
- Daily weight monitoring
- Daily fluid limitation
  - 2 liters per day
- Education focused on self-care
  - Smoking cessation, weight reduction, discontinue alcohol use, vaccination
  - Manage co-morbidities – diabetes, high blood pressure, atrial fibrillation
Life Vest & Cardiac Devices

- **Life Vest**
  - Often prelude to an implantable device
  - Non-invasive and continuous monitor
  - 98% first shock success rate

- **Implantable Cardioverter Defibrillator**
  - CABG or PCI must wait 3 months
  - AMI must wait 40 days

- **Cardiac Synchronization Therapy**
  - Biventricular pacing
  - 3 leads – right atrium, right ventricle, left ventricle
CRT- BiVentricular Pacing

EF < 35%. Wide QRS > 10 ms. Very complex algorithm.
Surgical Therapies for HF

- Percutaneous Coronary Intervention
- Coronary revascularization (CABG)
- Transcatheter aortic valve replacement (TAVR)
- Mitral valve repair or replacement
Medical management more complex.

- **Ejection Fraction (EF%) must be documented.**
  - New or documentation of known, or when will be performed

- **Discharged on**
  - Specific Beta Blocker
  - ACE-I or ARB therapy for left ventricular systolic dysfunction, EF (ejection fraction) \( \leq 40\% \)

- **Educated on**
  - Daily weights
  - Fluid limitations
  - Diet
  - Signs and symptoms
  - Follow up appointment
Patient Safety Indicators

1. Mortality
2. Readmission within 30 days
3. Patient Satisfaction
Wolff-Parkinson White

Sinus impulses bypass the AV node via an accessory pathway (AP) conduction.

- Uncommon - ~2 per 1,000 in the general population
- Can be right-sided, left-sided, anterior, or posterior – and sometimes more than a single AP.
- A very fast atrial fibrillation (250-300) – think WPW.
Accessory Pathway

Normal electrical pathways

Abnormal electrical pathway in Wolff-Parkinson-White syndrome
WPW

Three key signs:

- Delta wave which may be positive or negative
- QRS widening
- Short PR interval
Hypertrophic Cardiomyopathy
Hypertrophic Cardiomyopathy

Previously known as

- Hypertrophic obstructive cardiomyopathy – HCOM
- Idiopathic hypertrophic subaortic stenosis – IHSS

Number one cause of sudden cardiac death in young athletes (1-2%).

Inheritance is primarily autosomal dominant.

ECG changes

- Left ventricular hypertrophy pattern
  - Tall R waves
  - Large precordial voltages
- Deep, narrow “dagger-like” Q waves in lateral and inferior leads
- Giant T-wave inversion in apical HCM
- Left atrial enlargement
- Atrial fibrillation and SVTs are common
12 Lead ECG as a Screening Test

United States does not require
- Italy and Israel do

Issues
- Placement of leads
- 30% false positives

Ethical issues
- Consent for screening
- Who receives results
- Who makes the determination of risk with participation in activities

http://circ.ahajournals.org/content/130/15/1303
14 Element Screening

Medical history
- Verified by parents for high and middle school athletes

Personal history
- Chest pain, discomfort, rightness, pressure related to exertion
- Unexplained syncope, near-syncope
  - Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring during or after physical exertion
- Excessive and unexplained dyspnea, fatigue, or palpitations, associated with exercise
- Prior recognition of a heart murmur
- Elevated systemic blood pressure
- Prior restriction from participation in sports
- Prior testing for the heart, ordered by a physician
Family history

- Premature death (sudden and unexpected, or otherwise) before 50 years of age attributable to heart disease in ≥ 1 relative
- Disability from heart disease in close relative < 50 years of age
- Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members

Physical examination

- Heart murmur
  - Organic and unlikely to be innocent
  - Performed both supine and standing or with Valsalva maneuver
  - Specifically to identify murmurs of dynamic left ventricular outflow tract obstruction
- Femoral pulses to exclude aortic coarctation
- Physical stigmata of Marfan syndrome
- Brachial artery blood pressure
  - Sitting, bilateral
Pulmonary Embolus

Look at the sum of all in context with the clinical history.

- ECG is not diagnostic.
- Can strongly suggest before the V/Q or CT scan.

Old – $S_I-Q_{III}-T_{III}$ “classic” finding is neither sensitive nor specific
## Suspect PE?

- **New onset dyspnea, pleuretic**

- **Typically tachycardic**
  - Most common, seen in 44% of cases

- **RBBB**
  - Complete or incomplete

- **Right Ventricular strain pattern**
  - T wave inversion in V1, V2, V3, also V4
  - T Wave inversion II, III, aVF

- **Right axis deviation**
  - Extreme right axis may occur between 0 and -90, giving appearance of left axis (pseudo left axis)

- **Dominant R wave in V1**
  - Manifestation of acute right ventricular dilation

- **RA enlargement**
  - Peaked P waves in lead II

- **Wide S in lead I, subtle S in V6**

- **ST elevation in aVR**
Normal is considered less than half of the R-R (when the heart rate is ~70).

Conditions Predisposing for Long QT > Torsades
- Baseline long QT
  - >450 ms, esp > 500 ms
- Female gender
- Electrolyte disorder
  - Especially low K+ and Mg++
- Bradycardia < 50
- Structural heart disease
- Significant renal or hepatic dysfunction

Common causes:
- Medications
- Electrolyte imbalance
  - Hypokalemia
    - ST flattening, depression, develop U waves
  - Hypomagnesemia
    - Like hypokalemia
  - Hypocalcemia
    - Normal T wave after prolonged QT interval
- CNS catastrophes
  - Stroke, seizure, coma, intra-cerebral or brainstem bleeding
  - Can produce bizarre ST-T waves and some of the longest QT intervals
## Medications that prolong QT interval

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>sotalol</td>
<td>Betapace</td>
</tr>
<tr>
<td>quinidine</td>
<td>Quiniglute</td>
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<tr>
<td>*amiodarone</td>
<td>Cardarone, Pacerone</td>
</tr>
<tr>
<td>*procainamide</td>
<td>Procan, Pronestyl</td>
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<tr>
<td>*disopyramide</td>
<td>Norpace</td>
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<tr>
<td>nicardipine</td>
<td>Cardene</td>
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<tr>
<td>*ibutilide</td>
<td>Corvert</td>
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<td>*dofetilide</td>
<td>Tikosyn</td>
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<tr>
<td>trimethoprim-sulfa</td>
<td>Bactrim</td>
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<tr>
<td>*clarithromycin</td>
<td>Biaxin</td>
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<td>*erythromycin</td>
<td>EES, Erythrocin</td>
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<td>*methadone</td>
<td>Methadone, Dolophine</td>
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<tr>
<td>pseudoephedrine</td>
<td>Sudafed</td>
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<tr>
<td>tacrolimus</td>
<td>Prograf</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Generic name</th>
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</tr>
</thead>
<tbody>
<tr>
<td>albuterol</td>
<td>Ventolin, Proventil</td>
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<tr>
<td>levalbuterol</td>
<td>Xopenex</td>
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<tr>
<td>Salmeterol</td>
<td>Serevent</td>
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<td>*chlorpromazine</td>
<td>Thorazine</td>
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<td>Neosymphepine</td>
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<td>ondansetron</td>
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<tr>
<td>dobutamine</td>
<td>Dobutrex</td>
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<td>dopamine</td>
<td>Intropin</td>
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<tr>
<td>phenylpropanolamine</td>
<td>Dexatrim, Acutrim</td>
</tr>
</tbody>
</table>

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*Indicates medications that are frequently used for this purpose.
QTc by Bazett’s Formula

Step 1
• Find the square root of the R-R interval
• Measure the R-R interval (# of squares x 0.04) then press the square root sign on a calculator.

Step 2
• Measure the QT interval
• Change the QT interval from seconds to milliseconds (QT .44 secs = 440 ms)

Step 3
• Divide the QT interval in ms by the square root of the R-R interval to calculate the QTc.

Example:
Step 1
R-R is 19 squares x 0.04 = 0.76
Press the square root button
The square root of 0.76 is 0.87

Step 2
QT interval is .48 sec or 480 ms

Step 3
480 ÷ 0.87 = QTc of 552 (551.7) ms
References


