Conduction disorders

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Lecture for 5 course, update 2013
Cardiac Conduction

Sinus Node

• The Heart’s ‘Natural Pacemaker’
  – Rate of 60-100 bpm at rest
Cardiac Conduction
AV Node

- Receives impulses from SA node
- Delivers impulses to the His-Purkinje System
- Delivers rates between 40-60 bpm if SA node fails to deliver impulses
Cardiac Conduction

HIS Bundle

- Begins conduction to the ventricles
- AV Junctional Tissue:
  - Rates between 40-60 bpm
Cardiac Conduction
Purkinje Fibers

- Moves the impulse through the ventricles for contraction
- Provides ‘Escape Rhythm’: Rates between 20-40 bpm
ECGs Annotation

Normal Ranges in Milliseconds:

- PR (Q) Interval 120 – 200 ms
- QRS Complex 60 – 100 ms
- QT Interval 360 – 440 ms
Status Check

Match the term on the left with the description on the right

Click for Answer

- P-R Interval
  - Escape rate is 40-60 bpm

- AV Node
  - Connect His bundle to Purkinje network

- Purkinje Network
  - Normally 120-200 ms

- Bundle Branches
  - Depolarizes the Ventricles
Bradycardia Classifications

- Disorders of
  - Impulse Formation
- Impulse Conduction
Bradycardia Classifications

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

- Impulse Conduction
  - Slow or Blocked Conduction
Sinus Arrest

• Failure of sinus node discharge
• Absence of atrial depolarization
• Periods of ventricular asystole
• May be episodic as in vaso-vagal syncope, or carotid sinus hypersensitivity
  – May require a pacemaker
Sinus Bradycardia

- Sinus Node depolarizes very slowly
- If the patient is symptomatic and the rhythm is persistent and irreversible, may require a pacemaker
Brady/Tachy Syndrome

- Intermittent episodes of slow and fast rates from the SA node or atria
- Brady < 60 bpm
- Tachy > 100 bpm
- AKA: Sinus Node Disease
  - Patient may also have periods of AF and chronotropic incompetence
  - 75-80% of pacemakers implanted for this diagnosis
Bradycardia Classifications

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

**Impulse Conduction**
- Slow or Blocked Conduction
Mechanisms of Rhythm Disorders

Slowed or Blocked Conduction

- Impulse generated normally
- Impulse slowed or blocked as it makes its way through the conduction system
Cardiac conduction block

Block position:
Sinoatrial; intra-atrial; atrioventricular; intra-ventricular

Block degree
1. Type I: prolong the conductive time
2. Type II: partial block
3. Type III: complete block
Exit Block (Sinoatrial block)

- Transient block of impulses from the SA node
- Pacing is rare unless symptomatic, irreversible, and persistent
Atrioventricular (AV) Block

• AV block is a delay or failure in transmission of the cardiac impulse from atrium to ventricle.

• Etiology:
  Atherosclerotic heart disease; myocarditis; rheumatic fever; cardiomyopathy; drug toxicity; electrolyte disturbance, collagen disease
AV Block

AV block is divided into three categories:

1. First-degree AV block

2. Second-degree AV block: further subdivided into Mobitz type I and Mobitz type II, or a “high grade” block (2:1, 3:1)

3. Third-degree AV block: complete block
First-Degree AV Block

- PR interval > 200 ms
- Delayed conduction through the AV Node
  - Not an indication for pacing *Leave it alone!*
Second-Degree AV Block – Mobitz I

- Progressive prolongation of the PR interval until there is failure to conduct and a ventricular beat is dropped
- AKA: Wenckebach block
  - Usually not an indication for pacing
Second Degree AV Block Type 1 (Wenckebach)

- Increasing delay at AV node until a p wave is not conducted.
- Often comes post inferior MI with AV node ischemia
- Gradual prolongation of the PR interval before a skipped QRS. QRS are normal!
- No pacing as long as no bradycardia.
Second-Degree AV Block – 2:1 block

- Regularly dropped ventricular beats
- A “high grade” block,
- Usually an indication for pacing
- May progress to third-degree, or Complete Heart block (CHB)
Second-degree AV block

type II

Sudden loss of a QRS wave because p wave was not transmitted beyond AV node.
2nd degree heart block (2:1)
Second Degree AV Block 3:1 block

- Sudden loss of a QRS wave because p wave was not transmitted beyond AV node. May be precursor to complete heart block and needs pacing.
Third-Degree (Complete) AV Block

- No impulse conduction from the atria to the ventricles - atria and ventricles beat independently AND atria beat faster than ventricles:
  - Complete A – V disassociation
  - Atrial rate is faster than Ventricular rate
  - Usually a wide QRS as ventricular rate is idioventricular (distal block) or narrow QRS if AV is pacemaker (proximal block)
Complete (3rd degree) heart block
AV Block

Manifestations:

• First-degree AV block: almost no symptoms;
• Second degree AV block: palpitation, fatigue
• Third degree AV block: Dizziness, agina, heart failure, lightheadedness, and syncope may cause by slow heart rate, Adams-Stokes Syndrome may occurs in sever case.
• First heart sound varies in intensity, will appear booming first sound
AV Block

Treatment:
1. I or II degree I type AV block needn’t antibradycardia agent therapy
2. II degree II type and III degree AV block need antibradycardia agent therapy
3. Implant Pace Maker
Intraventricular Block

Intraventricular conduction system:

1. Right bundle branch
2. Left bundle branch
3. Left anterior fascicular
4. Left posterior fascicular
Intraventricular Block

Etiology:

• Myocarditis, valve disease, cardiomyopathy, CAD, hypertension, pulmonary heart disease, drug toxicity, Lenegre disease, Lev’s disease et al.

Manifestation:

• Single fascicular or bifascicular block is asymptom; tri-fascicular block may have dizziness; palpitation, syncope and Adams-stokes syndrome
Intraventricular Block

What happens to Right and Left ventricular depolarization if one bundle branch is blocked?

What do you suspect the QRS complex may look like?

- Two out of QRS's phase
- Wide QRS
Right BBB

Left BBB

R and R’ in Right and Left BBB often look like above
Right Bundle Branch Block (RBBB)

Right ventricle gets a delayed impulse

1. Depolarization spreads from the left ventricle to the right ventricle.

2. This creates a second R-wave (R’) in V1, and a slurred S-wave in V5 - V6.

3. The T wave should be deflected opposite the terminal deflection of the QRS complex. This is known as appropriate T wave discordance with bundle branch block. A concordant T wave may suggest ischemia or myocardial infarction.

4. Pacemaker if syncope occurs.

QRS is widened
V1 and V2 have rSR’
Left Bundle Branch Block (LBBB)

Left ventricle gets a delayed impulse

1. Depolarization enters the right side of the right ventricle first and simultaneously depolarizes the septum from right to left. This creates a QS or rS complex in lead V1 and a monophasic or notched R wave in lead V6.

2. The T wave should be deflected opposite the terminal deflection of the QRS complex. This is known as appropriate T wave discordance with bundle branch block. A concordant T wave may suggest ischemia or myocardial infarction.

3. Pacemaker if syncope occurs
Intraventricular Block

Therapy:

1. Treat underlying disease
2. If the patient is asymptomatic; no treat,
3. If progress to complete block, may need implant pacemaker if the patient with syncope
**Adult Bradycardia (With Pulse)**

3. Persistent bradyarrhythmia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Monitor and observe
   - Yes
   - No

5. Atropine
   - If atropine ineffective:
     - Transcutaneous pacing
     - Dopamine infusion
     - Epinephrine infusion

6. Consider:
   - Expert consultation
   - Transvenous pacing

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**Doses/Details**

**Atropine IV Dose:**
- First dose: 0.5 mg bolus
- Repeat every 3-5 minutes
- Maximum: 3 mg

**Dopamine IV Infusion:**
- 2-10 mcg/kg per minute

**Epinephrine IV Infusion:**
- 2-10 mcg per minute
Pacemaker

- Permanent - battery under skin
- Temporary - battery outside body
- Types
  - Transvenous
  - Epicardial - bypass surgery
  - Transcutaneous - emergency
- Modes
  - Asynchronous - at preset time without fail
  - Synchronous or demand - when HR goes below set rate
Pacemaker

In a dual-chamber pacemaker, one lead or electrical wire stimulates the right atrium and one stimulates the right ventricle to beat properly.
Pacemaker Problems:

• Failure to sense
• Failure to capture
Sudden cardiac death (SCD)

- Sudden cardiac death (SCD) is used to describe **cardiac arrest** with cessation of cardiac function, whether or not resuscitation or spontaneous reversion occurs.

- Patients who do not die after cardiac arrest should be said to have experienced **aborted SCD**.
Sudden cardiac death (SCD)

Definition by WHO

• Sudden collapse of cardiac function occurring within one hour of symptoms
Sudden cardiac death (SCD)

Pathophysiology

- The vast majority of cases of SCD are due to ventricular arrhythmias
- Ventricular tachycardia (VT) or ventricular fibrillation (VF) account for the majority of episodes
- This almost always occurs in the setting of underlying myocardial disease
- More than 80% of SCD events occur in individuals with coronary artery disease (CAD)
Sudden cardiac death (SCD)

Symptoms & signs

- Chest pain
- Dyspnea
- Fatigue
- Palpitations
- Syncope
Time References in Sudden Cardiac Death

Prodromes
- New or worsening cardiovascular symptoms
  - Chest pain
  - Palpitations
  - Dyspnea
  - Fatiguability

Days-to-months

Onset of terminal event
- Abrupt change in clinical status
  - Arrhythmia
  - Hypotension
  - Chest pain
  - Dyspnea
  - Lightheadedness

Up to 1 hour

Cardiac arrest
- Sudden collapse
  - Loss of effective circulation
    - Loss of consciousness

Minutes-to-weeks

Biological death
- Failure of resuscitation
  - OR
- Failure of electrical, mechanical, or CNS function after initial resuscitation
Major Causes of Sudden Cardiac Death

Ischemic heart disease
- Coronary artery disease with myocardial infarction or angina
- Coronary artery embolism
- Nonatherogenic coronary artery disease (arteritis, dissection, congenital coronary artery anomalies)
- Coronary artery spasm

Nonischemic heart disease
- Hypertrophic cardiomyopathy
- Dilated cardiomyopathy
- Valvular heart disease
- Congenital heart disease
- Arrhythmogenic right ventricular dysplasia
- Myocarditis
- Acute pericardial tamponade
- Acute myocardial rupture
- Aortic dissection

No structural heart disease
- Primary electrical disease (idiopathic ventricular fibrillation)
- Brugada syndrome (right bundle branch block and ST segment elevation in leads V1 to V3)
- Long QT syndrome
- Preexcitation syndrome
- Complete heart block
- Familial sudden cardiac death
- Chest wall trauma (commotio cordis)

Noncardiac disease
- Pulmonary embolism
- Intracranial hemorrhage
- Drowning
- Pickwickian syndrome
- Drug-induced
- Central airway obstruction
- Sudden infant death syndrome
SCD & ischemic heart disease

Incidence of VT and VF after ST-elevation MI

- VF: 4.2 %
- VT: 3.5 %
- Both VF and VT: 2.7 %

80 to 85 % of these arrhythmias occurred in the first 48 hours.
Hypertrophic cardiomyopathy

• Most common cause of SCD in young (age ≤ 35 y/o)
• SCD is primarily related to VT or VF.
• The mechanism of arrhythmia in this setting is not clear
• Autosomal-dominant inherited disease
Valvular disease

- Aortic stenosis (predominate)
- The mechanism of sudden death is unclear, and both malignant ventricular arrhythmia and bradyarrhythmia have been documented
Absence of structural heart disease

- Long QT syndrome
- Wolff-Parkinson-White (WPW) syndrome
- Commotio cordis
Long QT syndrome

- Prolonged QT interval
- Polymorphic ventricular tachycardia (VT) called torsade de pointes
**Torsade de pointes** This is an atypical, rapid, and bizarre form of ventricular tachycardia that is characterized by a continuously changing axis of polymorphic QRS morphologies.
QTc = QT interval ÷ square root of the RR interval (in msec)

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<th>1-15 yrs</th>
<th>Men</th>
<th>Women</th>
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<tr>
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<td>0.44</td>
<td>&lt;0.43</td>
<td>&lt;0.45</td>
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<tr>
<td>Borderline</td>
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<td>0.43-.045</td>
<td>0.45-.047</td>
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<tr>
<td>Prolonged</td>
<td>&gt;0.46</td>
<td>&gt;0.45</td>
<td>&gt;0.47</td>
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<tr>
<td>(upper one percent)</td>
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Causes of the Long QT Syndrome

**Congenital**
- Jervell-Lange-Nielsen syndrome
- Romano-Ward syndrome
- Idiopathic

**Acquired**

**Metabolic disorders**
- Hypokalemia
- Hypomagnesemia
- Hypocalcaemia
- Starvation
- Anorexia nervosa
- Liquid protein diets
- Hypothyroidism

**Bradyarrhythmias**
- Sinus node dysfunction
- AV block — second or third degree

**Antiarrhythmic drugs**
- Quinidine
- Procainamide or N-acetylprocainamide
- Disopyramide
- Amiodarone
- Sotalol
- Dofetilide, sematilide, ibutilide, bepridil, mibebradil

**Antimicrobial drugs**
- Erythromycin, clarithromycin, telithromycin, azithromycin (minor)
- Pentamidine
- Some fluoroquinolones (eg, sparfloxacin, gatifloxacin, levofloxacin, moxifloxacin)
- Other — Spiramycin, chloroquine, halofantrine, mefloquine

**Antihistamines**
- Terfenadine
- Astemizole

**Psychotropic drugs**
- Thioridazine
- Phenothiazines
- Butyrophenones
- Tricyclic or tetracyclic antidepressants
- Haloperidol
- Selective serotonin reuptake inhibitors
- Risperidone
- Very high dose methadone

**Other drugs**
- Vasodilators — Prenylamine
- Diuretics — Via electrolyte changes
- Serotonin antagonist — Ketanserin
- Motility drugs — Cisapride, domperidone
- Droperidol — may be safe at the low doses used by anesthesiologists (0.625 to 1.25 mg)
- Ranolazine
- HIV protease inhibitors
- Miscellaneous — Organophosphate insecticides, probucol, cocaine, terodiline, papaverine, Chinese herbs, chloral hydrate, arsenic trioxide, cesium chloride, levomethadyl

**Other**
- Mitral valve prolapse
- Myocardial ischemia or infarction
- Intracranial disease
- HIV infection
- Hypothermia
- Connective tissue diseases with anti-Ro/SSA antibodies
Wolff–Parkinson–White (WPW) syndrome

The type of pre-excitation syndrome:
- existence of an atrioventricular accessory pathway (bundle of Kent)

Atrial fibrillation (AF) with a rapid ventricular response was the most common

VT/VF may occur
Wolff–Parkinson–White syndrome

2. Wolff-Parkinson-White (preexcitation) syndrome

Impulses originate at SA node and preexcite peripheral conduction system and ventricular muscle via bundle of Kent without delay at AV node. (In type B, impulses may pass via posterior accessory bundle.)

After normal delay at AV node, impulses also arrive at ventricles via normal route to continue depolarization.

P wave is immediately followed by short delta wave, producing slurred upstroke on wide QRS with short or no PR interval.
Wolff–Parkinson–White (WPW)

A supraventricular rhythm originating in the SA node with normal & regular P-waves

**PR interval** is abnormally **short** (< 0.12 sec)

**QRS is wide** with a “slurred upstroke” (AKA the delta-wave)

Delta-waves are due to the accessory conduction pathway (bundle of Kent) from the atria to the ventricles, that bypasses the AV node

Must manifest a **tachycardia** at some point in time

Treatment: III class, Procainamide, radiofrequency ablation
WPW syndrome
Commotio cordis

• Refers to SCD that most often occurs in young athletes who have been struck in the precordium with a projectile object such as a baseball, hockey puck, or fist
• The most common arrhythmia is VF
4 rhythms that produce pulseless arrest:

• pulseless ventricular tachycardia (VT)
• ventricular fibrillation (VF)
• asystole
• pulseless electrical activity (PEA)
**Ventricular fibrillation**  There is a complete absence of properly formed QRS complexes and no obvious P waves. A recent onset (eg, within minutes) of the arrhythmia is suggested by the coarse morphology of the fibrillatory waves.
Asystole

Asystole is defined as a cardiac arrest rhythm in which there is no discernible electrical activity on the ECG monitor. Asystole is sometimes referred to as a “flat line.” Confirmation that a “flat line” is truly asystole is an important step. Ensure that asystole is not another rhythm that looks like a “flat line.” Fine VF can appear to be asystole, and a “flat line” on a monitor can be due to operator error or equipment failure.

The following are common causes of an isoelectric line that is not asystole:
1. loose or disconnected leads;
2. loss of power to the ECG monitor;
3. low signal gain on the ECG monitor.

Asystole for many patients is the result of a prolonged illness or cardiac arrest, and prognosis is very poor. Few patients will likely have a positive outcome and successful treatment of cardiac arrest with asystole will usually involve identification and correction of an underlying cause of the asystole.
## Agonal rhythm/asystole

<table>
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<tr>
<th>Parameter</th>
<th>Description</th>
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<tr>
<td>Ventricular rhythm</td>
<td>Two ventricular complexes to none</td>
</tr>
<tr>
<td>Ventricular rate</td>
<td>None</td>
</tr>
<tr>
<td>Atrial rhythm</td>
<td>None</td>
</tr>
<tr>
<td>Atrial rate</td>
<td>None</td>
</tr>
<tr>
<td>PRI:</td>
<td>None</td>
</tr>
<tr>
<td>QRS:</td>
<td>0.14 sec to none</td>
</tr>
</tbody>
</table>

![ECG waveform](image-url)
Asystole

- Ventricular rhythm: None
- Ventricular rate: None
- Atrial rhythm: None
- Atrial rate: None
- PRI: None
- QRS: None
Rhythm Strip During Episode of Sudden Death
AHA ACLS Adult Cardiac Arrest Algorithm

Shout for Help/Activate Emergency Response

Start CPR
- Give Oxygen
- Attach Monitor/Defibrillator

VT/VF

Yes

Rhythm Shockable?

No

PEA/Asystole

Shock

CPR 2 minutes/5 cycles
- Obtain IV/IO access

CPR 2 minutes/5 cycles
- Epinephrine every 3-5 min
- Consider advanced airway, capnography

Rhythm shockable?

No

Shock

CPR 2 minutes/5 cycles
- Amiodarone
- Treat reversible causes

Rhythm shockable?

No

CPR 2 minutes/5 cycles
- Obtain IV/IO access
- Epinephrine every 3-5 min
- Consider advanced airway

Rhythm shockable?

Yes

CPR 2 minutes/5 cycles
- Treat reversible causes

Rhythm shockable?

Yes

If no signs of return of spontaneous circulation (ROSC) go to

- If ROSC, go to Post-Cardiac Arrest Care

Core IV/IO Drugs Dosages:
- Epinephrine: 1 mg
- Vasopressin: 40 units
  - Can replace 1st or 2nd dose of epinephrine
- Amiodarone: 1st dose 500 mg
  2nd dose 150 mg

Go to
There are several important points that should be considered when initiating the pulseless arrest algorithm:

- High-quality CPR should be performed until the defibrillator is attached to the patient.
- Interruptions in chest compressions should be kept to a minimum.
- Rapid use of the defibrillator should be emphasized.
- If possible, use a manual defibrillator over an AED since the use of the AED can result in prolonged interruptions in chest compressions for rhythm analysis and shock administration.
Examples of cardioversion equipment

monitor

external paddles

adhesive pads
Defibrillation and the Shock

• Most defibrillators used today are biphasic. Biphasic means that the electrical current travels from one paddle to the other paddle and then back in the other direction.
• The biphasic shock also requires less energy to restore normal heart rhythm and is believed reduce skin burns and cellular damage to the heart.
• When using a biphasic defibrillator in VF and/or pulseless VT, you will use a dose of 120-200 Joules to shock.
• Start with 120J and increase the dosing in a stepwise fashion up to 200 Joules as needed.
To ensure safety during the shock

• To ensure safety during the shock, providers should always announce the following statement, “I am going to shock on three.
  – One, I’m clear…
  – Two, you’re clear…
  – Three, everybody is clear.”
Status Check

• What is the most likely rhythm disorder that might result in a patient getting a pacemaker?
  – Sinus node disease

• What are some symptoms a patient might complain of?
  – Fatigue, shortness of breath, palpitations, inability to perform activities of daily living, vertigo, syncope, racing heart at rest, slow pulse rate
What are some simple diagnostic tests used to make this diagnosis?

- 12-lead ECG, Ambulatory ECG (Holter)
Status Check
Identify the Rhythm

- Ventricular Tachycardia
- Sinus Bradycardia
- Complete Heart Block
- Atrial Fibrillation
- Ventricular Fibrillation
Status Check
Identify the Rhythm

• Ventricular Tachycardia
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