AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
1999

Cardiac Dysrhythmias II: Recognition and Treatment of Bradycardia and Conduction Blocks

This course is a continuation of Cardiac Dysrhythmias Part I and focuses on the bradyarrhythmias. The evaluation and treatment of bradyarrhythmias, conduction blocks, torsades, and drug-induced bradycardia will be reviewed, as well as problems related to technical devices, such as pacemakers and AICDs.

- Distinguish and treat proximal versus distal block.
- Recognize ischemia in the face of conduction abnormalities.
- Recognize and treat pacemaker problems.
- Recognize and treat drug-induced dysrhythmias.

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FACULTY

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Cardiac Dysrhythmias II: Recognition and Treatment of Bradycardia and Conduction Blocks

Objectives

- Distinguish and treat proximal versus distal block
- Recognize ischemia in the face of conduction abnormalities
- Recognize and treat pacemaker problems
- Recognize and treat drug-induced dysrhythmias

Outline

- Introduction and concepts
- Approach to patient with bradycardia
- Detection of AMI
- Pacemaker malfunction
- Drug-induced dysrhythmias
- Pediatric dysrhythmias

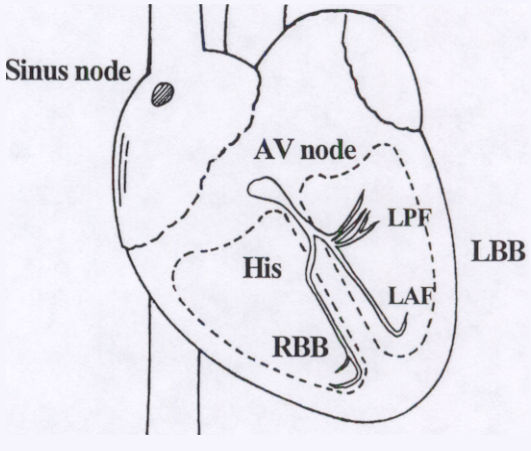
A SIMPLIFIED APPROACH TO BRADYCARDIAS

Introduction and Concepts

Normal Cardiac Conduction

In order to have a reasonable understanding of arrhythmias, and in particular the various types of bradyarrhythmias, a little basic anatomy and physiology is required, Under normal conditions, pacemaker activity of the heart comes from the sinus node, which is located in the right atrium. This is a specialized group of cells that undergoes spontaneous depolarization until threshold is reached and an impulse is generated that flows though the atrium to the AV node. In the AV node (the normal electrical connector between the atrium and ventricles), there is a slowing of conduction. From the AV node. impulses are then conducted via the bundle of HIS to the right and lefl bundle branches, and to the right and left ventricle, respectively. If an impulse goes down the normal conducting pathway, it will produce a normal or narrow QRS complex (i.e. < 0.12 msec or three small squares). Also, a normal PR interval will result (i.e. < 0.2 msec or five small squares).

If the sinus node does not work (for whatever reason) then the heart does not simply stop. Other pacemakers will take over the rhythm. In general the HIS bundle will fire at a rate of 40 to 60, followed by portions of the bundle branch further down. If the HIS is the pacemaker, there will be a narrow QRS complex. If the pacemaker is further down, the the QRS will be wide. Narrow complex rhythms tend to be more stable (i.e., asystole is rare). Wide complex bradycardic rhythms tend to be unstable (i.e., there are frequently periods of asystole).



Approach

Overview ***of the conduction system:***

- Sinus node - dominant pacemaker of the heart.
  - Baseline potential less negative than muscle tissue, undergoes spontaneous phase 4 depolarization responsible for automatic firing.
  - Slope of phase 4 depolarization depressed by vagal discharge (acetylcholine), slows firing of pacemaker cell.
  - Phase 4 potential markedly depressed (made more negative) by adenosine. may result in sinus arrest (only transient due to rapid metabolism of adenosine).
  - Sinus node firing depressed by digoxin (mediated via vagal stimulation).
  - Nodal tissue action potential mediated by calcium channels, therefore calcium antagonists result in negative chronotropy
- AV node - nodal tissue with similar physiology to sinus node.
  - Only connection from atrium to ventricles in normal hearts.
  - Subsidiary pacemaker - usually suppressed by sinus node activity.
  - Refractoriness of tissue extends past action potential - responsible for property of decremental conduction.
  - Decremental conduction - As AV node stimulated more rapidly, speed of conduction through node stows - responsible for Wenckebach physiology.
  - Effects of vagal stimulation; digoxin. calcium antagonists, adenosine similar effects on sinus node.
- His-Purkinje system -rapid, non-decremental conduction (i.e. all or none).
  - His bundle located in anterior portion of septum, divides into right and left bundles.
  - Left bundle - divides into anterior and posterior fascicles. Anterior fascicle is discrete tissue bundle, supplied by LAD. Block is common. Posterior fascicle is fan shaped, receives dual blood supply from LAD and RCA, therefore block is uncommon.
  - Right bundle - discrete, superficially located. Often transiently injured during right heart catheterization.
  - Vascular supply - related to MI
- Hierarchy of conduction system pacemakers.
  - Sinus node - spontaneous discharge 60 - 100 bpm
  - Junction - spontaneous discharge 40 - 60 bpm.
  - His-Purkinje-ventricular escape - 20 to 40 bpm.
- Blood supply of conduction system.
  - Sinus node - 60% from RCA, 40% from LCFX. (Proximal thrombosis in infarction may result in sinus arrest).
  - AV node - 90% from RCA. AV nodal block frequently seen in acute inferior MI secondary to RCA occlusion.
  - His-Purkinje system - More diffuse blood supply, 90% by LAD. Infranodal block results from more extensive infarction.

THERAPY OF BRADYCARDIC RHYTHMS

1. ABC's
2. Advanced airway and pacemaker ability to the bed side
3. Is the patient stable or unstable?
4. What is the rate- is there BP/pulse dissociation?
5. Is the QRS narrow or wide?
6. Are there P waves and how are the P waves related to the QRS complexes?
7. Could this be ischemia or drug induced?

Approach

Explanation of the Approach

1. **ABC's - you know the drill**
2. Advanced airway equipment and pacemaker ability to the bed side
   - always be prepared for profound bradycardia
   - call cardiology early-especially if the bradycardia is wide complex-they may need transvenous pacing and/or revascularization
3. Is the patient stable or unstable?
   - Treat the patient not the rate - remember that many patients can have a reasonable blood pressure but at the expense of profound vasoconstriction. The elderly patient with a rate of 40 but with a BP of 120 may still need to have her rate raised because she is ashen grey from vasoconstriction (and her central blood supply is also constricted to maintain her BP).
4. What is the rate?
   - Is there "rate/BP dissociation". If your patients blood pressure is 60/p and the rate is 55 then something else is going on (eg. a big Ml or drug overdose!).
5. Are the QRS complexes wide or narrow?

Determining if the QRS complexes are wide or narrow has enormous diagnostic and therapeutic implications/l

	Narrow QRS	***Wide QRS***
Level of block	AV node or high HIS	Below AV node
Response to atropine	Frequently responsive	Usually not responsive
"Stability" (i.e. propensity for profound bradycardia)	Generally stable	Generally unstable

6. Are there P waves and how are they related to the QRS?
   - The association of P waves to QRS complex is key to the correct diagnosis. If there are no P waves then there is either failure of the SA node or very fine atrial fibrillation seen as a flat base line.
   - If there are P waves are there more P waves than QRS complexes. If there is then there is a block at the AV node!
   - See the following summary of diagnoses for further diagnostic information
7. Could this be ischemia or drug toxicity?
   - If it is possibly ischemia give the patient aspirin and get a 12 lead ECG stat! Call cardiology.
   - If it is possibly drug toxicity treat supportively but now also consider specific therapy (see 2nd section by Dr. PACE)

TREATMENT FIRST -Diagnosis Second

- In the symptomatic patient then treat the rate first and worry about the exact diagnosis second. This is true since all symptomatic bradycardia may reasonably be treated initially the same way.

- In the simplest form one can think of treatment in the following way

  i. Try low dose incremental atropine
  ii. Try transcutaneous pacing
  iii. Try low dose epinephrine
  iv. Place a transvenous pacer

Approach

i. *Try low* dose incremental *atropine* narrow complex bradycardias are frequently responsive to atropine, wide complex less often. In reality, in the heat of battle, atropine is frequently tried first in all patients, this is defensible if done correctly.
   - Atropine 0.25mg IV and repeat up to 3 mg if required. Start with low doses to prevent a tachycardia exacerbating any ischemia which may be present.

Atropine may make a wide complex bradycardia worse, by increasing the block-so use with caution and prepare for an alternate intervention.

   - Paradoxical slowing of rate due to atropine has probably been way overstated. 0.25 mg is a low but acceptable starting dose.

ii. *Try transcutaneous pacing* - ACLS suggests transcutaneous pacing is a Class I intervention (ie. definitely helpful). Personally, I'm not convinced. Many patients fail to capture, assessment is difficult due to patient movement, and many have difficulty tolerating the shocks.
   - Transcutaneous pacing is painful and sedation is required for the majority of patients. Capture can be difficult in obese patients, COPD etc.
   - To assess capture, simply feel for the pulse but this can be difficult in a patient whose body jolts with pacer tiring. Reading capture off the monitor can also be difficult. Be sure to use the monitor with the pacer and take advantage of the filtering. Checking pulse, blood pressure, and LOC are clearly the best endpoints to evaluate efficacy, **Don't shock yourself on the pacer pads!**
   - Set the rate you want, and increase the energy output until capture occurs.
   - AP placement of the pads may be the most effective. Apex-base alignment is also acceptable.

iii. *Try low dose epinephrine* - Epinephrine may help patients that have failed atropine or pacing but use with care as this is powerful stuff. An epinephrine is best..
   - Put 1 mg of epinephrine (eg 1 ml of 1:1,000) into 250 mls of D5W (this results in a solution with 4 micrograms per ml) and run at 1 to 4 ml per minute (i.e. 4 -16 microgram minute) and titrate up as required.
   - Alternatively, if the patient is *about to arrest* and there is no time to set up an infusion:
     . Take a syringe of **1:10,000** epinephrine and give 0.25 mls (1/4 ml) very slowly. This is a high dose and may result in marked hypertension or tachycardia. ***Check the blood pressure and pulse frequently.***

iv. *Try transvenous pacing-Approaching* from the right external jugular using a J-wire to access the internal jugular and placing a balloon directed catheter tip at the base of the right ventricle under fluoroscopic guidance is the best way to accomplish this task. Other methods include electrocardiographic guidance. and blind placement, simply looking for ventricular capture on the ECG.

Approach

Diagnosis of Bradyarrhythmias

For bradyarrhythmias there are basically two broad groups of problems:

1) Failure of the sinus node
2) Failure of the AV node

Disorders of the Sinus Node

The normal sinus rate is frequently stated to be between 60 and 100. In reality normal rates well below 60 occur in healthy and fit people. Sinus node dysfunction occurs as a result of failure of impulse formation or propagation. This is manifest by a reduced rate, absence of sinus activity or episodes of fast and slow rates.

Pathological sinus bradycardia or arrest occurs with:

- aging
- hypothermia
- hypoxia
- acidosis
- drugs

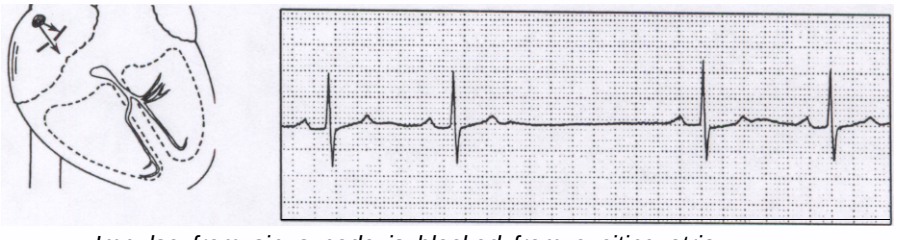
In certain patients, sinus node dysfunction is only manifest when they are taking certain cardioactive drugs (eg. digoxin, beta-blockers, calcium channel blockers). Treatment involves withdrawing slowing agents,

Sinus node exit block occurs when the sinus node fires but the impulse does not propagate into the atria.

There are three types of sinus node exit block: 1st, 2nd, and 3rd degree.

- 1st degree block is a delay from sinus node tiring to atrial capture and cannot be detected on an ECG, so don't worry about it.
- 2nd degree is an intermittent failure of propagation of sinus impulses, manifested as intermittent loss of P waves.
- 3rd degree sinus block is a dissociation between sinus node firing and atrial escape. This can not be read on a surface ECG.

exit block



Impulse from sinus node is blocked from exciting atria.

The brady-tachy syndrome is manifested as intermittent episodes of bradycardia and atrial tachyarrhythmias. The tachyrhythmias are frequently followed by a profound bradycardia that manifests as syncope.

Treatment for sinus node dysfunction

- These arrhythmias rarely need treatment in the ED. In general, symptomatic patients require consultation for permanent pacemaker placement.
- If the bradycardia is symptomatic in the ED, then atropine or pacing maybe required.

Approach

Disorders** of **the AV Node

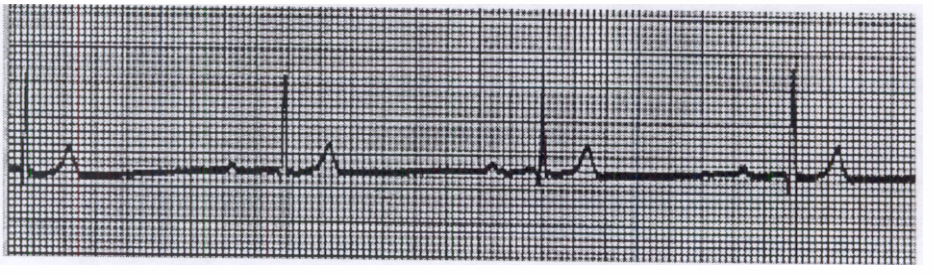
Bradycardia can occur as a result of dysfunction of the AV node.
AV node blockade is divided into three types:

1st degree AV block:

Better called "delayed AV conduction." This is manifest as a prolonged PR interval (>0.2 sec or five small squares). There is 1 :1 AV conduction and consistent PP and RR intervals.

. When the ***QRS is narrow*** the block is at the level of the AV node.

Example: 1st degree AV block



Causes and Treatment:

In general no treatment is required.

- Causes include: electrolyte disturbances, drugs (e.g. digoxin, beta-blockers, calcium channel blockers), inferior infarction. Treat the cause.
- In an MI patient, observation alone is generally enough.

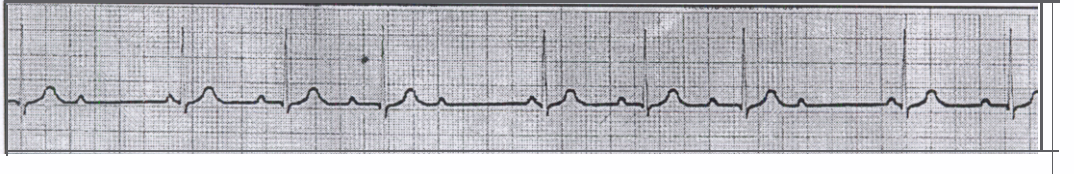
2nd Degree AV block:

There are two types of 2nd degree AV block: **Morbitz** Type I and II

Type I 2nd Degree AV block:

a progressive lengthening of the PR interval until there is failure of conduction. This is manifest by an increasing P-R interval and then failure of the P to be followed by a QRS complex. The cycle then repeats. Additionally, the R-R interval shortens as the P-R interval lengthens. *Usually the **block is in the AV node only so the QRS complex** is **narrow.*** This was described by Wenckebach without the aid of an ECG!! The man had skills!

Example: ***Type I 2nd Degree AV block:***



Causes and *Treatment:*

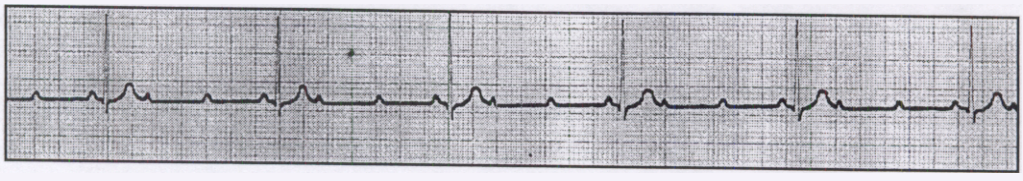
Generally *this is a benign condition that requires no treatment and resolves spontaneously.* It can occur in inferior MI which is usually transient and requires no treatment.

Approach

Type II 2nd degree AV block:

Conduction from the atrium to the ventricles ***fails suddenly*** and unexpectedly *without a preceding change in the PR interval.* It is usually due to a problem in the HIS-Purkinje system and therefore is *associated with a wide QRS.* This block has a high incidence of progression to AV block.

Example: *Type II 2nd degree AV block (narrow complex)*



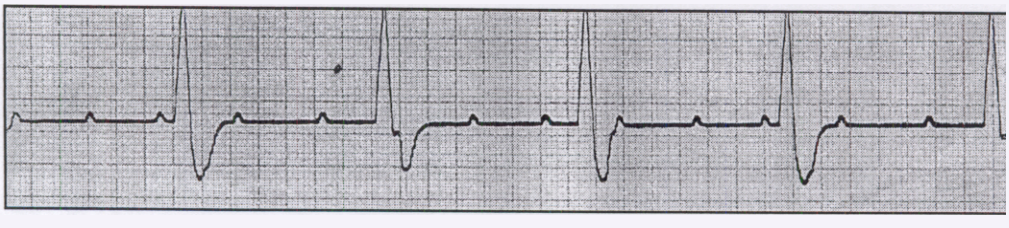
Causes and Treatment:
Anteroseptal infarction, degenerative disorders. In high degree block, two or more consecutive P waves are not conducted. The escape rhythm is usually slow and unstable requiring pacemaker insertion.

Third Degree A V block:
when no P waves propagate to the ventricles, third degree AV block is present. There are two types, a narrow and wide complex form.

- *The narrow form of third degree block* occurs from failure of the AV node, This usually occurs in the face of an inferior MI and is transient. It should be treated in the same manner as 2nd degree type I block. In the hemodynamically unstable patient then atropine or transcutaneous pacing is the best therapy.

- *The wide complex form* occurs due to a block lower than the AV node and is more serious. The pacemaker lies low in the Purkinje system or ventricle and is unstable. This block is caused by extensive disease of the conducting system *(for example an anterior MI).* **Treatment is mandatory pacemaker placement in the ED temporizing with a transcutaneous pacer and/or epinephrine until the equipment and skill is available to place a transvenous pacer.**

Example: ***Third Degree AV block***



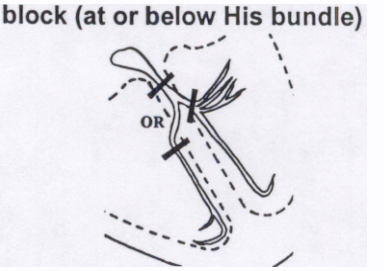
Generally wide complex 3rd degree block is atropine resistant

- If the ***QRS is wide*** the block is generally in the HIS bundle or Purkinje system.

Approach

SITE OF BLOCK: AV NODE

SITE OF BLOCK: Infranodal block (at or below His bundle)



AV block at the AV node - EKG and EP correlates.
Baseline QRS is usually normal duration,
Escape rhythm originates from junction or proximal HP system; therefore QRS is narrow with rate of 40 to 60 BPM.
May respond to atropine; junctional rate may accelerate,

Infranodal block - EKG and EP correlates,
Baseline EKG often shows BBB. bi- or trifascicular block, or alternating BBB's.
Escape rhythm originates from distal conduction system or ventricle; therefore escape complexes are wide. rates 20 to 40 BPM. Usually does not respond to atropine.
Treatment usually requires permanent pacing.

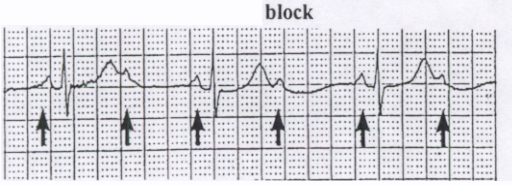
AV Conduction block - ECG and anatomic correlates.
First degree - PR prolongation only, usual site of delay is at AV node. Rarely of significance unless profound.
Type I 2^0AV block - usual site of block is at AV node. Features are result of decremental conduction.
Atrial rate fairly constant, Prolongation of PR interval, culminates in dropped QRS. Next PR after dropped QRS is short. Atypical appearance of Type 1 block is more common than typical Wenckebach block.
Type II 2^0 AV block - usual site of block is below the AV node (infra nodal block).
PR interval remains constant; P wave unexpectedly fails to excite QRS.
As block is in HP system, indicates instability of conduction system and potential progression to intermittent or permanent AV block.
2:1 Second degree AV block - cannot with certainty determine mechanism of block (nodal or infranodal) without EP study.
Advanced or high grade AV block - Intermittent AV block with 2 or more blocked P waves in succession.
Complete or third degree heart block - complete block of AV conduction, patient dependent on subsidiary escape pacemaker for ventricular systole.

2:1 AV block

Favors block @ node *Favors block below node*

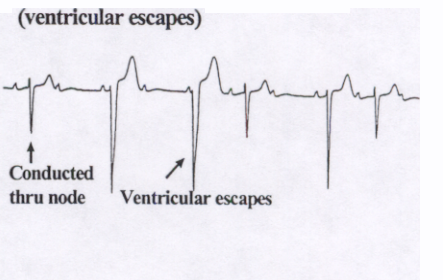
Normal QRS width
No fascicular or bundle branch block

Wide QRS - (IVCD)
LBBB
Bi or trifascicular block



HIGH GRADE AV BLOCK

Intermittent block below node
(ventricular escapes)



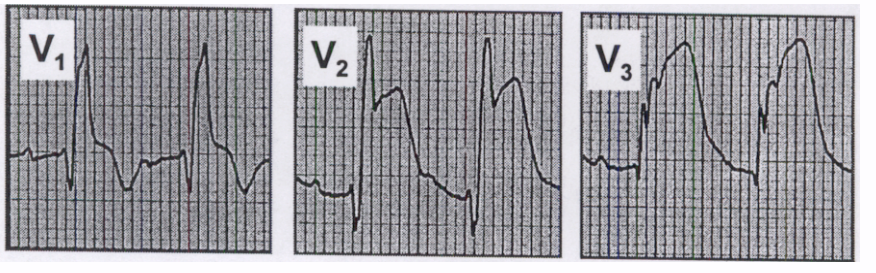
Approach

Detection **of Myocardial Ischemia**

AMI and RBBB

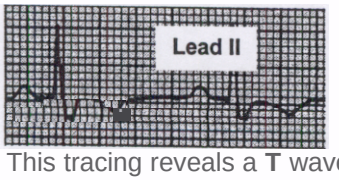
Initial QRS forces are not altered in RBBB and q waves can be seen.
Except true posterior AMI can not be seen due to positive forces VI - V3.
In complete BBB the T wave is directed opposite to main terminal deflection of the QRS complex.

Case 1
60 yo male with 2 hours of CP given tPA - 95% LAD lesion requiring emergency revascularization.



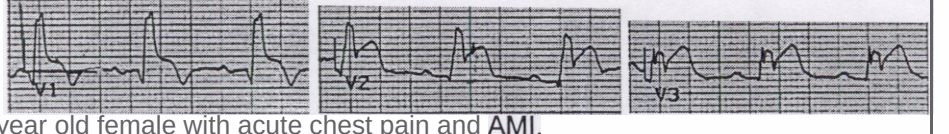
This tracing reveals obvious ST segment elevations and T wave orientation concordant with QRS.

Case 2



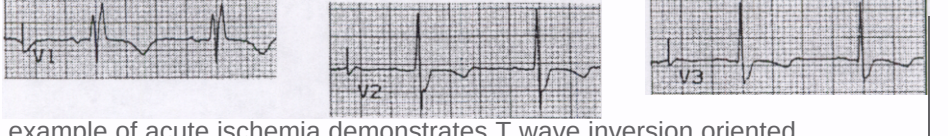
This tracing reveals a **T** wave orientation concordant with the QRS complex in limb lead 2 and represents ECG evidence of myocardial ischemia in the presence of RBBB.

Case 3



70 year old female with acute chest pain and AMI.
A little more subtle example of ST segment elevations and T wave orientation concordant with QRS.

Case 4



This example of acute ischemia demonstrates T wave inversion oriented concordant with the terminal, main deflection of the QRS complex.

AMI and LBBB

Q wave detection unreliable.

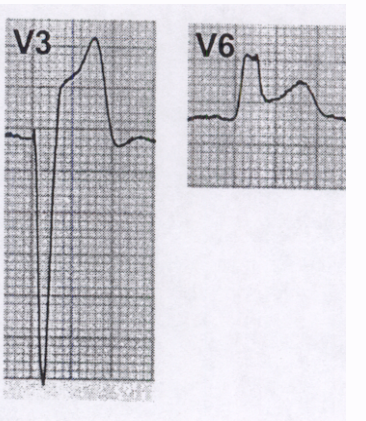
From GUSTO-I *(Sgarbossa, NEJM 1996)*

ST elevation > 1 mm concordant with QRS
ST depression > 1 mm V1, V2, V3
ST elevation > 5 mm discordant with QRS

Several recent studies (Li, Acad Emerg Med 1999; Ozment, Acad Emerg Med 1999; Shlipak. JAMA 1999) have challenged the utility of these findings essentially concluding that Sgarbossa's criteria are specific but not sensitive for the diagnosis of AMI. The emergency physician may therefore feel confident about the use of lytics for AMI when Sgarbossa's criteria are met but these findings may be present in only 15% or less of patients with AMI and LBBB.

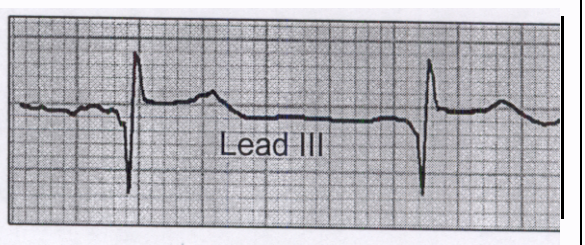
Case

73 year old male presents with three hours of chest pain **and** develops congestive heart failure. He rules in for AMI with CK-MB = 495 ng/ml. ECG to right shows ST elevation > 1 mm concordant with QRS in lead V_6 and ST elevation > 5 mm discordant with QRS in lead V_3.



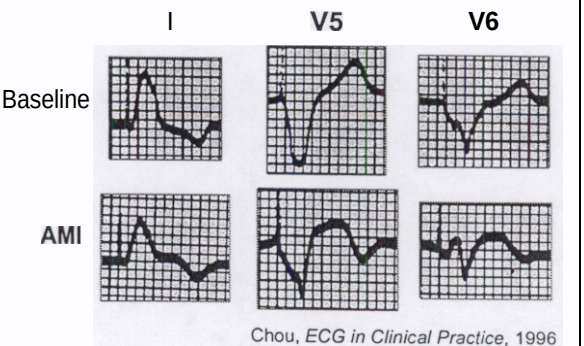
AMI and preexcitation

Significant ST segment changes concordant with main QRS deflection suggest ischemia. Q waves are unreliable because accessory bundle delta wave alters initial QRS deflection. Look for delta slurring and short PR to aid in diagnosis.



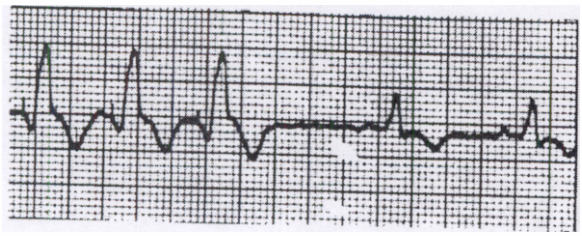
AMI and ventricular pacemaker

QRS complex is not helpful unless spontaneous beats are present and even then repolarization of native complexes may be altered due to presence and previous activity of pacemaker. *One* must rely on additional primary ST-T changes (few) as in LBBB.

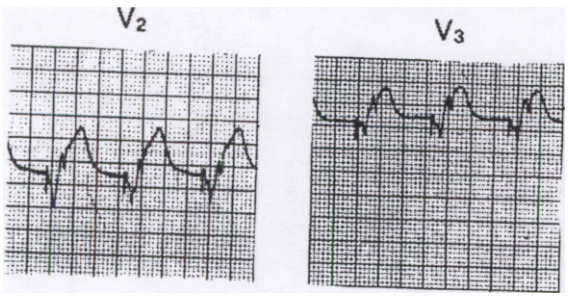


Detection of AMI

AMI and ventricular pacemaker (3 examples form Barold Card *Clinics*)
Lead aVL reveals "native beats which appear ischemic. This finding is suggestive but not diagnostic and repolarization patterns in native beats may be abnormal simply due to "pacemaker induced memory."



Anterior precordial leads demostrate marked ST segment elevations.



Acute inferior wall myocardial infarction

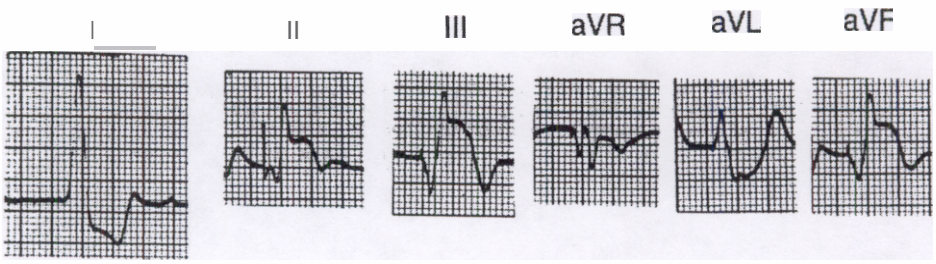
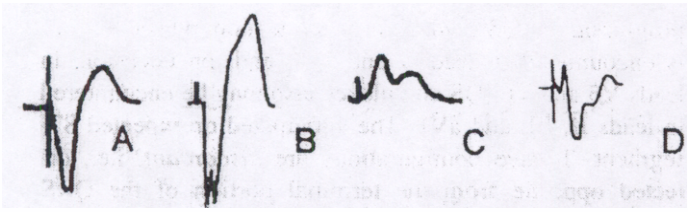


Diagram (from Kozlowski FH, et. al. *Acad Emerg Med 1998)* summarizing findings consistent with AMI in the presence of a ventricular pacemaker.



A - normal expected configuration
B -discordant ST segment elevation > 5mm
C – concorcant ST segment elevation > 1 mm
D – ST seqment depression > 1 mm in VI, V2, or V3

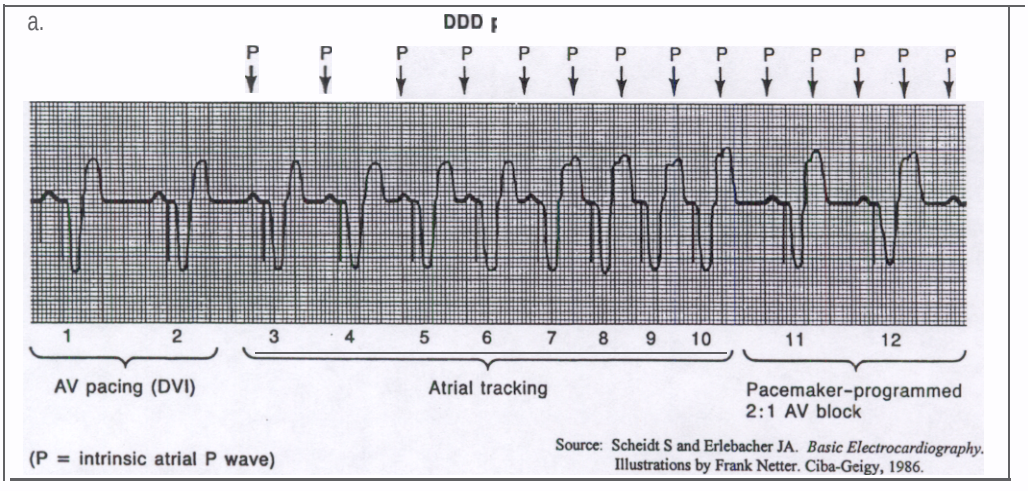
Detection of AMI

Pacemaker Malfunction

Pacing Modes

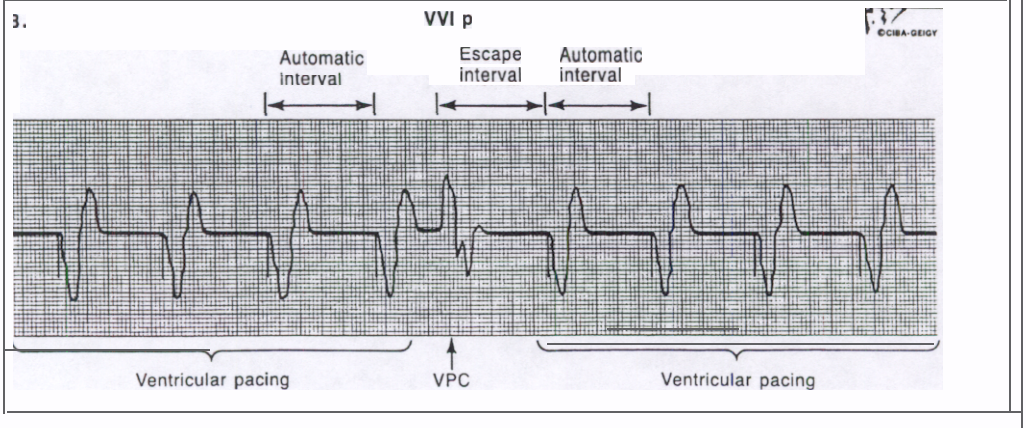
- DDD pacing is most commonly used today. Dual chamber pacing and sensing allows versatility and the advantage of preserved atrial contribution to ventricular filling. Exercise responsiveness is maintained if sinus node function is preserved and sinus rate increases with exercise. Atrio-ventricular synchrony is maintained thus avoiding pacemaker syndrome (see below). Dual chamber pacemakers are more complex than single chamber pacemakers and battery life may be shorter. Dual chamber devices may initiate a pacemaker mediated tachycardia and they are susceptible to cross-talk between chambers.

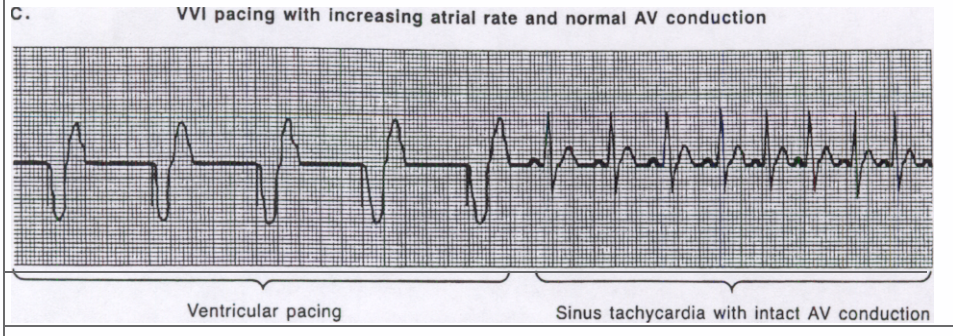
DDD Pacing

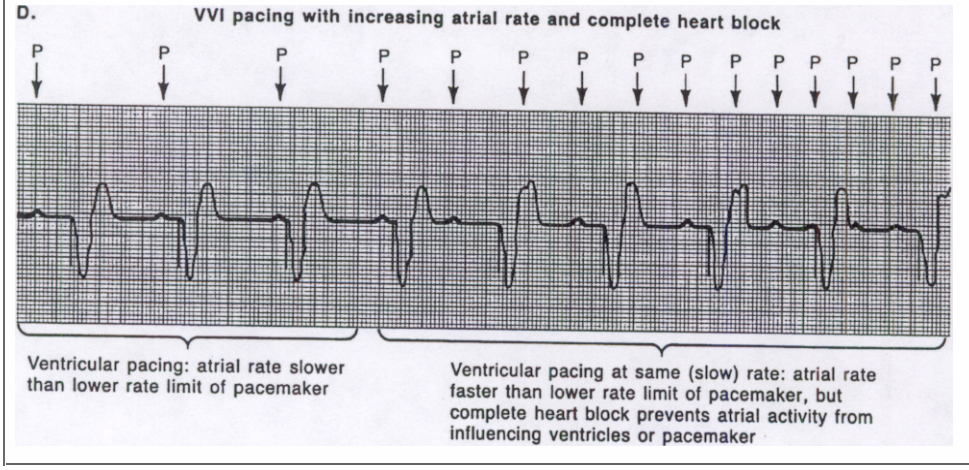


- WI pacing is another common mode for implanted pacemakers. Single chambe sensing and pacing reduces complexity, cost, frequency of follow-up and prolongs battery life. In patients with sick sinus syndrome, WI pacemakers are more often associated with atrial fibrillation, stroke, and overall mortality when compared to DDD mode. Patients with preserved atrial function may experience pacemaker syndrome as a result of atrial contraction against closed tricuspid and mitral valves.

WI Pacing







Source: Scheidt S and Erlebacher JA. *Basic Electrocardiography.* Illustrations by Frank Netter. Ciba-Geigy, 1986.

- Rate-Adaptive pacemakers (DDDR, WIR. DDIR, AAIR) allow for variable rate pacemaker output according to physiologic demands of the patient. This allows for exertional adjustments of the heart rate. Various types of sensors respond to physical, electrical, or chemical stimuli. Most commonly body motion is sensed which triggers increased rate of pacer impulses. Exercise changes in the QT interval, changes in respiratory rate, or even temperature of venous blood are stimuli for various sensors.

Standardized Pacemaker Nomenclature

I	II	III	IV	V
Chamber Paced	Chamber Sensed	Response to Sensing	Rate Modulation, Programmability	Antitachycardia Features
0 = none	0 = none	0 = none	0 = none	0 = none
A = atrium	A = atrium	I = inhibited	S = simple programmable	P = antitachycardia pacing
V = ventricle	V = ventricle	T = triggered	M = multi-programmable	S = shock
D = dual	D = dual	D = dual	C=communicating	D = dual
			R=rate modulation	

Pacemaker Complications -Malfunction

- Failure to pace or provide output.
  Component failure (rare), total battery depletion, lead fracture, lead disconnection, and oversensing may all result in failure of pacer output. Lead migration can result in lead fracture or even migration into the left ventricle.
- Failure to sense.
  Undersensing may be caused by lead dislodgment. poor lead position, lead insulation defect, or low-amplitude cardiac signal.
- Failure to capture.
  Lead dislodgment, lead insulation break, exit block, metabolic derangement, and battery depletion may result in failure to capture.
- Pacemaker Induced Dysrhythmia:
  Runaway Pacemaker is the result of inappropriate, rapid discharge of the device. Lead disconnection may be necessary in extreme circumstances. Older models will sometimes develop this complication with battery depletion.
  Pacemaker Mediated Tachycardia is a problem peculiar to dual chamber devices where a re-entrant tachycardia results for a retrograde P-wave being sensed by the pacemaker as a physiologic atrial depolarization. Atrial depolarization is tracked by ventricular pacing and an "endless loop" or re-entrant tachycardia occurs. This problem is often initiated by a PVC (see appendix). Emergent therapy requires magnet placement over the device to inhibit sensing and disrupt the cycle. Later, the device can be re-programmed to increase the ventricular refractory period.

Pacemaker Patient Evaluation

Standard patient evaluation includes assessment of hemodynamic status (including mental capacity and vital signs), cardiopulmonary examination, electrocardiography, electrolytes, and appropriate drug levels.

Pacemaker Malfunction - Unstable Patients

Unstable patients must have hemodynamic stability restored. *Bradycardic patients* may be treated with atropine. transcutaneous or transvenous pacing. If a problem with over-sensing is suspected (bradycardic patient with pacemaker that is not firing) magnet placement over the device will inhibit all sensing and convert pacemaker to VOO, AOO, or DOO function.

Unstable Patient

- Bradycardia
  - atropine
  - transcutaneous pacing
  - transvenous pacing
  - magnet placement

Tachycardic patients may be cardioverted or defibrillated as usual with special care to avoid paddle placement over the pacemaker metal box. Realize that external cardioversion or defibrillation may render an implanted pacemaker dysfunctional. *Pacemaker mediated tachycardia* is a re-entrant tachycardia seen with dual chamber pacemakers that may be treated with external magnet placement which inhibits pacemaker sensing and disrupts the tachycardia.

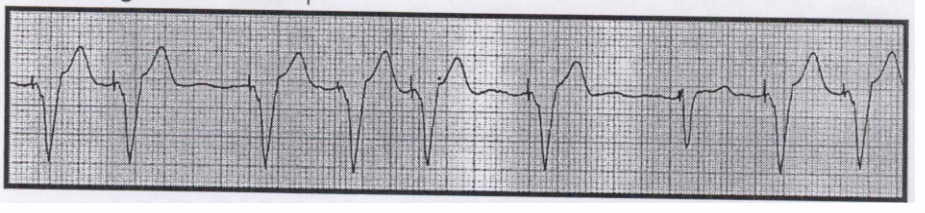
Unstable Patient

- Tachycardia
  - cardioversion/defibrillation paddle placement must avoid generator box or risk pacemaker dysfunction
  - pacemaker mediated tachycardia may be treated with magnet placement

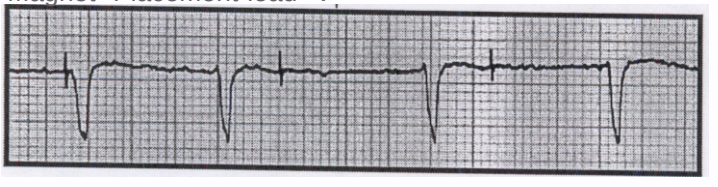
Pacer Malfunction Case Study

- 56 year old male with CAD and **DDD** pacer placed ‘or **brady/asystole** presents with palpitations and **mild dyspnea**
- **Meds include amiodarone, atenolol**
- **On exam he appears stable without chest pain or CHF**

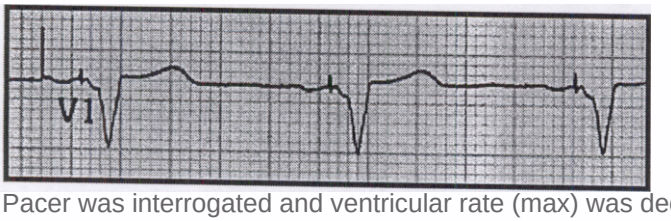
Presenting ECG – lead V_1



Magnet Placement-lead V_1



Atrial fibrillation resolved -lead V_1

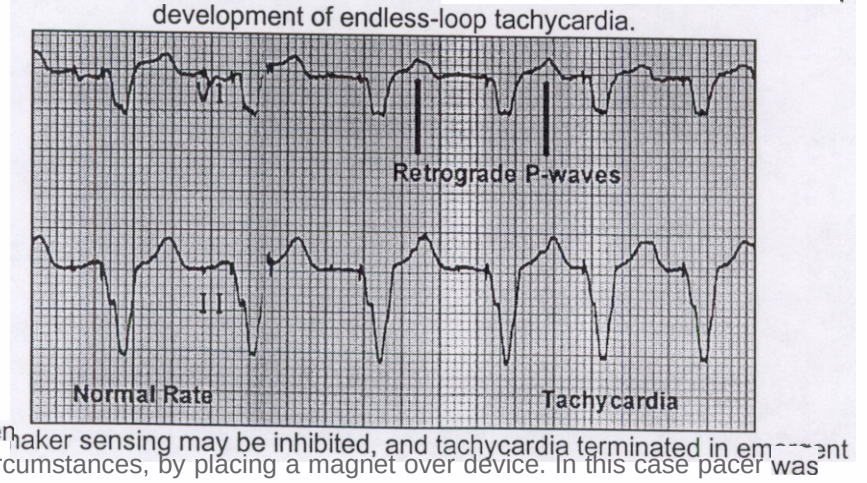


Pacer was interrogated and ventricular rate (max) was decreased

Pacemaker Mediated Tachycardia -Case History

58 year old male with history of coronary heart disease and recent palpitations with exercise. Tracing below was recorded during Bruce treadmill protocol,

Retrograde P-wave triggers ventricular firing, another retrograde P-wave, and development of endless-loop tachycardia.



Pacemaker sensing may be inhibited, and tachycardia terminated in emergent circumstances, by placing a magnet over device. In this case pacer was reprogrammed

Notes

Pacemaker Malfunction - Stable Patients

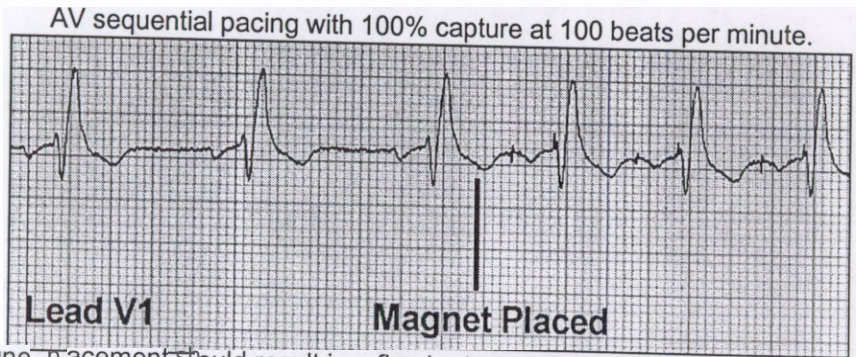
Stable patients should undergo the standard evaluation described above and will often require pacemaker interrogation by a cardiologist or technician. Telephonic interrogation is possible. Electrocardiography (with and without magnet placement) may help the emergency physician diagnose some causes of dysfunction. Sensing and capture may be evaluated if pacer spikes are seen on the ECG without magnet placement. A left bundle branch block with left axis deviation would be the normal appearance of a pacer lead in the right ventricle. Another axis may indicate lead migration away from cardiac apex and a right bundle branch block could indicate a left ventricular location. Magnet placement should result in a fixed rate pacer discharge where capture may be assessed. Magnet rates also help diagnose battery failures (according to manufacturer charts). Patients with significant presenting Symptoms (e.g., syncope) will need to be admitted until the pacemaker can be interrogated and the cause elucidated. ECG evaluation of ischemia is complicated by the typical LBBB configuration (with a superior QRS axis due to typical RV apex location of pacer lead) and even if intrinsic beats are present ST segments and T waves may be abnormal due to activity of pacemaker and myocardial "memory."

Stable Patient

- Pacemaker Interrogation
  - -cardiologist
  - -technician
  - -telephonic
- Admit **patients with significant symptoms (e.g. syncope) until PMK is interrogated and cause established.**

Pacemaker Testing with Magnet Placement
Case History

A 65 year old female with a DDD pacemaker presents with 4 days of intermittent light-headedness and feeling "woozy." Physical examination is unremarkable. 12-lead ECG reveals a normal sinus rhythm with right bundle branch block and left anterior fasicular block. Pacemaker magnet test during ECG monitoring reveals:



Magnet placement should result in a fixed rate pacer discharge where capture may be assessed. Magnet rates may diagnose battery failures (according to manufacturer charts).

Pacemaker Special Considerations

- **electrocautery**
- **transthoracic defibrillation**
- **magnetic resonance imaging**
- **therapeutic radiation**
- **non-medical devices**
  - **electric motors and arc welding**
  - **cell phones and security devices**

- *Electrocautery* may cause temporary sensing problems or reprogramming. Afler electrocautery is used in the vicinity of a pacemaker it should be checked for proper functioning.
- *Transthoracic* defibrillation may damage a pacemaker. Paddles should be placed away from the impulse generator.
- Magnetic *resonance imaging* will disable pacemaker sensing temporarily and may induce dysrhythmias. Patients with artificial pacemakers should not undergo MRI. Some pacemakers may be temporarily reprogrammed such that patients may safely undergo the procedure.
- *Therapeutic radiation* may cause damage to silicone and silicone oxide insulators within electronic circuit boards that renders pacemakers dysfunctional.
- *Non-medical equipment and devices* (e.g., heavy electric motors, arc. welding) that emit magnetic fields may temporarily disable pacemakers. Cellular phones and airport security have recently been implicated in occasional device malfunction.

Magnet Therapy

Unstable Patients

- Inappropriate, recurrent ICD Discharges
- Pacemaker-medicated tachycardia
- Symptomatic bradycardia in patient with implanted pacemaker that is not firing

Stable Patient

Patient with implanted pacemaker – ECG with and without magnet present.

Complications

- may shut off the old ICD's

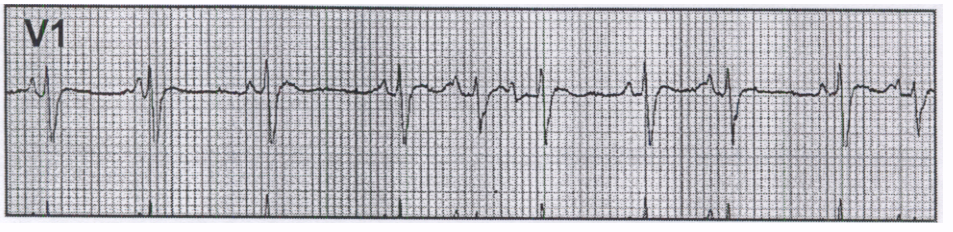
possible Ron T resulting in ventricular tachydysrhythmia (theoretical)
inhibiting appropriate ICD discharges
inhibiting appropriate ICD discharges

Pacemaker **Malfunction**

Drug Induced Dysrhythmias

Case 1 - Digitalis Toxicity

81 yo female with COPD - rhythm misdiagnosed as atrial fibrillation and patient give 0.5 mg of digoxin but level returns at 3.5 ng/ml. Rhythm is multifocal atrial tachycardia and represents a pitfall in caring for patients using digoxin.

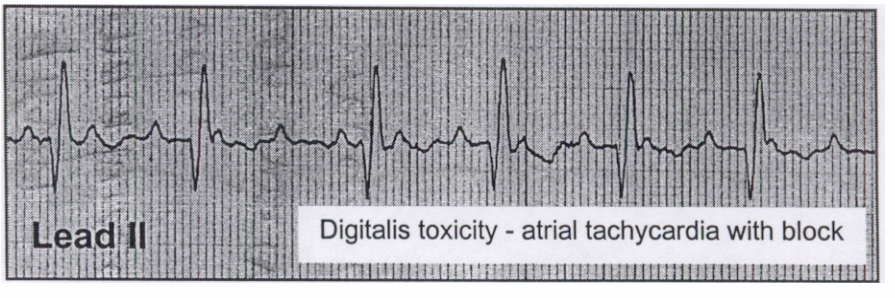


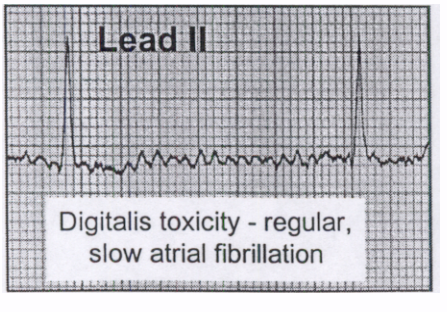
Digitalis effects on the ECG

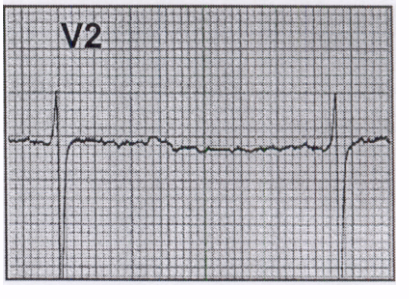
- ST segment depression
- Decreased amplitude and changes in direction of T wave
- Shortening of QT interval
- Increase U wave amplitude
- Atrial and ventricular ectopy
- Sinus brady and AV nodal blockade

Digitalis Toxic Rhythms

- Ventricular fibrillation
- Ventricular tachycardia and PVC's
- Severe bradycardia and 3^0 AV block
- Specific rhythms
  - Atrial tachycardia with block
  - accelerated junctional tachycardia
  - slow, "regular" atrial fibrillation
- Atrial fibrillation



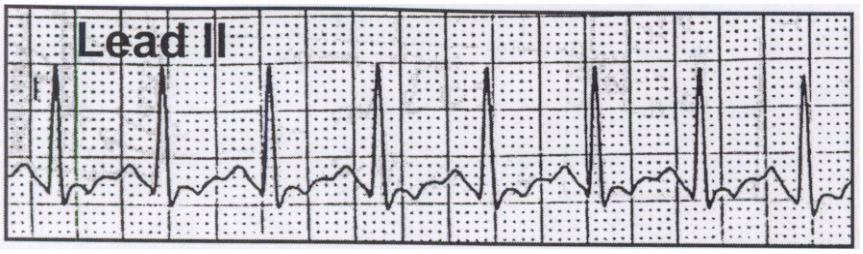


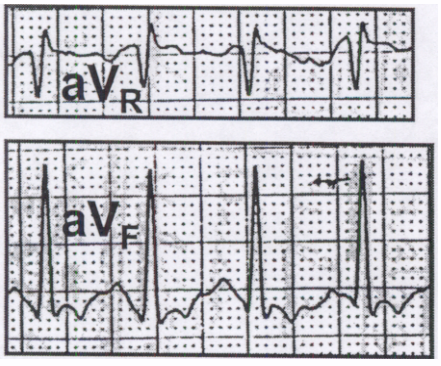


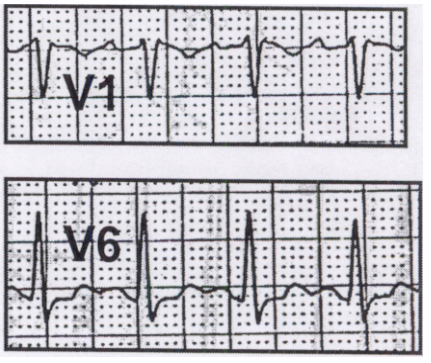
Therapy includes atropine, lidocaine, temporary pacing, and digoxin immune Fab fragments for ventricular tachycardia, ventricular fibrillation, hypotensive bradycardia, hypotensive bradycardia, or if unresponsive to conventional therapy, and level > 1ng/ml post-distribution,

Drug Induced Dysrhythmia

Case 2 -Cyclic antidepressant toxicity
14 yo female 3 hours ago attempted suicide with 1 gram amitryptiline and presents combative with episodes of apnea and intubated by paramedics





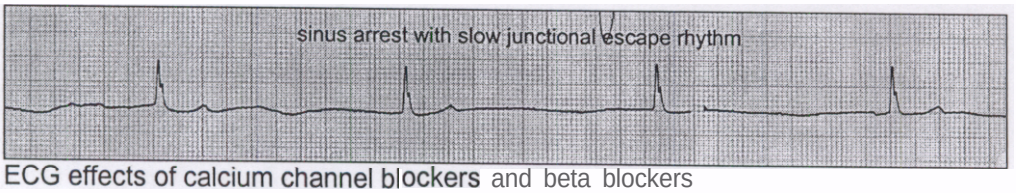


ECG Effects
- tachycardia
- QRS prolongation
- QT prolongatiion
- rightward terminal 40 ms in frontal plane

Therapy
- Alkalinization
- sodium bicarbonate
- hyperventilation
- lidocaine
- Fab fragments

Toxic Rhythms
- Ventricular fibrillation
- Ventricular tachycardia
- Sinus tachycardia

Case 3 - Calcium **channel blocker toxicity**
36 year old depressed male intentionally overdoses with atenolol and diltiazem in large quantities. He presents somulent, HR=30, and BP=68mmHg. ECG reveals:



ECG effects of calcium channel blockers and beta blockers
- sinus node depression (except nifedipine)
- prolongs AV node conduction

Cardiovascular effects of calcium channel blockers and beta blockers
- myocardial depression
- negative inotropy
- peripheral vasomotor depression

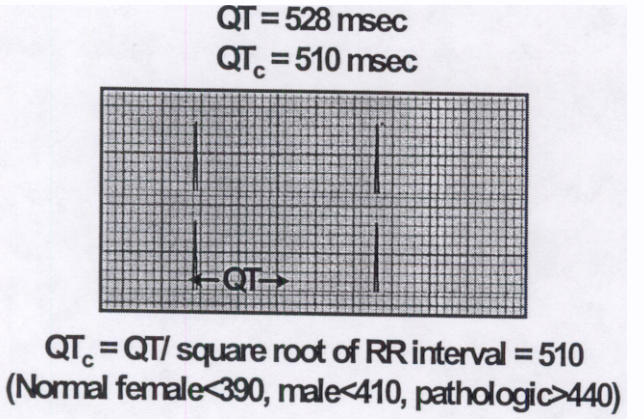
Therapy includes
- calcium chloride or calcium gluconate
- glucagon and epinephrine
- isoproterenol (for beta blockers) and temporary pacing

Drug Induced
Dysrhythmia

Prolonged QT Syndrome

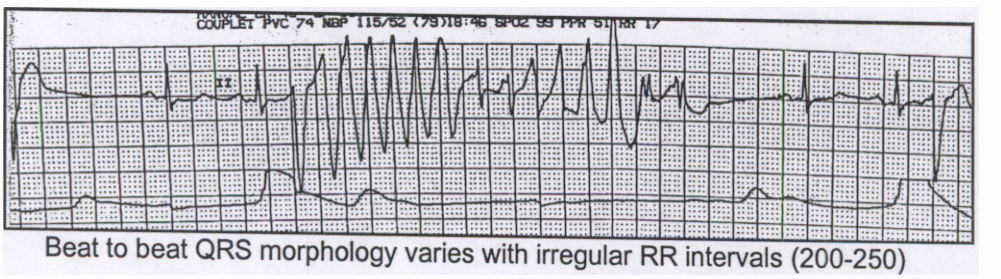
Case 1 - Syncope and seizure

22 yo female soldier with palpitations followed by syncope and tonic muscle contractions. Examination is normal. The ECG tracing demonstrates a prolonged QT interval. Further interviewing reveals the presence of an eating disorder and recent use of a high protein liquid diet. She is hospitalized and has no further symptoms. Eating disorder is treated and ECG QT interval normalizes.



Case 2 - Torsades de pointes

60 yo male with CAD and ventricular dysrhythmias uses procainamide and presents with syncope.



Differential diagnosis

- Polymorphic ventricular tachycardia
- WPW with atrial fibrillation
- rapid atrial fibrillation with BBB

Prolonged QT Syndrome - acquired causes

Drugs	Electrolytes	Intrinsic Cardiac
Antiarrhythmics	Hypokalemia	Myocarditis
Phenothiazines	Hypomagnesemia	Ischemia
Organophosphates	Hypocalcemia	Severe CHF
Liquid protein diets		Mitral valve prolapse
Organophosphates		
Tricyclic ntidepressants		

Therapy

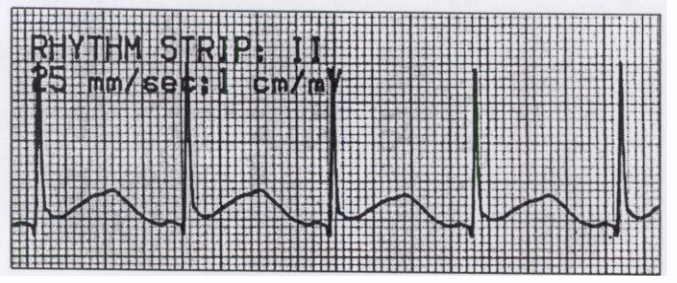
- Cardioversion
- Adrenergic stimulation and overdrive pacing
- Lidocaine
- Amiodarone

Prolonged QT

Case 3 -Adolescent seizure

18 yo male with first Seizure witnessed by father to be flaccid for 3-4 minutes with eyes deviated up and urinary incontinence. Mother and aunt both died of seizures in 30's and cousin has seizures. Returns to ED next day with recurrence of second seizure. Earlier had seen physician and referred to cardiologist for abnormal electrocardiogram. Admitted to hospital for observation.

Echocardiogram, GXT, and EEG are normal. Cousin has implantable cardioverter-defibrillator. 15 yo sister 2 months later was wearing event monitor showing ventricular tachycardia during a "seizure."



Diagnosis is Romano Ward Syndrome

Prolonged QT syndrome - congenital causes

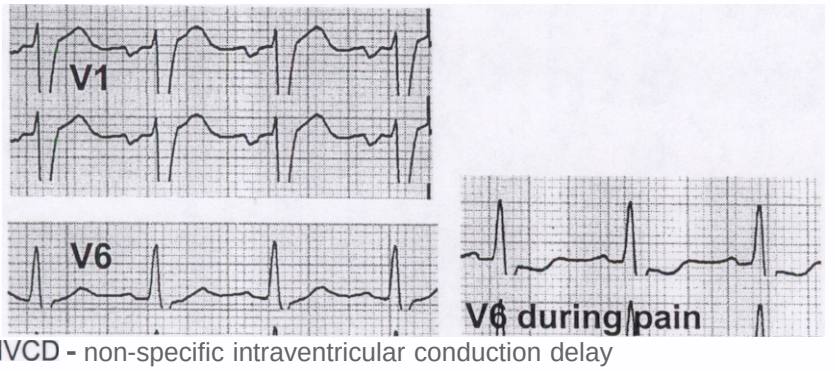
Syndromes	Mechanism
Lange-Dervell-Nielson	Imbalanced sympathetic tone
Romano-Ward Syndrome	Alteration of ion channels resulting in beta receptor variations

Therapy

- Cardioversion
- Beta blockade
- Magnesium
- Sympathectomy
- Implanted cardioverter-defibrillater

Case - Intraventricular conduction delay (IVCD)

71 yo male with diffuse CAD and angina



IVCD - non-specific intraventricular conduction delay

- Block occurs in distal segments of ventricular conduction tissue/muscle
- Due to drugs, ischemia, and fibrosis
- QRS > 100 ms and morphology not consistent with bundle branch block

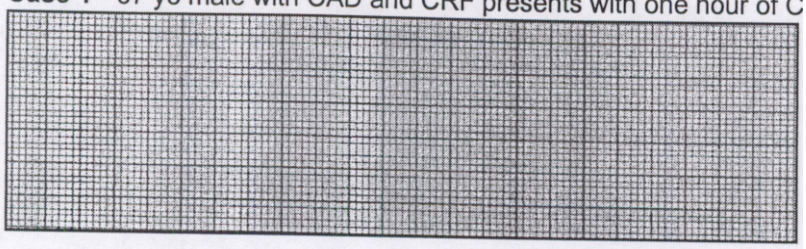
Non-specific intraventricular conduction delay - drug causes

- Cyclic antidepressant toxicity
- Hyperkalemia
- Type 1A antidysrhythmics
- Phenothiazines
- Antihistamines

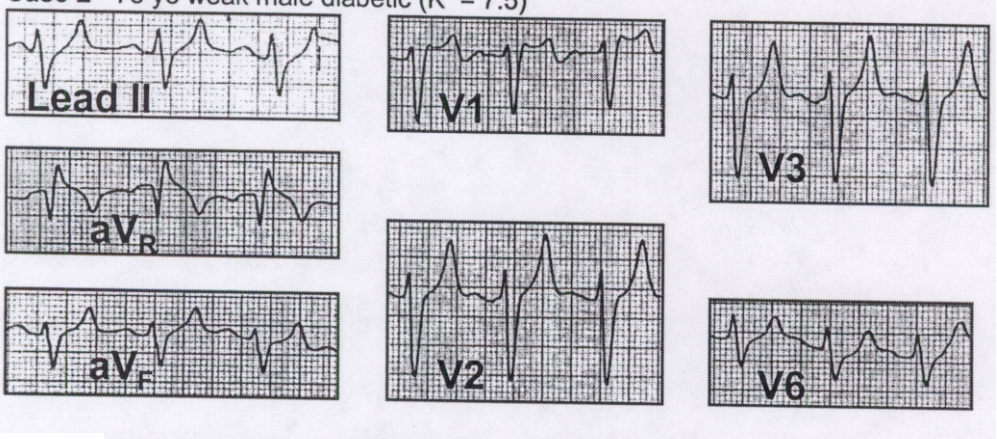
Prolonged QT and IVCD

Hyperkalemia

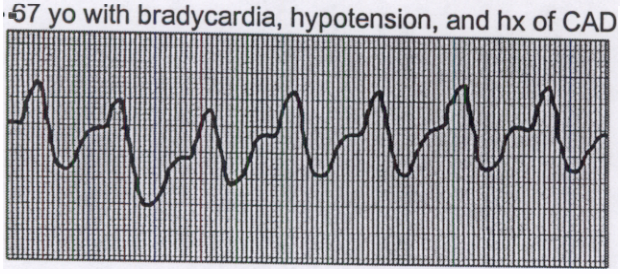
Case 1 - 67 yo male with CAD and CRF presents with one hour of CP (K^+ = 8.2)



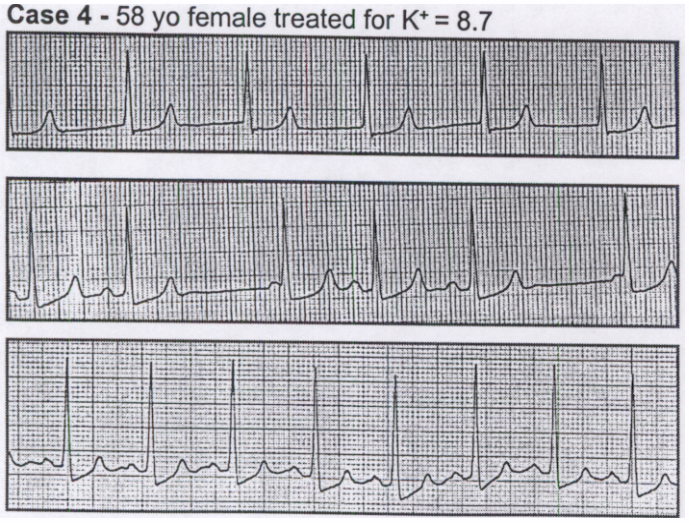
Case 2 - 78 yo weak male diabetic (K^+ = 7.5)



Case 3 - 67 yo with bradycardia, hypotension, and hx of CAD , CRF, CHF (K^+= 8.7)



Case 4 - 58 yo female treated for K^+ = 8.7



On arrival

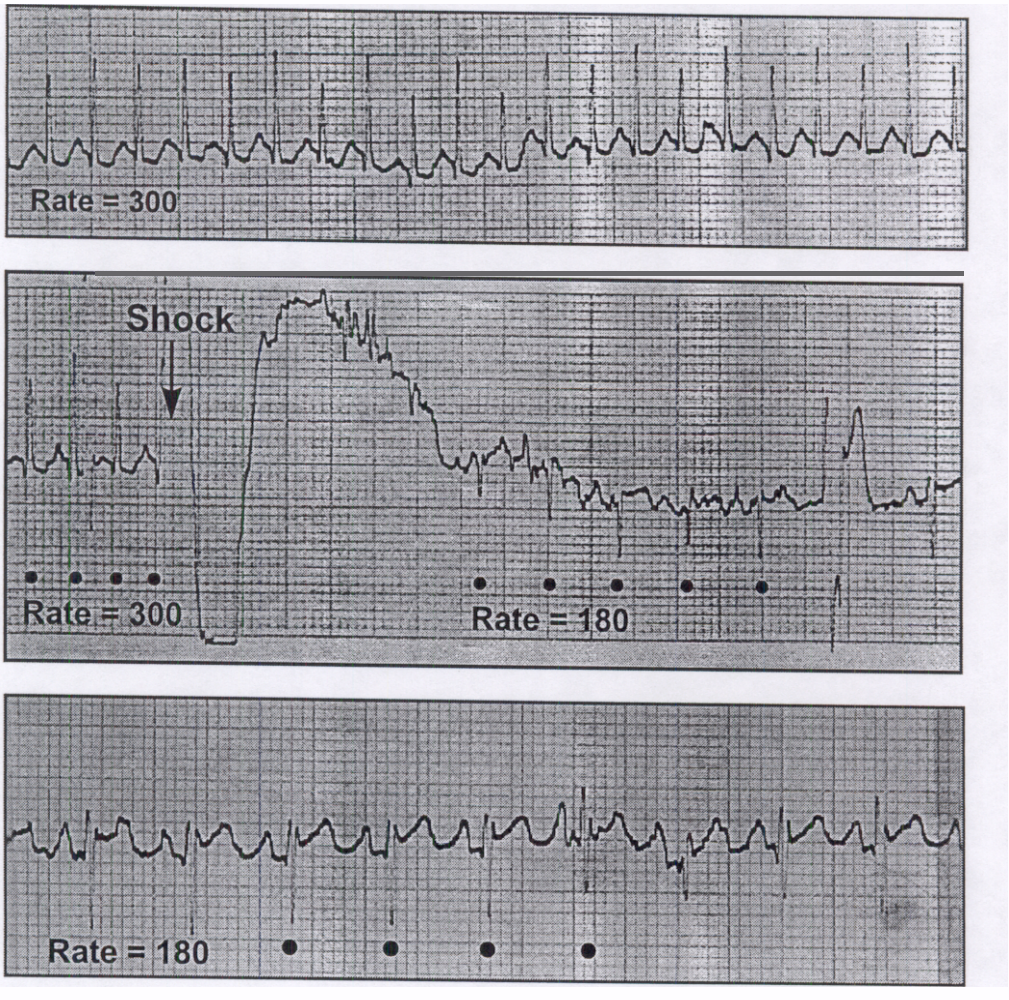
Ca, insulin, glucose, HCO_3

On discharge

Hyperkalemia IVCD

Pediatric Dysrhythmias

Case 1 ▪ Neonate
A 3 week infant presents with decreased appetite, urination, and stooling
Lethargic, ill appearing, grunting, cool to touch. and pale with pulses
HR=300, BP=65, RR=60, T=96.3
Chest retractions and hepatomegaly
No murmur and normal CXR



Outcome:
Infant is admitted to hospital and develops intemittent recurrent episodes of tachycardia treated with digoxin. Echocardiogram reveals no structural heart disease

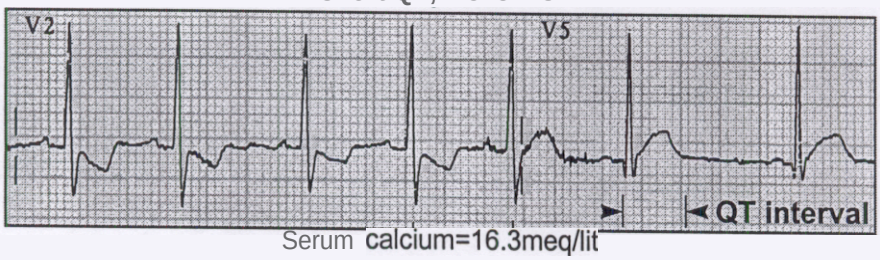
Pediatric Assessment
- General appearance
- Signs of heart failure or shock
- Heart Rate
  - count pulse. carddiac tachometer
  - ECG monitoring (MCL,)
  - 60 divided by RR interval
  - Large variation in normal by age

Therapy
- Unstable
  - Cardioversion at 0.5-l .O Joules/kg
- Stable
  - Vagal maneuvers
  - Adenosine
  - Lidocaine
- Other Drug Therapy
  - Digoxin
  - Propranolol
  - Verapamil

Case 2 -Vomiting in a toddler

20 month male with 3 weeks of vomiting, crankiness, and weakness admitted for dehydration. Has food allergies to corn, tomatoes, rice, wheat, and beans. Fussy, crying dehydrated child with irregular heart beat.

short QT, = 378 ms

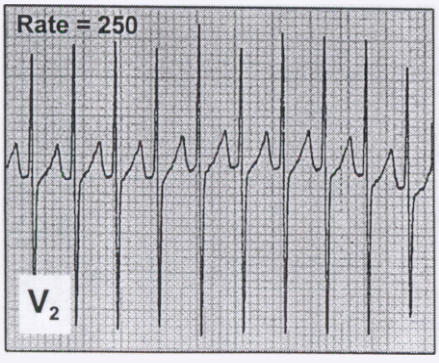


Serum calcium=16.3meq/lit
Final diagnosis = hypervitaminosis A

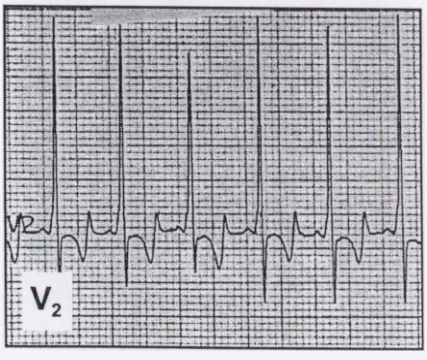
Case 3 - Infant with tachycardia

15 month old female with fever, congestion, and restlessness. On exam is well appearing in no distress with no murmur or hepatomegaly Vital signs: HR=250, RR=40, T=98.2. Rhythm converted after IV placement.

pre treatment



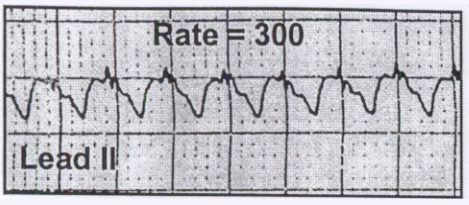
post treatment

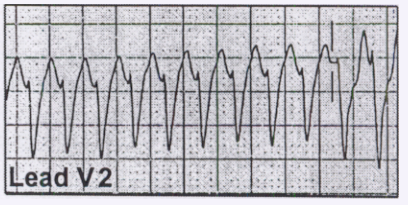


Pediatric Therapy
- Vagal maneuvers
- Lidocaine
- Procainamide
- Cardioversion
- Treat underlying

WPW Syndrome
- Short PR interval
- Wide QRS
- Delta wave
- Associated ST T changes
- Paroxysmal tachycardias

Case 4 -five month old with decreased appetite





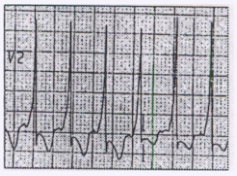
? Ventricular Tachycardia

Pediatric Causes

- Structural defect or myocarditis
- Prolonged QT syndrome
  - Congenital
  - Acquired
- Electrolyte abnormality
- Drug toxicity

Outcome

Ice Pack Therapy



WPW Syndrome

Pediatric Dysrhythmia -Summary

- Assessment
- Vagal maneuvers
- Adenosine 50-150 mcg/kg rapid
- Lidocaine 1mg/kg
- Cardioversion 0.5-2 Joules/kg

Appendix

Emergency Temporary Pacemaker Placement
Indications

- **Acute management of symptomatic bradycardia or heart block due to:**
  Electrolyte disturbance (e.g., hyperkalemia).
  Drug effect (digitalis, beta blockers, calcium antagonists, amiodarone).
  Myocarditis (e.g. Lyme disease, or viral myocarditis).
  Endocarditis (prophylactically with new bundle branch block or AV block).
  Sinus or AV node dysfunction post cardiac surgery.
- **Bridge to permanent pacemaker in symptomatic or unstable patient.**
- **In acute myocardial infarction** (ACC/AHA Guidelines) temporary pacing is:
  Highly recommended:
    Asystole.
    Symptomatic bradycardia (sinus with hypotension. or Type 1 second degree AV block with hypotension. not responsive to atropine or volume infusion).
    Bilateral bundle branch block (alternating bundle branch block, or RBBB with alternating LAFB and LPFB) (any age).
    New or indeterminate bifascicular block (RBBB with LAFB or LPFB, or LBBB) with first degree AV block.
    Mobitz II second degree heart block.
  Possibly helpful:
    RBBB with new or indeterminate fascicular block.
    RBBB with first degree AV block.
    LBBB. new or indeterminate.
    Incessant VT, for atrial or ventricular overdrive pacing,
    Sinus pauses > 3 seconds, not responsive to atropine.
    (For isolated BBB or fascicular block known to exist prior to MI, pacing is not indicated.)
  Potentially Harmful:
    First-degree heart block.
    Type I second-degree AV block with normal hemodynamics.
    Accelerated idioventricular rhythm with AV dissociation.
    Pacing may be indicated if medication deemed necessary, e.g. digoxin in advanced CHF.
- **AV block in acute MI**
  Pacing most often required in extensive anterior MI.
  Mortality predicated on extent of infarction and not heart block per se; however, pacing simplifies management (e.g. heart block in cardiogenic shock).
  AV sequential pacing may be useful in inferior MI with significant RV involvement - cardiac output dependent on atrial kick for sufficient RV filling.
  AV nodal block in inferior MI mediated by excessive vagal parasympathetic output (Bezold Jarisch reflex) - bradycardia but not necessarily hypotension is responsive to atropine.
  Alternatively, AV nodal block may be mediated through release of endogenous adenosine which may respond dramatically to theophylline or aminophylline (blocks adenosine response).
  Temporary pacing does not necessarily predict need for permanent pacing post MI -effects of persistent conduction defects more important in defining need for pacing.

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