PRACTITIONERS' GUIDANCE SERIES – XIV

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President's Remarks

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Editorial

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LEARNING ELECTROCARDIOGRAPHY

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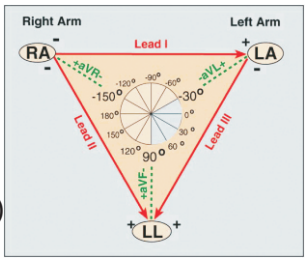
The Standard 12 Lead ECG

Clinical cardiac diagnosis depends mainly on patient's history and physical examination. Electrocardiogram can provide evidence to support a diagnosis and in some cases it is crucial for the patient management. With practice, interpreting the ECG is a matter of pattern recognition. However, the ECG can be analyzed from first principles if a few simple rules and basic facts are remembered. Let us revise some of the rules. A standard ECG is taken in 12 leads:-

➪ **Bipolar limb leads (frontal plane):**

- Lead I: RA (-) to LA (+)
- Lead II: RA (-) to LF (+)
- Lead III: LA (-) to LF (+)

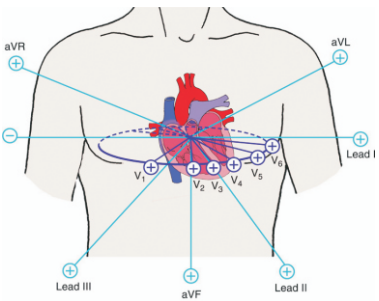


➪ **Augmented unipolar limb leads:**

- Lead aVR: RA (+) to [LA & LF] (-)
- Lead aVL: LA (+) to [RA & LF] (-)
- Lead aVF: LF (+) to [RA & LA] (-)

➪ **Unipolar (+) chest leads (horizontal plane):**

- Leads V1, V2, V3: (Posterior Anterior)
- Leads V4, V5, V6: Right Left, or lateral)



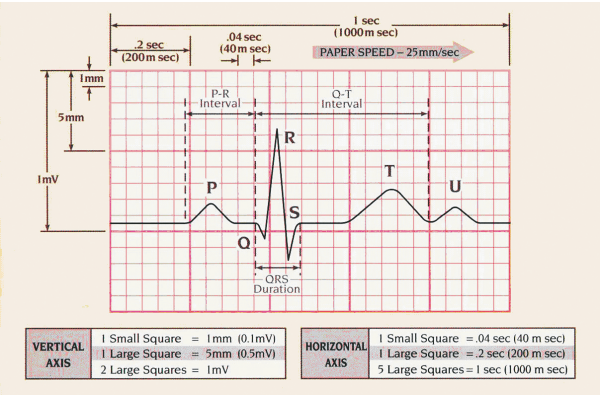
➪ **ECG Waves and Intervals:**

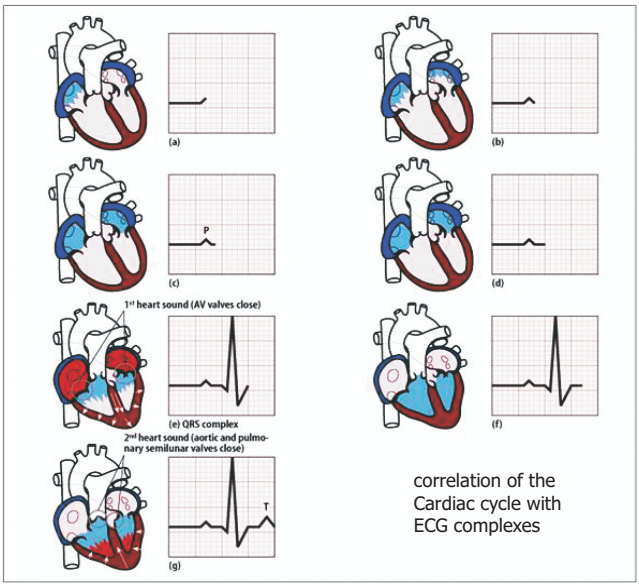
What do they mean?

- P wave: Sequential depolarization of the right and left atria
- QRS complex: Simultaneous right and left ventricular depolarization

- PR interval: Time interval from onset of atrial depolarization (P wave) to onset of ventricular depolarization (QRS complex)
- QRS duration: Duration of ventricular muscle depolarization
- QT interval: Duration of ventricular depolarization and repolarization
- RR interval: Duration of ventricular cardiac cycle
- PP interval: Duration of atrial cycle (an indicator of atrial rate)

Standard PQRST on ECG





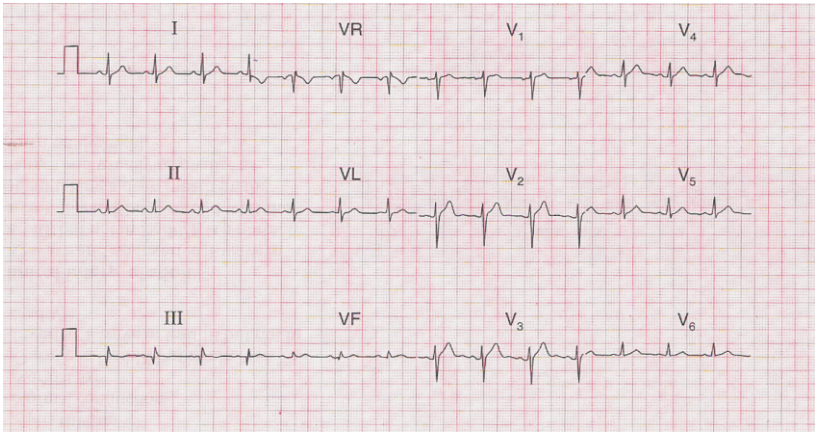
correlation of the Cardiac cycle with ECG complexes

LEARNING ELECTROCARDIOGRAPHY

- Cardiac muscle cells depolarize with a positive wave of depolarization, and then repolarize with a negative wave.
- Skin "leads" or electrodes have a positive and negative end.
- A positive wave form results from the wave of depolarization moving towards the positive end of the lead. A negative waveform is when a wave of depolarization is moving away from the positive electrode.
- ECG paper has 1 millimeter small squares - so height and depth of wave is measured in millimeters. 10 mm = 1.0 millivolt
- Horizontal axis is time.
  0.04 seconds for 1 mm (1 small box).
  0.2 seconds for 1 large box = 5 small boxes = 5 x .04 seconds.
- Lead nomenclature:

Limb Leads	**Chest Leads**	**Rhythm Strip**
I, II, III aVR, aVL, aVF	V1 to V6	Selected to give the best relationship of the P wave to the QRS

- A normal ECG



ECG interpretation: Look at three areas in order on each ECG.

- **Heart Rate**
- **Rhythm (Intervals)**
- **Axis**

Heart Rate

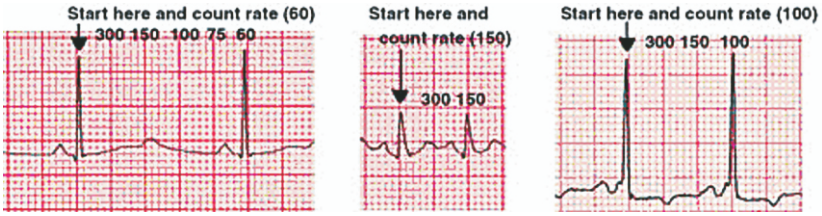
Normal rate for the SA node is 60-100 per minute.

SA node is the usual pacemaker. If SA node fails, other potential pacemakers are atrial pacemakers with inherent rates of 50-60, AV node (rate 40-60), or ventricles (rate 20-40). In certain pathologic conditions ectopic pacemakers can go much faster at rates 150-250 cycles/minute. There are three methods of calculating heart rate:

1. Most Common Method :

Find an R wave on a heavy line (large box) count off "300, 150, 100, 75, 60, and 50" for each large box you land on until you reach the next R wave. Estimate the rate if the second R wave doesn't fall on a heavy black line.

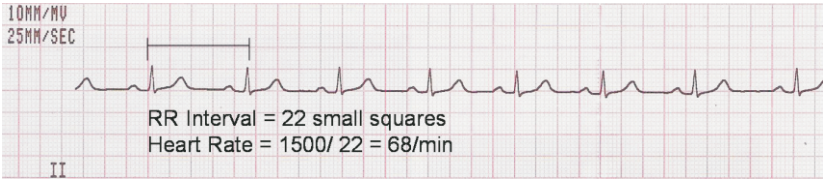
Rate calculation
Memorize the number sequence:
300, 150, 100, 75, 60, 50



2. Heart Rate Formula :-

Heart Rate = 1500 Divided by the RR interval

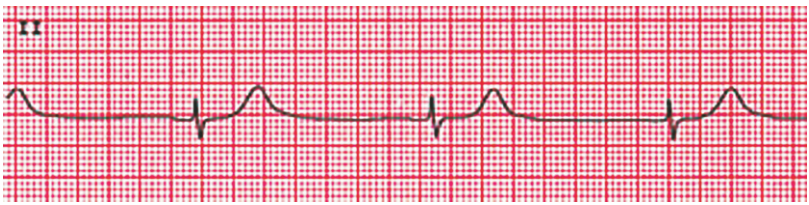
RR Interval = Number of small squares between two R waves



3. Mathematical method:

Use this method if there is a regular bradycardia, i.e. - rate < 50. If the distance between the two R waves is too long to use the common

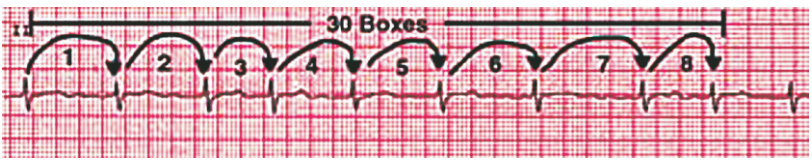
method, use the approach: 300 divided by number of large boxes between two R waves.



Count number of large boxes between first and second R waves=7.5. Thus, 300 divided by 7.5 large boxes = rate 40.

4. Six-second method:

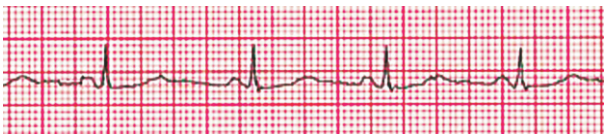
Count off 30 large boxes = 6 seconds (remember 1 large box = 0.2 seconds, so 30 large boxes = 6 seconds). Then, count the number of R-R intervals in six seconds and multiply by 10. This is the number of beats per minute. This is most useful if you have an irregular rhythm (like atrial fibrillation) when you want to know an average rate.



- Count 30 large boxes starting from the first R wave. There are 8 R-R intervals within 30 boxes. Multiply 8 x 10 = Rate 80.

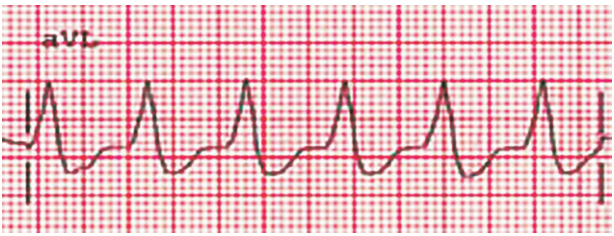
Rhythm (to include intervals)

The normal conduction pathway is: SA node —> AV node —> Bundle of HIS —> Bundle Branches. Now for some basics - "arrhythmia" means abnormal rhythm. Arrhythmia can be understood by realizing the existence of ectopic (out of place) foci (pacemakers) and understanding the normal conduction pathway of the heart. Very simply put, if the beat originates in the atria or AV node (supraventricular) the QRS is usually narrow (normal), because it comes from above along the normal pathway.



- QRS is Narrow (normal)

  If the beat is ventricular in origin the QRS is wide and bizarre because it doesn't come down the normal pathway.



- QRS is wide.

  Aberrancy is an exception to this rule - here it does actually follow the normal pathway (atria - AV node - ventricle) but for some reason one of the bundle branches is refractory to the beat and you get a wide QRS.

  A reasonable way to group arrhythmias is in four general groups. Let us briefly review these four groups, and then we will develop some common sense principles for evaluating rhythm (to include intervals).

Axis

- Axis is the direction of depolarization vector of the QRS complex.
  1. The left ventricle is thicker so the mean QRS vector is down and to the left. (The origin of the vector is the AV node with the left ventricle being down and to the left of this).
  2. The vector will point toward hypertrophy (thickened wall) and away from the infarct (electrically dead area).

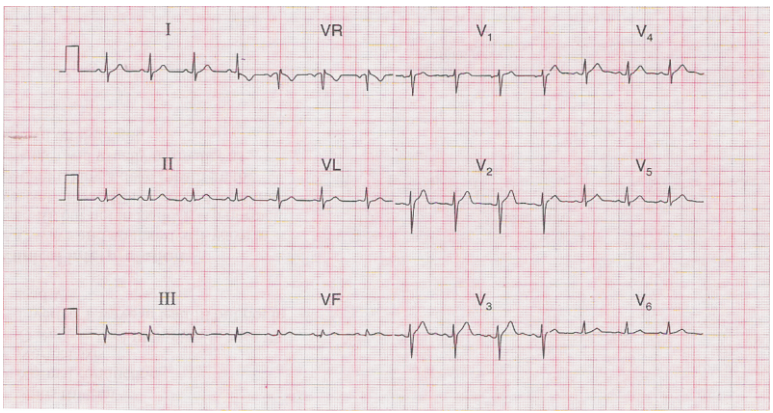
Normal axis	-30 to +90 degrees
Left axis deviation	-30 to -90 degrees
Right axis deviation	+90 to +/-180 degrees
Indeterminate (extreme) axis deviation	-90 to +/-180 degrees

- Since lead I and aVF are perpendicular to each other, you can use those two leads to quickly determine axis.
- You can repeat this process for any two leads, but I and aVF are the classic places to look. If you realize that there are two leads to

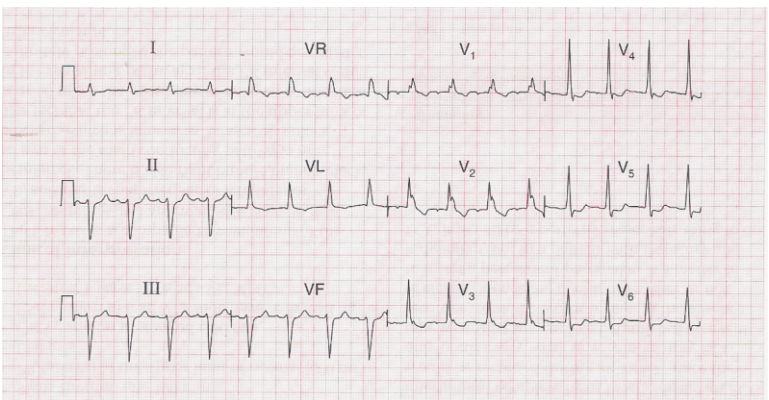
consider and a positive (+) or (-) orientation for each lead, there would be four possible combinations. Memorize the following axis guidelines.

Axis	Lead I	Lead aVF
Normal axis (0 to +90 degrees) Left axis deviation (-30 to -90) To be true	Positive	Positive
left axis deviation, lead II should be negative.		

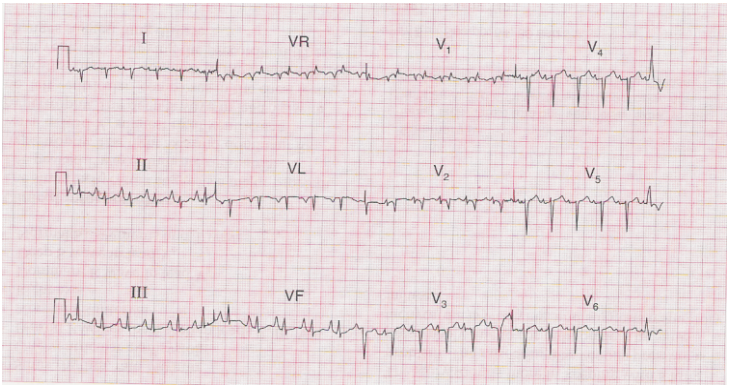
- Normal Axis



- Left axis deviation.



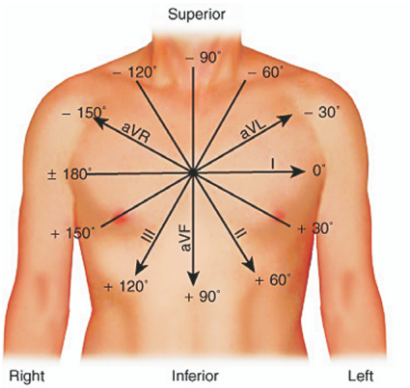
- Right axis deviation.

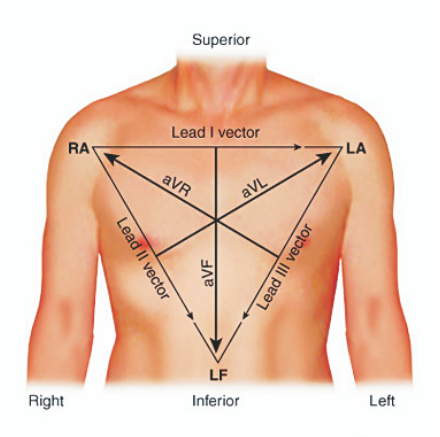


If the axis is shifted out of the normal quadrant, evaluate the reasons for this.

	Differential Diagnosis
Left axis deviation	LVH, left anterior fascicular block, inferior wall MI
Right axis deviation	RVH, left posterior fascicular block, lateral wall MI

-





A "Method" of ECG Interpretation

1. Measurements (usually made in frontal plane leads):

- ⇨ Heart rate (state atrial and ventricular rates, if different)
- ⇨ PR interval (from beginning of P to beginning of QRS)
- ⇨ QRS duration (width of most representative QRS)
- ⇨ QT interval (from beginning of QRS to end of T)
- ⇨ QRS axis in frontal plane (go to: "How To Determine Axis")

2. Rhythm Analysis

- ⇨ Find out the basic rhythm (e.g., normal sinus rhythm, atrial fibrillation etc.)
- ⇨ Identify additional rhythm events if present (e.g., "PVC's", "PAC's", etc)
- ⇨ consider all rhythm events from atria, AV junction, and ventricles

3. Conduction Analysis

- ⇨ "Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.

- The following conduction abnormalities are to be identified if present:
  - AV block: 1st, 2nd (type I vs. type II), and 3rd degree
  - Intraventricular blocks: bundle branch, fascicular, and nonspecific blocks
  - Exit blocks: blocks just distal to ectopic pacemaker site

4. Waveform Description

- Carefully analyze the 12-lead ECG for abnormalities in each of the waveforms in the order in which they appear: P-waves, QRS complexes, ST segments, T waves, and... Don't forget the U waves.
  - P waves: are they too wide, too tall, inverted (i.e., are they ectopic), etc.?
  - QRS complexes: look for pathologic Q Wave's.
  - ST segments: look for abnormal ST elevation and/or depression.
  - T waves: look for abnormally inverted T waves.
  - U waves: look for prominent or inverted U waves.

5. ECG Interpretation

- This is the conclusion of the above analyses. Interpret the ECG as "Normal", or "Abnormal". Occasionally the term "borderline" is used if unsure about the significance of certain findings. List all abnormalities. Examples of

"abnormal" statements are:

- Inferior MI, probably acute
- Old anteroseptal MI
- Left anterior fascicular block (LAFB)
- Left ventricular hypertrophy (LVH)
- Any rhythm abnormalities

6. Comparison with previous ECG

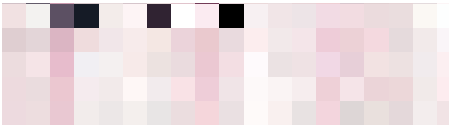
⇨ If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.

7. Normal Parameters

Rhythm	Sinus, Regular
P waves	Height < 3 mm and Width < 3 mm
PR Interval	3 to 5 mm (i.e. 0.12 to 0.20 sec)
Q wave	Depth < 1/4th R and Width < 1 mm
R Transition	V2-4
QRS axis	0 to110 (Young) -30 to 90 (Old)
ST segment	Same level as PR segment
T wave	Positive in most leads except aVR and V1-V2
U wave	Absent
QTc Interval	< 0.42 sec for males & < 0.44 sec for females
Other	Ectopic beats, Paced beats

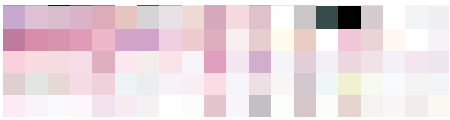
LEARNING ELECTROCARDIOGRAPHY

ECG 1: Calculate Heart Rate, Which waves are abnormal?



Heart Rate 75/min, T waves are abnormal

ECG 2: What is the Rhythm? Calculate Heart Rate.



Supraventricular Rhythm? Heart Rate Approximate 150/min

ECG 3: Calculate Heart Rate. Where is the P wave?

Heart Rate 220/min. There are no P waves.

Characteristics of the Normal ECG

1. Normal Measurements

- ⇨ Heart Rate: 60 - 90 bpm
- ⇨ PR Interval: 0.12 - 0.20 sec
- ⇨ QRS Duration: 0.06 - 0.10 sec
- ⇨ QT Interval (QTc < 0.40 sec)
  - ❍ Friedriecia's Formula: QTc = QT interval divided by Cube Root of RR in seconds
  - ❍ Practical Guide to QTc
    QT Interval should be less than half of the RR interval
  - ❍ The commonly used Bazett's correction is very inaccurate, specially at heart rates above 80 beats per minute.
- ⇨ Frontal Plane QRS Axis: +90 o to -30 o (in the adult)

2. Rhythm:

Normal sinus rhythm: The P waves in leads I and II must be upright if the impulse is coming from the sinus node.

3. Conduction:

Normal Sino-atrial (SA), Atrio-ventricular (AV), and Intraventricular (IV) conduction Both the PR interval and QRS duration should be within the limits specified above.

4. Waveform Description:

⇨ P Wave

It is important to remember that the P wave represents the sequential activation of the right and left atria, and it is common to see notched or biphasic P waves of right and left atrial activation.

- ❍ P duration < 0.12 sec
- ❍ P amplitude < 3 mm
- ❍ May see notched P waves in some leads

⇨ QRS Complex

QRS represents the simultaneous activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature.

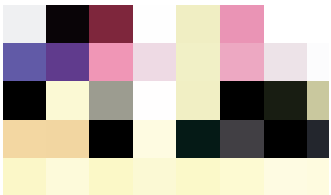
- ❍ QRS duration < 0.10 sec
- ❍ QRS amplitude is quite variable from lead to lead and from person to person. Two determinates of QRS voltages are:
  - ❒ Size of the ventricular chambers: Larger the chamber, larger the voltage
  - ❒ Proximity of chest electrodes to ventricles: Closer the electrode, larger the voltage)
- ❍ Frontal plane leads:
  - ❒ The QRS is mostly positive (upright) in leads I and II.
  - ❒ Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They are often seen in leads I and aVL when the QRS axis is to the left of +60°, and in leads II, III, aVF when the QRS axis is to the right of +60°. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.
- ❍ Precordial leads:
  - ❒ Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.
  - ❒ In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.
  - ❒ The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
  - ❒ Small "septal" q-waves may be seen in leads V5 and V6.

⇨ ST Segment and T wave

Sometimes ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T and followed by a rapid descent to the isoelectric baseline or the onset of the U wave.

The normal T wave is usually in the same direction as the QRS except in the right precordial leads. In the normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

- Normal ST segment elevation: ST segment elevation with concave upward appearance may also be seen in leads II, III, aVF and V2-V3. This is often called early repolarization, although it's a term with little physiologic meaning.
- Convex or straight upward ST segment elevation (e.g., leads II, III, aVF) is abnormal and suggests transmural injury or infarction.
- ST segment depression is often characterized as "up-sloping", "horizontal", or "down-sloping".
- Upsloping ST depression is often a normal variation.



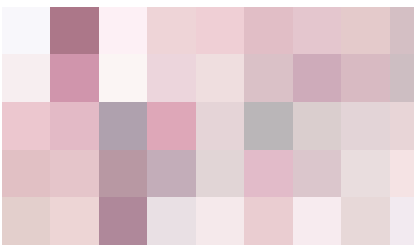
⇨ The normal U Wave: (the most neglected of the ECG waveforms)

- U wave amplitude is usually < 1/3 T wave amplitude in same lead
- U wave direction is the same as T wave direction in that lead
- U waves are more prominent at slow heart rates and in V2-V4.
- Origin of the U wave is related to after depolarization which follows repolarization.

ECG 4: Describe ST segment.

Left Axis , ST segment elevated in I, aVL, V1-4

ECG 5: Calculate Axis, What is the abnormal beat?



Left Axis, Abnormal beat is ventricular extrasystole

Abnormalities in the ECG Measurements

1. Heart Rate

In normal sinus rhythm, a resting heart rate of below 60 bpm is called bradycardia and a rate of above 100 bpm is called tachycardia.

< 60 bradycardia	> 100 tachycardia

In athletic individuals, the resting heart rate can even be upto 50 beats per min.

2. PR Interval is measured from beginning of P to beginning of QRS in the frontal plane

- ⇨ Normal: 0.12 - 0.20 sec
- ⇨ Short PR: < 0.10 sec
  - Pre-excitation syndromes:
    - ❒ WPW (Wolff-Parkinson-White) Syndrome: An accessory pathway connects the right atrium to the right ventricle. This is associated with a wide QRS complex and T wave inversions.
  - ❍ Ectopic atrial rhythms originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus P)
  - ❍ Normal variant
- ⇨ Prolonged PR: >0.20 sec
  - ❍ First degree AV block
    - ❒ PR interval is usually constant. There may be slower AV conduction, delayed conduction in bundle of His or bundle branches.
  - ❍ Second degree AV block (PR interval may be normal or prolonged; some P waves do not conduct)
    - ❒ Type I (Wenckebach): Increasing PR until nonconducted P wave occurs
    - ❒ Type II (Mobitz): Fixed PR intervals plus nonconducted P waves

Third degree complete AV block – the P waves and QRS complexes are dissociated (i.e., not married, but strangers passing in the night). Therefore there is no constant PR interval.

3. QRS Duration in frontal plane

⇨Normal: 0.06 - 0.10 sec

Prolonged QRS Duration (>0.10 sec):

- ❍ QRS duration 0.10 - 0.12 sec
  - ❒ Incomplete right or left bundle branch block, Nonspecific intraventricular conduction delay (IVCD).
- ❍ QRS duration > 0.12 sec
  - ❒ Complete RBBB or LBBB, Nonspecific IVCD, Ventricles ectopic rhythms (e.g., ventricular tachycardia, pacemaker rhythm)

4. QT Interval

It is measured from beginning of QRS to end of T wave in the frontal plane.

⇨ Normal: heart rate dependent (corrected QT = QTc = measured QT divided by Cube root of RR in seconds; upper limit for QTc = 0.44 sec)

⇨ Long QT Syndrome - (LQTS)

- ❍ It is based on upper limits for heart rate; QTc > 0.47 sec for males and >0.48 sec in females is diagnostic for hereditary LQTS in absence of other causes of increased QT.
- ❍ It usually indicates a state of increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death. The prototype arrhythmia of the Long QT Interval Syndromes (LQTS) is Torsade-de-pointes, a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline.
- ❍ Causes of LQTS include the following:

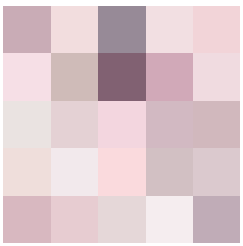
  Drugs (many antiarrhythmics, tricyclics, phenothiazines, antimalarials, quinolones and others), Electrolyte abnormalities(K+, Ca++, Mg++),CNS disease (especially sub-arrachnoid hemorrhage, stroke, trauma), Hereditary LQTS (e.g., Romano-Ward Syndrome), Disease (some post-MI patients) Coronary Heart disease (some post-MI patients)

5. Frontal Plane QRS Axis

- ⇨ Normal: -30 degrees to +90 degrees
- ⇨ Abnormalities in the QRS Axis:
  - ❍ Left Axis Deviation (LAD) : < -30 degrees (i.e., lead II is mostly negative)
    - ❒ Left Anterior Fascicular Block (LAFB) : rS complex in leads II, III, aVF, small q in leads I and/or aVL, and axis -45 degrees to -90 degrees
    - ❒ Some cases of inferior MI with Qr complex in lead II (making lead II 'negative')
    - ❒ Some cases of LVH and LBBB
    - ❒ Ostium primum ASD and other endocardial cushion defects
    - ❒ Some cases of WPW syndrome (large negative delta wave in lead II)
  - ❍ Right Axis Deviation (RAD) : > +90 degrees (i.e., lead I is mostly negative)
    - ❒ Left Posterior Fascicular Block (LPFB): rS complex in lead I, qR in leads II, III, aVF (however, must first exclude, on clinical basis, causes of right heart overload; these will also give same ECG picture of LPFB)
    - ❒ Many causes of right heart overload and pulmonary hypertension
    - ❒ High lateral wall MI with Qr or QS complex in leads I and aVL
    - ❒ Some cases of RBBB, WPW syndrome
    - ❒ Children, teenagers, and some young adults
  - ❍ Bizarre QRS axis: +180 degrees to -90 degrees (i.e., lead I and lead II are both negative)
    - ❒ Consider limb lead error (usually right and left arm reversal)
    - ❒ Dextrocardia, Complex congenital heart disease
    - ❒ Some cases of ventricular tachycardia

ECG 6: What is the rhythm? Calculate the QRS width.

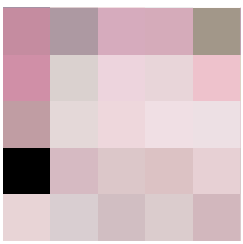
Ventricular rhythm. QRS complex is 200ms



ECG 7: Describe the ST segment

ST segment horizontally depressed in V3-6

ECG 8: Describe the T waves

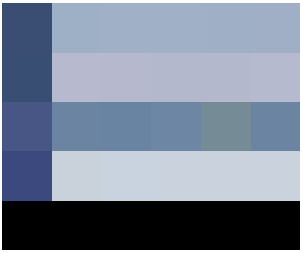


T waves tall and peaked in V2-6

ECG Rhythm Abnormalities (Supraventricular)

1. Premature atrial complexes

- ⇨ Occur as single or repetitive events and have unifocal or multifocal origins.
- ⇨ The ectopic P wave is often hidden in the ST-T wave of the preceding beat.
- ⇨ The P'R interval is normal or prolonged because the AV junction is often partially refractory when the premature impulse enters it.



A "ladder" diagram is an easy way of conceptualizing the conduction of impulses through the heart, and the resulting complexes (i.e., P waves and QRS waves).

- ❍ Outcome 1: Nonconducted (blocked); i.e., no QRS complex because the PAC finds AV node still refractory. (See PAC labeled 'a' in the diagram on the previous page)
- ❍ Outcome 2: Conducted with aberration; i.e., PAC makes it into the ventricles but finds one or more of the conducting fascicles or bundle branches refractory. The resulting QRS is usually wide, and is sometimes called an Ashman beat (see PAC 'b' in the diagram on the previous page)

- Outcome 3: Normal conduction; i.e., similar to other QRS complexes in the ECG. (See PAC 'c' in the diagram on the previous page)

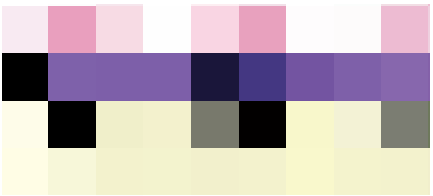
⇨ In the diagram seen above, the PP interval has increased (slower heart rate), and this results in increased refractoriness of all the structures in the conduction system (i.e., wider boxes). PAC 'b' now can't get through the AV node and is non conducted; PAC 'c' is now blocked in the right bundle branch and results in a RBBB QRS complex (aberrant conduction); PAC 'd' is far enough away to conduct normally. Therefore, the fate of a PAC depends on 1) the coupling interval from the last P wave and 2) the preceding cycle length or heart rate.

2. Premature Junctional Complexes (PJC)

⇨ Similar to PAC's in clinical implications, but occur less frequently.

⇨ The PJC focus, located in the AV junction, captures the atria (retrograde) and the ventricles (antegrade). The retrograde P wave may appear before, during, or after the QRS complex; if before, the PR interval is usually short (<0.12 s).

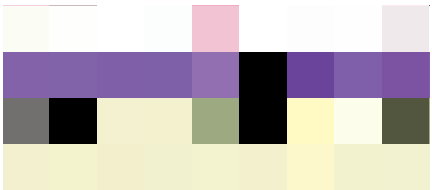
3. Atrial Fibrillation (A-fib)



⇨ Atrial activity is poorly defined; may see course or fine undulations or no atrialactivity at all. If atrial activity is seen, it resembles an old saw.

- ⇨ Ventricular response is irregularly irregular and may be fast (HR >100 bpm, indicates inadequate rate control), moderate (HR = 60-100 bpm), or slow (HR < 60 bpm, indicates excessive rate control, AV node disease, or drug toxicity).
- ⇨ A regular ventricular response with A-fib usually indicates complete AV block with an escape pacemaker originating in the AV junction or ventricles (i.e., must consider digoxin toxicity or AV node disease).
- ⇨ The differential diagnosis includes atrial flutter with an irregular ventricular response and multifocal atrial tachycardia (MAT), which is usually irregularly irregular.

4. Atrial Flutter (A-flutter):



- ⇨ Regular atrial activity with a "clean" saw-tooth appearance in leads II, III, aVF, and usually discrete 'P' waves in lead V1. The atrial rate is usually about 300/ min, but may be as slow as 150-200/min or as fast as 400-450/min.

5. Ectopic Atrial Tachycardia and Rhythm:

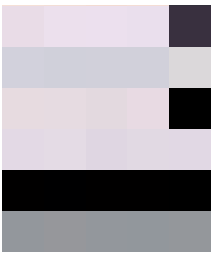
- ⇨ Ectopic, discrete looking, unifocal P' waves with atrial rate <250/min.
- ⇨ Ventricular response may be 1:1 or with varying degrees of AV block.
- ⇨ Ectopic atrial rhythm is similar to ectopic atrial tachycardia, but with HR < 100 bpm.

6. Multifocal Atrial Tachycardia (MAT) and rhythm:

- ⇨ Discrete, multifocal P' waves at rate of 100-250/min, with varying P'R intervals
- ⇨ At least 3 different P wave morphologies to be present in a given lead.
- ⇨ Ventricular response is irregularly irregular (i.e., often confused with A-fib).
- ⇨ May be intermittent, alternating with periods of normal sinus rhythm.
- ⇨ Most often seen in elderly patients with chronic or acute medical problems like, exacerbation of chronic obstructive pulmonary disease.
- ⇨ May be also seen with digitalis toxicity.

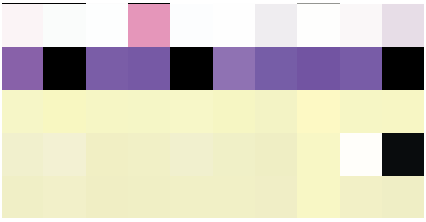
7. Paroxysmal Supraventricular Tachycardia (PSVT) :

These arrhythmias are circus movement or reciprocating tachycardias because they utilize the mechanism of reentry. The onset is sudden, usually initiated by a premature beat, and the arrhythmia also stops abruptly - which is why they are called paroxysmal. They are usually narrow-QRS tachycardias unless there is preexisting bundle branch block or rate-related aberrant ventricular conduction. There are several types of PSVT depending on the location of the reentry circuit.



8. Junctional Rhythms and Tachycardias:

- **Junctional Escape Beats:** These are passive, protective beats originating from subsidiary pacemaker cells in the AV junction (usually in the Bundle of His). The pacemaker's basic firing rate is 40-60 bpm; junctional escapes are protective events that occur whenever the primary pacemaker (i.e., sinus node) defaults or the AV node blocks the atrial impulse.
- **Junctional Escape Rhythm:** This is a sequence of 3 or more junctional escapes occurring by default at a rate of 40-60 bpm. There may be AV dissociation or the atria may be captured retrogradely by the junctional pacemaker. In the ECG example below the retrograde P waves are not seen and must be hidden in the QRS complex; the significant "Q" wave with ST elevation in the bottom strip suggests an acute MI.
- **Accelerated Junctional Rhythm:** This is an active junctional pacemaker rhythm caused by events that perturb pacemaker cells (e.g., ischemia, drugs, and electrolyte abnormalities). The rate is 60-100 bpm).
- **Non-paroxysmal Junctional Tachycardia:** This usually begins as an accelerated junctional rhythm but the heart rate gradually increases to above 100 bpm. There may be AV dissociation, or retrograde atrial capture may occur. Ischemia and digitalis intoxication are the two most common causes.



ECG 9: Describe the rhythm abnormality

Atrial fibrillation with LBBB

ECG 10: Describe the rhythm abnormality

Atrial flutter with 2:1 conduction

ECG 11: Describe the rhythm abnormality

Ventricular Tachycardia

ECG Rhythm Abnormalities (Ventricular)

1. Premature Ventricular Complexes (PVCs) :

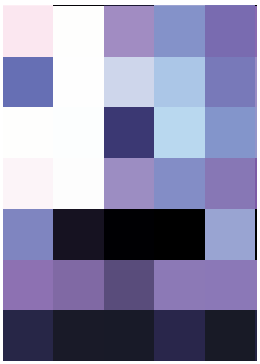
- ⇨PVCs may be unifocal or multifocal. Multifocal PVCs have different sites of origin, which means their coupling intervals are usually different.
- ⇨PVCs may occur as isolated single events or as couplets, triplets, and salvos (4-6 PVCs in a row), also called non-sustained ventricular tachycardias.
- ⇨PVCs may occur early in the cycle (R-on-T phenomenon), after the T wave (as seen above), or late in the cycle - often fusing with the next QRS (fusion beat). R-on-T PVCs may be especially dangerous in an acute ischemic situation, because the ventricles may be more vulnerable to ventricular tachycardia or fibrillation.
- ⇨Not all PVCs are followed by a pause. If PVC occurs early enough (especially if the heart rate is slow), it may appear sandwiched in between two normal beats. This is called interpolated PVC. The sinus impulse following the PVC may be conducted with a longer PR interval because of retrograde concealed conduction by the PVC into the AV junction slowing subsequent conduction of the sinus impulse.

2. Aberrancy vs. Ventricular Ectopic:

- ⇨ Aberrant Ventricular Conduction: defined as the intermittent abnormal intraventricular conduction of a supraventricular impulse. The phenomenon comes about because of unequal refractoriness of the bundle branches and critical prematurity of a supraventricular impulse (see diagram of "Three Fates of PACs"). With such critical prematurity, the supraventricular impulse encounters one

bundle branch (or fascicle) which is responsive, and the other which is refractory, and is consequently conducted with a bundle branch block or fascicular block pattern.

- ECG clues to the differential diagnosis of wide QRS premature beats:
  - Preceding ectopic P wave (i.e., the P' of the PAC) usually hidden in the ST-T wave of the previous beat favors aberrant ventricular conduction.
  - Analyze the compensatory pause: A complete pause favors ventricular ectopy (i.e., no resetting of the sinus pacemaker; next sinus impulse comes on time). An incomplete pause favors aberration (i.e., because supraventricular premature beats are more likely to reset the sinus node's timing).



Analyze the QRS morphology of the funny-looking beat. This is one of the most rewarding of the clinical clues, especially if lead V1 (or the MCL1 monitored lead in intensive care units) is used. Since aberrancy is almost always in the form of a bundle branch block morphology, V1 is the best lead for differentiating RBBB from LBBB; RBBB creates a positive deflection, and LBBB, a negative deflection. Therefore, the first order of business is to identify the direction of QRS forces in V1.

If the QRS in V1 is mostly positive the following possibilities exist: rsR' or rSR' QRS morphologies suggest RBBB aberrancy >90% of the time!

Monophasic R waves or R waves with a notch or slur on the down stroke of the R waves suggests ventricular ectopy > 90% of the time (see below)!

3. Ventricular Tachycardia:

- Sustained (lasting >30 sec) vs. nonsustained
- Monomorphic (uniform morphology) vs. polymorphic vs. Torsade-de-pointes
  - Torsade-de-pointes: a polymorphic ventricular tachycardia associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200bpm and ventricular fibrillation is a consequence.
- Presence of AV dissociation vs. retrograde atrial capture
- Presence of fusion QRS complexes called Dressler beats, which occur when supraventricular beats get into the ventricles during the ectopic activation sequence.
  - Differential Diagnosis: just as for single premature funny-looking beats, not all wide QRS tachycardias are ventricular in origin.

Monomorphic Ventricular Tachycardia

4. Differential Diagnosis of Wide QRS Tachycardias :

⇨ ECG Clues:

- Regularity of the rhythm: If the wide QRS tachycardia is sustained and monomorphic, then the rhythm is usually regular (i.e., RR intervals equal); an irregularly-irregular rhythm suggests atrial fibrillation with aberration or with WPW pre-excitation.
- A-V Dissociation strongly suggests ventricular tachycardia! Unfortunately AV dissociation only occurs in approximately 50% of ventricular tachycardias (the other 50% have retrograde atrial capture or "V-A association"). Of the patients with AV dissociation, it is only easily recognized if the rate of tachycardia is <150 bpm. Faster heart rates make it difficult to visualize dissociated P waves.
- Fusion beats or captures often occur when there is AV dissociation and this also strongly suggests a ventricular origin for the wide QRS tachycardia.
- QRS morphology in lead V1 or V6 as described above for single premature funny looking beats is often the best clue to the origin, so go back and check out the clues!

Step 1: Absence of RS complex in all leads V1-V6?

If Yes: It is ventricular tachycardia!

Step 2: No: Is interval from beginning of R wave to nadir of S wave >0.1s in any RS lead?

If Yes: It is ventricular tachycardia!

Step 3: No: Are AV dissociation, fusions, or captures seen?

If Yes: It is ventricular tachycardia!

Step 4: No: Are there morphology criteria for VT present both in leads V1 and V6?

If Yes: It is ventricular tachycardia!

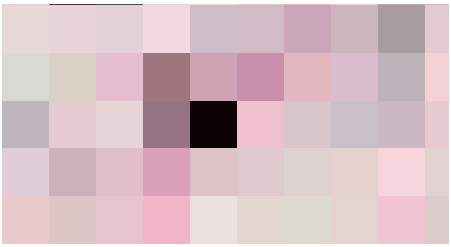
If NO: Diagnosis is supraventricular tachycardia with aberration!

5. Accelerated Idio-Ventricular Rhythms (AIVR):

- ⇨ It is an "active" ventricular rhythm due to enhanced automaticity of a ventricular pacemaker. (Reperfusion after thrombolytic therapy is a common causative factor).
- ⇨ Ventricular rate 60-100 bpm (anything faster would be ventricular tachycardia).
- ⇨ Usually benign, short lasting and do not require any therapy.

6. Idioventricular Rhythm:

- ⇨ A "passive" escape rhythm that occurs by default whenever higher-lever pacemakers in AV junction or sinus node fail to control ventricular activation.
  - ❍ Escape rate is usually 30-50 bpm (i.e., slower than a junctional escape rhythm).
  - ❍ Seen most often in complete AV block with AV dissociation.

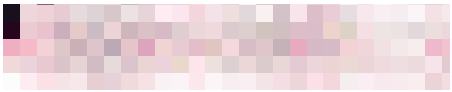


Accelerated Idio-Ventricular Rhythms (AIVR)

ECG 12: Describe the odd beats

Sinus rhythm with ventricular extrasystole

ECG 13: Describe the odd beats



Atrial fibrillation with ventricular tachycardia, extrasystole

ECG 14: Describe the rhythm abnormality

Atrial fibrillation with rapid ventricular rate

ECG Conduction Abnormalities

Introduction:

Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and their fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction (1st degree), intermittent conduction failure (2nd degree), or complete conduction failure (3rd degree). This section considers conduction disorders in the anatomical sequence that defines the cardiac conduction system; so let's begin . . .

1. Sino-Atrial Exit Block (SA Block):

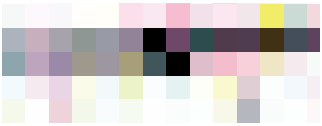
- Type I (SA Wenckebach): the following 3 rules represent the classic rules of Wenckebach, which were originally described for Type I AV block:
  - PP intervals gradually shorten until a pause occurs (i.e., the blocked sinus impulse fails to reach the atria)
  - The pause duration is less than the two preceding PP intervals
  - The PP interval following the pause is greater than the PP interval just before the pause
- Type II SA Block:
  - PP interval is fairly constant until conduction failure occurs.
  - The pause is approximately twice the basic PP interval

2. Atrio-Ventricular (AV) Block

⇨ Possible sites of AV block:

- AV node (most common), His bundle (uncommon), Bundle branch and fascicular divisions.

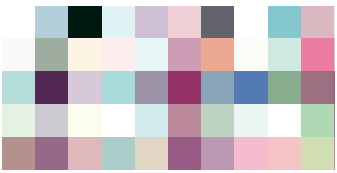
⇨ 1st Degree AV Block: PR interval > 0.20 sec; all P waves conduct to the ventricles.



⇨ 2nd Degree AV Block:

In "classic" Type I (Wenckebach) AV block the PR interval gets longer (by shorter increments) until a nonconducted P wave occurs. Type I AV block is almost always located in the AV node, which means that the QRS duration is usually narrow, unless there is preexisting bundle branch disease. The RR interval of the pause is less than the two preceding RR intervals, and the RR interval after the pause is greater than the RR interval before the pause. These are the classic rules of Wenckebach (atypical forms can occur).

Type II (Mobitz) AV block: There must be two consecutive constant PR intervals before the blocked P wave to diagnose Type II AV block (i.e., if there is 2:1 AV block we can't be sure if its type I or II). The RR interval of the pause is equal to the two preceding RR intervals. Type II AV block is almost always located in the bundle branches, which means that the QRS duration is wide indicating complete block of one bundle; the nonconducted P wave is blocked in the other bundle.



Intermittent 2:1 AV Block with Junctional Escape

⇨ Complete (3rd Degree) AV Block

- ❍ Usually see complete AV dissociation because the atria and ventricles are each controlled by separate pacemakers.
- ❍ Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the pacemaker focus, usually in the AV node.
- ❍ Wide QRS rhythm suggests a ventricular escape focus (i.e., idioventricular rhythm).

3^0 AV Block with Junctional Escape Focus

3. AV Dissociation (independent rhythms in atria and ventricles):

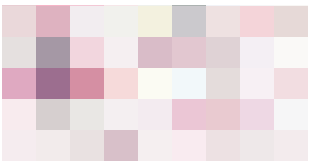
- ❍ Not synonymous with 3rd degree AV block.
- ❍ May be complete or incomplete. In complete AV dissociation the atria and ventricles are always independent of each other. In incomplete AV dissociation there is either intermittent atrial capture from the ventricular focus or ventricular capture from the atrial focus.
- ❍ There are three categories of AV dissociation (categories 1 & 2 are always incomplete AV dissociation):
  - ❒ Slowing of the primary pacemaker (i.e., SA node); subsidiary escape pacemaker takes over by default:
  - ❒ Acceleration of a subsidiary pacemaker. e.g. VT.
  - ❒ 3rd degree AV block with escape rhythm from junctional focus or ventricular focus.

4. Right Bundle Branch Block (RBBB):

- ❍ "Complete" RBBB has a QRS duration >0.12 sec
- ❍ Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized after the left ventricle. This means the following:
  - ❒ Terminal R' wave in lead V1 (usually see rSR' complex)

- ❒ Terminal S waves in leads I, aVL, V6 indicating late rightward forces
- ❒ Terminal R wave in lead aVR indicating late rightward forces

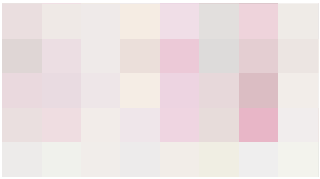
- ❍ The frontal plane QRS axis in RBBB should be around 90° If left axis deviation is present, think about left anterior fascicular block, and if right axis deviation is present, think about left posterior fascicular block in addition to the RBBB.
- ❍ "Incomplete" RBBB has QRS duration of 0.10 - 0.12 sec with the same terminal
- ❍ QRS features. This is often a normal variant.QRS features. This is often a normal variant.
- ❍ The "normal" ST-T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards. If the ST-T waves are in the same direction as the terminal QRS forces, they should be labeled primary ST-T wave abnormalities.
- ❍ In a patient with RBBB. ST-T wave abnormalities may be related to ischemia, infarction, electrolyte abnormalities, medications, CNS disease, etc. (i.e., they are nonspecific and must be correlated with the patient's clinical status).



5. Left Bundle Branch Block (LBBB):

- ❍ "Complete" LBBB" has a QRS duration >0.12 sec
- ❍ Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented leftward and posteriorly because the left ventricle is depolarized after the right ventricle.

- ❒ Terminal S waves in lead V1 indicating late posterior forces
- ❒ Terminal R waves in lead I, aVL, V6 indicating late leftward forces; usuallybroad, monophasic R waves, poor R progression from V1 to V3 is common.



The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards; in leads with terminal S forces the ST-T should be upwards. If the ST-T waves are in the same direction as the terminal QRS forces, they should be labeled primary ST-T wave abnormalities. In the above ECG the ST-T waves are "normal" for LBBB; i.e., they are secondary to the change in the ventricular depolarization sequence.

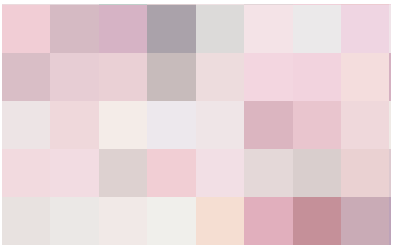
"Incomplete" LBBB looks like LBBB but QRS duration = 0.10 to 0.12 sec, with less ST-T change. This is often a progression of LVH.

6. **Left Anterior Fascicular Block (LAFB):** is the most common intraventricular conduction defect
   - ❍ Left axis deviation in frontal plane, usually -45 to -90 degrees
   - ❍ rS complexes in leads II, III, aVF
   - ❍ Small q-wave in leads I and/or aVL
   - ❍ QRS duration usually <0.12 sec unless coexisting RBBB
7. **Left Posterior Fascicular Block (LPFB):** Very rare intraventricular defect
   - ❍ Right axis deviation in the frontal plane (usually > +100 degrees)
   - ❍ rS complex in lead I
   - ❍ qR complexes in leads II, III, aVF, with R in lead III > R in lead I
   - ❍ QRS duration usually <0.12 sec unless coexisting RBBB

- Must first exclude (on clinical grounds) other causes of right axis deviation such as cor-pulmonale, pulmonary heart disease, pulmonary hypertension, etc., because these conditions can result in the identical ECG picture!

8. Bifascicular Blocks:

- RBBB plus either LAFB (common) or LPFB (uncommon)
- Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.)



The above ECG shows classic RBBB (note rSR' in V1) plus LAFB

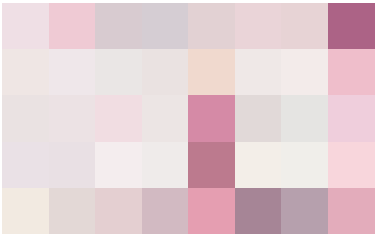
9. Nonspecific Intraventricular Conduction Defects (IVCD):

- QRS duration >0.10 sec indicating slowed conduction in the ventricles
- Criteria for specific bundle branch or fascicular blocks not met
- Causes of nonspecific IVCD's include:
  - Ventricular hypertrophy (especially LVH), Myocardial infarction (so called peri-infarction blocks), Drugs: class IA and IC antiarrhythmics (e.g., quinidine, flecainide), Hyperkalemia.

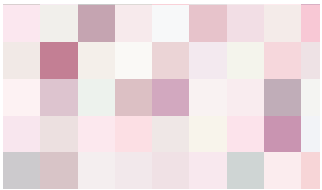
10. Wolff-Parkinson-White Preexcitation:

Although not a true IVCD, this condition causes widening of QRS complex and, therefore, deserves to be considered here

- QRS complex represents a fusion between two ventricular activation fronts:
  - Early ventricular activation in the accessory AV pathway (Bundle of Kent)
  - Ventricular activation through the normal AV junction, bundle branch system
  - ECG criteria include all of the following:
    - Short PR interval (<0.12 sec)
    - Initial slurring of QRS complex (delta wave) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway
    - Prolonged QRS duration (usually >0.10 sec)
    - Secondary ST-T changes due to the altered ventricular activation sequence



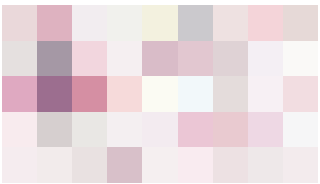
- QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).
- Delta waves, if negative in polarity, may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.



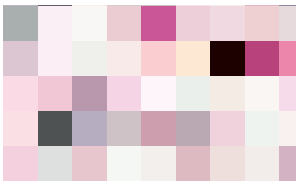
ECG 15: Describe the rhythm abnormality

Second degree 2:1 block and Bifascicular block

ECG 16: Describe the rhythm abnormality



First degree AV block with RBBB

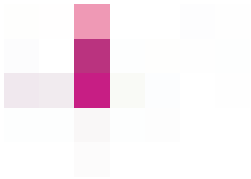


ECG 17: Describe the rhythm abnormality

Second degree Mobitz Type 2 and Bifascicular block

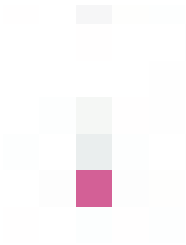
Atrial Enlargement

1. Right Atrial Enlargement (RAE)



- P wave amplitude > 2.5 mm in II and/or > 1.5 mm in V1 (these criteria are not very specific or sensitive)
- Better criteria can be derived from the QRS complex; these QRS changes are due to both the high incidence of RVH when RAE is present, and the RV displacement by an enlarged right atrium.

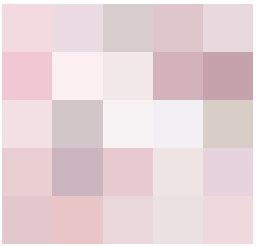
2. Left Atrial Enlargement (LAE)



- P wave duration > 0.12 s in frontal plane (usually lead II)
  - Notched P wave in limb leads with the inter-peak duration > 0.04 sec
  - Terminal P negativity in lead V1 > 0.04 sec, depth > 1 mm.
  - Sensitivity = 50%
  - Specificity = 90%

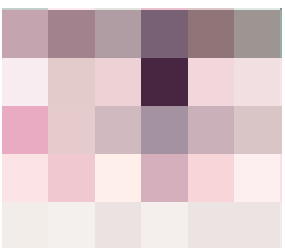
3. Bi-Atrial Enlargement (BAE)

- ⇨ Features of both RAE and LAE in same ECG
- ⇨ P wave in lead II >2.5 mm tall and >0.12 sec in duration
- ⇨ Initial positive component of P wave in V1 >1.5 mm tall and prominent P-terminal force



ECG 18: Describe the P waves, Diagnosis?

Right atrial hypertrophy



ECG 19: Describe the P waves, Diagnosis?

Left atrial hypertrophy, probable ischemia

Ventricular Hypertrophy

1. Introduction :

➪ The ECG criteria for diagnosing right or left ventricular hypertrophy are very insensitive (i.e., sensitivity -50%, which means that -50% of patients with ventricular hypertrophy cannot be recognized by ECG criteria). However, the criteria are very specific (i.e., specificity >90%, which means if the criteria are met, it is very likely that ventricular hypertrophy is present).

2. Left Ventricular Hypertrophy (LVH)

- ❍ QRS amplitude (tall R-waves in LV leads, deep S-waves in RV leads)
- ❍ Left ventricular strain pattern, i.e. T inversion in V4-V6
- ❍ Leftward shift in frontal plane QRS axis
- ❍ Evidence for left atrial enlargement (LAE)

Estes Criteria for LVH ("diagnostic", > 5 points; "probable", 4 points)

ECG Criteria	Points
Voltage Criteria (any of): R or S in limb leads >20 mm S in V1 or V2 > 30 mm R in V5 or V6 > 30 mm	3 points
ST-T Abnormalities: Without digitalis With digitalis	 3 points 1 point
Left Atrial Enlargement in V1	3 points
Left axis deviation	2 points
QRS duration 0.09 sec	1 point
Delayed intrinsicoid deflection in V5 or V6 (> 0.05 sec)	1 point

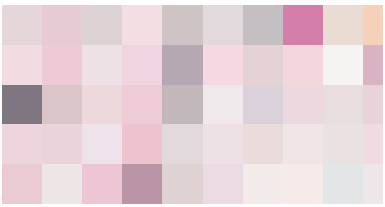
➪ **CORNELL Voltage Criteria for LVH**
(sensitivity = 22%, specificity = 95%)

- ❍ S in V3 + R in aVL > 28 mm (men)
- ❍ S in V3 + R in aVL > 20 mm (women)

➪ **Other Voltage Criteria for LVH**

- ❍ Limb-lead voltage criteria:
  - ❒ R in aVL >11 mm or, if left axis deviation,
  - ❒ R in aVL >13 mm plus S in III >15 mm
  - ❒ R in I + S in III >25 mm

  Chest-lead voltage criteria:
  - ❒ S in V1 + R in V5 or V6 > 35 mm



3. Right Ventricular Hypertrophy (RVH)

➪ General ECG features include:

- ❍ Right axis deviation (>90 degrees)
- ❍ Tall R-waves in RV leads; deep S-waves in LV leads
- ❍ Slight increase in QRS duration
- ❍ ST-T changes directed opposite to QRS direction
- ❍ May see incomplete RBBB pattern or qR pattern in V1
- ❍ Evidence of right atrial enlargement (RAE)

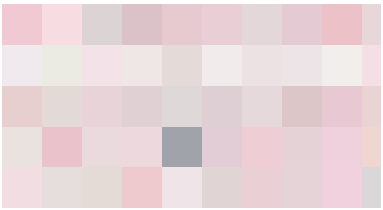
➪ Specific ECG features:

- ❍ Any one or more of the following (if QRS duration <0.12 sec):
  - ❒ Right axis deviation (>90 degrees) in presence of disease causing RVH

- ❒ R/S ratio > 1 and negative T wave in V1
- ❒ R > 6 mm in V1
- ❒ R/S ratio in V5 or V6 < 1

❍ ST segment depression and T wave inversion in right precordial leads is seen in severe RVH

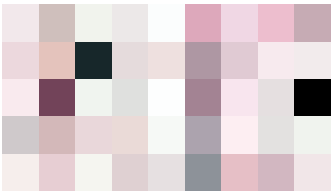
❍ Seen in pulmonary stenosis and pulmonary hypertension.



4. Biventricular Hypertrophy (difficult ECG diagnosis to make)

⇨ In the presence of LAE any one of the following suggests this diagnosis:

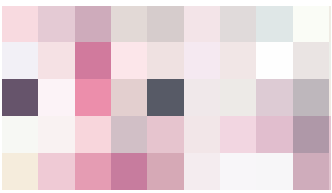
- ❍ R/S ratio in V5 or V6 < 1, S in V5 or V6 > 6 mm, RAD (>90 degrees), Criteria for LVH and RVH both met.
- ❍ LVH criteria met and RAD or RAE present.



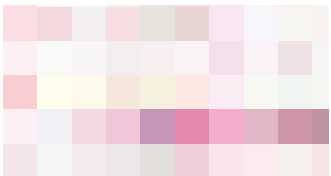
ECG 20: Guess the ventricle abnormality

Left ventricular hypertrophy

ECG 21: Guess the ventricle abnormality



Severe Right ventricular hypertrophy



ECG 22: Describe the abnormality

Old inferior myocardial infarction

Myocardial Infarction

1. Introduction

Rupture of an atherosclerotic plaque followed by acute coronary thrombosis is the usual mechanism of acute MI. The ECG changes reflecting this sequence usually follow a well-known pattern depending on the location and size of the MI. MI's resulting from total coronary occlusion result in more homogeneous tissue damage and are usually reflected by a Q-wave MI pattern on the ECG. MI's resulting from subtotal occlusion result in more heterogeneous damage, which may be evidenced by a non Q-wave MI pattern on the ECG. Two-thirds of MI's presenting toemergency rooms evolve to non-Q wave MI's, most having ST segment depression or T wave inversion.

- ⇨ Most MI's are located in the left ventricle. In the setting of a proximal right coronary artery occlusion, however, up to 50% may also have a component of right ventricular infarction as well. Right-sided chest leads are necessary to recognize RV MI.
- ⇨ In general, the more leads of the 12-lead ECG with MI changes (Q waves and ST elevation), the larger the infarct size and the worse the prognosis. Additional leads on the back, V7-9 (horizontal to V6), may be used to improve the recognition of true posterior MI.
- ⇨ The left anterior descending coronary artery (LAD) and it's branches usually supply the anterior and anterolateral walls of the left ventricle and the anterior two-thirds of the septum. The left circumflex coronary artery (LCX) and its branches usually supply the posterolateral wall of the left ventricle. The right coronary artery (RCA) supplies the right ventricle, the inferior and true posterior walls of the left ventricle, and the posterior third of the septum. The RCA also gives off the AV nodal coronary artery in 85-90% of individuals; in the remaining 10-15%, this artery is a branch of the LCX.

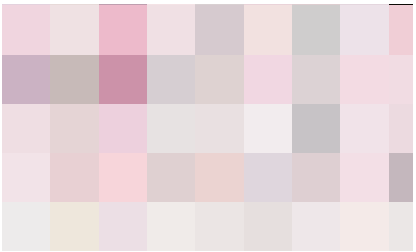
- Usual ECG evolution of a Q-wave MI; not all of the following patterns may be seen; the time from onset of MI to the final pattern is quite variable and related to the size of MI, the rapidity of reperfusion (if any), and the location of the MI.
  - Normal ECG prior to MI
  - Hyperacute T wave changes - increased T wave amplitude and width; may also see ST elevation
  - Marked ST elevation with hyperacute T wave changes (transmural injury)
  - Pathologic Q waves, less ST elevation, terminal T wave inversion (necrosis)
  - Pathologic Q waves have duration >0.04 s or >25% of R-wave amplitude)
  - Pathologic Q waves, T wave inversion (necrosis and fibrosis)

    Pathologic Q waves, upright T waves (fibrosis)

2. Inferior MI Family of Q-wave MI's

(Inferior, true posterior and right ventricular MI's)

- Inferior MI
  - Evolving ST-T changes in leads II, III, aVF



- ⇨ True posterior MI
  - ❍ ECG changes are seen in anterior precordial leads V1-3, but are the mirror image of an anteroseptal MI:
    - ❒ Increased R wave amplitude and duration (i.e., a "pathologic R wave" is a mirror image of a pathologic Q)
    - ❒ R/S ratio in V1 or V2 >1 (i.e., prominent anterior forces)
    - ❒ Hyperacute ST-T wave changes: i.e., ST depression and large, inverted Twaves in V1-3
    - ❒ Late normalization of ST-T with symmetrical upright T waves in V1-3
  - ❍ Often seen with inferior MI (i.e., "inferoposterior MI")
- ⇨ Right Ventricular MI

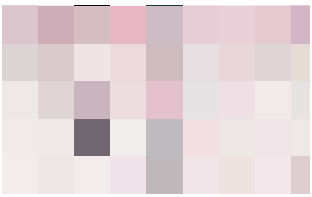
  (Only seen with proximal right coronary occlusion; i.e., with inferior family MI's)

  ECG findings usually require additional leads on right chest (V1R to V6R, analogous to the left chest leads)

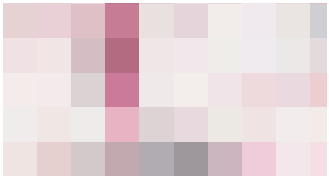
  ST elevation, >1mm, in right chest leads, especially V4R (see below)

3. Anterior Family of Q-wave MI's

- ⇨ Anteroseptal MI
  - ❍ Q, QS, or qrS complexes in leads V1-V3 (V4)
  - ❍ Evolving ST-T changes



- Anterior MI (similar changes, but usually V1 is spared; if V4-6 involved call it "anterolateral")
- High Lateral MI (typical MI features seen in leads I and/or aVL)



Anterolateral Myocardial Infarction

4. MI with Bundle Branch Block

- MI + Right Bundle Branch Block

  Usually easy to recognize because Q waves and ST-T changes are not altered by the RBBB

- MI + Left Bundle Branch Block
  - Often a difficult ECG diagnosis because in LBBB the right ventricle is activated first and left ventricular infarct Q waves may not appear at the beginning of the QRS complex (unless the septum is involved).
  - Suggested ECG features, not all of which are specific for MI include:
    - Q waves of any size in two or more of leads I, aVL, V5, or V6 (See below: one of the most reliable signs and probably indicates septal infarction, because the septum is activated early from the right ventricular side in LBBB)
    - Reversal of the usual R wave progression in precordial leads
    - Notching of the down stroke of the S wave in precordial leads to the right of the transition zone (i.e., before QRS changes from a predominate S wave complex to a predominate R wave complex); this may be a Q- wave equivalent.

- ❒ Notching of the upstroke of the S wave in precordial leads to the right of the transition zone (another Q-wave equivalent).
- ❒ "Primary" ST-T wave changes (i.e., ST-T changes in the same direction as the QRS complex rather than the usual "secondary" ST-T changes seen in uncomplicated LBBB); these changes may reflect an acute, evolving MI.

5. Non-Q Wave MI

- ⇨ Recognized by evolving ST-T changes over time without the formation of pathologic Q waves (in a patient with typical chest pain symptoms and/or elevation in myocardial-specific enzymes)
- ⇨ Although it is tempting to localize the non-Q MI by the particular leads showing ST-T changes, this is probably only valid for the ST segment elevation pattern
- ⇨ Evolving ST-T changes may include any of the following patterns:
  - ❍ Convex downward ST segment depression only (common)
  - ❍ Convex upwards or straight ST segment elevation only (uncommon)
  - ❍ Symmetrical T wave inversion only (common)
  - ❍ Combinations of above changes

6. The Pseudo infarcts

- ⇨ These are ECG conditions that mimic myocardial infarction either by simulating pathologic Q or QS waves or mimicking the typical ST-T changes of acute MI.
  - ❍ WPW pre-excitation (negative delta wave may mimic pathologic Q waves) IHSS (septal hypertrophy may make normal septal Q waves "fatter" thereby mimicking pathologic Q waves)
  - ❍ LVH (may have QS pattern or poor R wave progression in leads V1-3)
  - ❍ RVH (tall R waves in V1 or V2 may mimic true posterior MI)
  - ❍ Complete or incomplete LBBB (QS waves or poor R wave progression in leads V1-3)

- Pneumothorax (loss of right precordial R waves)
  Pulmonary emphysema and cor pulmonale (loss of R waves V1-3 and/or inferior Q waves with right axis deviation)
- Left anterior fascicular block (may see small q-waves in anterior chest leads) Acute pericarditis (the ST segment elevation may mimic acute transmural injury)
- Central nervous system disease (may mimic non-Q wave MI by causing diffuse ST-T wave changes)

7. Miscellaneous Abnormalities of the QRS Complex:

⇨ The differential diagnosis of these QRS abnormalities depend on other ECG findings as well as clinical patient information

⇨ Poor R Wave Progression - Loss of or no R waves in leads V1-3 (R < 2mm):

- Normal variant (if the rest of the ECG is normal)
- LVH (look for voltage criteria and ST-T changes of LV "strain")
- Complete or incomplete LBBB (increased QRS duration)
- Left anterior fascicular block (should see LAD in frontal plane)
- Anterior or anteroseptal MI
- Emphysema and COPD (look for R/S ratio in V5-6 <1)
- Diffuse infiltrative or myopathic processes
- WPW pre-excitation (look for delta waves, short PR)

⇨ Prominent Anterior Forces - defined as R/S ration >1 in V1 or V2

- Normal variant (if rest of the ECG is normal)
- True posterior MI, RVH, Complete or incomplete RBBB, WPW.

ECG 23: Describe the abnormality

ST Segment Abnormalities

1. Introduction

⇨ The specificity of ST-T and U wave abnormalities is provided more by the clinical circumstances in which the ECG changes are found than by the particular changes themselves. Thus the term, nonspecific ST-T wave abnormalities, is frequently used when the clinical data are not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant! It is the responsibility of the clinician providing care for the patient to ascertain the importance of the ECG findings.

⇨ Factors affecting the ST-T and U wave configuration include:

- ❍ Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes)
- ❍ Drugs (e.g., digoxin, quinidine, tricyclics, and many others)
- ❍ Electrolyte abnormalities of potassium, magnesium, calcium
- ❍ Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
- ❍ Metabolic factors (e.g., hypoglycemia, hyperventilation)
- ❍ Atrial repolarization (e.g., at fast heart rates the atrial T wave may pull down the beginning of the ST segment)
- ❍ Ventricular conduction abnormalities and rhythms

⇨ "Secondary" ST-T Wave changes (these are normal ST-T wave changes solely due to alterations in the sequence of ventricular activation)

- ❍ ST-T changes seen in bundle branch blocks (generally the ST-T polarity is opposite to the major or terminal deflection of the QRS)
- ❍ ST-T changes seen in fascicular block
- ❍ ST-T changes seen in nonspecific IVCD

- ST-T changes seen in WPW pre-excitation
- ST-T changes in PVCs, ventricular arrhythmias, and ventricular paced beats

⇨ "Primary" ST-T Wave Abnormalities (ST-T wave changes that are independent of changes in ventricular activation and that may be the result of global or segmental pathologic processes that affect ventricular repolarization)

- Drug effects (e.g., digoxin, quinidine, etc)
- Electrolyte abnormalities (e.g., hypokalemia)
- Ischemia, infarction, inflammation, etc
- Neurogenic effects (e.g., subarrachnoid hemorrhage causing long QT)

2. Differential Diagnosis of ST Segment Elevation

⇨ Normal Variant "Early Repolarization" (usually concave upwards, ending with symmetrical, large, upright T waves)

⇨ Ischemic Heart Disease (usually convex upwards, or straightened)

- Acute transmural injury - as in this acute anterior MI
- Persistent ST elevation after acute MI suggests ventricular aneurysm
- ST elevation may also be seen as a manifestation of angina (coronary artery spasm)Prinzmetal's (variant)
- ST elevation during exercise testing suggests extremely tight coronary artery stenosis or spasm (transmural ischemia)

⇨ Acute Pericarditis

- Concave upwards ST elevation in most leads except aVR
- No reciprocal ST segment depression (except in aVR)
- Unlike "early repolarization", T waves are usually low amplitude, and heart rate is usually increased.
- May see PR segment depression, a manifestation of atrial injury

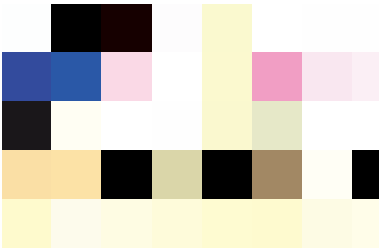
⇨ Other Causes:

- Left ventricular hypertrophy (in right precordial leads with large S-waves)

- ❍ Left bundle branch block (in right precordial leads with large S-waves)
- ❍ Advanced hyperkalemia
- ❍ Hypothermia (prominent J-waves or Osborne waves)

3. Differential Diagnosis of ST Segment Depression

❒ ST segment depression is characterized as horizontal, up sloping or down sloping



Note: "Up sloping" ST depression is not an ischemic abnormality

- ❍ Non Q-wave MI
- ❍ Reciprocal changes in acute Q-wave MI (e.g., ST depression in leads I & aVL with acute inferior MI)

⇨ Normal variants or artifacts:

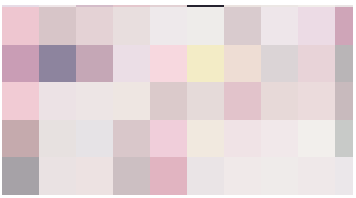
- ❍ Pseudo-ST-depression (wandering baseline due to poor skin-electrode contact)
- ❍ Physiologic J-junctional depression with sinus tachycardia (most likely due to atrial repolarization)
- ❍ Hyperventilation-induced ST segment depression

⇨ Ischemic heart disease

- ❍ Subendocardial ischemia

- ⇨ Nonischemic causes of ST depression
  - ❍ RVH (right precordial leads) or LVH (left precordial leads, I, aVL)
  - ❍ Digoxin effect on ECG
  - ❍ Hypokalemia
  - ❍ Mitral valve prolapse (some cases)
  - ❍ CNS disease

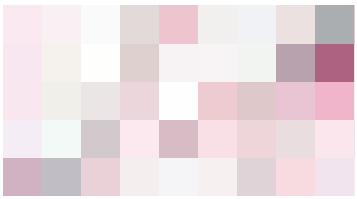
RBBB, LBBB, WPW,



ECG 24: Describe the abnormality

Gross horizontal ST depression in V2-6, probable ischemia

ECG 25: What is your diagnosis?



Hypertrophic cardiomyopathy

T Wave Abnormalities

1. Introduction

- ⇨ T wave represents the recovery period of the ventricles, when they recruit their spent electrical forces (repolarization). T wave is the most labile wave in the ECG. We notice the direction, shape and height of T waves.
- ⇨ Normal T wave is usually in the same direction as the QRS except in the right precordial leads. It is asymmetric with the first half moving more slowly than the second half.

In normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR. The other leads are

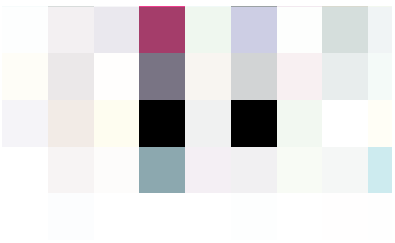
- ⇨ variable depending on the direction of the QRS and the age of the patient.

2. Differential Diagnosis of T wave Abnormalities

- ⇨ Sharply pointed T waves are seen with myocardial infarction
- ⇨ Notching of T waves is seen in children and in pericarditis.
- ⇨ Tall T waves
  - ❍ Myocardial ischemia, infarction,
  - ❍ Cerebrovascular accidents
  - ❍ LV overload with severe mitral regurgitation
  - ❍ Hyperkalemia.
  - ❍ Normal variant.
- ⇨ Flattened T waves
  - ❍ Obesity (Normalize with reduction of weight)
- ⇨ T Wave Inversion :
  - ❍ Q wave and non-Q wave MI
  - ❍ Myocardial ischemia
  - ❍ Post infarction evolution

- Subacute or old pericarditis
- Pericardial effusion
- Myocarditis or Traumatic Myocardial contusion
- Subarrachnoid hemorrhage
- Mitral valve prolapse, pneuthorax, hyperventilation
- RVH and LVH with strain
- Cardiomyopathy
- Digoxin effect - Inverted T as a 'Reverse correct sign'
- Electrolyte imbalance
- Normal variant

ECG 26: Describe the Twave abnormality, what is the probable cause?

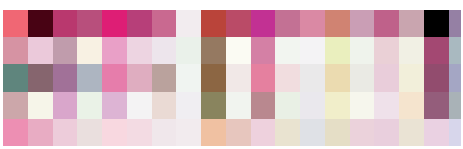


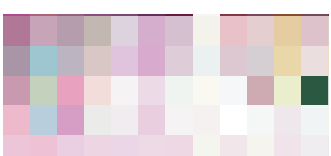
Tall, peaked Twaves due to hyperkalemia

Nice Seeing "U" Again

1. Introduction:

The U wave is the only remaining enigma of the ECG, and probably not for long. The origin of the U wave is still in question, although most authorities correlate the U wave with electrophysiologic events called "afterdepolarizations" in the ventricles. These afterdepolarizations can be the source of arrhythmias caused by "triggered automaticity" including torsade de pointes. The normal U wave has the same polarity as the T wave and is usually less than one-third the amplitude of the T wave. U waves are usually best seen in the right precordial leads especially V2 and V3. The normal U wave is asymmetric with the ascending limb moving more rapidly than the descending limb (just the opposite of the normal T wave).





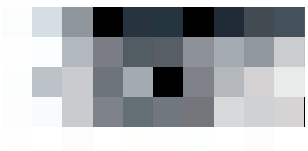
2. Differential Diagnosis of U Wave Abnormalities

Prominent upright U waves

- Sinus bradycardia accentuates the U wave
- Hypokalemia (remember the triad of ST segment depression, low amplitude
- T waves, and prominent U waves)
- Quinidine and other type 1A antiarrhythmics
- CNS disease with long QT intervals (often the T and U fuse to form a giant "T-U fusion wave")
- LVH (right precordial leads with deep S waves)
- Mitral valve prolapse (some cases)
- Hyperthyroidism

Negative or "inverted" U waves

- Ischemic heart disease (often indicating left main or LAD disease)
  - Myocardial infarction (in leads with pathologic Q waves)
  - During episode of acute ischemia (angina or exercise-induced ischemia)
  - Post extrasystolic in patients with coronary heart disease
  - During coronary artery spasm (Prinzmetal's angina)
- Nonischemic causes
  - Some cases of LVH or RVH (usually in leads with prominent R waves)
  - Some patients with LQTS



QT Prolongation with deep T inversion

APPLICATION OF NANOTECHNOLOGY IN MEDICINE

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PRACTITIONERS' GUIDANCE SERIES - XIV

Application of Nanotechnology in Medicine

Richard Feynman

Introduction

Nano(Greek: dwarf, one billionth of something)medicine took its roots when Nobel Physicist Richard Feynman (1959) predicted the emergence of a new Science called Nanotechnology. He said, 't here were no fundamental physical reasons that materials could not be fabricated by manoeuvring individual atoms'.

Richard Feynman who worked on Manhattan Project at Los Almos made a statement that 'there is plenty of room at the bottom', and proposed using machine tools to make smaller machine tools. They can be used in turn to make still smaller machine tools, and so on all the way down to the atomic level. He visualized that it is 'a development which cannot be avoided'/

Utilizing that technology, very small machine tools could be produced and introduced into the human body to undertake cellular repairs at the molecular level. Nanomachine tools, nanodevices and nanorobats that are designed, and manufactured could help in undertaking a variety of atomically precise microscopic instrumentation. Nanotechnology deals with structures one to one hundred nanometers (nm) in scale. The advances that have occurred in the field of nanotechnology in the recent years have made significant impact in all scientific fields. The technique has potential medical applications.

APPLICATION OF NANOTECHNOLOGY IN MEDICINE

Nanomedicine

Nanomedicine refers to the medical diagnosis, monitoring and treatment at the level of single molecules or molecular assemblies providing structure, control, signaling, homeostasis and motility in the cells of nanoscale of 100 nm or less (1). Nanomedicine is the application of nanotechnology to medicine and it utilizes the molecular tools and molecular knowledge of the human body. It is possible to provide therapy at a molecular level and improve human health by utilizing molecular tools and molecular knowledge of the human body.

Conventional drugs therapy surfers from major limitation of adverse effects due to the non-specificity their action. Even improper dosage affects their efficacy. It is now possible to design the drugs with greater degree of cell specificity and provide therapy at a molecular level. Nanomedicine is bringing about a change in the methodology by which drugs and sustained release preparations are delivered (2). Thus it provides targeted drug delivery. It improves efficacy and minimizes adverse effects of the drugs. Tools are being developed to take images of structures at a molecular level and to undertake high speed measurement of the molecular assemblies. Such tools are likely to control and manipulate a variety of nanoscale structures in living cells.

Nanomedicine helps in better understanding of the pathophysiology of different diseases at the molecular scale and the dynamic behaviour of abnormally functioning cellular machinery. It enables to provide specific treatment of diseases. Miniaturized medical tools will provide more accurate, more controllable, more versatile, more reliable, faster approaches to enhance the quality of human life (3). By using nanoscale-structured materials, simple nanodevices, nanomedicine has addressed itself into many medical problems.

Newer methods of drug delivery systems based on nanotechnology methods are being developed. These are being used in the treatment of cancer, diabetes, viral infections, fungal disorders and in gene therapy. The advantage of this mode of therapy is targeting the drug to the site of disease, and increased safety profile. In addition nanotechnology is utilized in the diagnostic procedures by using contract agents, fluorescent dyes and magnetic nanoparticles (4).

Nanomedical devices

Some of the examples of nanomedical devices are as follows:

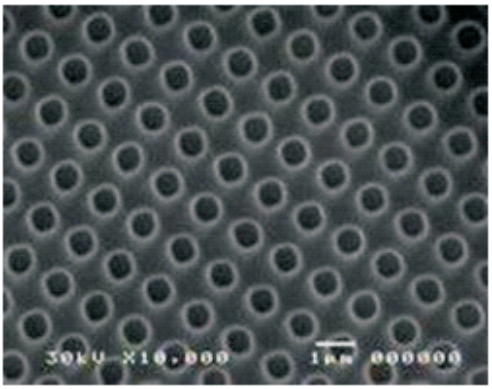
i) **Nonoporous biocapsules:** Tiny chambers can be created within single crystalline silicon wafers containing biological cells, with a surface perforated with very tiny holes or nanopores as small as 20 nm in diameter (5). These pores though allow small molecules such as oxygen, glucose, and insulin to pass into the surrounding biologic environment. They do not allow entry of larger molecules such as immunoglobulins and cells into the chamber. Thus it is able to make immunoisolation. It enables encapsulated rat pancreatic cells to receive nutrients and remain healthy for weeks. It is able to secrete insulin and allow its passage through the pores while remaining hidden from immune system. If such an arrangement was not there, it would have been attacked and rejected the foreign cells. Nanopores can be utilized to protect transplanted tissues form the host immune system while retaining the benefits of transplantation. This device has a place in the treatment of type 1 diabetes mellitus.

Nanoporous membrane with 24.5-nm pores.
Courtesy: Leon L, Desai TA: Nanoporous biocapsules, IEEE Transactions
Biomedical Engineering 2001: 48; 1335-41

Nanoporous biocapsules containing pig islet cells could be implanted under the skin of diabetic patients to restore the body's glucose control feedback loop without being affected by immunosupprrsants (6). This observation has given an opportunity to introduce encapsulated new cells to body

exhibiting deficiency diseases and neurologic disorders having deficiency of neurotransmitters as seen in Alzheimer's disease and Parkinson's disease.

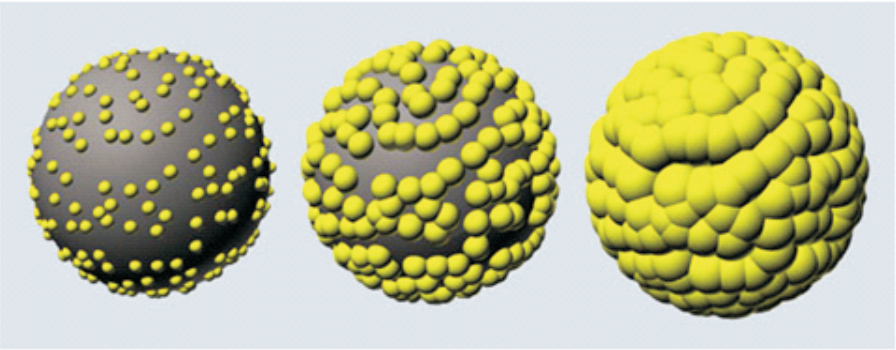
ii) **Nanosieves**: It is possible to regulate the flow of materials that is occurring through nanopores by utilizing an artificial voltage-gated molecular nanosieve. They contain tiny tubules of an internal diameter of 1.6 nm. Positive charging of these tubules, excludes positive ions and facilitates transportation of negative ions through the membrane. In presence of a negative voltage, only positively charged ions pass. Voltage-gated nanosieves are able to control transportation of ions such as enzymes, antibodies and other proteins (7).



Nanosieves
Courtesy: Huang K et al
Photon-nanosieve for ultra broad band and
Large angle- ofview holograms, Wiley online library 10 Apr 2017

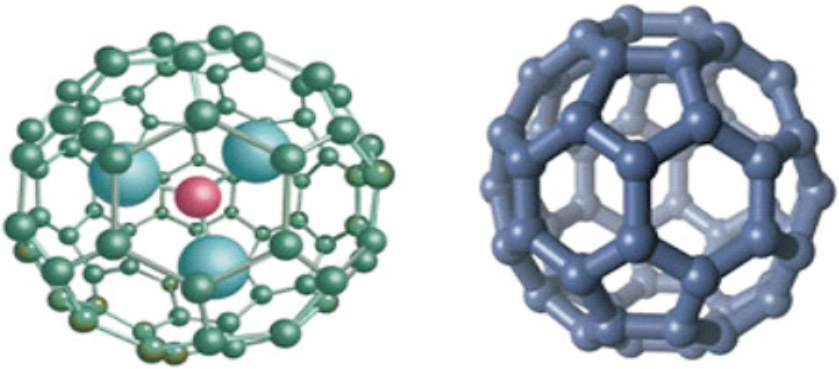
iii) **Nanoshells:** Nanoshell is a platform for nanoscale drug delivery. Nanoshells made of gold-coated silica nanospheres can be embedded in a drug-containing tumour-targetted hydrogel polymer and then injected into the body. It will concentrate near tumour cells. On heating with an infrared laser from a source outside the body, the nanoshells selectively absorb a specific infra-red frequency, melt and release the drug at a specific location. Thus they exhibit thermo-ablative property. These nanoshells may be utilized in the treatment of diabetes mellitus where the injected nanoshell polymer can release a pulse of insulin on heating the skin using a tiny infrared laser (8). Such a system can stay in the body for several months and it takes away the need for regular injections of insulin several times a day. The

same approach is being made in the treatment of micrometastases.



Courtesy: Genome News Network (N Halas)

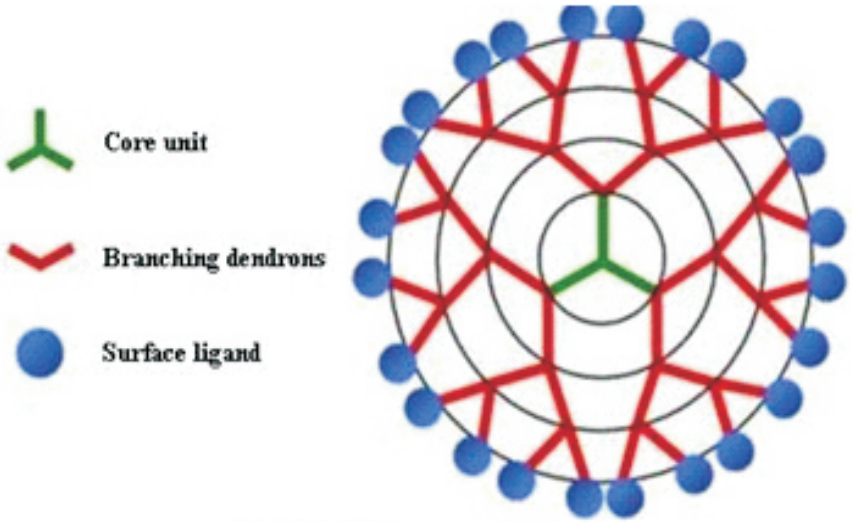
iv) Fullerene-based pharmaceuticals: Fullerenes or bucky balls are carbon allotropes. Soluble derivative of fullerenes having 60 carbon atoms ('the buckminister') arranged in a shape of a truncated icosahedrons, can find a place in pharmatherapy. As they exhibit good biocompatibility and low-toxicity, they can act as antiviral agents, antibacterial agents, antimitotic agents, antioxidants and antiapoptosis agents (3).



Metallofullerene and empty cage fullerene
Source: Zhou Z Liposome formulation of fullerene-based molecular diagnostic and therapeutic agents Pharmaceutics 2013; 5: 525-51

v) **Tectodendrimers:** Tectodendrimers are a single core, tree-shaped synthetic molecules formed within a regular branching structure called dendrimer to which several dendrimer modules are attached, each of which is able to function as a therapeutic nanodevice. These nanodevices can recognize the diseased cells, their location and drug delivery and report the response. Chemotherapeutic-releasing dendrimer modules attached to the core dendrimer can be made to recognize cancer cells and release cytotoxic agents to destroy such cells specifically, without affecting healthy normal cells (9).

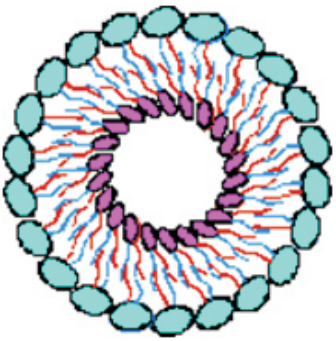
Dendrimers can replace conventional viral vectors in gene therapy. They enter the cells by endocytosis, where the DNA gets transported into nucleus for transcription of the applied gene. They do not stimulate any immune reaction. Dendrimers have found a place as contrast agents for imaging.



Basic dendrimer components

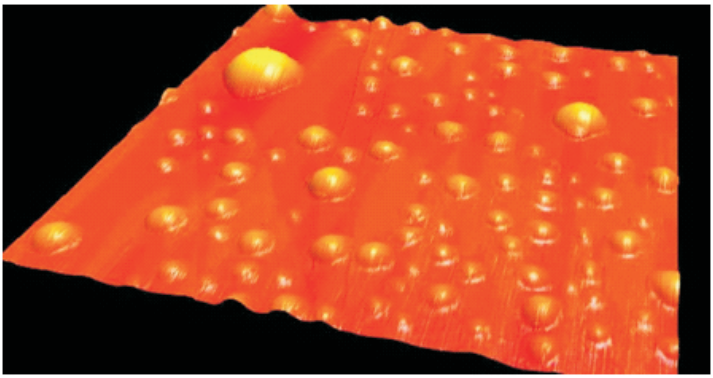
Courtesy: Lee K; Designing dendrimeres for biomedical applications
University of California, Irvine, Biomedical Engineering

vi) **Nanosomes:** Nanosomes acting as probes encapsulated by biologically localized embedding (PEBBLE) are being utilized for diagnosis and therapy of brain tumours. Polyethylene glycol coated silica nanoparticles along with targeting antibody and contrast gadolinium are used to access specific areas of brain affected with tumour (10). The binding of nanoparticles specifically to the tumour cells with the targeting aids and the contrast helps in proper delineation of the growth with magnetic resonance imaging. Later laser can destroy the cells loaded with nanoparticles by heat generated by iron oxide following absorption of infra red light.



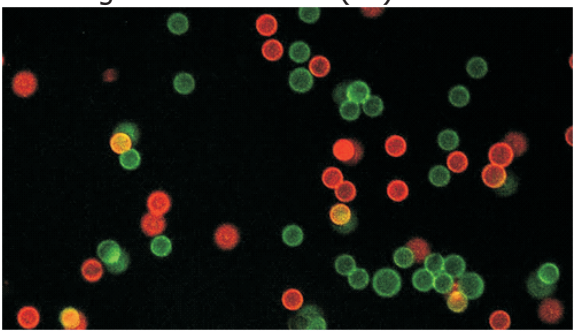
Structure of a nanosome
Courtesy: Elsom Research Innovative Biotechnology

vii) **Nanobubbles:** Nanoscaled bubble-like structures referred to as nanobubbles can be utilized to incorporate anti-cancer drugs. These bubbles remain stable at room temperature. They get coalesced to form microbubbles when heated to physiological temperature within the body. They are able to target the tumour tissue and deliver the drug selectively under the influence of ultrasound exposure. This enables an increased uptake of the drug by the tumour cells. Doxorubicin has been administered by incorporating it into nanobubbles. It has shown to reach the tumour tissue through leaky vasculature and get accumulated there (11).It leads to formation of microbubbles by coalescing of nanobubbles and it can be seen by ultrasound techniques. When the site is focused with high-intensity focused ultrasound (HIFU), there is disruption of the microbubbles leading to release of the drug. It gets concentrated in the target cells.



Nanobubbles at the interface between hydrophobic silica and water.
Image Credits: Virginia Tech

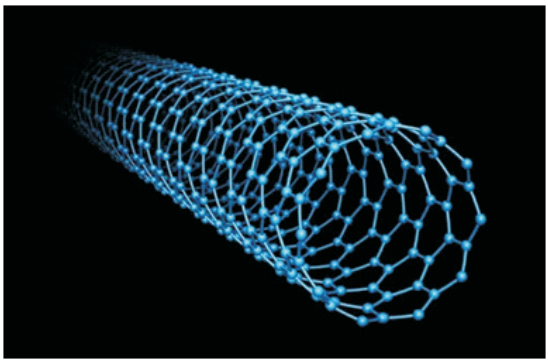
viii) Quantum dots: Quantum dots are 2-10 nm sized-nanocrystals which can be made to fluorescence on stimulation by light. They consist of an inorganic core, an inorganic shell and an aqueous organic coating to which biomolecules are conjugated. They can be used for diagnosis and treatment. They can be used as highly sensitive probes after tagging with biomolecules. Quantum dots conjugated with polyethylene glycol and antibody to prostate specific antigen (PSA) are able to get accumulated prostate tumour tissue (12). Quantum dots can be used to image sentinel node in cancer. This will enable to stage the tumour and guide treatment regimen. Quantum dot probes proved real time imaging of the sentinel node with Near Intra-red fluorescence system. It is able to produce much brighter fluorescence (13).



Quantum dots

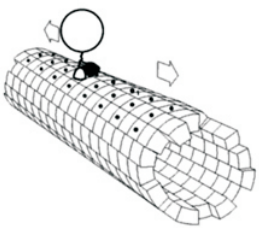
Courtesy: Morrison G How quantum dots supercharge farming, medicine and solar too

ix) Nanotubes: Nanotubes are tubular structures like a sheet of graphite rolled into a cylinder capped at one or both ends by a buckyball. These carbon tubes may be single-walled, or multi-walled. The former has an internal diameter of 1-2 nm and the latter 2-25 nm. These tubes possess greater strength and stability. Hence they can be used as stable drug carriers (14). Carbon nanotubes are made more soluble by incorporation of carboxylic or ammonium groups to their structure. They can be used to transport peptides, nucleic acids and other drug molecules. Amphotericin B nanotubes are able to provide an increased drug delivery to the interior of cells.



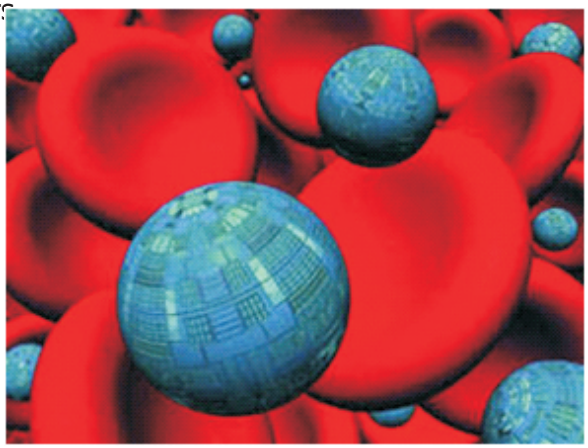
Single walled carbon nanotubes

x) Biorobots: Biorobots are engineered bacterial biologic robots. They are constructed from 300 highly conserved genes to act as a functional microbe (15). These synthetic microbes can be made to produce vitamins, hormones, enzymes, or cytokines that are found in lesser amount in human body. They can be made to absorb and metabolize poisons and toxic substances into harmless end products.



Biorobots

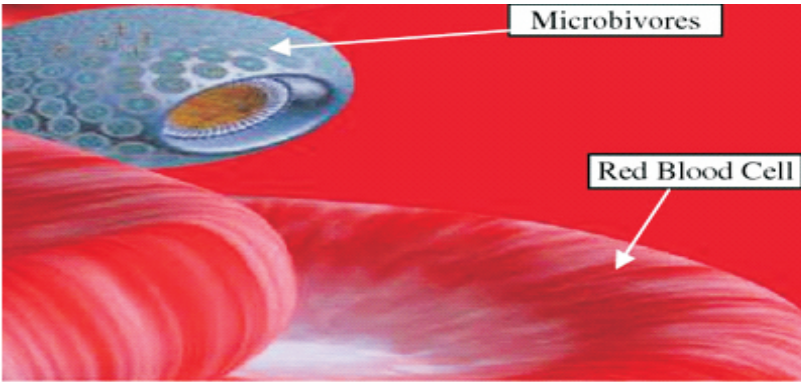
xi) **Respirocytes:** Respirocyte is an artificial mechanical red blood cell. It is a spherical diamondoid nanorobot, and is able to deliver 236 times more oxygen to the tissue per unit volume than natural red blood cells and to maintain carbonic acidity (16). They possess sensors on the surface which can detect changes in the environment and the onboard nanocomputer helps in regulation of intake and output of oxygen and carbon dioxide molecules. Respirocytes function similar to that of natural haemoglobin-filled red blood cells. Respirocytes may replace transfusable blood substitutes. An infusion of one litre of 50 per cent respirocytes saline suspension can keep the patient oxygenated up to four hours.



Respirocytes

Courtesy: Respirocytes-synthetic red blood cells , Steemit

xii) **Microbiovore:** Microbiovore is an artificial mechanical white blood cell capable of destroying haematogenously distributed pathogenic microbes (3).This spheroidal nanorobot attracts the microbiologic pathogens which get bound to the surface of microbiovore, then get internalized and minced, and discharged into the blood stream as amino acids, fatty acids, nucleotides and sugurs. They are likely to function as efficient, and fast-acting phagocytes, and may eliminate septicaemic infections at a faster rate than the natural defense mechanisms with antibiotics (17).



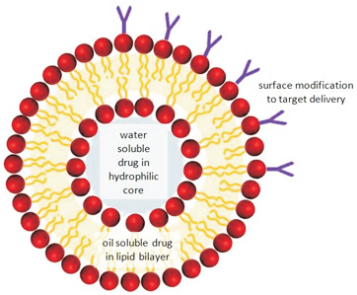
Microbiovores
Courtesy: Semantic Scholar

xiii) Liposomes: Liposomes are the original models of nanoscaled drug delivery devices. They are in use over 4 decades. They are the earliest examples of nanoscaled drug delivery devices. They are spherical nanoparticles. They consist of lipid bilayer membranes with an aqueous interior. Liposomes may be unilamellar with a single lamella of membrane or multilamellar with multiple membranes. They have been used as effective drug delivery systems. They have shown to be more efficient and safe as compared to convential preparations in administering anti-cancer drugs, amphotericin, and hamycin. Liposomes can be loaded with water-soluble drugs in the aqueous compartment and lipid soluble drugs in the lipid membrane (18). . The drawback of liposomes is their rapid degradation and clearance by hepatic macrophages. This reduces the duration of action of the drug. Attempts have been made to prolong their circulation time by coating liposomes with a non-ionic surfactant, polyoxyethylene or polyethylene glycol (stealth liposome) (19, 20). This prevents opsonisation of liposomes and their uptake by macrophages. \

Liposomes can be targeted to specific organ or tissue. Immunoliposomes have been produced by conjugating liposomes with an antibody directed towards the tumour antigen (21).Such agents can be injected into the body and they reach the

target tissue and get accumulated at the site of action. This enhances the drug delivery to the target tissue and reduces the unwanted effects.

Ligand-directed liposomes are produced by conjugating with specific ligands directed towards target tissues (19). There is an over-expression of folate receptors in ovarian tumors. Liposomal drug can be conjugated with folate and its action becomes directed to the tumour (22). In the treatment of leishmaniasis, liposomal hamycin has been tried, wherein liposomal hamycin is conjugated with mannosyl human serum albumin and is targeted towards macrophages (23).



Liposome
Courtesy: Entegris

Visceral leishmaniasis: Various lipid formulations of amphotericin B (liposomal amphotericin B, amphotericin B lipid complex and amphotericin B colloidal dispersion) exhibit preferential uptake by reticuloendothelial cells of liver, spleen and bone marrow in viscerla leishmaniasis (VL). This has enabled targeted drug delivery to the leishmania parasites with marked decrease in adverse effects. It has enabled administration of a large dose of the drug over a short period of time.

Liposomal amphotericin B administered intravenously in various doses exhibited good results

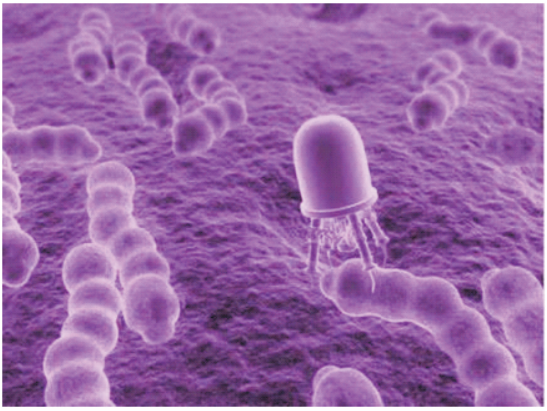
dose	cure rate (percent)
6 mg/kg for 10 days (24)	100
3.75 mg/kg for 10 days (25)	89
7.5 mg/kg as single dose (26)	90

The response to conventional amphotericin B was compared with liposomal amphotericin B and amphotericin B lipid complex. The results were comparable. The liposomal preparations exhibited lower rate of nephrotoxicity and lower infusion reactions (27).

preparation	dose and duration of therapy	cure rate (%)
amphotericin B	1 mg/kg on alternate days for 30 days	96
liposomal amphotericin B	2 mg/kg for 5 days	96
amphotericin B lipid complex	2 mg/kg for 5 days	92

A comparative study of response to liposomal amphotericin B as a single dose of 5 mg and a similar dose administered for 5 days showed a cure rate of 91 and 93 per cent respectively (28). Single dose administration showed good tolerance and safety.

xv) Chromosome replacement: A nanorobot controlled chromosome replacement therapy is a cytosurgical procedure wherein chromosomes can be extracted from a particular diseased cell and insert new ones in their place (24). The chromosome has to be produced outside the patient's body using the patient's individual genome as the blue print. The replacement chromosome is demethylated so as to express only the appropriate exons that are active in the cell type to which the nanorobot has been directed.



Nanorobot
Courtesy: Nanorobots and its medical applications, Material Science 2019

Thus nanomedicine is able to create nanomachinery of living cells and control and manipulate these molecular and supramolecular assemblies in living cells, combat disease-states and improve the health of individual. Nanotechnology has raised great hopes in medicine and many potential benefits have been visualized. However the safety of nanomedicine has to be worked out. However nanomedicine will play a crucial role in the treatment of human diseases in the years to come.

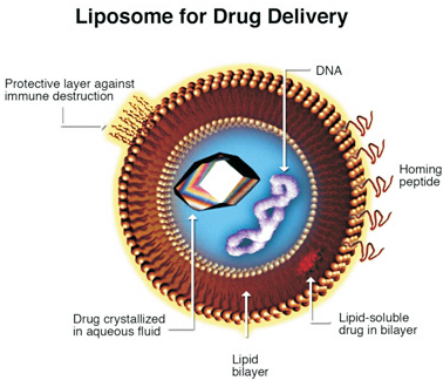
Nanoscale drug delivery

Commonly drugs are administered by oral or parenteral (intravenous, intramuscular and subcutaneous) routes. In addition the drugs are administered through the skin, and mucus membrane, by inhalation, by intranasal, spinal, and intraocular instillation, per rectal and per vaginal routes, and by implantation. Practice has shown that they may not be always most effective way for a particular treatment. A need has been felt to introduce novel delivery systems to administer new biologic agents containing bio-blood proteins, bio-vaccine and nucleic acids and other formulations. Utilization of such technologies offers better compliance by the patients (30).

Effective drug delivery

The nanostructure-mediated drug delivery technology incorporates one or more of the following substances: biologics, polymers, silicon-based metals, carbon-based materials or metals. These materials structured in microscale are now in nanoscale formats. Particle size determines the efficiency of drug delivery to different parts of the body. Particles less than 100 nm diameter can be effectively delivered into pulmonary system (31). Microparticles of 100 nm are absorbed with greater efficacy by the gastrointestinal system (32). Transcutaneous permeation of vaccine is better when the particle size is 50 nm or less (33).

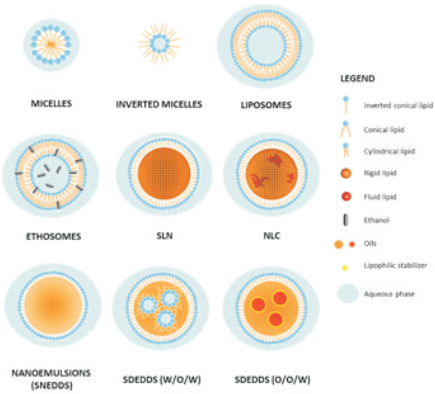
Nanostructure-mediated drug delivery system aims to target their drugs with nano precision (34). They can be utilized in gene delivery vectors and in stabilization of drug molecules that would have degraded very quickly (35). Utilizing these controlled drug delivery devices it is possible to obtain better drug distribution with reduced drug toxicity (36).



Source: en.wikipedia.org

Precise delivery

Many drugs encounter difficulty to reach their targets due to anatomical features such as blood-brain barrier, the branching pathways of bronchial tree and tight epithelial junctions at the skin (37). These barriers can be overcome by using nano-structured drug carriers.



Types of lipid-based nanocarriers
SLN" solid lipid nanoparticles
Source: www.researchgate.net

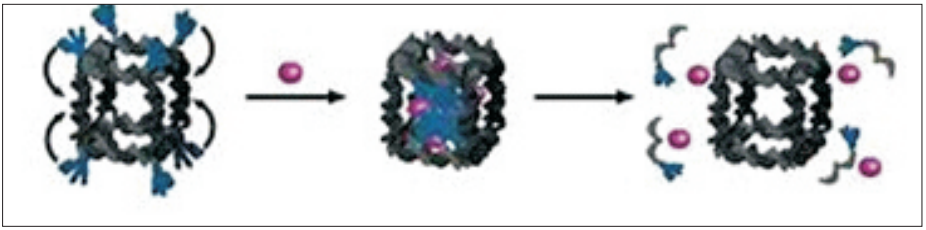
Nanostructure-mediated drug delivery has the advantage of delivering drug molecules directly into the cells (38). The chemotherapeutic agent can be delivered into the solid tumours located within healthy tissue (39). It is possible to release the drug by precise control of the drug carrier architecture. Three kinetic profiles belonging to zero order, first order and Higuchi are utilized for the purpose. They facilitate a steady release of drug so as to maintain its level constant in the body while the drug is being administered (37).

The nanostructure drug delivery forms may be in the form of solid or hollow spheres, tubes, solid or porous particles, micelles, nanoshells or dendrimers (branched structure).

Drug delivery technology

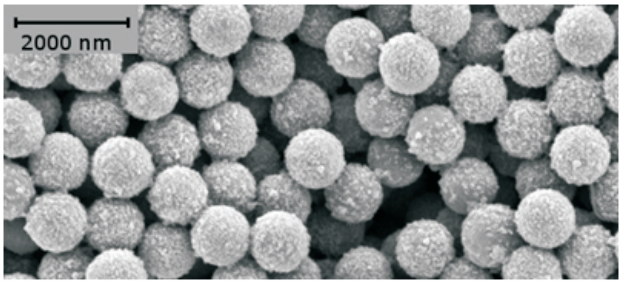
1. **Biologic nanostructures:** There are many examples of biologic nanostructures that have been developed for drug delivery. They include lipid nanotubes, lipid nanospheres, lipid nanoparticles, lipid emulsions, circular peptides, polysaccharides and nuclei acid nanostructures and

viral nanoparticles (8). Liposomes are hollow vesicles and they are able to entrap and release drug molecules. Vesosome, a multi-compartment drug delivery vehicle facilitates an extended drug delivery (40)



DNA-based biological nanostructure for controlled
Drug delivery
Source: www/kurzwaiai/net

1. **Polymer structures:** Polymeric materials exhibit biocompatibility, biodegradability and fractionalization capacity. The drug molecules can be entrapped within the polymer following fractionalization and structural manipulation of polymer material. It allows greater control of the pharamacokinetic behaviour of the active drug molecule and it can be released from the carrier at a constant steady level in a zero order fashion (41).



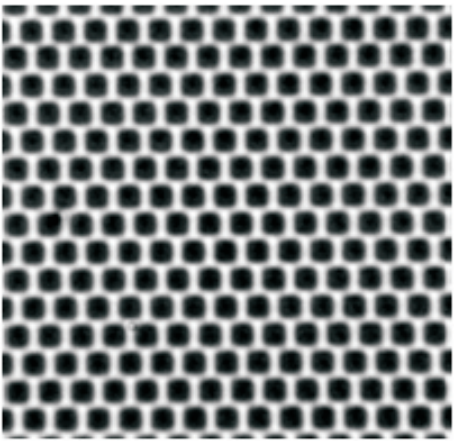
Personalized polymer particles
Source: www.ikerlatpolymers.es

Polymer nanoparticles consisting of polylactic acid (PLA), polyglycolic acid (PGA) or a copolymer of PLA and PGA have been experimentally tried for delivery of proteins and genes, vaccines, anticancer drugs and ocular drugs (37). Polyethylene glycol (PEG) is capable of

entrapping biologic materials and act as a more stable drug carrier. Stealth liposome is a liposome coated with PEG and it exhibits prolonged circulation times (42). Tablets coated with hydroxypropyl methylcellulose phthalate (HPMCP) nanoparticles exhibit a decreased rate of release with a zero-order release kinetcs (43).

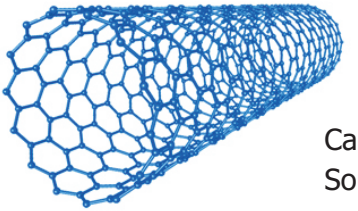
Another example of polymers is dendrimers which are highly branched macromolecules. Drug molecules can be incorporated into dendrimers (44).

1. **Silicon-based structures**: Porous silicon and silica or silicon dioxide are utilized for drug delivery. They come in the form of nanopores, nanoparticles and nanoneedles. A controlled delivery of the drug can be achieved by allowing the drug to flow constantly out of the pores. The release kinetics is near zero-order. Platinum, artificial growth factor and antibiotics can be embedded in the porous silicon delivery system.



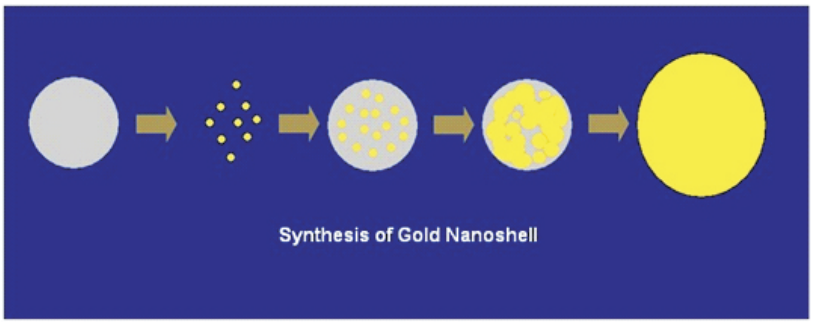
Microporous silica
Source: www.silicon-membranes.com

1. **Carbon-based structures**: Nanotubes and Fullerenes exhibit spherical, hollow, carbon-based, cage-like architectures. Their structure resembles the geodesic domes of Buckminster Fuller. The configurations include single-wall nanotubes, multiwall nanotubes and C60 fullerenes. They can be utilized to deliver vaccines and genes (45).



Carbon nanotubes
Source: large.stanford. edu

5. **Metal structures**: Hollow thin metal nanoshells around a core of silica nanoparticles are utilized for drug delivery (46). They can be embedded within polymeric drug carriers. The metal nanostructures act as thermal release triggers after irradiation with infrared light or excited by an alternating magnetic field (47).



Source: sites.google.com

New drug-delivery system with nanostructural delivery architecture has shown promising results to deliver the drug to the specific targets. They are also capable of sustained release of drug. Nanoscale drug can enter cells and act locally without exhibiting toxic effects. Nanoscale drug delivery technique is likely to affect the aetiologic condition of the disease and bring about relief to the patient.

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MANAGEMENT OF SURVIVORS OF SUICIDAL ATTEMPT AND ITS PREVENTION

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PRACTITIONERS' GUIDANCE SERIES - XIV

MANAGEMENT OF SURVIVORS OF SUICIDAL ATTEMPT AND ITS PREVENTION

C R CHANDRASHEKAR

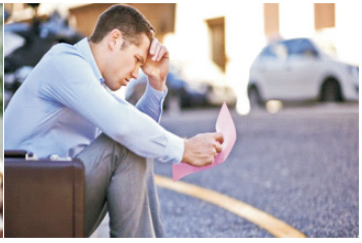
Suicide is one among top 10 causes of death all over the world. One person commits suicide every 40th second. Nearly 1 million people die every year in the world by committing suicide. In India during 2018, 1,34,516 people died. Maharashtra topped and Karnataka occupied 5th place with 11,561 suicidal deaths. Chennai was on the top of the list followed by Bengaluru in 2016. Suicide deaths are increasing in young persons and is the second cause of death in age group of 15 to 29 years. For every suicide death, there may be 30 cases of deliberate self-harm (DSH) or Para suicide or Non – futile suicidal attempts. Doctors especially lady doctors are at high risk for suicidal attempts compared to general population.

Though every religion says that attempting suicide is bad and is a sin, individuals are going ahead to die. Hindu religion scriptures warn its followers that the 'souls' of persons who end their life by suicide have to run around aimlessly as they are refused **salvation** or entry into the other world. Not only they suffer, they also make 'souls' of their ancestors suffer. In the future new life, they are made to suffer from poverty, diseases and bad life events. This belief also does not act like a deterrent.

What makes one to commit suicide

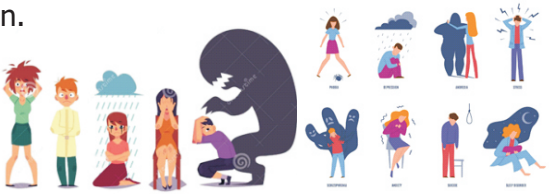
1. Frustration:

Frustration arising out of losses, failure, financial constraints, family disputes, loss of job or prolonged unemployment, insults, injustice done to her/him, social rejection or isolation, prolonged, painful and socially stigmatizing diseases or disability or any kind of physical or mental suffering.



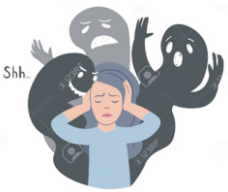
2. Mental Disorders:

Depression: 40% of cases end up in suicidal attempts. 60% suicides are due to depression.



Alcohol/Drug abuse and dependence:





Personality disorders like Borderline personality, Impulsive, Antisocial personality disorder.

3. Social Factors:

Poverty, disparity between rich and poor, more value for money and materials, class and caste discrimination, Lack of social cohesion, poor social support system, Media, irresponsible reporting of crime, violence and suicidal cases, migration, decreased values and norms, poor law enforcing system, cheating and other social evils.

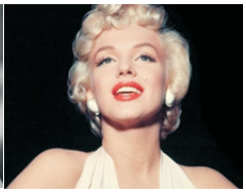



Risk Factors

1. Genetic predispositions.
2. Repeated traumatic life events.
3. Age factor: High risk in persons between 15 to 29 years and elderly group.
4. People who live alone, away from their families and who think that they are isolated /rejected by others.


5. Ideas of helplessness and hopelessness and a thought that suicide is an answer or remedy for their problem.
6. Media – Models: --- celebrities or others committing suicide.






1. Guilt feeling about past mistakes/wrong decisions / unlawful/ unethical acts.
2. Sex: More women make suicidal attempts than men but more men die because of suicide.

DSH – Para suicides are also more in girls.

Indians who committed suicide Indian Mythological stories:



1. Dakshayini – Wife of Shiva
2. Rama, Lakshmana and Seetha
3. Madri – Wife of Pandu (Mahabharatha)

Individuals who thought of Suicide:



1.Hanuman 2.Arjuna 3.Duryodhana

4.Many saints and sanyasis and to attain **salvation**


➢ **Sathi System:**
Following the death of the husband, wife would jump into the pyre and die.

➢ **Garuda System:**
Security guards of the king would kill themselves if they fail to protect the king.

Suicide accepted as part of the culture in ancient India. Saints, old people, and others who lost interest in living were allowed to die by 'Prana Thyaga'

- Agni Pravesha (Fire)

- Jala Pravesh (Water)

- BhooPravesha (Bhoo / Samadhi)



- Prayopavesha (Fast unto death)

- Committing suicide in holy places to attain mukthi or **salvation** to please the God or deity.

- Self-punishment for having committed crime or violating religious norms or ethical norms, 'DaivaDroha', 'Dharma Droha' or 'SamajaDroha'.

FACTS ABOUT SUICIDES IN INDIA

Year	Total Deaths	Rate / One Lakh population / Year
1967	38,820	7.8
2010	1,34,600	11.4
2011	1,35,585	11.2
2012	1,35,445	11.2
2013	1,34,799	11.0
2014	1,31,000	10.6
2018	1,34,516	11.6

In India on an average 12 persons commit suicide and die per every one lakh population every year.

Depending on the reporting - recording efficiency, the rates keep varying. During 2015, the following is the recording

State / U.T		**Per 1 Lakh population**
PUDUCHERY	:	43.2
SIKKIM	:	37. 5
TAMILNADU	:	22.8
KERALA	:	21.6
KARNATAKA	:	17.4

Male: Female Ratio – 68% and 32 %.

Men : 25. 8/1 Lakh

Women: 16.4/1Lakh
Farmers: 10,349 in 2018

But suicidal attempts are more in women. Among total suicidal deaths in women in the world, 27% are Indian women.

Causes of death as reported in death note as on 2014 in India:

1. Family and marital disputes : 28% of total cases
2. Diseases – ill health : 23%
3. Failures : 3.2%
4. Financial Crunch : 2%
5. Poverty : 2%

Age:

15 to 30 years - 34% of total cases

31 to 45 years - 34% of total cases

60 years - < 1%

Students committing suicide are increasing. They constitute 6% of all cases.

Method of committing Suicide:

Hanging : 42%

Fire : 26%

Water : 6%

Tablets / Poisons : 7%

Falling from Heights : 1%

Para Suiciders (Where intention to die is less but intention to put pressure on others or to show their anger, dissent) take overdose of any medicine/Substance like harpik, TIK 20 and wrist slashing. Para Suicides are common in adolescent boys and girls and young women.

Extended Suicide:

When a person decides to commit suicide, he/she may persuade the spouse and children to commit suicide. All together they may consume poison, jump into water or set fire, drive fast and make accident.

Death Note/Suicide Note:

A few write death note to the concerned people and mention the reasons for committing suicide. They may blame someone/family/ society for the act. Some may say that it is their decision and do not blame anybody.

Altruistic Suicide:

Some commit suicide for the cause of others, religion/Spiritual beliefs. Some voluntarily die for the sake of their organization or for their country (Suicide Bombers).

Management of Survivors of Suicidal attempt:

Once the medical emergencies are taken care and the person is out of danger, doctors have to talk to the individual, family members, friends and colleagues to find out the cause of the suicidal attempt and prevent the repetition of the act.

Talk to the family members and other significant people and collect information about the suicidal attempt.

a) At what time and where the act was committed:

The time and place reveal the intention and its severity to die. For example:

- Late Night, Mid Night, Early in the morning

  Inside the toilet, room or other lonely places of the house or outside the house – lonely place where there would not be anybody. This means strong intention to die.

- Day time, in the hall or living room or in front of the house, in front of people means less intention to die. The attempt could be para suicide.

b) Method of Suicide:

Hanging, Setting fire (Ablaze), gunshot, drowning, consuming large number of tablets, drinking acid, jumping from height means severe intention to die. Consuming few tablets, small quantity of TIK 20, Folidol, Harpik, Dettol or wrist slashing is Para suicide.

c) Communication about the act to others:

No communication, No indirect communication (Like tomorrow I may not see you, this is my last dinner, you will never meet me again) means severe intention.

Direct or subtle communication: I am going to die. It's enough/cannot bear this insult anymore, you will repent for my absence, life is not worth living. I wish that I am dead and gone. I won't be there to bother you. You won't see me again means less intention.

d) Death Note:

Death note written and brought to the notice of the concerned: Less intention. Death note written and hidden from the notice of others means severe intention.

e) Earlier attempts to commit suicide and the nature.

f) History of suicide in the family / close relatives.

g) History of depression in the past one month or more.

The following are the symptoms:

i. Food intake and sleep is disturbed
ii. Being dull, withdrawn and avoiding people, poor communication, prefer to be lonely.
iii. Crying Spells
iv. Expressing ideas of hopelessness, worthlessness and helplessness, expression of pessimistic ideas. Low Self esteem
v. Vague physical complaints and consulting doctors and getting investigated. Taking tablets to get sleep
vi. Less efficient, no regards to work, neglecting personal hygiene, work and responsibilities.
vii. Students: Not attending classes and tests and getting low marks.
viii. Being irritable, Sulking, blaming others for trivial or no reasons.
ix. Expressing inability to complete the tasks assigned to him or her.

x. Unexplainable fear, anxiety, nervousness
xi. Suspicion that others may harm or pass negative comments
xii. Hearing derogatory voices and comments, death wish, desire to end life.
xiii. History of alcohol, and drug use/abuse.
xiv. History of any abnormal and strange talk, ideas, behaviours, responses and how long they lasted. Any consultation from psychiatrist or psychiatric hospital in the past.
xv. Individual personality, attitude, general knowledge, self-confidence, coping abilities and skills to manage problems and challenges. Any history of breaking down under pressure or difficult situations/challenges of life.
xvi. History of physical ailments or diseases he individual had, or has been having. Treatment details have to be obtained.
xvii. Unstability in the workplace or family life.

Also get the responses of family members and friends – colleagues regarding the individuals suicide attempt.

- Are they shocked?
- Are they angry? Do they feel insulted?
- Are they anxious / Fearful?
- Are they sad and depressed?
- Are they unconcerned?
- Are they judgmental? Say that he/she is weak, bad and mad or a fool?

Do they support and want to help the individual? Are they aware of the issues and problems which made him/her to commit suicide?

Explain and educate them that suicidal behaviour is a common behaviour all over the world. They should sympathise and empathise with the person. Help him, improve his coping skills to manage his/ her affairs efficiently. They should also take precautions to prevent relapse of suicidal act.

Talk to the individual:

Make note of his or her responses:

- Is she/he unhappy for surviving? Does he/she say 'why did you save me?'' I should have died'
- Is he apologetic, shy or guilty for the act of suicide?
- Is he angry with himself and others? Does he blame himself or others for the act?
- Does he express his desire to make another attempt to die?
- Is accepting food, willing to take care of his personal hygiene? Is he sleeping well?
- Is he comfortable, restless, fearful, fidgety, or remorseful?
- Reassure him/her that you and other hospital staff will do the best to make him comfortable.
- Reassure him/her that you will talk to his family and friends to help and support him.
- They will be helpful to solve the problem and make him happier and comfortable.
- Ask him whether and when he will be able to talk to you regarding the issues, problems, situations or people who made him to decide to die
- Ask him/her to give a detailed account of the issues and circumstances which made him to decide to commit suicide. How long it took for him to plan and execute the act. The chain of thoughts and emotions which accompanied has to be explored.
- Ask him/her whether he/she would like to blame self or others or the society for the same? Let him/her describe the problems, what he did to solve the problem? What was the result? Who supported? who did not support? Who criticised him? Who ill-treated him? Let him discuss his coping methods and how they failed to give results?
- Ask him/her to describe his experiences, thoughts and emotions after surviving from the suicidal act.
- Whether he/she feels shameful, guilty or dejected and feels bad for not succeeding in the attempt to die or he/she is worried about the complications in his/her health or worried about the reactions and responses of family/friends/colleagues or about

unsolved/unresolved problems? How to face them or about his future?

- Administer the following questionnaire:

More than 5 yes answers reveal that the person is emotionally disturbed. (Depression and Anxiety disorders)

Sl.No.	Item	Yes	No
1	Is your appetite less?		
2	Is there problem getting sleep? or sleep for 6-7 hours.		
3	Is there fear/Anxiety/anticipating more problems?		
4	Is there trembling of hands/legs?		
5	Do you have boredom/Feel sad?		
6	Do you get angry which you cannot control?		
7	Do you have difficulty in thinking?		
8	Are you getting more negative thoughts?		
9	Do you think that you are useless?		
10	Do you think that you are a burden to family and society?		
11	Have you lost interest in everything?		
12	Do you have pain in the body? Feel weak?		
13	Do you want to make one more attempt to die?		
14	Do you think that your future is bleak?		
15	Do you think that you are not cared by family		

If the person is depressed, prescribe following drug and dosage

1. Anti-depressant drug

Escitalopram: Start with 10mg increase to 20mg,
or
Fluoxetine: 20mg → 40mg
or
MITRAZEPINE: 15mg → 30mg

2. Minor Tranquillizers:

Clonazepam 0.5mg to 1 mg at bedtime
or
Lorazepam 1mg to 2mg.

Counselling:

Spend 30 to 45 minutes talking to the person.

1. Identify issues/Problems/Conflicts
   They may be in the area of family, finance, relationship, education, occupation or society
2. Asses the coping skill of the individual. Ask what he had done earlier and what he would do now. Give suggestions to improve his/her coping skill.
3. Get the support of the family and friends.
4. Suggest outside agencies to seek guidance and help.
5. Refer to a Psychiatrist or psychologist for further help.

General Instructions to the patient and the family:

1. The individual should not be left alone. Someone should stay with him/her all 24 hours. If admitted, hospital staff should keep vigil 24 hours.
2. Avoid access to lethal weapons, substances, medicines.
3. Take care of the basic needs of the person – food, clothes, shelter
4. The Individual should be engaged in healthy recreational activities like music, sports, reading, watching good TV program, religious activities, meeting good friends and relatives.
5. Regular Follow up

Para Suicides:

With no intention to die, Para suiciders enact a drama of deliberate self-harm with the intention of

* Putting pressure on family members/concerned persons to yield to their demands like

  "If you don't give me mobile, I am going to die"

  "If you insist on following your advice, 1 will end my life"

"If you prevent me from meeting my friend, I will cut my wrist to bleed onto death"

"If you don't take back the show-cause notice, I will set fire to myself"

"If you don't love me, I will drink poison"

"If the government neglects our demands, I or we will die"

* Protesting and showing one's resentment

"You have insulted me in front of my classmates, I will commit suicide"

"Take me back to work otherwise I will jump from the terrace"

"How dare are you to scold me, I will teach you a lesson, I will leave a death note that you are responsible for my death and drink TIK 20"

"If you don't compensate for the loss, I will die"

Because of media influence, many adolescents and young people use DSH and brought to treatment to the hospitals and clinics. Handle them with care! They sulk. They do not communicate properly. They blame the parents or others. They threaten that they would try again to die. They are adamant and believe that they are right and their demands are right and should be fulfilled immediately. Try to persuade them that they should not use 'Suicidal act' to blackmail others. They should be patient and persuade others to help them. Many of these individuals have low frustration tolerance and poor communicators. Their coping skills are inadequate. They have to be taken up for counselling to acquire these skills. They have to be referred to professional counsellors if needed.

Prevention of Suicide:

Majority of suicides are preventable. Family members, friends, colleagues, doctors can identify individuals who are in distress, depressed, angry for some reasons and counsel them. Emotional support can definitely prevent suicide.

Identify and talk to people who have high risk for suicide or Para Suicide.

MANAGEMENT OF SURVIVORS OF SUICIDAL ATTEMPT AND ITS PREVENTION

1. People who are facing negative life events like death of a family member, close friend or a person whom the individual adored.
2. People who are facing unexpected loss, failure, in exam, competitions, business, love affair etc.
3. Individuals living alone, living away from the family.
4. Patients who are suffering from socially stigmatizing illness like HIV, fits, Leprosy.
5. Patients who are having life threatening and painful illness like cancer.
6. Individuals who are suffering from mental diseases like depression, schizophrenia, personality disorder, puerperal psychosis, acute psychosis etc.
7. People who are abusing or addicted to alcohol and other intoxicating substances.
8. Women who are subjected to domestic violence, dowry harassment, sexual abuse or whose husbands having extra – marital relationship
9. Children/adolescents who are addicted to video games.
10. Individuals who are subjected to open punishments and insults.
11. Women who are subjected to rape or any kind of sexual assaults.

We have to ask all these individuals:

- Do you have death wish?
- Do you feel that life is not worth living?
- Do you feel that it is better die than live?
- Do you get suicidal ideas?
- Have you planned to commit suicide and how you would do it?

If he/she says 'yes' to the above questions, we have to take him/her to for intense counselling. We have to inform and involve the family members and friends to give him/her emotional support and to face the adverse situation/problem. Keep 24 hours vigil and prevent the act of suicide. We have to help him/her to improve one's life skills and attain emotional stability.

PREVENT SUICIDE: SAVE LIFE

SUICIDE AND LAW

I.P.C (INDIAN PENAL CODE)

Section 309: Attempt to commit suicide:

Whoever attempts to commit suicide and does any act towards, the commission of such offence shall be punished with simple imprisonment for a term which may extend to one year or with fine or both.

Section 306: Abetment to suicide:

If any person commits suicide, whoever abets the commission of such act (forces/helps) shall be punished with imprisonment which may extend to 10 years and also liable to for fine.

Section 305: Abetment of suicide of child or insane person:

If any person under 18 years of age, any insane person, any mentally retarded or any person in a state of intoxication commit suicide, whoever abet the commission of such suicide shall be punished with death or imprisonment for life or for a term not exceeding 10 years and also liable to fine.

NEW MENTAL HEALTH CARE ACT – 2017

SECTION 115

Notwithstanding anything contained in section 309 of IPC, any person who attempts to commit suicide shall be presumed, unless proved otherwise, to have severe stress and shall not be tried and punished under the said code. The appropriate government shall have a duty to provide care, treatment and rehabilitation to a person having severe stress and who attempts to commit suicide to reduce the risk of recurrence of attempt of suicide.

EUTHANASIA – Mercy Killing

Patients with life threatening ailments, Patents with severe pain and disability, having no hopes of relief or cure may think of ending their life. Patients who are in ICU – semi conscious or unconscious /coma, not responding to treatment and who are on life saving treatment, then relatives may wonder how long to wait for death? They may request the doctors to stop the life-saving treatment and discharge the patient so that death come early. Some may request the doctors to give some drug

or do something so that patient dies quickly. Thus, in euthanasia, there are two types:

Active Euthanasia (Something is done to bring death)
Passive Euthanasia (Nothing is done except to withdraw the life saving measures)

Active euthanasia is considered as illegal and unethical and equivalent to murder or suicide. Therefore, except one/two countries like Netherlands, Belgium no country permits active euthanasia. But many countries including India have permitted to do passive euthanasia.

Living Will: Any person can write a will which he/she can request for passive euthanasia in case he/she develops life threatening illness with no reliable and effective intervention.