Cardiac Arrhythmias

Basic Electrophysiology

Impulse Formation

Automatic impulse formation (rhythmicity)

Inherent property of nodal and Purkinje tissue

Descending gradient of rhythmicity from SA node downward

Thus three potential locations of impulse formation

a) Primary center (Sinoatrial Node). This collection of tissue is located at the junction of the superior vena cava and the right atrium. Under ordinary conditions, this forms impulses at a rate of from 60 to 80 per minute.

b) Secondary center (Atrioventricular Node). Its superior portion is in the interatrial septum, the middle portion is in connective tissue at the AV junction, and the inferior portion is in the membranous portion of the interventricular septum. The inherent rate of impulse formation is from 40 to 60 per minute.

c) Tertiary centers (Purkinje Tissue throughout in ventricular musculature). Impulses are formed at a rate of less than 40 per minute.

The center that forms impulses at the fastest rate will become the pacemaker of the heart. Thus, under ordinary conditions, the sionatrial node is the pacemaker of the heart.

Telologically this defense in depth design prevents asystole by:

Providing instantly available standby pacemaker

In addition lower centers are under much less neurogenic control.

Shift of pacemaker from SA node to lower center produces an ECTOPIC rhythm or ECTOPIC BEAT

Mechanisms responsible for shift:

1. Passive transfer of pacemaker

   a) Here the primary pacemaker falls below the inherent rate of the lower pacemaker and the lower center then attains control of the rhythm.

   b) The lower center thus escapes for one or more beats.

   c) Such beats or rhythms are properly called ESCAPE BEATS or RHYTHMS.

2. Active transfer of pacemaker

   a) Here lower center rhythmicity increases -- change in local excitability.

   b) Here lower center's rate of impulse formation exceeds that of higher center.

   c) Result: ECTOPIC BEATS or ECTOPIC Rhythm.

   d) Mechanism may be physiological but often pathological - e.g., digitalis intoxication, hypoxia.

Refractory Period

Immediately after excitation of any portion of the myocardium (including nodal and Purkinje tissue) there is a period of time during which the tissue will not respond to impulse stimuli. Termed: Absolute Refractory Phase.

Immediately thereafter; Relative Refractory Phase. During this period impulses may or may not produce excitation or conduction. Consequently response may be

a) normal,
b) delayed,
c) incomplete, or
d) absent.

Duration:

Absolute + Relative Refractory Sum = Refractory period

Refractory period is proportional to cycle length
(i.e., fast rate, short refractory period).

Refractory period longest in AV Junctional Tissue
Hence AV node weak link in conduction between SA node and ventricle.

Interference

Term used to describe the mechanism involved when conduction of one impulse delays or prevents conduction of another.

For example the spread of an impulse may be completely stopped because it reaches an area which is still in the refractory phase from a previous conducted beat.

Because of the inherently longer refractory period of the AV nodal tissue it is a frequent site of interference. May occur with:

a) Rapid discharge of single pacemaker, e.g., SA node:

2nd impulse may be either slowed or stopped because of partial refractoriness of AV node.

b) Simultaneous discharge of two pacemakers, e.g., SA node and AV node.

Atria depolarized by antegrade conduction from above.

Atria also depolarized by retrograde conduction from below.

Each wave leaves refractory tissue in its wake.

When two meet they cancel for further spread impossible.

P waves produced in this manner termed atrial fusion beat or fusion P wave. Contour intermediate between normal and retrograde P.

Fusion P wave an example of a trial interference.

As long as interference continues the SA and AV node discharge independently - resultant rhythm: Atrio ventricular dissociation

Imp. Mechanism of interference per se is physiological. It may or may not be associated with pathological arrhythmias.

AV Block

N.B. AV interference and dissociation must not be confused with AV block. The refractory period is normal in interference.

AV block occurs when the refractoriness of the junctional tissue is abnormally prolonged.

On basis of severity AV block may be classified.

1° AV Block All impulses conducted but slower than normal. PR interval exceeds 0.21 sec.

2° AV Block (incomplete or partial)

Occasional dropped beats

Common Type

Wenckebach Progressive lengthening of PR and finally failure of impulse to reach ventricle.

Uncommon Type

Constant prolonged PR with occasional failure of AV node to conduct.

3° AV Block (complete block)

Complete AV Dissociation

Auricles and ventricles beat independently.

Autonomic Nervous Tone

Sympathetics - acceleration
Vagus - depression

Enervate SA and AV node

Rt. Vagus to SA node
Lt. Vagus to AV node

The Sino-Atrial Arrhythmias

Ordinary sinus rhythm
Sinus bradycardia
Sinus tachycardia
Sinus arrhythmia
Wandering pacemaker
Sinus arrest
Sinus block

Ordinary sinus rhythm - Any fairly regular rate of the atria between 60 and 100 with normal P waves and variation of the cycle length of less than 0.16 sec.; a PR interval greater than 0.12 sec. is called ordinary sinus rhythm. The upper limit of sinus rate has been defined as 100 but this does not necessarily define normalcy. For example, at birth the average range of rate is from 110 to 150. On the other extreme, well conditioned athletes who are "normal" often have heart rates of less than 60.

Sinus bradycardia-A sinus rhythm with a rate of 60 or less. Occurs when there is increased vagal tone, with increased CNS pressure, jaundice, pancreatitis, retroperitoneal masses, posterior myocardial infarctions, athletes in good condition, especially swimmers.

Sinus tachycardia - A sinus rhythm with a rate greater than 100. Usually, in adults, sinus tachycardia does not exceed 140. However, in infants and children the rate may go as high as 230 and still be sinus tachycardia.

Causes are: fever, anoxia, congestive heart failure, thyrotoxicosis, psychoneuroses, anemia, shock, acute rheumatic fever, stresses in general.

It is important to realize that during sinus tachycardia, as with other types of tachycardia, transient ST and T wave changes may occur. Also, intraventricular block may develop during the fast heart rate.

Sinus arrhythmia - A sinus rhythm with a P-P variation of (±) 0.16 sec. This is a very common finding. In a reported series of 50,000 ECGs taken on hospital patients, 8,302 had sinus arrhythmia. This diagnosis would perhaps be even more prevalent in a young, normal population. Some sinus arrhythmias seem to be related to the respiratory cycle; others are not.

Wandering pacemaker There are two types:

A) Within the sinus node - the site of impulse formation shifts to less active parts of the sinus node (remember that the node is relatively large). With this arrhythmia the P wave amplitude changes and the P-P interval changes. As long as the PR interval is greater than 0.12 sec. and the P wave contour is essentially upright it is assumed that the pacemaker is in the sinus node.

B) With shift to the AV node - in this type the PR interval shortens to less than 0.12 sec. and the P waves may become retrograde. Therefore this is both a sinus and a nodal arrhythmia.

Neither form of wandering pacemaker has clinical significance. Both are thought to be caused by vagal activity and can usually be abolished by atropine.

Sinus arrest or sinus standstill The P waves are absent and the pause in atrial activity is not a multiple of the sinus cycle. This condition may occur with severe potassium intoxication. Occasionally it results from carotid sinus massage or other maneuvers to stimulate the vagus. Vagal induced sinus arrest will usually be reversed by atropine. With sinus arrest unless secondary mechanisms take over the pacing function, cerebral vascular insufficiency and death may result. A sharp frappe on the chest will often stimulate the heart to resume beating. Always try this before resorting to external cardiac massage, electrical pacing, or thoracotomy.

Sinus block The atria are not activated by the sinus impulse in this arrhythmia because the impulse fails to leave the sinus node. Although the impulse is formed normally, there is impaired conduction (block) in the junctional tissue between the sinus node and the atria. Since sinus activity per se does not produce an ECG deflection, no P wave is seen on the conventional tracing.

With sinus block, P waves are dropped at multiples of the sinus cycle. This observation tends to differentiate sinus block from sinus arrest. Sinus block is thought to be caused by vagal activity and can be prevented by giving anticholinergic drugs. Wenckebach periods can occur at the sinus node and are characterized by acceleration of the P wave rate followed by a dropped P wave. (The mechanism of the Wenckebach phenomenon is discussed under the section on AV block.)

AV Nodal Rhythms

Nodal escape beats
Nodal premature beats
Nodal rhythm including nodal tachycardia
Reciprocal rhythm

In AV nodal rhythms, the impulse arises in the AV node, travels downward and initiates ventricular depolarization, and travels upward or retrograde to initiate atrial depolarization. Since the spread of the atrial depolarization wave in AV nodal rhythms is opposite that of sinus initiated depolarization, the P wave is altered and usually inverted with retrograde conduction.

From the electrocardiogram four locations of nodal impulse formation are defined:

A) High nodal - PR less than 0.12 sec., P wave is inverted and fall in front of the QRS

B) Middle nodal - P wave is lost in the QRS complex

C) Low nodal - P is inverted and falls after the QRS

D) Coronary sinus nodal - (Included in high nodal classification by some electrocardiographers). Although retrograde conduction takes place, the nodal impulse center is high and the retrograde depolarization produces an upright P wave. The P contour is abnormal and the PR interval is o.10 sec. or less.

AV nodal escape beats These occur when the rate of the primary pacemaker drops below the inherent rate of the AV node or when the sinus impulse does not reach the AV node for other reasons. Thus it is an example of passive ectopic beat formation and can be considered a normal protective mechanism of the heart. Its salient ECG features are:

1. The presence of delayed beats
2. The QRS contour is normal or changed slightly
3. The P wave may be absent, retrograde, or upright with a PR interval of less than 0.12 sec.

Premature AV nodal beats Are beats that occur before the expected sinus beat. These have retrograde P waves or upright P waves of abnormal contour with short PR intervals. The QRS complexes are normal or slightly abnormal.

AV nodal rhythm. Since the inherent rate of the AV node is from 60 to 35 beats/min., nodal rhythms of the passive type usually fall in this rate range. Nodal tachycardia and nodal rhythms with rates greater than 60 are usually due to active mechanisms such as mechanical stimulation during cardiac catheterization, digitalis intoxication, and electrolyte imbalance. Nodal rhythms are usually perfectly regular, the QRS contour is normal or slightly changed, and the P waves, if visible, are retrograde.

Atrial Response in Persisting AV Nodal Rhythm
Variable - Basis of further classification

a) With complete atrial capture as above
P waves inverted either preceding or following QRS unless lost in QRS

b) With incomplete or transient atrial capture (intermittant atrial interference) (vide infra)

c) Without atrial capture, i.e., AV dissociation - due to

1. interference (v.i.)
2. AV Block (v.i.)

AV dissociation due to interference

It has been mentioned that the atria and ventricles are independent except for a single bridge of tissue connecting them. Conduction of impulses occurs over this bridge in the normal situation but can be altered or actually prevented by two mechanisms - interference and block. When control of the

two chambers is separated, dissociation is said to be present. It is important to understand that both forward conduction and retrograde conduction must be blocked or one of the pacemakers will regain control and dissociation will no longer be present.

It is also quite important to be able to distinguish dissociation due to block and dissociation due to interference electrocardiographically. For example, the treatment for ventricular tachycardia of the interference-type is quite different from the treatment of ventricular tachycardia with complete heart block. The use of quinidine or procaine amide with the latter type of ventricular tachycardia could cause death.

In interference dissociation, conduction over the bridge of tissue connecting the atria and ventricles is prevented by the tissue being in a transient refractory state. Conduction through this bundle is normal during the rest of the cardiac cycle. With complete AV interference dissociation, a lower pacemaker is stimulated to form an impulse before the impulse from the primary pacemaker reaches it. When the normal sinus impulse arrives at the AV bridge it encounters refractory tissue produced by repolarization initiated by the lower pacemaker. Thus for that moment conduction does not take place and the atria have been depolarized by a sinus impulse; the ventricles have been depolarized by a lower impulse. As long as the dissociation is complete there is independence of the two chambers and no impulses are conducted. More commonly the atria and ventricles beat at slightly different rates. Therefore sinus impulses occasionally reach the AV bridge when it is not in a refractory state and conduction occurs. This arrhythmia is termed incomplete AV interference dissociation and the conducted beats are called capture beats. These conducted capture beats transiently disrupt the rhythm of the lower pacemaker. Occasionally the arrhythmia begins as incomplete AV dissociation and the atrial rate and lower pacemaker rate are pulled into synchrony and the arrhythm becomes complete AV dissociation.

In summary:

Complete AV interference dissociation: P waves are normal and there is only slight variation in the sinus rate. However, the sinus rate and the lower pacemaker rate are essentially the same and no capture beats occur. If the lower pacemaker is in the AV node the QRS complexes will be normal or only slightly altered. This condition is most often caused by digitalis excess.

Incomplete AV interference dissociation - P waves are normal and there is often slight variation in the sinus rate. The sinus rate and the lower pacemaker rate are different with the lower pacemaker being the fastest. Capture beats occur which disrupt the regularity of the lower pacemaker. If the lower pacemaker is in the AV node the QRS complexes will be normal or only slightly altered.

Ventricular tachycardia (interference type) - This can be either complete or incomplete dissociation. The pacemaker is in the ventricles and therefore the QRS complexes are bizarre. This arrhythmia is discussed in detail later.

Reciprocal rhythm - An unusual sequence in which an impulse from the AV node depolarizes the atria by retrograde conduction, delayed conduction occurs in the AV node, and then the ventricles are stimulated by anterograde conduction from the atria. Thus the source of stimulus for the ventricles alternates between the AV node and the atria.

Wandering pacemaker to the AV node - (Discussed under Wandering Pacemaker - sinus)

Atrial ectopic rhythms

Atrial premature systole
Atrial tachycardia
Atrial tachycardia with block
Atrial flutter
Atrial fibrillation

Atrial premature systole The impulse is formed in the atrial tissue rather than the sinus node and by definition occurs before the expected sinus impulse. On the electrocardiogram a premature P wave is produced having a PR interval longer than 0.12 sec. and having a contour different from that of the normal sinus P wave. The conduction of the atrial impulse after it is formed may be normal or abnormal (even to the degree of not being conducted. A premature P wave without a subsequent QRS complex is also considered to be an atrial premature systole.

Atrial tachycardia This can be thought of as a series of atrial ectopic beats occurring at a rate of 150-250/min. with perfectly regular cycles. The P waves have an abnormal contour and are usually upright in leads I, II, and AVF. There is an isoelectric period between P waves. When the arrhythmia begins suddenly and ceases suddenly it is called paroxysmal atrial tachycardia. PAT is sometimes converted to sinus abruptly by vagal maneuvers but is often not affected. (Discussed under paroxysmal tachycardia).

Paroxysmal atrial tachycardia with block. The atrial rate usually ranges from 150-250/min. (Note that the term tachycardia refers to the atrial activity and the ventricular rate can be under 100.) PAT with block is frequently a manifestation of digitalis intoxication and is therefore treated by omitting digitalis preparations, replacing potassium. Typically, with vagal maneuvers there is an increase in the block with no change in the atrial rate.

Atrial flutter. In this condition the atria form impulses from a single ectopic center in a rate range of 250-400/min. On the electrocardiogram flutter waves typically have a saw-tooth appearance with no isoelectric period between waves. The mean axis of these waves tends to be directed downwards and anteriorly.

Not all of the impulses formed in the atria are transmitted to the ventricles. The refractory period of atrial tissue is shorter than for AV nodal tissue and some of the atrial impulses are prevented from traversing the AV tissue by the mechanism of interference. Flutter may have a regular or irregular ventricular response. With the regular type the ratio of flutter waves to ventricular complexes in most often an even ratio. With the irregular type (flutter with varying "Block") odd ratios may be present transiently.

With vagal maneuvers, changes in the rate may occur in steps. For example the F:QRS ratio might go from 2:1 to 4:1, with slowing of the QRS rate by a factor of two. When vagal stimulation is stopped, the original ratio is frequently resumed.

Atrial fibrillation. There has been theoretical disagreement as to the pathogenesis of atrial fibrillation. For many years the circus theory of Sir Thomas Lewis was accepted. More recently Prinzmetal has presented evidence that in atrial fibrillation impulses arise from multiple ectopic foci without any apparent pattern. In both theories, areas of differential conduction must be presumed to be present.

In AF, 400 to 600 impulses per minute are formed. On the electrocardiogram continuous, small undulations appear and the ventricular response usually ranges from 100 to 180/min. If the multiple ectopic foci theory is accepted, the atrial arrhythmias may be thought of as forming a continuum with rates varying from unity (premature atrial systole) to 600 impulses/min. (atrial fibrillation). The ventricular response with atrial fibrillation is totally irregular. Interference at the AV node limits the response in a manner similar to that of atrial flutter.

Clinical consequences of atrial fibrillation: increased incidence of embolization, less cardiac reserve, and a tendency to develop tachycardia. Since most of ventricular filling does not depend on atrial systole, patients with AF and slow ventricular rates may have few cardiac symptoms. Their exercise capacity is decreased, however, and many clinicians believe that AF per se will cause congestive heart failure occasionally.

Paroxysmal atrial fibrillation may occur in apparently normal persons but such instances are quite rare. Persistant AF strongly suggests underlying heart disease and is the hallmark of rheumatic heart disease and is not infrequent in coronary artery disease. In the apparent absence of the latter, manditory to rule out cryptic thyrotoxicosis.

Ventricular Ectopic arrhythmias

Premature ventricular systole
Fusion beats
Ventricular tachycardia
" flutter
" fibrillation

Premature ventricular systole

1. QRS duration .12 sec. or greater.
2. QRS configuration bizarre.
3. 2° T wave changes.
4. Retrograde conduction thru AV uncommon.
5. Retrograde P waves rare.
6. SA rhythm undisturbed.
7. 1st P after PVC falls on refractory tissue in either AV node or ventricle hence no ventricular response.
8. Produces pause till next P released which does fire ventricle.
9. The duration of the R-R interval between the two normal QRS on either side of the PVC is equal to two sinus cycles.
10. The pause was therefore compensatory.
11. If sinus rate slow and PVC early enough, AV node may have recovered and next P wave fires ventricle.
12. This produces no compensatory pause.
13. Such a beat termed interpolated.

Significance of PVC's

1. 90% have none

2. Well known tho in anoxia, after infarction etc., they may herald ventricular fibrillation.

3. PVC's with QRS duration greater than 0.16 sec. suggest an illfunctioning myocardium.

4. Multifocal PVC's highly suggestive of organic heart disease.

Coupling

Bigeminy and trigeminy are descriptive terms that apply to coupled beats. Bigeminy is an ectopic beat coupled to a normal beat. Trigeminy is two coupled ectopic beats or other combinations of three beats.

Mechanisms postulated to explain the occurrence and timing of ventricular ectopic beats:

A) Activation of a dormant focus with subsequent sporadic impulse formation.

B) Re-entry This mechanism is dependent on differential areas of conduction. Due to impaired conduction in an area of tissue, an impulse could be delayed and protected while depolarization occurs in the rest of the chamber. Recovery occurs and the delayed impulse triggers ectopic depolarization. This mechanism could be used to explain fixed coupling.

Parasystole:

Term used to describe the simultaneous activity of two independent impulse formation centers in the heart when one is "protected" from the other. The ectopic parasystolic center is characteristically situated in the ventricle and is protected by unidirectional conduction so that it is free to compete with the SA node for dominance. It fires at its own rate and requires no triggering impulse. Hence ventricular response in parasystolic rhythms:

1. PVC's have inconstant relation to sinus triggered beats.

2. Follow each other by a multiple of parasystolic rate.

3. Frequently form fusion beat.

Significance

PVC's produced by parasystolic foci almost always associated with organic heart disease. In contrast coupled beats benign.

Fusion beat: When a wave of depolarization initiated by a normally conducted impulse combines with a wave of depolarization initiated by an ectopic focus, a fusion beat is produced. Fusion beats occur in both ventricular tissue and atrial tissue. On the electrocardiogram, fusion beats appear as transition complexes between normal and abnormal. For example, ventricular fusion beats have short PR intervals, QRS complexes which are abnormal when compared to the sinus beats but not as bizarre as the PVC's, and secondary T wave changes. There is never a compensatory pause with a ventricular fusion beat.

Ventricular tachycardia This important arrhythmia occurs when a ventricular ectopic center usurps control of the cardiac rhythm from higher centers. By definition the ventricular rate is greater than 100 but the same mechanism produces slower rates of similar gravity. In the majority of cases interference dissociation due to complete heart block is present and very rarely the rhythm is not truly dissociated because retrograde activation of the atria occurs. It can also occur with atrial fibrillation and flutter.

This arrhythmia is characterized by bizarre QRS complexes (like a series of PVC's) occurring at a rate greater than 100. If P waves are visible, they are usually not related to the QRS complexes except when normal conduction occurs. This momentary reversion to a conducted rhythm occurs when a sinus impulse arrives when the ventricular tissue has recovered from the refractory state and is called a capture beat. The ventricular rhythm is essentially regular until a capture beat disrupts it.

Electrocardiographically ventricular tachycardia may be confused with supraventricular tachycardia and aberrant ventricular conduction. The presence of ventricular fusion beats helps to confirm the diagnosis of ventricular tachycardia because this implies a combination of a normally conducted impulse and an impulse initiated by an ectopic ventricular focus has occurred.

In summary, the salient ECG features of ventricular tachycardia:

1. QRS complexes which are slurred and widened occurring at a rate of 100 to 200 with approximately regular rhythm.

2. P waves undisturbed in their sinus rhythm (see exceptions above)

3. The presence of capture beats and fusion beats are confirmatory findings.

Clinically ventricular tachycardia is most often associated with recent myocardial infarction. It also occurs with severe coronary insufficiency without infarction, during cardiac catheterization, after electrical shock, with quinidine intoxication, and in terminal states from many conditions.

Ventricular flutter represents progression of the arrhythmia toward a more chaotic state. Ventricular flutter waves resemble a sine curve and have rates greater than 250. Immediately prior to death the cardiac rhythm may become totally disorganized and irregular undulations of the baseline appear. This terminal electrical activity is called ventricular fibrillation.

Artifacts

Telemetry

Noise - About same both channels

Inversely proportional to distance

Spikes - From switches and solenoids near transmitter

Appear same on both channels

Patient Artifacts

Muscle potentials - Small irregularly recurring spikes of different sizes

Motion artifacts - Pressure on electrodes: sharp to slow

Shift of baseline

Frequently affects on channel more than other

Usually accompanied by muscle potentials

U. S. NAVAL SCHOOL OF AVIATION MEDICINE
CARDIOLOGY COURSE
PART I

INTRODUCTION

Fundamentals of Electricity

Pertinent to ECG

1. Conductivity - term used to describe how well or how poorly a substance conducts electricity.

   Infinite

   Finite

2. Resistance reciprocal of conductivity

   $R = \frac{1}{\text{conductivity}}$ measured in ohms.

3. Ohms Law $E=IR$

   E = EMF (def: push or driving power (potential))

   I = Current (def: amount of electricity flowing)

   R = Resistance (def: obstruction to flow)

   Except under rather special conditions (electrostatic charges), current (I) and potential (EMF) are intimately interrelated.

   In ECGology the current flow is so small that it is convenient to confine our discussion almost exclusively to potentials.

Detection & Measurement of Electrical Forces

Extremely high potentials

   Spark, arc, lightning

Smaller potentials

   Measure with VOLTMETER

   Indicator (needle, mirror etc) attached to coil of wire suspended in magnetic field.

   Left hand rule: coil and hence indicator is deflected by the interaction of the magnetic force set up by the electricity in the coil and the magnetic field in which coil suspended.

Characteristics of a Voltmeter

1. Two terminals + and -
2. When + terminal more positive than - terminal, a positive reading is obtained.
3. When + terminal more negative than - terminal, a negative reading is obtained.
4. Voltmeter measures potential difference between two points.
5. This is an algebraic difference:

(Plus terminal voltage)-(Negative Terminal Voltage=Reading

Positive Pole	Negative Pole	Meter Reading
+2	0	+2
+2	-2	+4
-2	+2	-4
0	-2	+2
0	+2	-2
+2	+2	0
-2	-2	0
+1002	+1000	+2
+5	+3	+2

Types of Voltmeters

1. Coil and mirror

   Extremely sensitive

   capable of measuring very small potentials

   very limited in frequency response and the more sensitive the more limited.

2. String galvanometer

   very sensitive

   fair frequency response

   best example of left hand rule

3. Amplification prior to measurement (VTVM)

permits variety of measuring tech

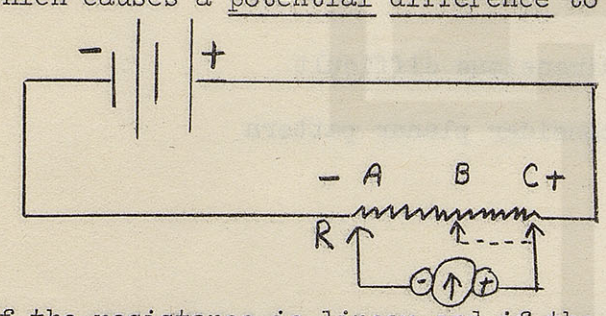
1. direct writer ECG machine
2. Cathode ray oscilloscope
3. Oscillograph

Resistance, Potentials, and Bathtubs

With rare exception when electricity is conducted through a substance the conductivity is finite and it presents a definite though usually small resistance to its flow.

In copper or silver wire resistance is small (conductivity great). The voltage drop over a considerable length may be so small it is difficult to measure. Still the voltage drop occurs.

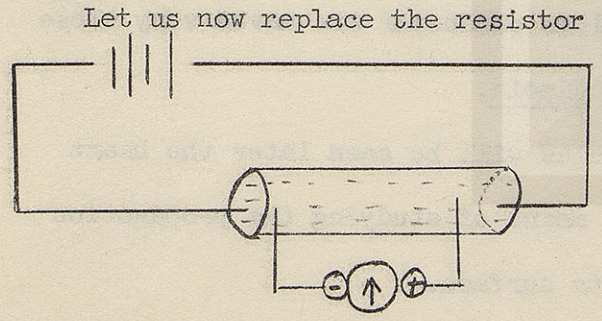
Stated in another way, the wire has a definite resistance (measured in ohms per foot) which causes a potential difference to appear along its length.



The resistance (R) in the sample circuit shown here causes a potential drop to occur along its length. If a measurement were taken at AC it would be found to be greater than AB. If the resistance is linear and if the distance AB=BC; then the potential difference measured at AC would be twice that at AB.

The resistance of a finite conductor permits it to develop and maintain potential differences. These potential differences can be measured with a suitable voltmeter or ECG machine.

Let us now replace the resistor in the circuit with a volume conductor (tap water or saline in a plastic tank). Measurements can again be made of the potential differences either at the surface or within the tank.



The tank behaves exactly like resistor as far as A & B are concerned. Measurements taken along electrode axis will show potential difference (voltage drop) which is proportional to distance and conductivity.

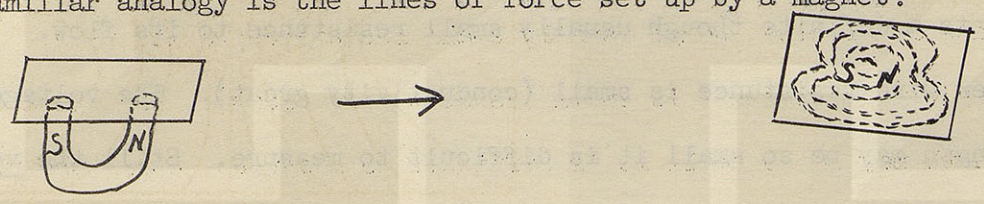
Volume Conductors

The path of an electric current in a volume conductor has been extensively studied.

Path is dependent upon:

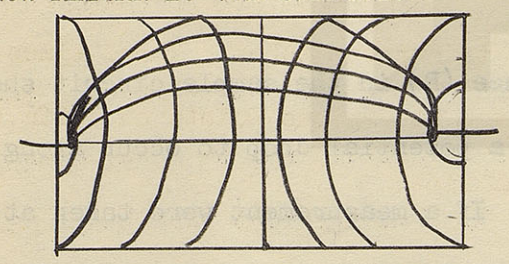
1. shape of vol. conductor
2. conductivity of medium
3. position of source electrodes in tank

Most familiar analogy is the lines of force set up by a magnet:



and easily demonstrated with iron filings on paper above the poles of the magnet.

NOW RETURN TO VOL CONDUCTOR



3 dimensions difficult

∴ consider planar pattern

Current travels between the two points in the volume conductor in spagetti like tubes as noted above. Voltage can be measured between any 2 points in vol. conductor of map. With voltmetor with many readings find that along definite lines voltage is constant. These are the lines of isopotential and are perpendicular to current tubes.

Dipole in Volume Conductor

If the two point sources of the electricity in the volume conductor are moved very close together (instead of being far apart as above) they form a dipole.

Since the body behaves like a vol. conductor and since as will be seen later the heart behaves like a dipole this technique provides a very clear means of studying the propagation of the heart's electrical forces through the body and to its surface.

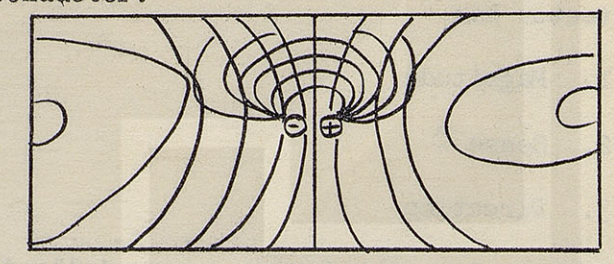
Conductivity of living tissues relatively poor hence, body can be treated as a volume conductor. Only problem is its representation in suitable contour and shape.

The two points from which the electricity originates are usually termed:

Source & Sink

Current flows from source to sink. In electrocardiography we are particularly interested in the path which it follows through the volume conductor.

Dipole in Volume Conductor



Again current flows in tubes from source to sink and fills volume conductor. All tubes begin at sink and end at source.

Again lines of isopotential exist which extend at right angles to current tubes. These lines also extend to boundaries thus can determine potential difference at various points on surface.

Relation of current and voltage to generator driving source and sink:

When EMF ↑ Current (I) ↑

and when EMF ↓ current ↓

likewise

when EMF small - potential difference is small.

Field Determenants

When dipole strength constant (EMF & I constant) the factors which control the current lines and their path are:

1. the orientation of the dipole - position of heart - e.g. PNX
2. the contour & dimensions of the boundaries of the col. cond.
   (Anatomical differences of chest contour)
3. The conductivity of the media of V.C. (Pericardial effusion, Pleural effusion)

Scalar Values

Scalar values are algebraic quantities such as + 1, + 3, - 5, etc

They are the readings on the voltmeter or ECG machine

They give no idea of direction.

Vectors

A vector has

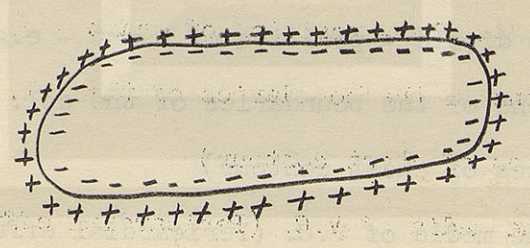
1. Magnitude
2. Sense $\pm$
3. Direction

Useful to describe forces - especially interacting forces.

Electro-physiology of an excitable cell

A. Properties of living cells

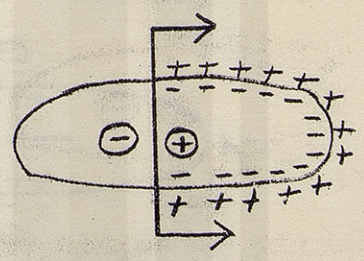
1. Semipermeable membrane which limits ionic transfer
2. Insulating or dielectric membrane. Note: 1. and 2. may be identical.
3. Mechanism for active transfer of ions, especially sodium. "Sodium pump". This transfer may actually involve movement of bound ions. Potassium transfer is probably passive.
4. Resting, or polarized, state characterized by cations outside the cell in excess of anions.
5. Therefore, outside of cell is 75-100 mv. positive compared to inside.
6. This is a stable condition; the membrane prevents current flow despite this difference in potential
7. Diagramatically:



B. Depolarization:

1. Stimulus leads to local loss of dielectric and, in part, semi-permeable properties of the membrane.
2. Current flow occurs as Na enters and K leaves the cell. Transient overshoot.
3. Thus, a dipole is produced; lines of current and lines of isopotential appear.
4. The membrane adjacent to such a dipole is, in turn, depolarized. Thus dipole progresses along the cell.
5. Diagramatically:

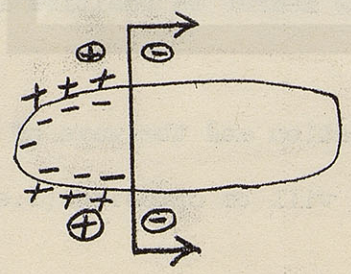
6. Depolarized portion is negative with respect to the yet undepolarized portion.
7. <u>Important</u> The wave of depolarization has positive leading edge and a negative trailing edge



8. Perpendicular to the direction of propagation the polarity is zero.

C. Repolarization:

1. Restoration of semi-permeable and dielectric properties of membrane. Also sequential.
2. Active ionic transfer becomes effective, as Na leaves, K enters cell and anions are retained in cell. Transient overshoot.
3. Trans-membrane potential and cellular excitability re-established.
4. Diagramatically:



5. Wave of repolarization has negative leading edge and a positive trailing edge.

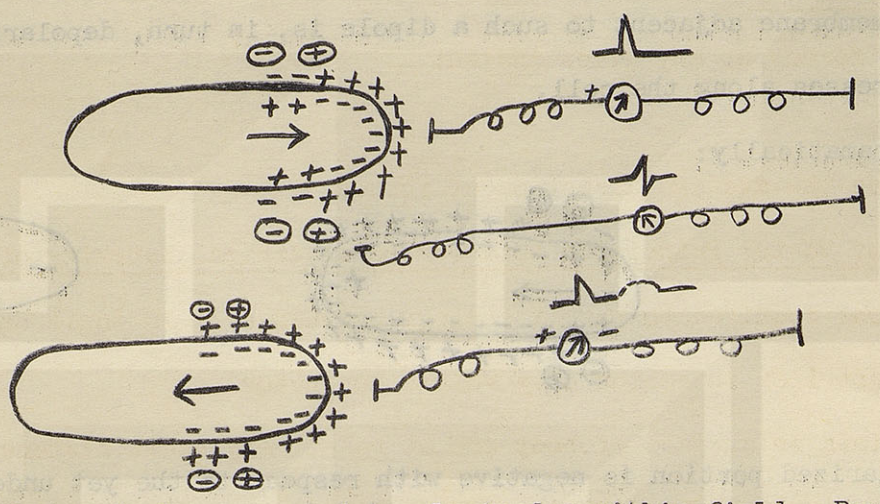
6. Repolarization requires biological work and reflects health of cell. In contrast depolarization is an all or nothing response.

Recording of electrical events:

1. Unipolar lead. Negative terminal at zero potential; positive terminal within electrical field.

   a. Depolarization

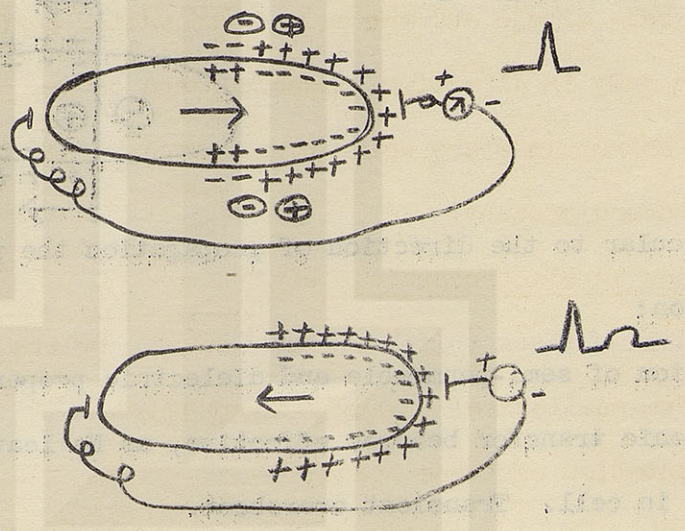
   b. Repolarization



2. Bipolar leads: Both terminals connected to electrodes within field. Resultant is algebraic difference between electrodes.

   a. Depolarization

   b. Repolarization



3. General rule: positive pole of dipole nearer to positive terminal produces positive deflection.

4. General Rule: when wave of depolarization and the wave of repolarization move in same direction, their recorded deflections will be opposite (i.e. if one positive, the other will be negative).

5. Coorelary: If move in opposite directions, their recorded deflections will be of same polarity.

Vectorial Representation

Depolarization and Repolarization forces may be described vectorially.

Instantaneous Vector:

Depicts forces at any given instant

Mean Vector

Describes the average direction of forces.

Application of Vector Principles to describe electrical activity of heart:

Assumptions required to permit analogy between cell in ionic bath and heart in body.

1. Heart lies in center of a volume conductor
2. Points on surface of body are at relatively great distance from heart.
3. The simultaneous activity of multitude of dipoles within heart may be expressed in terms of a single dipole and instantaneous vector.
4. This average dipole and the associated vector quantity remain centrally located, changing only in direction, magnitude and sense.
5. These assumptions are only approximately true, but permit accurate clinical electrocardiography.

Conventional leads in electrocardiography

1. Standard limb leads (bipolar limb leads). Derived by Einthoven with postulate that limbs from equilateral triangle about heart.
   a. Orientation of these leads:

	pos terminal	neg terminal
lead I	LA	RA
II	LL (F)	RA
III	LL	LA

b. I + III = II

c. Limbs are effectively linear conductors

d. The two legs are electrically equivalent.

e. Einthoven's triangle; triaxial reference figure.

Unipolar leads: positive terminal connected to an exploring electrode; negative terminal at zero potential, i.e. at center of dipole.

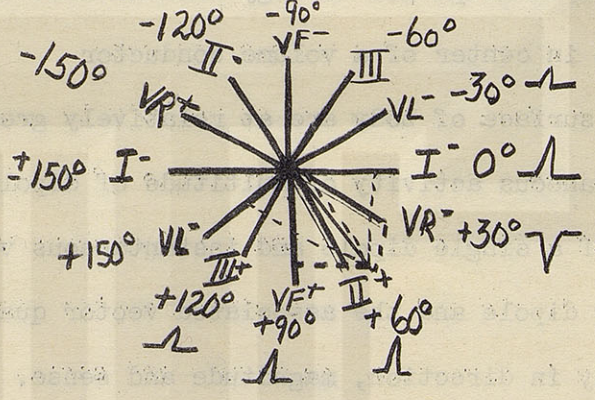
1. Wilson's central terminal. Kirchoff's Law. Leads VR, VL, VF.

2. Goldberger's modification. Leads aVR, aVL, aVF.

   a. aVR = 3/2 VR.

Standard and unipolar limb leads represent frontal plane of body. Hexaxial figure of reference.

1. Interrelationship of mean frontal plane vector to limb lead electrocardiogram.



Discrepancies of Sensitivity

a. Goldberger unipolar extremity leads (aVR, aVL, aVF) are only 87% as sensitive as standard bipolar leads (I, II, III)

b. Goldberger leads 50% more sensitive than Wilson V bal.

c. GPG Box corrects voltage of unipolars, inverts aVR. New leads: VVL, VVR, VVF.

Unipolar precordial leads: Electrodes in plane about chest sample electrical forces in horizontal plane of body.

1. Placement of precordial electrodes

   V_1 - 4th ICS, right sternal border

   V_2 - 4th ICS, left sternal border

   V_4 - MCL at level of apex beat, or in 5th ICS

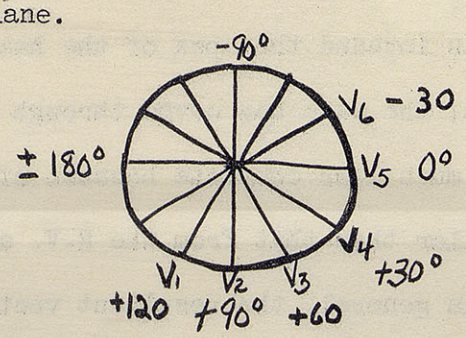
   V_3 - midway on line between V_2 and V_4

   V_5 - left ant. axillary line at level of V_4

V_6 - left mid axillary line at level of V_4

a. Approximation of a horizontal plane.

2. Diagrammatic representation:



3. Proximity error

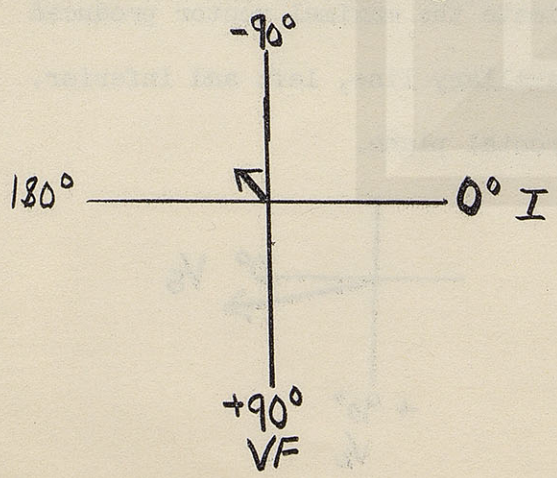
Summary:

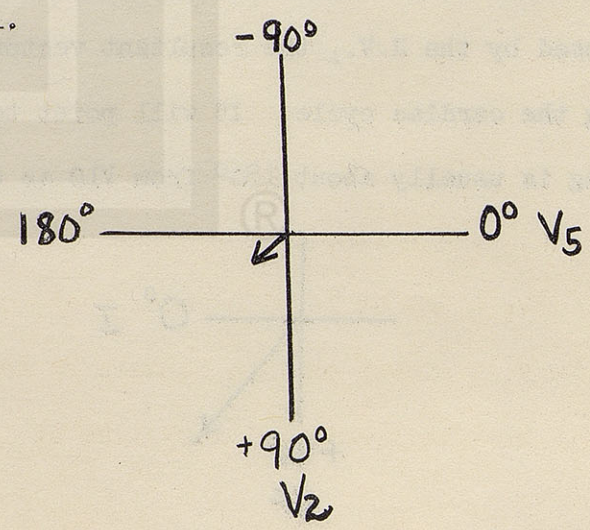
1. The 12 lead conventional ECG consists of 6 leads in frontal plane (FP) and 6 leads in horizontal plane.
2. In both planes the leads form a hexaxial reference system. Thus in essence a bihexaxial reference system.

The vectorial representation of ventricular depolarization

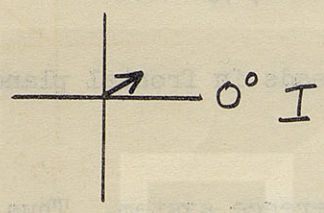
For convenience of description, the process of depolarization of the human ventricles can be depicted by 6 distinct vectors. These instantaneous vectors are chosen because they are most helpful and have characteristic relations between each other. They are the instantaneous vectors at 10, 20, 30, 40, 50, and 70 milliseconds after the onset of depolarization. They will be referred to as V10, V20, etc.

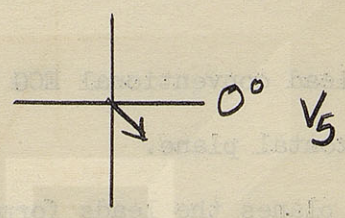
V10 is representative of the initial forces of depolarization which arises exclusively from the septum. Since this structure is supplied chiefly by the left bundle branch, the earliest activity is in the left subendocardioum and spreads, therefore, from the left side of the septum to the right. If one keeps in mind the anatomical position of the septum, it is clear that the vector which describes this field will point anteriorly, slightly superiorly and to the right. The magnitude will be small.



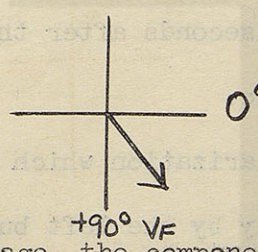


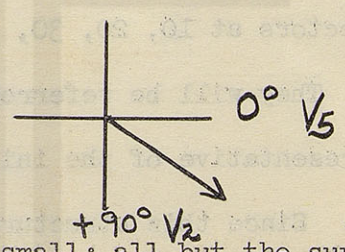
V20. At this point, most of the central portion of the septum has depolarized and the wave of excitation has invaded the apex of the heart. Because the right ventricle is much thinner than the left, the wave has moved through this wall to the surface. On the left side this process is much less complete because of the thickness of the wall. The component from the L.V. is smaller than that from the R.V. and this is the only time in the whole cycle when this is true. In general, the resultant vector points to the left and anteriorly.



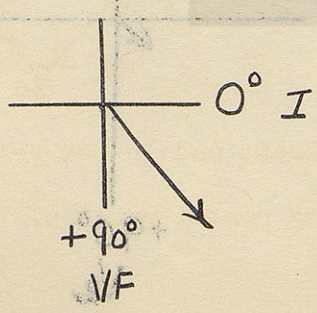


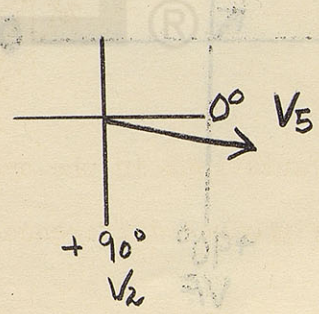
V30. At this stage, most of the free wall of the right ventricle is depolarizing. The R. V. component will point to the right, anteriorly and inferiorly. The component from the L.V. will be much greater in magnitude and will point to the left inferiorly and posteriorly. The resultant of these two will point left, slightly anteriorly and inferiorly.



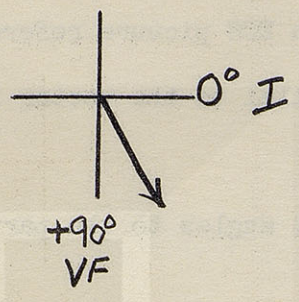


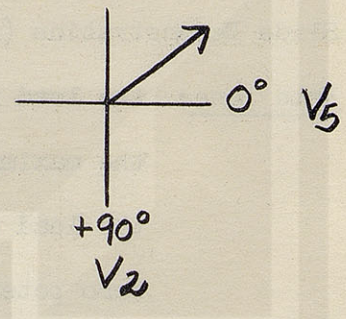
V40. At this stage, the component from the R.V. is small; all but the superior portion of the R.V. has been depolarized. It will thus point to the right, superior and anteriorly. At the same time the great mass of the L.V. is still depolarizing. This is largely the lateral wall and the lateral portion of the anterior and diaphragmatic surface. Since it is largely unopposed by the R.V., the resultant vector will approximate the maximal vector produced during the cardiac cycle. It will point toward the midaxillary line, left and inferior. Its heading is usually about 180° from V10 as seen in the frontal plane.



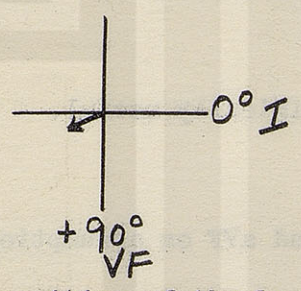


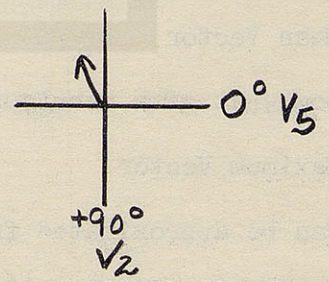
V50. Now, the depolarization process has swept over the anterior and lateral portions of the left ventricle and will be progressing more posteriorly. The magnitude of the forces from the left ventricle is still rather larger although the maximal vector for this complex will have already occurred. The right ventricle meanwhile is contributing a small, usually insignificant force superiorly and to the right. The resultant vector will be posterior, inferior, and to the left.





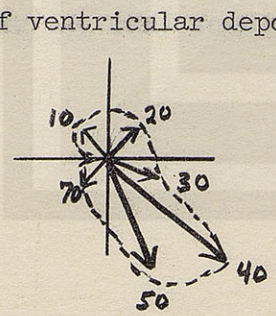
V70. The vectorial representation of the electrical forces occurring at this time are generally quite variable. This is largely determined by the mass and location of the muscle fibers around the base of the left ventricle. The resultant vector will almost invariably be posterior but may vary slightly from right to left and from superiorly to inferiorly.

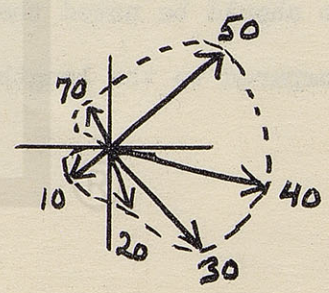




Obviously the anatomic position of the heart will effect materially the headings of the component portions of the electric field.

If one joins the heads of these instantaneous vectors by a continuous line, the so-called vector loop has been made. Actually, the loop may be defined as the locus of the heads of all the instantaneous vectors of ventricular depolarization.





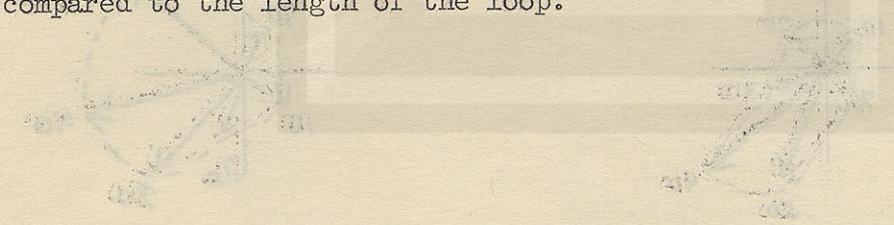
Note that minor variations in the anatomic position of the heart and in the relative electrical preponderance may produce differences from this pattern among normal persons.

Derivation of ECG from Instantaneous Vectors

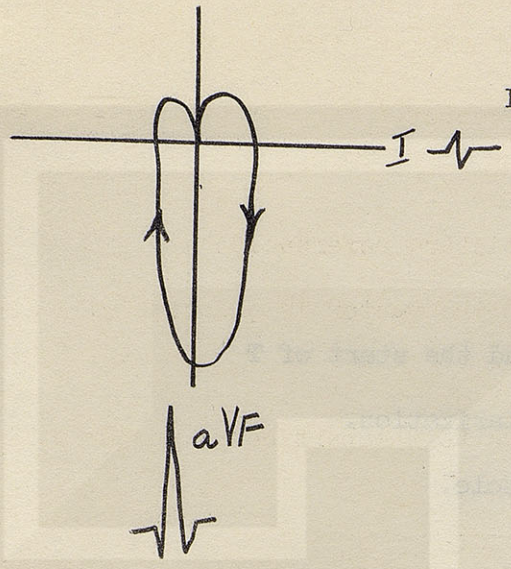
1. Projection of the vector on each lead. To accomplish, draw perpendicular from each lead of vector.
2. Repeat for each "significant" instantaneous vector.
3. Works equally for P, QRS, ST, and T.
4. Slide Demonstration (Also available in ECG picture reference)
5. Note Bene: the lead most nearly parallel to the vector in question will record the maximal deflection.
   the lead which is at right angles to the particular vector will be at zero potentials.

Derivation of Vectors from ECG

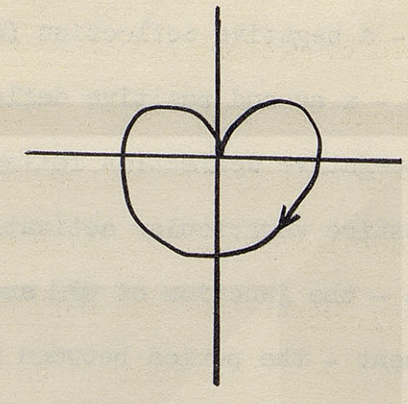
1. Instantaneous vectors.
   Possible only if 2 or more ECG leads are simultaneously recorded.
   If have x y & z axes can find spatial vectors.
2. Mean Vector
   Somewhat of a nebulous concept, but clinically useful.
3. Maximum Vector
   Can be approximated from R in Lead I and aVF on assumption that peak of R occurred simultaneously in both leads. Subject to error.
4. Best method for obtaining average vector
   Approximation obtained by determining the transitional lead (s) in each plane and placing vector perpendicular to each in direction of positive deflections.
   It should be noted that this method postulates that the width of the loop is small compared to the length of the loop.



Since this usually is true it is justifiable and clinically useful

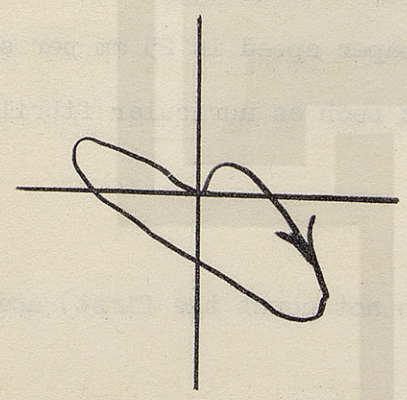


But:



Here no transition and all leads are essentially R=S. Fortunately unusual

Another complication: The MV, MV^1 type of loop:



Often seen S_1 S_2 S_3, RVH, and occasionally in RBBB.

Conventions in Clinical Electrocardiology

a. Paper speed - 25 mm./sec.

b. vertical markings - fine lines at 0.04 sec. intervals; broad lines at 0.2 sec. intervals.

c. one terminal designated as positive, i.e. current entering this terminal leads to an upward deflection of stylus.

d. Standard deflection: 1.0 cm = 1.0 mv. = 10 mm 1 mm = 0.1 mv.

Standard terminology of ECG tracing:

P wave - initial complex - atrial activation

PR interval - from start of P to start of QRS

PR segment - from end of P to start of QRS

Q wave - an initial negative deflection in QRS

R wave - the first positive deflection in QRS

S wave - a negative deflection following R

R' wave - a second positive deflection

S' - a negative deflection following R'

QRS - entire ventricular activation comples

J point - the junction of QRS and ST

ST segment - the period between the end of QRS and the start of T

T wave - the deflection follwoing ST; vent. repolarization.

QT interval - Q (or R) through T; electrical systole.

Method for determining rate:

First determine relationship of auricular and ventricular depolarization.

The following rules apply to the standard ECG whose paper speed is 25 mm per second

1. Average rate - very useful in irregular rhythms such as auricular fibrillation.

   1 in = 2.5 cm - 25 mm - 1 sec.

   6 in = 6 sec.

   Hence count number of complexes in 6 inches (do not count the first) and multiphy by 10.

   Or if shorter lead (less than 6 inches) count number in 3 inches and multiply by 20.

2. Rate based on 2 complexes

   This useful to obtain extremes in rate.

   A. Measure the R to R time in seconds and divide into 60.

      i.e., R to R = 1 sec.

      $\frac{60}{1}$ = 60 beats per minute.

B. Approximation by large blocks (5 mm or 0.20 sec)

If rate were fast enough so that a complex fell on each successive heavy line (i.e., each 0.20 sec) then

$$\frac{60}{.2} = 300 \text{ beats per minute.}$$

Hence:

1 block = 300

2 blocks = 150

3 blocks = 100

4 blocks = 75

5 blocks = 60

6 blocks = 50

By inspection and interpolation then one may determine the approximate rate of a tracing.

C. The 1500 Factor

The paper moves at rate of 25 mm/sec. Hence in one minute 25 x 60 = 1500 mm or small blocks, pass under stylus each minute.

To determine rate, divide the R to R distance in mm (or number of small blocks) into 1500

For example:

R to R = 50 small blocks

$$\frac{1500}{50} = 30 \text{ beats/min.}$$

Amplitude Measurements

Measure from upper edge of baseline (TP segment) to peak of complex or lower edge of baseline to lowest point of complex.

U.S. NAVAL SCHOOL OF AVIATION MEDICINE
CARDIOLOGY COURSE FOR STUDENT FLIGHT SURGEONS

PART II

Normal Electrocardiograms

Our knowledge of the normal electrocardiogram is based on empirical clinical correlations observed over many years plus the fairly recent intuitive concepts which have resulted from our increased knowledge of electrophysiology. Where the two approaches conflict we usually accept the empirical observations. Until comparatively recently our knowledge of the electrocardiogram was based on a large number of tracings taken on symptomatic, clinically abnormal patients and a small number of records on normal patients. The normal electrocardiogram was thus interpreted as any tracing which did not demonstrate the characteristics of the known abnormal tracings. With the increase in the number of tracings taken on normal asymptomatic individuals, our knowledge of the normal variations possible has been augumented and our criteria for the diagnosis of normal and abnormal have become more flexible. The recent discovery of bundle-branch-block conduction defects in young, healthy men given a routine electrocardiogram provides a good illustration of the problem presented by discovering this defect in an elderly patient without any definite history of heart disease. The trend today is to place more credence in changes from previous records rather than making a categorical diagnosis on the basis of a single tracing. This presumes the existence of a baseline record taken at an age when the subject is believed to be normal.

Thus, there are many variants in the electrocardiograms of normal individuals which may be misinterpreted as evidence of heart disease. The vector concept aids in preventing misinterpretation of normal variations and a means of defining the normal changes in serial electrocardiograms.

Interpretation of the record is best accomplished by the following of a definite routine:

Critique

In attempting to ascertain whether a tracing is normal or abnormal, one must have a clear knowledge of normal variants. In addition, one should have a definite plan of attack or a routine for reading a tracing. If one does not adapt such a plan it is exceedingly easy to miss a very obvious and easy diagnosis. Similar reasoning accounts for the check-off lists in all Navy aircraft.

ECG Check-Off

1. Preliminary Survey of Recort to Determine:

Artifacts

AC interference - lack of ground-cable pick up

Muscle tremor - tense patient - cold patient

Wandering baseline - dirty electrodes, poor contact

Mixed leads - carelessness

Sufficient leads available - All ECGs should have at least 12 leads

Check for correct mounting and designation

Drugs - particularly digitalis and quinidine.

2. Standardization:

In a properly taken ECG the standardization voltage will appear in each lead. Since the standardizing voltage is 1.0 m.v. (millivolts), the deflection of the baseline should be 1 cm. or 10 mm or 10 small 1 mm blocks. If it is less than 1 cm, the amplitude of the recorded deflections will be less than "normal" and conversely if greater than 1.0 cm they will be falsely greater than norma. This is quite important in comparing serial tracings - especially when attempting to evaluate changes in T and ST.

3. Rate:

First determine relationship of auricular and ventricular depolarization.

Is each P followed by a QRS? If not are the two independent?

Determine auricular rate

Determine ventricular rate

Normal sinus rhythm 60 to 100

Less than 60 - bradycardia

Greater than 100 - tachycardia

4. Amplitude Measurements

Actually a measure of voltage of a given complex

Usually measured in mm rather than millivolts

With normal standardization:

0.001 volt = 1 millivolt = 1 cm = 10 mm

hence: 0.1 m.v. = 1 mm

Deflections above baseline positive, below negative

Positive deflections measured from top of baseline to nadir of complex

Negative deflections from bottom of baseline to lowest peak of complex.

Evaluation of the individual complexes and segments as to:

Amplitude, duration, configuration. While this purely mechanical measurement and observation seems dull, it is nevertheless true that a diagnosis of abnormality can be established on the basis of abnormal duration and amplitude alone in about half of the abnormal ECGs seen.

5. Rhythm (Sequential order of appearance):

Identify P and QRS in each lead

Determine whether P and QRS spacing is regular or irregular

Note reaction of each P and QRS - constant or variable

P wave present and of uniform configuration

QRS contour uniform or variable

Sinus arrhythmia: phasic rate fluctuation in otherwise NSR.

Wandering atrial pacemaker and transient noday rhythm are

not unusual and do not necessarilly suggest underlying disease.

The Individual Complexes of the ECG

I The P Wave,

The resultant of the contribution of right and left atrium.

A. Duration: (measure on concave side)

0.10 = 0.1 sec.

Greater than 0.12 sec. abnormal

Suggests hypertrophy or disease.

B. Amplitude

2.0 mm = 0.5 mm

Greater than 3.0 mm abnormal (hypertrophy)

C. Mean P Vector (Frontal Plane)

+15 to +75 (95%)

6000 USAF Males - 16 to over 45 yrs

+45 mode

Tends to follow QRS axis

Little or no relation to age in adults

Vector to right of +90° may indicate dextrocardia, ectopic atrial rhythm, or right atrial enlargement; vector less than -30° may occur with left atrial enlargement.

Horizontal plane: variable projection usually results in biphasic, upright, or inverted P waves of low amplitude in precordial leads.

Prominent inverted P in V_1 frequently seen in marked pectus excavatum.

II Auricular T waves

represent atrial repolarization. Depression of PR segment and often ST segments; greatest in leads where P is prominent and rate rapid. May cause difficulty in measuring PR or may be misinterpreted as pathologic ST depression. Usually last in QRS. Best seen in complete heart block.

Heading T_A - Approximately 180^o opposite to P wave.

Recall that if wave of depolarization and repolarization follow same path that their recorded deflections will be opposite in direction.

III PR segments

passage of excitation through the slowly conducting AV node

differ from PR interval (excitation of auricle plus junctional) which is P wave plus PR segment.

Absent PR segment in W-P-W.

IV PR interval

Time from onset of P to onset of QRS.

Duration - (0.12 to 0.20) wide individual variation and varies with heart rate, age, build (tables available) with some tendency to increase with age. Difficult to measure without simultaneous leads for both P and QRS may be isoelectric at start or finish.

Rule: measure longest in frontal plane.

Prolongation of A-V conduction by various processes delay PR interval, notably rheumatic carditis (serial changes important)

Shortening (below 0.12) in W-P-W and nodal rhythm when conduction does not proceed through the A-V node.

Prolonged PR (greater than 0.20)

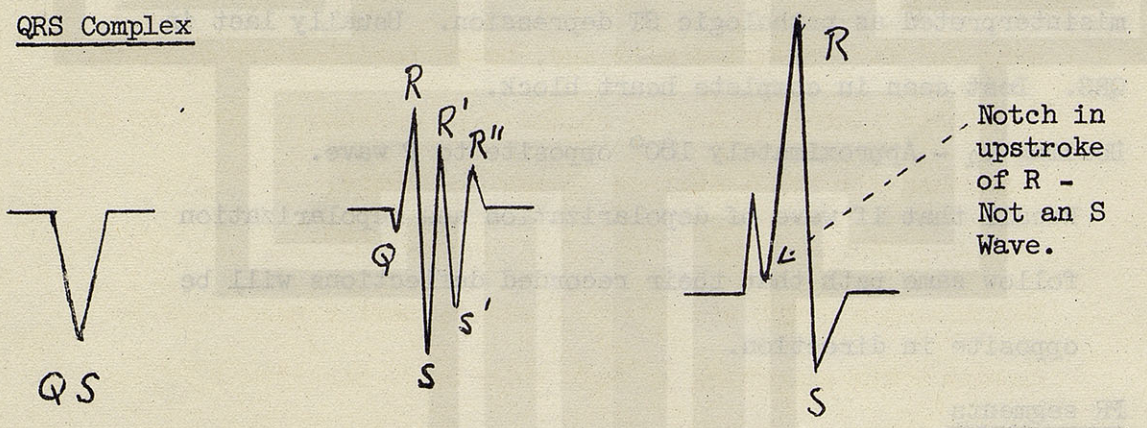
By definition first degree heart block

Frequently physiological in athletes

Here not fixed. Shortens with standing or exercise.

Thus, if prolonged and fixed - abnormal.

V <u>QRS Complex</u>



A. Mean spatial vector:

1. Frontal plane: usually 0° to $+90^{\circ}$; less frequently may occur normally in range from -30° to $+120^{\circ}$
   a. Left axis deviation: -30° to -90°
   b. Right axis deviation: $+120^{\circ}$ to -90°
2. Horizontal plane: 0° to -60°
3. Limitations of spatial vector approach-chiefly the horizontal plane
   a. Variations in chest size and shape may cause striking deviations of vectorial representation in certain of the precordial leads.
   b. The location of the heart in the chest may contribute to unusual patterns.

c. In rare instances immediately subjacent ventricular potentials may be transmitted to precordial leads, rather than the effects of an average vector which is transmitted to all leads.

d. Sensitivity of precordials differ from one to another depending on proximity of electrode to heart. This causes amplitude to be independent of vectoral heading (proximity error).

B. QRS Heading

Mean QRS vector varies with age, heart position and disease.

Infants plus 130° due to relative prominence of RV - do not mistake for RVH - changes at age 1-2 years.

At 2-5 years - plus 50° Puberty +67°

Dextrocardia - QRS and T 90° to right, best single clue to dextrocardia according to Sodi Polaris is inverted P in lead I.

Thousand Aviator Study:

QRS Heading in F.P.

Av. Age	Year	Heading
24.6	1942	+60.8 ± 26.9
36	1952	+47.9 ± 29.1
42	1958	+41.6 ± 34.5

In the men with marked LAD

Cholesterol higher

More weight gain

Higher blood pressure.

C. QRS Amplitude

Based on Simonson study of 960 healthy men and women

(97.5 percentile cut off)

Maximum amplitude of normal:

R_{V_6} = 20 mm

S_{V_1} + R_{V_5} = 33 mm in women

S_{V_1} + R_{V_5} = 36 mm in men over 30 years

S_{V_1} + R_{V_5} = 44 mm in men under 30 years

Sokolow: Maximum Normal

Ra_{VL} = 11 mm

Ra_{VF} = 20 mm

Gubner

R_1 + S_3 = 25 mm

Not often exceeded but when occurs a reliable guide to L.V.H.

Low Voltage

Said to present if all FP leads are less than 5 mm

or all HP leads less than 7 mm

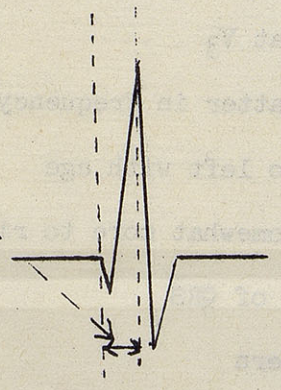
Factors to consider in assessing amplitude:

1. Amplitude diminishes with age.
2. Amplitude greater in thin chested
3. Amplitude affected by standardization
4. Amplitude diminished in
   a. emphysema
   b. pericardial effusion
   c. plural effusion

D. Intrinsicoid Deflection.

V_1 = 0.035

V_5 V_6 = 0.045



E. Q Waves

a. FP normal limits (except aVR and aVL)

25 % of following R wave

.03 sec. duration

b. HP normal limits

V_5 and V_6 up to 3 mm and 0.03 sec.

F. QRS Duration

1. Measured only repeat only in F.P.

2. Value recorded is max duration found in a F.P. lead

3. Usually 0.07 to 0.09 sec. Approx 3% normals 0.10 sec or more

4. Longest normal durations seen in $S_1S_2S_3$ syndrome

5. In a given lead either initial or terminal portion of QRS may be isoelectric

6. H.P. duration 0.10 to 0.02 sec. longer than F.P. because septal forces are essentially dead anterior hence do not record in F.P. leads.

G. QRS in HP

1. Proximity error

2. R wave progression. Actually R/S amplitude ratio

a. Rarely is V_1 a QS complex but more common in female.

b. A decrease in the R/S amplitude ratio suggests an old anterior wall infarction.

3. Transition at V_3

   a. More scatter in frequency distribution in young

   b. Moves to left with age

   c. Women somewhat more to right than men.

H. Normal Variants of QRS

1. $S_1S_2S_3$ pattern

   a. Incidence 150/1000 (Lamb) but diminishes with age.

   b. Initial and maximal forces normal

   c. Terminal forces right, posterior and superior

   d. Do not confuse with RVH or RBBB

   e. QRS duration 0.09 sec. or more. Occasionally up to 0.12 sec.

   f. Terminal forces thought to be derived from crista supraventricularis

2. $S_1S_2S_3$ with $R'V_1$

   a. Incidence 15/1000 (Lamb)

   b. Essentially same as $S_1S_2S_3$

   c. Rarely if ever R' in V_2

   d. Frequently a terminal S' in V_1

VI ST SEGMENT

1. Conceptually isoelectric

2. Actually normally elevated 1.0 to 3.0 mm in healthy young males in V_1, V_2, or V_3

3. Normal deviation is indirection of T wave and as a rule the longer the T, the greater the elevation.

4. ST depression in normal subjects is rare.

5. ST deviation opposite in direction of T is usually abnormal.

6. ST may be depressed physiologically by T_A
7. The normal ST when elevated is concave from above
8. When bowed or convex from above it is rarely normal
9. After exercise, ST tends to follow the S wave. Such ST depression is physiological

VII T WAVE

1. T best evaluated in relation to QRS. If QRS small, T will be small and vice versa
2. In lead I, V_5 , or V_6 T should never be less than 1/10 of the accompanying R wave
3. Inverted T in V_1 (or even V_2 in young) is to be expected
4. T is never, repeat never, flat or inverted in lead I or V_5
5. Hence in F.P. T heading is abnormal when it exceeds $+85^{\circ}$
6. Tv_6 is always of greater positive amplitude than Tv_1
7. T amplitude diminishes with age and glucose administration
8. Largest Ts found in V_2 V_3 and V_4 where the 97.5 percentile cut-off in males is 10.44, 11.06, and 10.49 respectively.
9. Minimum Ts in V_2, V_3, and V_4 is 1.67, 2.14, and 1.59 (same criteria as #7 from Simonson)
10. Thousand Aviators

T Vector

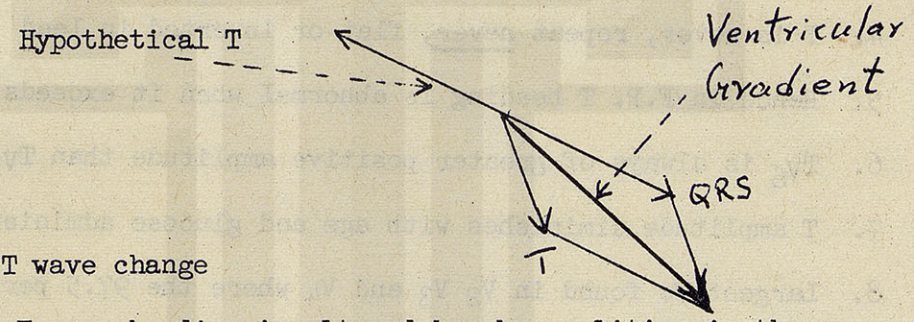
	Age	Heading
1942	24	$+ 43.1 \pm 18.2$
1952	36	$+ 41.1 \pm 18.4$
1958	42	$+ 39.1 \pm 19.2$

11. Exercise increases magnitude of T vector and causes a rightward and anterior shift.

12. Inversion of T wave in V_2 and V_3 is not uncommon in anxious neurotics (N.C.A.) and may well be related to hyperventilation or excessive symptathetic - para sympathetic tone.

VIII Ventricular Gradient

1. Theoretically the T vector should be equal and opposite to that of the QRS
2. Actually this far from true.
3. The Ventricular gradient is the graphic expression of all the forces (known and unknown) which deviate the T forces from its hypothetical course.



4. Primary T wave change

   Here the T wave heading is altered by abnormalities in the cells undergoing repolarization, e.g. ischemic T waves

5. Secondary T wave change

   Here T wave heading change is caused by the abnormal sequence of ventricular depolarization, e.g. PVC LBBB

   The observation that flat T waves in Lead I and II are abnormal is based strictly on empirical observations and it is worth noting that less than 0.5% in over 5000 known normals demonstrated a flat T. The tracing should be repeated since many extracardiac effects will cause a flattening of T waves, i.e. smoking, drinking cold water, tachycardia.

IX QRS-T ANGLE

A less precise, but clinically useful means of obtaining similar information.

1. Normally 0° to 45° difference between QRS and T in frontal plane. Unusually this angle may be as great as 60°. T wave vector tends to vary less than QRS in normal subjects.
2. When QRS axis vertical or horizontal the QRS-T angle is normally wider.
3. QRS-T angle tends to remain constant with aging. T remains fixed while QRS swings.
4. All comments on QRS-T angle confined to F.P. In H.P. and spatially QRS-T angle is much wider.

X QT INTERVAL

Varies with rate

Shortened QT interval seen with digitalis administration and with hypercalcemia

Lengthening of QT interval occurs with hypokalemia, quinidine (or Procaine amide) administration, and with hypocalcemia.

XI U WAVES

Upright undulation following T waves and may represent supernormal phase

Premature contractions occur here

Differentiate from P waves in arrhythmias

Abnormal if inverted - seen in ischemic heart disease

Increase in amplitude after administration of adrenalin

XII INTERPRETATION OF FINDINGS

Remember ECG is a record of electrical potentials and do not impute more to record than it shows

Clinical interpretation plus ECG rather than ECG as the primary evidence

ECG is a record of past or present events and seldom affords much information as to future events in the heart

Where a question of abnormality exists repeat or serial tracings are extremely important.