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Interpretation of the Electrocardiogram

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Abstract
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The E.C.G. is particularly useful in the diagnosis of cardiac rhythm and myocardial infarction. It may reveal evidence of conduction defect, hypertrophy of the myocardium or pericarditis and may also indicate therapeutic response in certain electrolyte disturbances. On the other hand, non-specific E.C.G. changes are common.

INTERPRETATION OF THE ELECTROCARDIOGRAM

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ELECTROPHYSIOLOGY

Like every living cell a cardiac muscle fibre at rest is polarised; the cell membrane has a positive charge on its outer surface and a negative charge within. When stimulated the polarity reverses and the intracellular potential becomes positive. This rapid change (depolarisation) is followed by a slower recovery phase (repolarisation) until the cell is again in its resting or polarised state. As this activity spreads along the muscle cell an action potential is produced which may be detected by connecting suitably placed micro-electrodes to a recording galvanometer.

The E.C.G. is a method of recording these electrical forces which occur in heart muscle. The instrument is designed to produce a positive or upward deflection on the tracing when an impulse passes towards an electrode and vice versa. At rest, i.e. when no current is flowing, the base line of the tracing is horizontal or iso-electric.

GENESIS OF THE E.C.G.

In a normal heart the sino-atrial node originates the cardiac impulse. This impulse spreads over both atria to reach the atrioventricular node. It then passes through the bundle of His and down the main conducting bundles on the left and right of the interventricular septum. The ventricular muscle is stimulated as the impulse passes from endocardial to epicardial surface through the specialised conduction tissue of Purkinje.

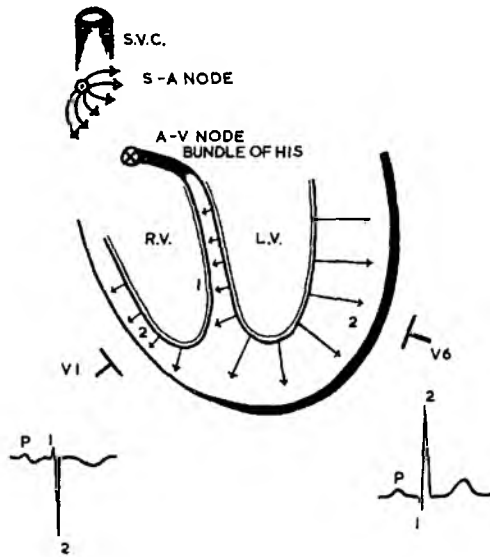These events are illustrated diagramatically in FIG. 1. The ventricular septal activation

FIG. 1

Conduction pathway of the heart to show the genesis of the ECG recorded from 2 electrodes one over the right ventricle (V1) the other over the left ventricle (V6).

(b) unipolar limb leads
   aVR — right arm
   aVL — left arm
   aVF — left leg
(c) unipolar chest leads
   V1 — fourth right interspace adjacent to sternum
   V2 — fourth left interspace adjacent to sternum
   V3 — mid-way between V2 and V4
   V4 — fifth left interspace in mid clavicular line
   V5 — in same horizontal plane as V4 but in the anterior axillary line
   V6 — as for V5 in mid-axillary line

Unipolar leads record electrical forces at the site of the exploring electrode on the individual limb or position on the chest. These routine leads provide a satisfactory survey of the electrical forces over different parts of the heart in the frontal (limb leads) and horizontal (chest leads) planes for routine recording but if more extensive cover is necessary additional leads may be recorded on the right (V3R, V4R), round the back of the chest (V7 to V9) and in higher interspaces.

in the normal heart passes from the left to the right side. Thereafter, both ventricles are activated simultaneously but the wave form produced by stimulation of the left ventricle predominates over that of the right ventricle since its muscle mass is always greater.

E.C.G. waves are designated by the letters:— P. Q. R. S. T. U. (FIG. 2); small letters may be used for relatively small waves, e.g. q, r, s. The P wave is produced by atrial depolarisation; the QRS complex is produced by depolarisation of the ventricular septum and muscle of both ventricles while the ST segment represents complete ventricular depolarisation. Subsequent recovery (or repolarisation) is denoted by the T wave and U wave.

The routine E.C.G. consists of 12 leads :
(a) standard limb leads
   Lead I — right arm to left arm
   Lead II — right arm to left leg
   Lead III — left arm to left leg

These leads are bipolar and record the difference of potential between two limbs in such a way that relatively positive electrical forces in the left arm (Lead I) and left leg (Leads II and III) are represented as upward deflections on the E.C.G.
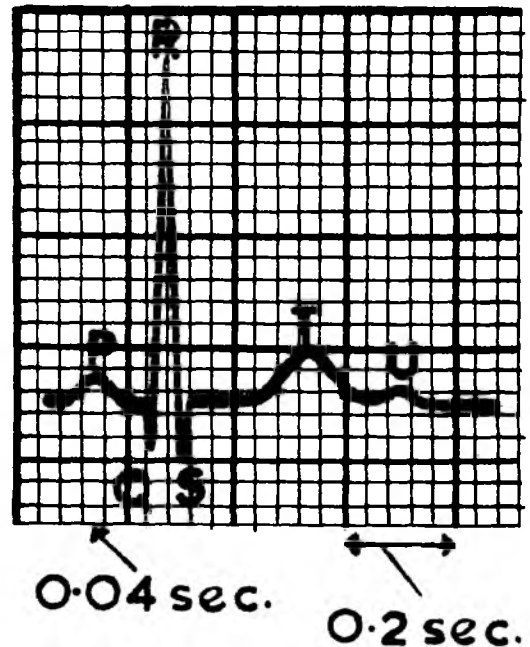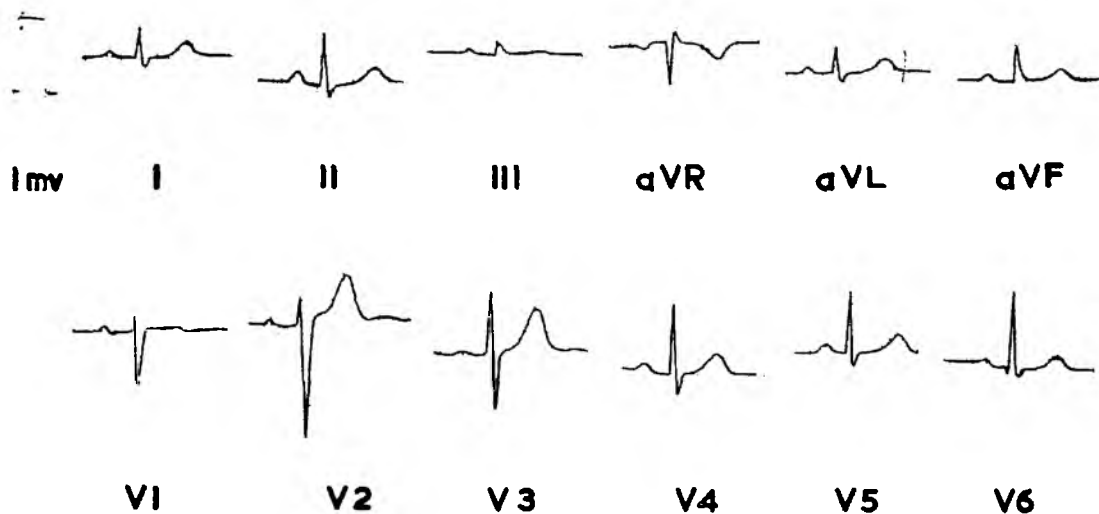

FIG. 2

Normal ECG complex.

FIG. 3

Normal electrocardiogram.

When these leads are related to the electrical forces in the normal heart it will be apparent that the significance of the different E.C.G. waves varies with the lead in which they are recorded (FIG. 3). A few examples will make this clear :

1. AVR faces the cavities of the heart. The electrical force from the SA node spreads over the atria away from the electrode and the P wave is therefore negative. Depolarisation of the septum and the ventricles also passes away from the electrode and produces a deep negative wave (Q). The T wave is also negative.

2. V1 usually faces the heart in the region of the right atrioventricular junction. The electrical force from the SA node may pass towards the electrode producing an upright P wave or may be intermediate and produce a diphasic P as illustrated; the electrical force through the ventricular septum is towards the electrode and the initial ventricular deflection is therefore a small r wave. As the ventricles are activated, the greater mass of the left ventricle generates the dominant electrical force away from the electrode and this is reflected in the deep S wave. The T wave is also negative.

3. V6 faces the epicardial surface of the left ventricle; the initial electrical force through the ventricular septum is away from the electrode so that an initial small Q wave precedes the tall R wave produced by left ventricular activation.

ARTEFACTS

An E.C.G. tracing may be distorted by electrical currents which are not produced by the heart, e.g. muscle movements of the body (shivering), faulty contact between skin and electrode, electromagnetic disturbances in the environment. The patient should therefore be relaxed, comfortable and warm; electrodes should be clean and firmly applied and the instrument must be adequately earthed.

MEASUREMENT AND TIMING OF E.C.G. WAVES

E.C.G. tracing paper is squared in millimetres with bold lines every 5 mm. (FIG. 2). In order to ensure uniformity, all records should be standardized so that an impulse of 1 mv. produces a deflection of 10 mm. in height on the tracing. Records from the same patient at different times and those from other patients may then be compared. Each mm. length represents a time interval of 0.04 second and a bold line occurs every 0.2 second. The PR interval or atrioventricular conduction time represents the time interval between the onset of atrial and the onset of ventricular depolarisation. It is measured from the start of the P wave to the beginning of the Q wave of the ventricular complex. In adults the normal range for the PR interval is between

15

0.12 and 0.20 second (3 and 5 small squares).
The normal QRS complex is less than 0.10
second and seldom more than 0.08 second (2
small squares).

HEART RATE

Heart rate is most readily calculated by
counting the number of bold lines between
comparable points on successive cardiac cycles
(usually the peaks of the R waves). This
number, divided into 300 (300/5ths of a
second per minute) gives the heart rate per
minute, i.e. 3 = 100 per minute; 4 = 75 per
minute; 5 = 60 per minute; 6 = 50 per
minute. If the points do not co-incide with
a bold line, the number of small squares may
be counted and this number divided into 1500
(1500 times 0.04 second = 1 minute). When
the rhythm is irregular, the heart rate may be
calculated by multiplying the number of car-
diac cycles between 30 bold lines (6 seconds)
by 10.

E.C.G. INTERPRETATION

The normal E.C.G. pattern in the different
leads varies to some extent with the position
of the heart in the chest, obesity, chest and
spinal deformities, the phase of respiration
and the height of the diaphragm. There is
therefore a wide range of normality which has
to be taken into account before changes in the
pattern can be attributed to heart disease. It
must also be recognised that the normal
E.C.G. per se does not exclude serious under-
lying heart disease.

DISORDERS OF RATE, RHYTHM AND CONDUCTION

Interpretation of an E.C.G. should always
be correlated with the patient's age, clinical
features and current or previous digitalis ad-
ministration. One should always look over
the whole record since the rate and rhythm
need not necessarily remain constant. At this
initial survey one can also see if the rhythm
is regular or irregular, whether the ventricular
complexes are consistent in shape or whether
bizarre forms are present. The next step is to
look for P waves and check their relationship
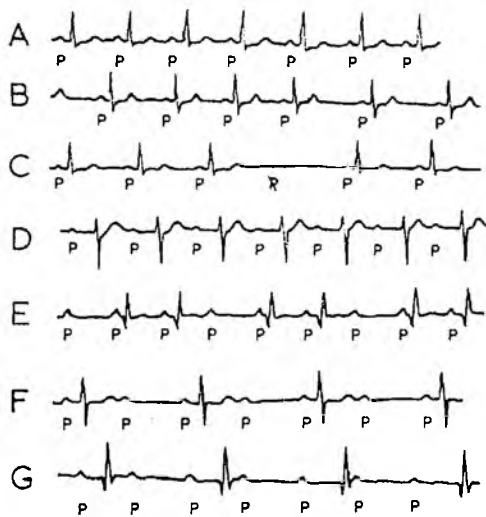to the QRS complexes. This is usually seen
most clearly in leads II and V1.

FIG. 4

A. Sinus rhythm, rate 88/min. PR 0.16 sec.
B. Sinus arrhythmia.
C. Sino-atrial block.
D. 1st degree heart block. PR 0.32 sec.
E. 2nd degree heart block; Wenckebach periods.
F. 2nd degree heart block (2 : 1 block).
G. Complete heart block, atrial rate 88/min.
   Ventricular rate 42/min.

Normal sinus rhythm (FIG. 4A) is present
when P waves precede each QRS complex;
the PR interval remains constant (within the
normal range); the ventricular complexes occur
regularly and the cardiac rate is between 60
and 100 per minute. Sinus rhythm with a
rate less than 60 per minute is a sinus brady-
cardia and when over 100 per minute is con-
sidered to be a sinus tachycardia. Variation
of the heart rate with respiration, becoming
faster with inspiration and slower with expir-
ation, is called sinus arrhythmia (FIG. 4B). It
is a normal variant and should not be confused
with sino-atrial block which is abnormal. Sino-
atrial block is caused by failure of the SA node
to initiate a cardiac impulse resulting in
absence of a complete cardiac cycle (FIG. 4C).

Atrioventricular block which is also abnor-
mal may occur in various forms. First-degree
atrioventricular block is present when the PR
interval exceeds 0.2 second (FIG. 4D). Second-
degree AV block occurs in two forms. The
first is characterized by a series of cardiac cycles
in which there is progressive lengthening of
the PR interval until an impulse is blocked
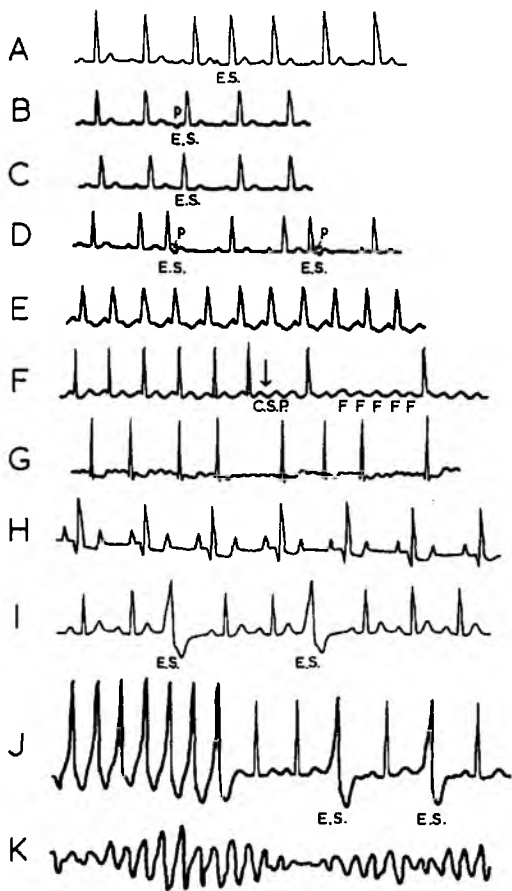and fails to initiate a QRS complex (FIG. 4E).

FIG. 5

A. Atrial ectopic.
B. Nodal ectopic; inverted P wave preceding QRS complex.
C. Nodal ectopic; P wave incorporated in QRS complex.
D. Nodal ectopics; inverted P wave following QRS complex.
E. Paroxysmal atrial tachycardia, rate 158/min.
F. Atrial flutter 2 : 1 block, atrial rate 300/min. Ventricular rate 150/min. Carotid sinus pressure (C.S.P.) increased the block and revealed the characteristic "saw-toothed" flutter waves (F).
G. Atrial fibrillation, ventricular rate approximately 100/min.
H. Paroxysmal atrial tachycardia with block, atrial rate 166/min.
I. Ventricular ectopics.
J. Ventricular tachycardia, rate 200/min. reverting to sinus rhythm. Two ventricular ectopics with the same configuration as the tachycardia are present.
K. Ventricular fibrillation.

This sequence is known as a Wenckebach period. Following each blocked beat the sequence is repeated. Wenckebach periods may be short or long but are usually constant in any particular patient. In the other form of second-degree atrioventricular block the P waves are regular and the PR interval is constant but the impulse is blocked usually after every second atrial beat (2 : 1 atrioventricular block) (FIG. 4F) or less frequently in a more complex pattern (3 : 1, 3 : 2). *Third-degree or complete heart block* is characterised by complete independence of P waves and QRS complexes (FIG. 4G). The atrial rate is usually normal while the ventricular rate, although regular, is slow at about 40 per minute or less. The ventricular complexes often have a bizarre shape since their focus of activation lies outwith the normal conduction pathway.

ATRIAL ARRHYTHMIAS

Irregularities of the rhythm may arise from ectopic foci in the atria or AV node. These *supraventricular ectopics* have normal QRS complexes but the P wave may be distorted and the PR interval depends on the distance between the ectopic focus and the AV node (FIG. 5A). When the ectopic focus is in the AV node itself the P waves are inverted in leads II, III and AVF and may precede (FIG. 5B) or follow (FIG. 5D) the ventricular complex. This depends on the delay in retrograde conduction into the atria. Sometimes the P wave cannot be seen (FIG. 5C), since it is incorporated into the ventricular complex. A rapidly recurring series of supraventricular ectopic beats constitute a *supraventricular (atrial or nodal) tachycardia* (FIG. 5E). The rate may vary from 150 to 250 per minute but is perfectly regular in any particular case. P waves which are often small and of abnormal shape, may be difficult to identify. The ventricular complexes have a normal configuration. If the tachycardia continues for more than a few hours secondary myocardial ischaemia many cause ST segment and T wave changes.

Atrial flutter, like paroxysmal atrial tachycardia, is due to a rapid series of impulses arising from an ectopic focus within the atria. Atrial flutter distorts the base line of the E.C.G. by so-called "saw-toothed", regularly recurring flutter waves at a rate of 200 to 350 per minute (FIG. 5F). The ventricles can seldom respond to such a rapid rate but are

activated by every second or third flutter wave. The ventricular rate is usually regular (2:1, 3:1 or 4:1) and the QRS complexes have a normal configuration although the ST segments are distorted by the flutter waves. Sometimes the ventricular response varies with resultant irregularity of the heart rate.

Atrial fibrillation is characterised by completely disordered atrial activity (Fig. 5G). The E.C.G. tracing shows an irregular base line which may vary from coarse irregular waves to almost a flat line. The ventricular response is totally irregular.

Before leaving atrial arrhythmias, mention should be made of *paroxysmal atrial tachycardia* (PAT) *with block* (Fig. 5H). This is uncommon and when present it is usually due to digitalis toxicity. The atrial rate is about 180 per minute (120 to 250) but in contrast to atrial flutter there are iso-electric intervals between peaked P waves.

VENTRICULAR ARRHYTHMIAS

Ventricular ectopics are readily distinguished from those of supraventricular origin by their broad, bizarre shape and inverted T waves (Fig. 5I). This is because their conduction pathway to the rest of the myocardium is through ventricular muscle rather than through normal conducting tissue. Ventricular beats arising from the same focus have the same bizarre shape in any particular lead but when they arise from different foci the complexes vary in shape. Ventricular ectopics may occur singly or in runs of 2 or 3 in rapid succession. When a ventricular ectopic follows each normal beat this is known as coupled rhythm.

A rapidly recurring series of ventricular ectopic beats constitute *ventricular tachycardia* (Fig. 5J). The rate usually varies between 150 and 200 per minute and is slightly irregular. P waves may sometimes be detected occurring at an independent slower rate. Ventricular tachycardia is a dangerous arrhythmia usually due to serious myocardial disease. It may herald the onset of *ventricular fibrillation* (Fig. 5K) with cessation of ventricular contraction.

CAROTID SINUS PRESSURE

The electrocardiographic interpretation of tachycardia is not always easy but can often be clarified by the response to vagal stimul-ation produced by carotid sinus massage. Carotid stimulation should be performed with the patient recumbent while an E.C.G. trace is being recorded. The carotid sinus is at the bifurcation of the common carotid artery. This point lies just below the angle of the jaw and once the vessel has been palpated, gentle massage is applied postero-medially in the line of the vessel with either the thumb or two or three fingers. The vessel lumen should not be obliterated. Each side should be massaged separately, since one side is frequently more sensitive than the other. Sinus tachycardia responds with temporary slowing of the heart rate whereas a supraventricular tachycardia will cease abruptly or remain unaffected. With atrial flutter the ventricular response is temporarily slowed to reveal the characteristic "saw-toothed" flutter waves which previously may have been obscured by the ventricular complexes (Fig. 5F). Very occasionally, atrial flutter may revert to sinus rhythm. PAT with block responds to carotid sinus pressure in the same way as atrial flutter. The ventricular rate in atrial fibrillation is sometimes slowed temporarily but ventricular tachycardia is unresponsive.

CARDIAC HYPERTROPHY

1. *Atrial Hypertrophy*. Atrial hypertrophy may be revealed by the size and shape of the P waves. Tall peaked P waves (over 2.5 mm. in height), seen in leads II, III and AVF and in the right chest leads, suggest *right atrial hypertrophy* (Fig. 6B). Broadened bifid P waves (longer than 0.12 second) usually seen best in leads I, II, aVR and aVL suggest *left atrial hypertrophy*. These P wave changes which are sometimes transient may result from temporary atrial hypertension, but are seldom sufficiently marked to constitute certain evidence of hypertrophy of the atrial walls.

2. *Ventricular Hypertrophy*. The amplitude of the QRS complex is increased by ventricular hypertrophy but it may be affected by many other factors such as body build and the closeness of the heart to the chest wall. In contrast, significant degrees of ventricular hypertrophy may be present before the amplitude of the QRS complex affords certain E.C.G. confirmation of its presence.

Left ventricular hypertrophy. In an adult of normal build, left ventricular hypertrophy is suggested by the following criteria:

(a) a combined amplitude of the S wave in V1 and the R wave in V5 or V6 exceeding 35 mm. (FIG. 6A).

(b) an R wave in aVL exceeding 13 mm.

(c) an R wave in aVF exceeding 21 mm.

None of these figures is absolute and not all of them need be present in any one case. In children and thin adults similar large voltage complexes may be normal variants.

Right ventricular hypertrophy. The E.C.G. changes of right ventricular hypertrophy are less striking since right ventricular hypertrophy seldom exceeds the bulk of the normal left ventricle. Nevertheless, leads over the right ventricle (VI, V3R, V4R) may show a dominant R wave instead of the normal S wave (FIG. 6B). These additional leads should always be recorded when right ventricular hypertrophy is suspected but is not revealed in lead VI.

In more advanced cases of ventricular hypertrophy, repolarisation is abnormal. The ST segment may be depressed and the T wave asymmetrically inverted in leads over the left ventricle in left ventricular hypertrophy (FIG. 6A) and in leads over the right side of the heart in right ventricular hypertrophy (FIG. 6B). Some T wave inversion is a normal variant in leads V4R, V3R and VI so that these changes are only significant of right ventricular hypertrophy when marked or extend to lead V2 or V3. The ST segment and T wave changes are due to relative ischemia or replacement fibrosis and constitute a manifestation of "strain" on the relevant ventricle. Sometimes a "strain" pattern occurs without electrocardiographic evidence of ventricular hypertrophy.

BUNDLE BRANCH BLOCK

Complete bundle branch block produces broad, notched or slurred QRS complexes (their duration measures 0.12 sec. or more) and abnormalities of the ST segments and T waves in all leads. These changes are due to:

1) the excitation wave passing through atypical pathways in the myocardium on the side of the blocked conduction bundle.

2) asynchronous activation of the two ventricles.

3) abnormal repolarisation after delayed ventricular activation.

The shape of the complex depends upon whether the left or right main bundle branch is blocked.
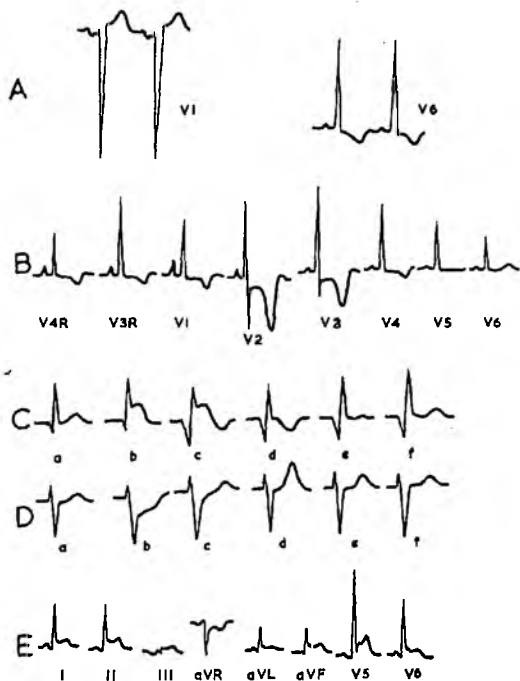

FIG. 6

A. *Left ventricular hypertrophy SV 1 + RV 6 > 35mm.*

B. *Right ventricular hypertrophy. Note that an R wave is the sole deflection in the ventricular complexes over the right side of the heart. The P wave in V 1 is tall and peaked (P pulmonale); T wave inversion extends to V 4.*

C. *Serial electrocardiographic patterns from an electrode overlying a myocardial infarction (compare D below).*
   (a) Normal complex.
   (b) ST elevation (concave downwards).
   (c) Pathological Q wave; ST elevation; T wave inversion.
   (d) ST segment isoelectric.
   (e) T wave low upright.
   (f) Persisting pathological Q wave; T wave normal.

D. *Serial electrocardiographic patterns from an electrode facing the opposite side of the heart to an infarcted area (compare C above).*
   (a) Normal complex.
   (b) Reciprocal ST depression.
   (c) ST depression less marked.
   (d) Peaked T wave.
   (e)(f) Normal complex.

E. *Acute pericarditis ST elevation (concave upwards) in all leads except a VR.*
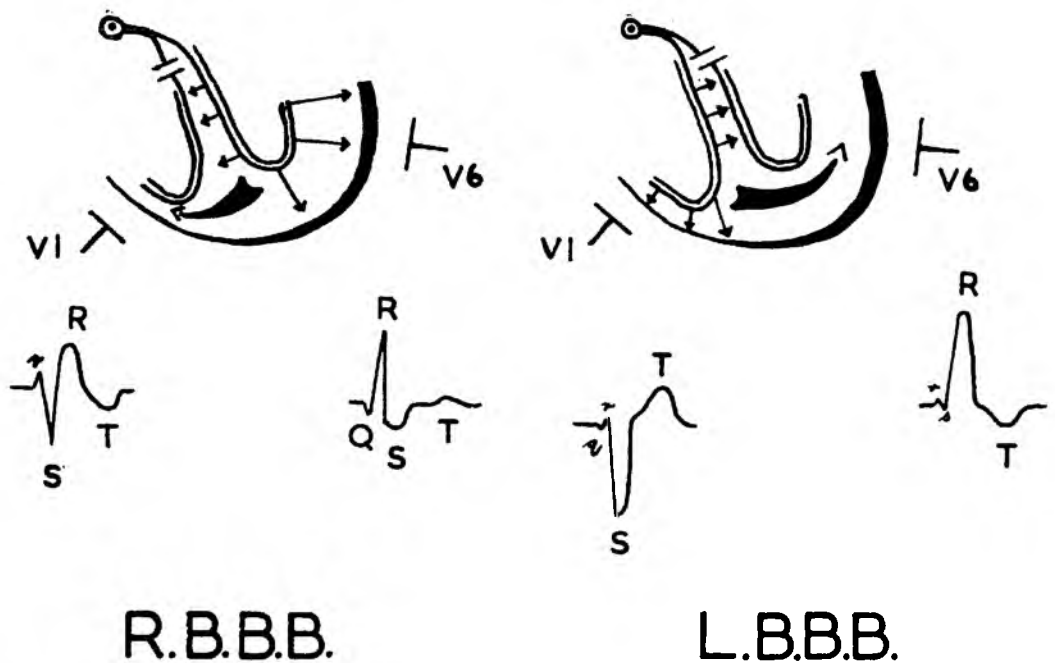
R.B.B.B.   L.B.B.B.

FIG. 7

*Genesis of the ECG complex in right bundle branch
block and left bundle branch block as recorded from
2 electrodes one over the right side of the heart
(V 1) the other over the left (V 6). (see text)*

In *right bundle branch block* delayed activation of the right ventricle causes a tall secondary R wave in the right ventricular leads and a slurred terminal S wave in the left ventricular leads (FIG. 7).

In *left bundle branch block* the initial small Q wave of septal activation is absent in the left ventricular leads and late activation of the left ventricle delays the peak of the R wave which is notched or slurred on the upstroke by earlier right ventricular activation. This also causes notching or slurring of the QS complex in the right ventricular leads. These changes are best seen in the chest leads (FIG. 7)

MYOCARDIAL INFARCTION

The E.C.G. usually develops characteristic changes after a recent myocardial infarction.

In leads overlying an infarcted area these changes, in order of their appearance, are:—
1) elevation of the ST segment, 2) the appearance of a pathological Q wave, 3) symmetrical inversion of the T wave.

The recognition and interpretation of these changes becomes clearer if the mechanism of their origin is understood.

CHANGES IN THE ST SEGMENTS

As noted previously a resting normal muscle has a positive surface charge but when stimulated or injured this surface charge becomes negative (the negative current of injury). An anoxic area on the epicardial surface of the heart will therefore have a constant negative charge while surrounding healthy muscle will carry a normal positive charge. This difference of potential between injured and normal
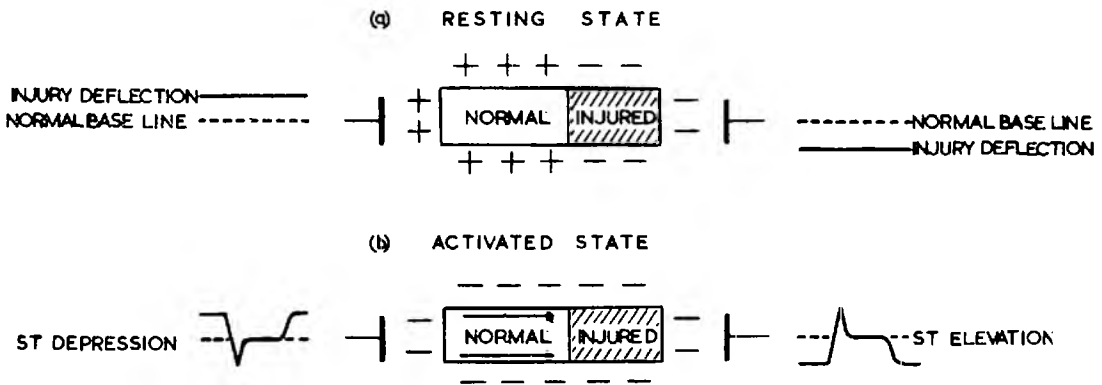
FIG. 8

Diagram of cardiac muscle :

*(a) to demonstrate the depressed base line re-
corded from an electrode overlying injured
myocardium in the resting state.*

*(b) to show ST elevation in a lead overlying in-
jured myocardium in the activated state.
Arrows indicate the direction of the stimulus.
Reciprocal ST depression is present in a lead
facing uninjured myocardium. (see text)*

myocardium is reflected in the E.C.G. as a
depression of the normal base line in leads
overlying the injured area and as an elevation
in leads over normal myocardium (FIG. 8A).
When the healthy muscle is activated it also
becomes negative and current will cease flow-
ing. The depressed base line therefore re-
turns to the normal iso-electric level giving the
impression of an elevated ST segment (FIG.
8B). When healthy muscle is restored to its
resting state the negative current of injury
reappears and the base line again becomes
depressed.

PATHOLOGICAL Q WAVES

Pathological Q waves of myocardial infarc-
tion exceed 0.04 second in duration and may
be deep. They only appear when the in-
farct involves the whole thickness of the
ventricular wall (transmural infarction) and do
so because the infarcted myocardium, being
dead, is "electrically inactive". Leads over-
lying an infarcted area of left ventricle there-
fore record electrical forces of exactly the same
pattern as those within the cavity of the ven-
tricle, i.e. the deep Q wave of aVR in the
normal E.C.G. (vide supra).

SYMMETRICAL T WAVE INVERSION

This does not appear until recovery of the
infarcted muscle has begun.

E.C.G. changes of myocardial infarction
follow a sequence, the time intervals of which
vary within wide limits. ST elevation usually
disappears within the first few days while the
pathological Q waves may decrease in size but
usually persist for years and often remain as a
permanent abnormality. Inverted T waves
which develop as the ST elevation subsides
usually become upright during the first few
months but also may persist for years. This
sequence of events is illustrated in FIG. 6C.
Leads facing the opposite side of the heart may
show reciprocal ST depression during the
acute stage and tall peaked T waves as re-
covery takes place (FIG. 6D).

The position of the infarcted area can be
localised by the leads in which the infarct
pattern develops.

Anterior myocardial infarction is shown in
the chest leads and normally in leads I and
aVL.

Inferior myocardial infarction is shown in
leads II, III and aVF.

When an infarct does not involve the
whole thickness of the ventricular wall (intra-

mural infarction) the changes affect only the ST segments and T waves. Q waves do not develop.

Although the characteristic E.C.G. changes of myocardial infarction develop within the first few hours of its occurrence this is not invariable and diagnosis then depends on the clinical picture and elevation of certain serum enzyme levels. It must be recognised that in some patients E.C.G. confirmation of a myocardial infarction may never occur while in others it may only develop after an interval of days. Additional leads in higher interspaces or round the back of the chest may be required to detect and localise an infarct.

ANGINA PECTORIS AND MYOCARDIAL ISCHAEMIA

Angina pectoris is usually diagnosed from the patient's history of exertional chest pain. The E.C.G. is usually normal but may show evidence of myocardial ischaemia, especially if recorded while the patient has pain. When the history is equivocal and the resting record negative an exercise test may establish the diagnosis (vide infra). The E.C.G. feature of acute myocardial ischaemia is plateau type ST depression (1 mm. or more) in leads overlying the ischaemic area. In ischaemia, in contrast to infarction, muscle injury is intermittent and confined to the sub-endocardial region of the ventricles. As a consequence, E.C.G. leads facing the surface of the heart record the changes of uninjured muscle and show ST depression during phases of ischaemia. Anterior ischaemia is best shown in the chest leads, inferior ischaemia in leads II, III and aVF. Biphasic or symmetrically inverted T waves may develop in the same leads during recovery from an ischaemic episode and provide confirmatory evidence of the diagnosis.

EXERCISE TEST

A resting record is taken and then the patient is exercised by climbing repeatedly over steps, by climbing stairs or by using a bicycle ergometer or tread mill. The amount of exercise is determined for each patient, consideration being taken of age, sex, weight and general clinical state. An exercise test should not be carried out in elderly people or those in poor physical condition. It is value-less when patients are having digitalis or other similar drugs which affect the E.C.G. and is unnecessary when ST segment and T wave changes are already present. Exercise should be stopped if anginal symptoms or other features of distress develop. The E.C.G. is repeated during or immediately after the exercise, after five minutes rest and subsequently at five minute intervals if the record is not returning to normal. Development of ST depression of over 1 mm., especially if followed by inversion or diphasia of the T wave, constitutes a positive test. The changes are usually transitory but occasionally persist for over an hour.

PERICARDITIS

Acute pericarditis, like acute myocardial infarction, produces ST elevation but in contrast to myocardial infarction, the elevation is concave upwards, the changes are more widespread and occur in all the limb leads reflecting epicardial potentials as well as in the chest leads (Fig. 6E). Pathological Q waves are absent unless there has been associated myocardial infarction. During recovery the ST elevation disappears and is replaced by T wave inversion before returning to normal.

ELECTROLYTE ABNORMALITIES

Disturbances of the electrolyte balance can produce most bizarre E.C.G. changes, particularly affecting the ST segment and T waves. The most frequently encountered electrolyte disturbance concerns potassium. *Hyperkalaemia* (Fig. 9A) produces tall peaked T waves and reduces the height of the R wave. The QRS complex is broadened and P waves may disappear. The extent of these changes depends on the severity of the electrolyte upset. *Hypokalaemia* (Fig. 9B) is associated with prominent U waves, flattening of the T wave, ST depression and prolongation of the PR interval.

DIGITALIS

Digitalis often affects the E.C.G.; the changes may mask or simulate changes of underlying heart disease and before interpreting the record it is essential to know whether a patient has taken this drug. The most
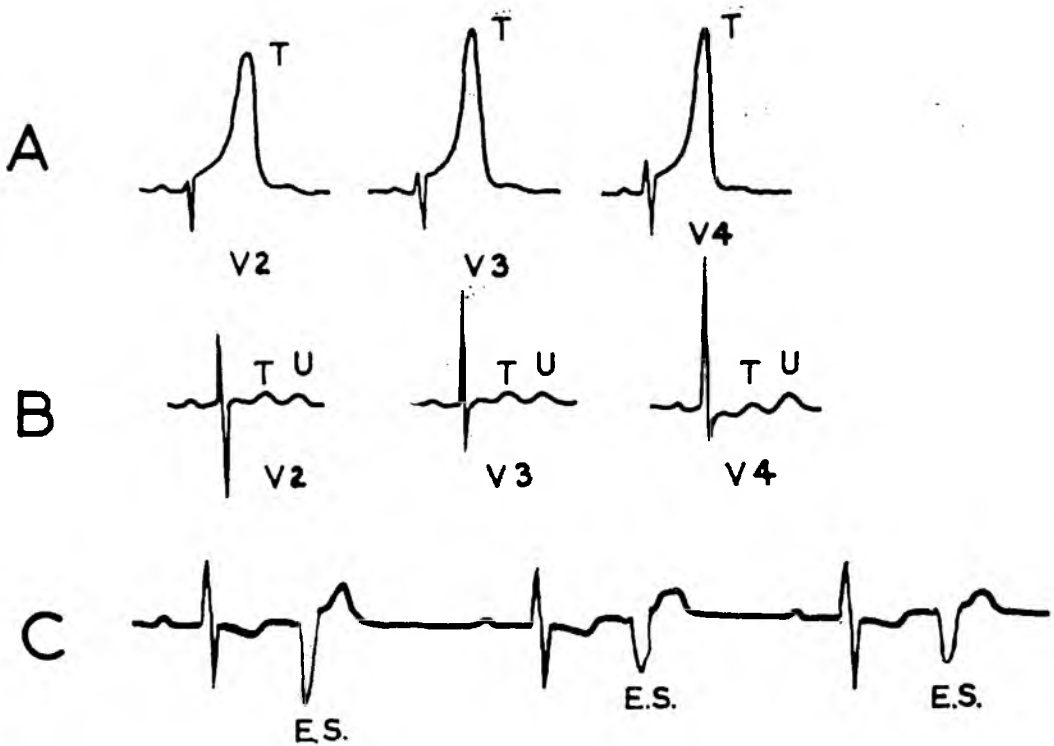
FIG. 9

A. *Hyperkalaemia. Tall peaked T waves; diminished amplitude of R waves.*

B. *Hypokalaemia. Prominent U waves; flattening of T waves; slight ST depression (V 4).*

C. *Digitalis toxicity. Coupled ventricular ectopics of multifocal origin; prolongation of PR interval; sagging ST segment.*

common change is sagging of the ST segment which may mimic myocardial ischaemia. Bradycardia, prolongation of the PR interval and occasional ventricular ectopics are also common. More serious toxic effects of digitalis are infrequent in the absence of overdosage or hypokalaemia secondary to inadequately controlled diuretic therapy. In these circumstances ventricular ectopics become more frequent, they may be coupled (FIG. 9C), multifocal in origin or progress to ventricular tachycardia. Complete heart block or paroxysmal atrial tachycardia with block may develop.

SUMMARY

In this paper the more common E.C.G. abnormalities encountered in clinical practice have been outlined and explained in an attempt to clarify their interpretation.