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AN APPRAISAL OF THE ELECTROCARDIOGRAPHIC EXERCISE TEST

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A good history is usually sufficient to establish the diagnosis of angina pectoris or coronary insufficiency. At times, however, the pain is atypical and the history doubtful; objective confirmation of the diagnosis then becomes desirable. In such cases, the electrocardiographic changes seen in response to exercise may provide this confirmatory evidence.

HISTORY OF THE EXERCISE TEST

In 1908, Einthoven[1] recorded the first electrocardiogram (ECG) after exercise; he noted the change in the ECG of his old laboratory porter after he had climbed a few stairs. The following year Nicolai and Simons[2] reported the changes after exercise in a patient who had angina pectoris. Changes occurring spontaneously during attacks of angina pectoris were recorded by Bousfield in 1918,[3] and Cowan and Ritchie in 1922.[4] In those early days of electrocardiography the changes after exercise were sometimes recorded fortuitously, since several flights of stairs had to be climbed to reach the electrocardiographic laboratory.

In 1931, Wood, Wolferth and Livezey[5] first specifically used the test to provoke attacks of angina pectoris; they investigated the changes both in normal subjects and in patients with angina pectoris. Although no untoward occurrences took place, they considered it a dangerous procedure 'to induce anginal attacks indiscriminately'. It was in 1932 that Goldhammer and Scherf[6] first recommended the use of moderate exercise followed by an ECG as an aid in the diagnosis of cardiac pain. Further observations were published by these authors in the following year,[7] and subsequently by many others.

In 1942, Master and his associates[8] standardized the performance of the test, which is now known as the Master 2-step exercise test.

THE PERFORMANCE OF THE EXERCISE TEST

There are 2 methods of performing the exercise test — a standardized and a non-standardized method.

The Standardized Method

Master and his associates[8] standardized the test according to sex, weight and age. Using these parameters, tables were constructed, based on the return of blood pressure and pulse rate to normal within 2 minutes. The exercise is performed on a special standardized 2-step apparatus and the patient is required to do a certain number of ascents and descents in 1½ minutes.

These principles may well be questioned, for it has not been shown that coronary-artery disease, once present, runs a course which is more severe in the female; nor has it been shown that ECG changes following exercise parallel those of pulse rate and blood pressure. Furthermore, although coronary-artery disease is usually more prevalent and more marked in the older age groups, in the individual as such it may be very severe in a woman of 40 years and not at all evident in a man of 80 years. According to Master's criteria, a man aged 50 years and weighing 160 pounds should do 20 ascents on the standardized apparatus in 1½ minutes, whereas a woman of the same age and weight is required to do only 16 ascents, despite the possibility that the coronary-artery disease may be so advanced in the man that only a few steps may bring on angina, whereas the particular woman may be capable of undergoing much greater exercise and thus require far more than 16 ascents before either ECG changes or angina become manifest. In addition, other factors, such as emotion and training, may influence the outcome of the test and will affect any attempt to standardize it.

More recently, Simonson and Keys[9] recommended a double Master 2-step exercise test — twice the number of ascents recommended, over twice the period. The diagnostic criteria remain the same. This further underlines the difficulties and questionable value of a standardized procedure.

Nevertheless, although the validity of the exercise test as described by Master may be questioned, it should be stated that the Master 2-step test is recognized in many centres and is commonly used as a routine procedure in many ECG laboratories.

The Non-standardized Method

Scherf recommended originally,[7] and still recommends today,[10] that the amount of exercise the patient is required to perform be adapted to the needs of the particular individual. The patient is subjected to approximately the exertion that has been known to bring on an attack of angina pectoris. This does not mean that the patient is exercised indiscriminately until such time as he develops pain. If, for example, the patient has pain after only the slightest exertion, he may be asked to do a few knee-bends or sit up and down a few times; whereas a patient who has pain only after severe exertion may be asked to climb several flights of stairs rapidly. The form of exertion is unimportant, since the object of the test is to increase the demand for coronary blood flow where an inadequate flow is suspected; the induced coronary insufficiency and resultant inner-layer myocardial ischaemia or injury may precipitate ECG changes that are diagnostic.

If no changes are noted, and if the patient's condition warrants it, the exercise test may be repeated after a suitable interval (usually 1 hour) with a cautious increase in the amount of exercise.

The following procedure is thus recommended in the performance of the exercise test:

1. The patient must not be in pain. The history and physical examination must not suggest an impending myo-

cardial infarction or acute pulmonary embolism. The patient must not be in congestive cardiac failure.

2. An ECG is recorded at rest and must be normal or at most equivocal in respect of coronary-artery disease. There must be *no* tachycardia.

3. The test is preferably performed before a meal, since physiological variants are more likely to occur after a meal. If the patient relates a history of angina pectoris after meals, the test should then be performed before a meal and, if negative, repeated after a meal.

4. The exercise test is performed according to the *non*-standardized method.

5. If pain, substernal discomfort, a feeling of faintness, or pallor develop during the performance of the test, the exercise is stopped immediately. Exercise to the point of pain is hazardous and unjustifiable. The attendance of a physician is mandatory.

6. The ECG is recorded immediately after the exercise and at 2-minute intervals for 6 minutes, or until such time as it returns to the resting configuration.

7. The ECG changes should be observed in at least 1 precordial and 1 extremity or bipolar lead. Changes in the precordial leads are usually best seen in those with the tallest R waves — commonly V_4 or V_5. Changes that occur in standard lead I are usually the most significant of those seen in the standard leads.

THE INTERPRETATION OF THE EXERCISE TEST

There are certain ECG changes following exercise which are always pathological, and others which can only be regarded as normal physiological variants. Nevertheless, the transition between what is normal and abnormal is extremely difficult, if not impossible, to define, and there is a considerable degree of overlap between the two. Since it is never possible to rule out false negative tests, i.e. a normal ECG does not necessarily exclude coronary-artery disease, it is best to establish stringent criteria in order to avoid labelling borderline physiological variants as abnormal.

Changes following exercise may affect all components of the record — the P wave, the PR segment, the QRS complex, the ST segment, the T wave and the U wave; in addition, abnormal rhythms may occur (Table I). The changes that occur after exercise are the same as those that may be seen during spontaneous pain.

Changes Affecting the P Wave

Following exercise there is a tendency for right axis deviation of the P wave, so that the P waves in standard leads II and III tend to become taller. These are normal physiological variants.

Changes Affecting the PR Segment

Atrial depolarization is normally followed by atrial repolarization, i.e. as a T wave follows the QRS complex, so a corresponding 'T' wave normally follows the P wave. This atrial T wave is known as the Ta or Tp wave and is normally opposite in direction to the P

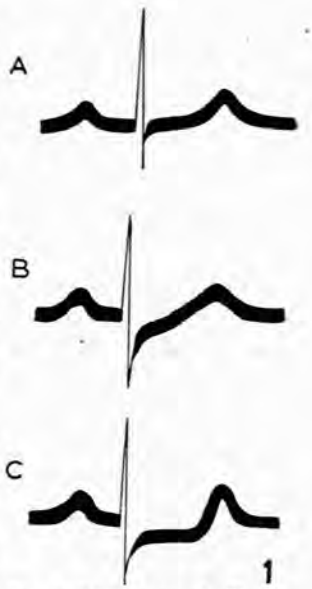

Fig. 1. Diagrammatic illustration showing: (*A*) normal QRST complex, (*B*) junctional ST-segment depression, and (*C*) plane ST-segment depression.

TABLE I. INTERPRETATION OF THE ELECTROCARDIOGRAPHIC EXERCISE TEST

Component of ECG	*Abnormal*	*Usually abnormal*	*Physiological or diagnostically uncertain*
P wave			Right axis deviation (taller in standard leads II and III)
PR segment			Downward slope
QRS complex	Left bundle-branch block	Right bundle-branch block	Right axis deviation
ST segment	Depression of 2 mm. or more in the precordial leads	Depression of 0·75 - 2 mm. in the precordial leads	Junctional depression
		Any degree of plane or sagging depression	
	Depression of 1·5 mm. or more in the extremity leads	'Horizontality'	
		Sharp-angled ST-T junction	
T wave		Inversion in standard lead I	Inversion in other leads
		T in standard lead I lower than T in standard lead III	
		Increase in height by 5 mm. or more in lead V_4	
		Symmetrical T waves—upright or inverted	
U wave	Inversion		
Ventricular extrasystoles	Multifocal	Unifocal:	Isolated unifocal
		In 'showers'	
		In bigeminal rhythm	
		In a patient over 40 years	
	Post-extrasystolic T-wave change		
	Post-extrasystolic U-wave change		

wave. It is usually masked by the ensuing QRST deflection and is therefore best seen following *isolated* P waves such as are found during periods of complete heart block (Fig. 3).

Following exercise, the Tp deflection normally becomes more pronounced and may cause the PR segment to have a downward slope (Fig. 4, ½-minute and 2-minute tracings). It may cause junctional depression of the ST segment, especially with short PR intervals, thus producing a false positive ST depression.

Changes Affecting the QRS Complex

There is a normal tendency for right axis deviation of the QRS complex following exercise.

The development of abnormal widening of the QRS complex is regarded as a positive test. The appearance of left bundle-branch block after exercise is always pathological. Frank right bundle-branch block is usually abnormal, but may occasionally be found in normal individuals.

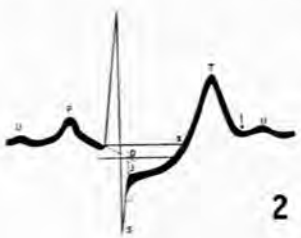

Fig. 2. Diagram illustrating methods of measuring (1) true and false depression of the ST segment, and (2) the degree of horizontality of the ST segment. (1) The line of the sloping PR segment is continued until it meets, at point O, a vertical line drawn from the junction of the QRS and ST deflections; the distance O—J indicates the true amount of ST-segment depression. (2) A horizontal line drawn from the beginning of the QRS deflection is continued till it meets the T wave at point X. This distance, Q—X, is expressed as a percentage of the QT interval (measured from the beginning of the QRS complex to the end of the T wave—arrow); Q—X is greater than 50% of QT in the majority of true-positive tests.

Changes Affecting the ST Segment

ST-segment depression is frequently seen following exercise and it is the interpretation of this sign that has caused the most difficulty and occasioned the greatest controversy.

ST-segment depression may be interpreted both quantitatively and qualitatively, i.e. both the amount and type of depression should be considered. There are 2 principal types of ST depression:

(*a*) The whole or nearly all of the ST segment is depressed, resulting in horizontality, or plane depression, of the ST segment (*C* in Fig. 1; ½- and 2-minute tracings in Fig. 4; Fig. 5). Normally, the ST segment merges smoothly and gradually with the ascending limb of the T wave, so that a definite separation between the two is difficult or impossible to define (*A* in Fig. 1; Fig. 5); the horizontality or plane depression will result in a sharp-angled ST-T junction. This form of ST-segment depression or configuration should always be regarded with suspicion and is nearly always abnormal (Figs. 1, 4 and 5). Indeed, even if there is no depression, but the ST segment has the appearance of horizontality with a sharp-angled ST-T junction, the change is usually abnormal. Measurement of the degree or amount of horizontality has been attempted by Lepeschkin and Surawicz[12] (for details, see Fig. 2).

Fig. 3. Isolated P waves from a case of complete A—V block. The Tp wave can be seen as a negative deflection after each P wave. A QRS complex has been artificially placed over the second Tp deflection and illustrates how an ensuing ST segment could be depressed to the extent of 0·5 mm. by the Tp deflection.

Fig. 4. ECG recording during an exercise test; standard lead I. Note: normal upright U wave in the control tracing and an inverted U wave in the 2-minute tracing; superimposition of the P wave on the U wave during the tachycardia of the ½-minute tracing; 1·25 mm. *plane* depression of the ST segment with *sharp-angled ST-T junction* in the ½-minute and 2-minute tracings, contrasting with the smooth ST-T transition in the control tracing; the downward slope of the PR segment in the ½-minute and 2-minute tracings. There is also a prolonged PR interval in the control and 4-minute tracings. *Comment:* Correction of the ST-segment depression to allow for the false depression of the Tp deflection (*cf.* Fig. 2) reveals a true depression of 0·75 mm. This amount of depression is usually, though not invariably, diagnostic. However, the plane configuration and the presence of an inverted U wave makes this test positive and diagnostic.

(*b*) The other form of ST-segment depression affects the proximal part or junction of the QRS deflection with the ST segment (*B* in Fig. 1). This junctional form of depression should be interpreted with caution, since it may sometimes be found in the normal individual. This is from the effect of the Tp deflection (see below), which becomes more marked after exercise (*cf.* Fig. 3).

ST-segment deviation is best seen in the precordial leads with the tallest R waves — usually V_4 and V_5.

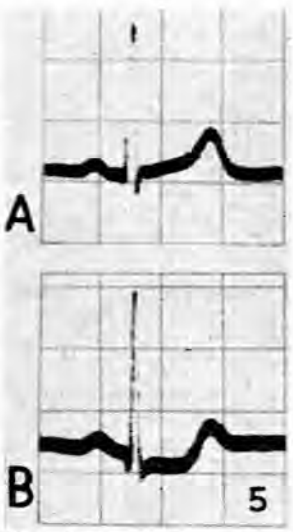

Fig. 5. A. normal QRST complex. Note the smooth transition from ST segment to T wave. *B.* QRST complex of coronary-artery disease. Note plane depression of the ST segment and the sharp-angled ST-T junction.

The amount of depression that is considered definitely abnormal is the most disputed point in the interpretation of the exercise test. Figures in excess of 0·5,[8] 0·75,[12,13] 1·0,[14-16] 1·5,[17-20] and 2[21-28] mm. depression in the precordial leads V_4 and V_5 have all been considered as definitely abnormal. The matter is further complicated by the fact that it is at times difficult to judge the position of the baseline as a reference point from which to measure the depression, and the depressing effect of the Tp deflection must also be taken into account. The best baseline or

isoelectric level to use is the UP segment (Fig. 2), but this is often obscured during the tachycardia which so frequently follows exertion (½-minute recording in Fig. 4). In such cases the baseline is measured from the junction of the PR segment with the QRS complex. Owing to the depressing effect of the Tp deflection, the PR segment may have a downward slope and the amount of ST-segment depression may be more than that caused by coronary insufficiency alone.

To avoid the inclusion of this false depression of the ST segment, Lepeschkin and Surawicz[22] have devised a procedure in which the downward slope of the PR segment is continued to meet a vertical line extended from the junction (J) of the QRS complex with the ST segment (Fig. 2). This level is taken as the true baseline and these authors, in a well-controlled investigation, have found that, by using a criterion of 0·75 mm. depression below this baseline, only 16% false positives occurred as compared with 26% using the same procedure with Master's criterion of 0·5 mm. depression. Nevertheless, 16% is still a high percentage of false positives. Other reports[23,24] have also shown that ST-segment depression of 1 mm. or more may be found in normal individuals.

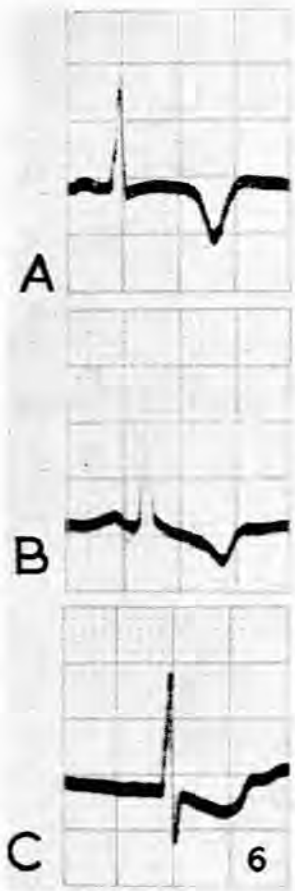

Fig. 6. A. T wave in coronary-artery disease. Note the symmetry and pointed vertex. *B.* T wave in hypertensive heart disease. Note asymmetry and absence of pointed vertex. *C.* T wave with digitalis effect. Note asymmetry and straight downward slope of the ST segment.

The position was best evaluated by Scherf[23] who stated that, since it is impossible to eliminate false negative tests, i.e. since a negative result or normal ECG does not exclude cardiac disease, it becomes imperative to avoid false positive tests, consequent incorrect diagnoses, and possible iatrogenic disease. Thus Scherf deliberately set extremely stringent criteria to avoid making incorrect diagnoses, so that a positive test should be based on incontestable standards. The test is therefore only considered diagnostically positive when the ST-segment depression is 2 mm. or more in the precordial leads and 1·5 mm. or more in the extremity leads. This is well seen in Fig. 7, which illustrates the changes occurring during a spontaneous attack of angina pectoris. Nevertheless, any depression of between 0·75 mm. and 2 mm. in the precordial leads should be regarded with suspicion, since it is usually, though not inevitably, abnormal.

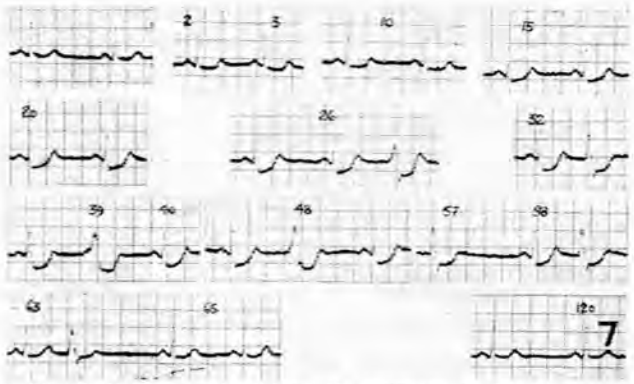

Fig. 7. ECG (standard lead II) recorded during a spontaneous attack of angina pectoris. Figures indicate the number of seconds after the onset of pain. Note the ST depression of 3 mm. during the period of 26 - 48 seconds after the onset of pain; also the frequent ventricular premature beats. This amount of ST-segment depression is always diagnostic of coronary insufficiency. It is noteworthy that the Master 2-step exercise test in this individual was consistently negative and that the changes recorded above occurred only during spontaneous attacks of pain. (Courtesy Dr. H. J. L. Marriott and The Williams and Wilkins Co., Baltimore.)

Changes Affecting the T wave

The T waves in the precordial and augmented limb leads frequently become diphasic, isoelectric or inverted, following exercise. The T wave, however, is the most unstable component of the ECG recording; changes of this deflection may occur with hyperventilation, heavy meals, anxiety, smoking, drinking iced water, changes in bodily position, and decrease in blood pressure. Variations also occur with age and race. They are found so frequently as normal variants that if they occur as isolated phenomena their diagnostic import is uncertain.

Despite this, there are certain T-wave changes that are frequently suggestive of coronary-artery disease and, although not in themselves definitely diagnostic, are pointers towards the presence of coronary insufficiency. The T wave of coronary insufficiency has symmetrical limbs and a sharp pointed vertex (Fig 6 *A*); the ST segment usually shows an upward convexity. The T-wave configuration from other causes usually shows asymmetrical limbs without a peaked vertex (Fig. 6 *B* and *C*).

In the presence of a dominantly positive QRS deflection in lead I, frank inversion of the T wave in that lead is usually abnormal.[12] Furthermore, in the presence of a dominantly positive QRS deflection in lead I, a T wave in lead I that is lower than a T wave in lead III is likewise frequently abnormal.

Occasionally the T wave becomes taller, pointed, and symmetrical following exercise. If the increase in height in lead V_4 is 5 mm. or more than the resting value, it should be regarded with suspicion and is usually abnormal.[12]

Changes Affecting the U Wave

The U wave is a small deflection occurring just after the T wave (Figs. 2 and 4). It is best seen in the precordial leads reflecting the transition zone — usually V_2 - V_4. It

is normally in the same direction as the T wave. The deflection may be so small as to make accurate recognition extremely difficult, and with tachycardia the U wave may be superimposed on the following P wave — this makes recognition impossible (½-minute tracing in Fig. 4).

An inverted U wave, i.e. a U wave which is opposite in direction to the T wave, is diagnostic of cardiac disease, especially of coronary-artery or hypertensive origin. When it develops after exercise it always constitutes a positive response and indicates cardiac ischaemia[12] (Fig. 4). Occasionally, it is the only abnormal finding.[25]

Changes in Rhythm Following Exercise

Sinus tachycardia normally follows exercise. It may occasionally precipitate left or right bundle-branch block (see above).

The presence of *multiform ventricular premature beats* is diagnostic of cardiac disease. When they develop in response to exercise they constitute a positive test.

Unifocal ventricular premature beats may occasionally be found in the normal subject. Nevertheless, their presence after exercise usually means abnormality, especially if they occur in 'showers', if they give rise to short runs of bigeminal rhythm, if they occur in a person over 40 years of age, or if they persist for several minutes or longer.

ADDITIONAL FACTORS TO BE CONSIDERED

The Duration of ECG Abnormalities after Exercise

ECG changes, particularly ST-segment depression, from coronary insufficiency tend to last longer than those caused by physiological variants. Although there are exceptions, normal variants or false-positive changes usually last less than 2 minutes, whereas pathological or true-positive changes commonly last 5 minutes or longer.[12]

A depression of, say, 0·75 mm. in the precordial leads, which is usually, though not definitely, diagnostic of cardiac ischaemia, is considerably strengthened as a criterion of positivity when the change lasts for 5 minutes or longer.

The Effect of Digitalis on the Exercise Test

The exercise test cannot be interpreted with confidence in the presence of digitalis effect. Digitalis itself may markedly influence the ST segment (Fig. 6 *C*) and positive tests have been reported in patients taking digitalis, in whom there was no evidence of coronary-artery disease.[26,27]

Hypertension and the Exercise Test

The exercise test should be interpreted with caution in the presence of hypertension, since this may at times mimic the effects of coronary insufficiency. Following exercise, a pattern of left ventricular hypertrophy — QRS changes only — may change to one of 'strain' — ST-segment depression with T-wave inversion; U-wave inversion may also occur. Such T-wave change, however, shows asymmetrical limbs without a pointed vertex (see above and Fig. 6 *B*).

Relationship of ECG Changes to the Development of Pain

While patients who develop pain on exercise usually develop abnormal ECG changes, this correlation is not invariable. The appearance of abnormal ECG changes does not necessarily correlate with that of pain. Although the patient should not be exercised to the point of pain by intent, when pain is precipitated as a result of the exercise test it may occur long after the appearance of abnormal changes and may disappear long before such changes have regressed. Abnormal changes, which cannot be reproduced by exercise, may be found during spontaneous pain (Fig. 7).

Dangers of the Exercise Test

Although the element of danger in the exercise test can never be entirely eliminated, the incidence of reported fatalities are infinitesimal and the cause-and-effect relationship often questionable. Instances of myocardial infarction precipitated by the exercise test have been

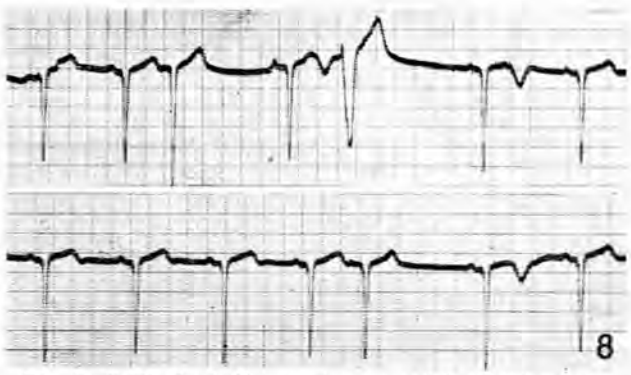

Fig. 8. ECG (lead V_1, continuous strip) showing post-extrasystolic T-wave inversion. The 3rd cycle in the upper strip and the 5th cycle in the lower strip are atrial extrasystoles; the 5th ventricular complex in the upper strip is a ventricular extrasystole. Note the marked inversion of the T wave in the normal sinus complex *following* each extrasystole.

reported where the basic requirements for the performance of the test were not fulfilled. These cases are often quoted without critical evaluation. In particular, the case reported by Grossman and Grossman[28] is worthy of comment. The patient gave a typical history of myocardial infarction 4 months previously; the so-called borderline tracing showed (*a*) a tachycardia of 100 per minute, (*b*) a sagging ST-segment depression with sharp-angled ST-T junction in standard lead II, (*c*) obvious U-wave inversion in leads I, V_2, V_4 and V_5. The diagnosis of coronary-artery disease was thus well established both clinically and on ECG, and the performance of the test was unnecessary. The case reported by Friedberg[29] likewise shows U-wave inversion in the 'normal' control tracing.

The test is reasonably safe, provided the basic precautions, outlined above, are taken.

The "Poor Man's Exercise Test"

Levine has facetiously styled the chance finding of an extrasystole with post-extrasystolic T-wave changes in the control tracing as a "poor man's exercise test", since it gives immediate evidence of abnormality and obviates the necessity for a further, more expensive, exercise test.

The abnormality consists of a T-wave change in the *first sinus beat following* an atrial or ventricular extrasystole (Fig. 8). The T wave usually becomes inverted,

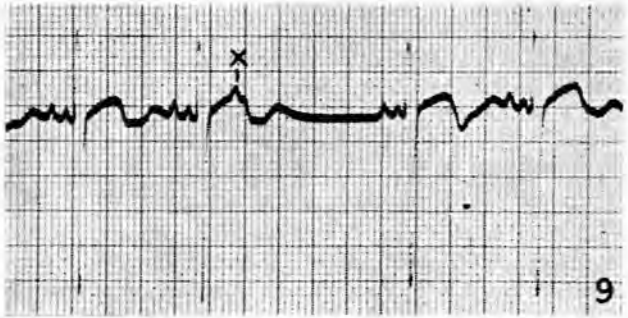

Fig. 9. ECG (lead V_1) showing a blocked atrial extrasystole — the ST segment of the second QRST complex is deformed by the superimposition of the ectopic atrial 'P' wave (X) occurring so early that the A—V nodal tissues are still refractory and therefore unable to conduct the impulse to the ventricles. Note the inversion of the T wave in the sinus beat following the blocked extrasystole. This demonstrates that it is not the extrasystole *per se* which causes the post-extrasystolic T-wave inversion, but rather the pause which it occasions. There is also intra-atrial block — P-wave duration = 0·14 seconds.

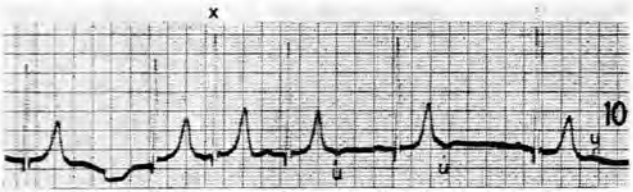

Fig. 10. ECG (standard lead II) showing post-extrasystolic U-wave inversion. X represents an atrial extrasystole. Note the U-wave inversion in the 2 sinus beats following the extrasystole.

but may become taller than normal; any change is significant. Similar changes have been observed following *blocked* atrial extrasystoles[30] (Fig. 9). Thus, it is not the extrasystole *per se* that causes this change, but rather it is the pause it occasions that evokes the change.

Occasionally, U-wave inversion may also be observed following extrasystoles (Fig. 10).

The above signs are nearly always diagnostic of cardiac disease.

I wish to express my thanks to Dr. H. J. L. Marriott and the book publishers, The Williams and Wilkins Company, Baltimore, Md., USA, for permission to use Fig. 135 from Marriott's 'Practical electrocardiography' reproduced here as Fig. 7.

I should also like to express my thanks to the Photographic Unit of the Department of Medicine, University of the Witwatersrand, for the photographic reproductions.

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