THE LESSER ELECTROCARDIOGRAPHIC SIGNS OF CARDIAC PAIN

BY

WILLIAM EVANS AND COLIN McRAE*

From the Cardiac Department of the London Hospital

Received March 20, 1952

When the cardiographic diagnosis of cardiac infarction was first described a characteristic pattern was assigned to the pathological tracing; it consisted of deviation of the S-T segment, inversion of the T wave, and often the presence of significant Q waves (Herrick, 1919; Pardee, 1920; Parkinson and Bedford, 1928). The next few years saw a search for leads that would portray the lesser changes of myocardial injury more clearly than the orthodox limb leads, and so chest lead electrocardiography came into use (Wolferth and Wood, 1932).

Among patients with a characteristic history of cardiac pain there continue to be many in whom the cardiogram appears to be normal on casual examination. Closer scrutiny of such tracings frequently shows changes that, although small, do not tally strictly with the slight variants found in a tracing traditionally accepted as physiological.

We determined to examine such changes more closely and especially to watch the progress of patients exhibiting them, in order to apportion to each its true significance, and to test their value in the diagnosis of cardiac pain.

Some eight changes meriting such assessment came to our notice and they include the presence of a wave in the P-Q period, notching of the QRS complex, addition of an R1 wave, depression of the S-T segment, a low T wave, a blunt T wave, terminal dipping of the T wave, and an inverted U wave.

In the investigation of each cardiographic sign the same procedure was followed. The electrocardiograms were recorded deliberately with three machines (Cambridge, Elmqvist, and Sanborn) lest a slight change might be an instrumental artefact. Leads I, II, III, CR1, CR4, and CR7 were recorded in all cases while III R, intermediate bipolar chest leads, and V leads were taken in the majority. The tracings from 156 healthy subjects were first examined to determine whether a particular sign might be a normal variant. If it was absent in the healthy group it was then sought in the tracings from patients in whom there was a characteristic history of cardiac pain but an electrocardiogram that had previously been regarded as normal. Whenever one of the lesser cardiographic signs was present in these patients, other evidence was sought to give proof that their symptom was true cardiac pain. Such evidence was considered to be present if a one-time physiological cardiogram had been recorded fortuitously when chest pain was not a symptom, if a previous or subsequent tracing had shown undisputed changes of cardiac infarction, if cardiac anoxæmia following physical exercise had produced cardiographic changes accepted as typifying cardiac ischæmia (Masters *et al.*, 1942; Wood *et al.*, 1950), or if death had occurred unexpectedly from cardiac infarction and before an opportunity had presented to test the significance of the cardiographic sign. These minor electrocardiographic changes will now be discussed in turn.

A WAVE IN THE P-Q PERIOD

When a small upright deflection is found in the final phase of the P-Q period it appears to be part of the QRS complex, but it can hardly be designated an R wave because the downward deflec-

* Holding a grant from the Research Fund of the Royal Australasian College of Physicians.

FIG. 1.—A wave in the P-Q period. A short pointed wave appears before the primary R wave in leads II and III. (A) is from a patient with aortic incompetence and (B) from a healthy subject.

tion that may immediately follow it is undoubtedly a Q and not an S wave. In most of the cases in which we have identified the wave the P-R period has not been long enough to allow an auricular T wave to appear, nor has it the same form, being much more pointed than the Ta wave. Again, when this wave appeared earlier in the P-R period it has never seemed an integral part of the P wave itself, while its appearance in auricular fibrillation confirms that it is independent of the P wave. Although we found this wave, seldom measuring more than 1 mm. in height, in patients with thyrogenic, hypertensive, and coronary artery disease, and in those with bundle branch block, we sometimes found it in our series of healthy subjects (Fig. 1). We were able to conclude that the presence of this wave in the P-Q period of the cardiogram of a patient subject to chest pain does not indicate a myocardial injury.

NOTCHING OF THE QRS COMPLEX

According to the New York Heart Association (1940) a deflection is slurred if one of its limbs or its peak shows an obvious local thickening; the term should not be applied to the slight thickening at the apex of the wave or just where it leaves or returns to the base-line. A notched deflection is characterized by spikes on one or other of its limbs, while a splintered deflection is one which shows two or more apices. We believe that all three changes reflect the same mechanism, namely an interruption in the progress of the electrical impulse through the myocardium and that the distinction between them is one of degree only. If an apparent slur is examined under magnification it will often be seen as a minute notch. We have found it more convenient to refer to all three changes as *notching* and to designate the grades of notching as slight, moderate, or prominent.

The relationship of notching of the QRS complex to myocardial damage has been discussed in the past (Carter, 1914 and 1918; Wedd, 1919; Wilson and Herrmann, 1920). The New York Heart Association stated that slurring, notching, or splintering of the QRS was abnormal if it appeared in two leads and nearer the peak than the base in one of them. Bayley (1946) stated that such changes usually, although not invariably, indicated death of a portion of the myocardium. In 95 patients in whom patchy fibrosis of the myocardium was found at necropsy, Weinberg *et al.* (1950) reported notching of the QRS in 32 per cent, but without specifying the affected leads; they stated that notching, other than in lead III, was almost invariably a sign of myocardial damage and they emphasized a statement made before by one of them (Katz, 1946) that notching was more likely to be significant if it happened nearer the apex than the base of the complex, was present in multiple leads, and was obvious. It had no significance if the change was slight or was associated with a tracing of low voltage.

Notching of the QRS in lead III was common in our healthy series and was found in more than one-third of the 156 cases. On the other hand, notching in leads I and II is uncommon, 11 and 13 among 2000 cases (Hall *et al.*, 1942), 13 and 4 among 500 cases (Stewart and Manning, 1944), and 23 and 35 among 1000 cases (Graybiel *et al.*, 1944). In our series (see Table I) prominent notching in leads I and II was never met with either alone or in combination, nor was it seen in all three limb leads. Stewart and Manning found two examples of notching in these three leads, but they did not specify its degree. The incidence of notching in the limb leads I and II, low though it is, is probably too high to regard the sign as unequivocal evidence of myocardial damage unless it is associated with a similar change in the chest leads.

TABLE I
THE INCIDENCE AND DEGREE OF NOTCHING OF THE QRS COMPLEX IN SEPARATE OR COMBINED LEADS IN 156
HEALTHY SUBJECTS

Leads	I	II	III	I and II	I and III	II and III	I, II, and III
Degree							
Slight	2	—	25	—	1	4	1
Moderate	1	—	14	—	—	3	1
Prominent	—	—	17	—	—	1	—
Total	3	None	56	None	1	8	2
Leads	CR1	CR4	CR7	I and CR1	III and CR1	II, III, and CR1	I, III, and CR4
Degree							
Slight	10	1	1	1	21	2	1
Moderate	2	—	—	—	4	—	—
Prominent	1	—	—	—	4	2	—
Total	13	1	1	1	29	4	1

FIG. 2.—Notching of the QRS in cardiac pain. Prominent notching in CR7 is a lone sign in (A), taken three years before (B), which shows added signs of infarction in the form of a deep Q and an inverted T wave in leads III and III R.

It was common to find notching in CR1 (45 out of 156) and approximately the same incidence was found for CR2 and CR3, and the change was often present in two or all three leads. We know that notching in these leads can be a pathological sign because we have seen it develop along with other graphic evidence of cardiac infarction and persist as the only sign when the cardiogram had otherwise resumed a normal pattern. It is true that such notching was on the ascending limb of the R wave, but this does not wholly differentiate the pathological from the physiological curve, and in the meantime it cannot be regarded as having diagnostic value. Notching of the QRS in CR4 was only found once in our healthy series when it was very slight and associated with similar notching in leads I and III; in this instance there was also notching in CR3 and CR5. With this exception and recently in a healthy youth of 22, not included in our series, who showed slight notching in CR7, the change was not found in leads CR4 to CR7. There is agreement that notching can take place over the so-called transition zone (Wilson *et al.*, 1944; Rosenman and Reynolds, 1950) which varies in position and width. Our case of notching in CR4 and CR5 occurred when both leads showed an Rs complex.

On the other hand, among patients with chest pain, we have found several examples of notching of CR7 and less frequently of CR4 when this lead showed a qR pattern. The notching is usually on the downstroke of the R wave and it is often associated with similar notching in a limb lead. Such a change may be a lone cardiographic finding or appear alongside other slight abnormalities like S-T depression. We regard the sign as indicating myocardial damage, and cardiac infarction in a patient with chest pain (Fig. 2). We have also found this sign in patients showing left ventricular preponderance from hypertension, most of whom had cardiac pain; even in cases of hypertension without pain we regard the sign as pointing to coronary arterial disease.

DEPRESSION OF THE S-T SEGMENT

A deformity of the S-T interval showing as a depression is a common finding in early myocardial injury from coronary artery disease. Such depression may not extend obviously below the isoelectric level and usually only part of the segment is involved. Thus, the change may happen in the early, middle, or distal portions of the S-T interval and much or even the whole of the segment may not fall below the baseline (Fig. 3).

FIG. 3.—Diagram showing the different forms of S-T depression in cardiac pain.

Depression of the early portion of the S-T. Such depression was not met with in the healthy series, but was sometimes seen in patients with chest pain whose electrocardiograms at one time were regarded as normal or only showing equivocal changes, but where cardiac infarction was ultimately proved to be the cause of the pain. The deformity in this group was of two kinds. The first was a small dip after the start of the segment resulting in a *sickle depression* (Fig. 4). This was made more obvious when an R1 wave immediately preceded it. In the second kind the depression is earlier and affecting the returning limb of the S wave producing a *claw depression*. Like the first variety this is not a common deformity. It may be difficult of interpretation and it should not be confused with a broad S wave which always forms an angle at the point where it joins the S-T segment (Fig. 5). In the pathological claw depression on the other hand, the S-T is depressed below the isoelectric level from its very commencement at the peak of the S whence it passes upwards as an upward convex curve to reach the isoelectric line (Fig. 6).

Depression of the middle portion of the S-T. This deformity too was not found in the healthy series, but appeared in those patients with cardiac pain and minor cardiographic changes. The resulting curve which is in the form of a *trough depression* (Fig. 7) is again seen to better advantage when an R1 precedes it.

Depression of the distal portion of the S-T. This deformity is noteworthy in that the segment may not at any point reach below the isoelectric line. It deserves the term depression, however, because the terminal portion is depressed compared with the corresponding portion of the normal segment. As a result, the segment is linear as far as the more acute angle that it forms with the ascending limb of the T wave; this limb is unusually steep because its formation is delayed. We have named this deformity the *plane depression* and we regard it as the most valuable of the lesser cardiographic signs of cardiac pain (Fig. 8 and 9). Sometimes the whole segment may be displaced

FIG. 4.—Sickle depression of S-T in cardiac pain. This deformity in CR7 is the only abnormal sign in the resting cardiogram. The exercise test gave a positive result.

FIG. 5.—Broad S wave in health simulating claw depression of S-T. The returning limb of the S wave is wide but is not depressed.

FIG. 6.—Claw depression of the S-T in cardiac pain. This deformity in leads II, III, and CR7, along with lowering of the T in III R, are the only abnormal signs in the resting cardiogram. The exercise test gave a positive result (A).

downwards while retaining this characteristic flattening, when its significance is the same and the difference is one of degree only.

An exaggerated form of this depression is met with where the end of the S-T interval dips below the isoelectric level, making with the ascending limb of the T wave a curve resembling the lapwing in flight, and for this reason we have referred to it as *wing depression*. In patients with cardiac pain this deformity was less common than the plane variety when the cardiogram was recorded at rest, but it was common as part of the abnormal curve that followed cardiac anoxæmia induced by physical exercise (Fig. 10).

S-T depression in III R. A comparison of the S-T segment in lead III with that seen in III R (lead III during deep inspiration) proved valuable in the detection of lesser ischæmic changes. A code of normality has been nominated for III R (Evans, 1951) and this concerns the Q and T waves in addition to the S-T segment. A depression of the S-T does not appear in health except in the rare instances when the heart moves upwards during inspiration (paradoxical movement of the diaphragm) (Fig. 11), and sometimes when the heart hangs vertically in the chest. This depression of the S-T in III R may affect any part of the segment and it was found either in company with other minor cardiographic effects of cardiac ischæmia or as a lone sign (Fig. 12 and 13). The presence of the sign depends on the site of the infarct, so that it was present when the injury was situated in the lateral, posterolateral, and postero-inferior parts of the left ventricle, and occasionally in the septum, but not when the infarct was placed anteriorly. The usefulness of III R, therefore, depends on its sensitiveness to changes in three out of the five areas in the heart that are commonly affected by infarction.

THE R1 WAVE

By definition the R1 wave is the upright wave immediately following the S wave, but the designation is not used here to describe the large secondary R wave found in leads over the right ventricle in patients with right bundle branch block. When studying the less obvious cardiographic changes in patients with chest pain we found the R1 wave in many of them; it was present either in company with other minor though significant changes or was the only obvious change. We next looked for it in the healthy series of 156 cases and found it in 45 of them (Fig. 14). The particular leads that showed R1 are listed in Table II, and when the incidence of R1 in single or combined leads was compared

with that found in cases with chest pain the two groups could not be distinguished. It caused some surprise that the presence of R1 should prove to be less significant than prominent notching of the QRS complex, but the analysis of the two series of cases does not justify the conclusion that the presence of the wave by itself signifies myocardial injury.

FIG. 7.—Trough depression of the S-T in cardiac pain. This deformity in leads II, III, III R, and CR7 in (A) is replaced by more obvious signs of cardiac infarction in (B), recorded 15 months later.

FIG. 8.—Plane depression of the S-T in cardiac pain. This deformity in leads I and CR7 with a low T in CR4 (A), gave way to more obvious changes three years later (B).

FIG. 9.—Plane depression of the S-T in cardiac pain. This deformity in leads I, II, CR4, and CR7 (A) was followed eight months later by more obvious signs of infarction (B).

CHANGES IN T WAVES

The low T wave. It is known that the height of the T wave varies among healthy subjects and from lead to lead. This variation applies not only to its absolute amplitude, but also in its relationship to the height of the R wave. One would expect that between the normal upright and the abnormal flat T wave there should be an intermediate stage where a lowered T could be regarded as an abnormal sign. The difficulty lies in the definition of the lower limits of normal. Dressler (1943) tried to overcome this by comparing the amplitude of T waves in different leads, but Goldberger (1947) has disputed the accuracy of this procedure in the case of limb leads. Leatham (1950) calculated the height of the T wave in chest leads and its relation to the height of the R wave; in

TABLE II
INCIDENCE OF R1 WAVE IN THE LIMB AND THREE CHEST LEADS OF 156 HEALTHY SUBJECTS

Leads	I	II	III	I and II	I and III	II and III	I, II, and III	
Incidence	2	6	7	—	—	6	—	
Leads	CR1	CR4	CR7	I and CR7	II and CR7	III and CR7	CR4 and CR7	I, II, and CR7
Incidence	6	5	2	2	2	1	1	1

CR4 the average height of the T was 7.9 mm. and this was 26 per cent of the average amplitude of the R wave. The corresponding figures for CR7 were 3.3 mm. and 29 per cent. We obtained similar figures in a further series of healthy subjects, but the variation proved so wide as to make such measurements of little value.

Although a low T wave is met with in CR4 in health its presence in a patient with chest pain should always be regarded with suspicion and another tracing should be recorded after exercise. Should the low T wave be a pathological sign in the cardiogram at rest, exercise should produce a positive test (Fig. 8, 15, and 16). On the other hand a negative test is expected if the low T is part of a physiological cardiogram. A low T wave is unusual in CR7 in health and for this reason it is a valuable sign; this does not apply to V7 where the normal T is expected to be low. A low T in lead I may occur in health, but when it is associated with a low T in CR4 and especially in CR7 it is likely to be significant.

The blunt T wave. A blunted T wave, where the customary sharp peak is pruned giving it a domed appearance, is not necessarily a low T wave for the change is confined to the summit of the

FIG. 10.—Wing depression of S-T in cardiac pain. A plane depression in the resting CR4 cardiogram (A) changed to wing depression after exercise (B).

FIG. 11.—Paradoxical movement of diaphragm. In a case where left phrenic nerve had been divided in the treatment of tuberculosis, inspiration caused the left dome of diaphragm to move upwards and inversion of the T wave in III R.

FIG. 12.—Depression of S-T in III R in cardiac pain. This deformity is a lone sign in (A) recorded two months before the more obvious signs of cardiac infarction appeared (B). Patient died during an attack of pain six months later.

FIG. 13.—Depression of S-T in III R in cardiac pain. A lowering of terminal portion of S-T and further inversion of T are the only abnormal signs in the resting cardiogram. A positive exercise test was obtained (A).

wave, although for that reason the wave naturally loses a little of its stature. Indeed, this slight lowering distinguishes it from a blunted tall T wave which is occasionally met with in health. From their study of the allied deformity of notching of the T wave, Dressler *et al.* (1951) concluded that such a deformity had the same significance as T wave inversion; and that slurred, flat-topped, and triphasic T waves were closely related to notched T waves and had the same meaning. A blunted and slightly depressed T wave in CR4 or CR7 was not met with in our healthy series, and although it was not a frequent finding in those cases with chest pain and equivocal cardiographic changes, its presence even as a lone finding told of a myocardial injury (Fig. 17 and 18) and for that reason we regard it as an important sign.

Terminal dipping of the T wave. We have been hesitant about including this change among the less obvious cardiographic signs, because its recognition is not difficult as a rule when it gives to the T wave a diphasic conformation. It is when this deformity is minimal in a tracing taken either early in the illness of cardiac infarction or during recovery that it may assume importance in that it

FIG. 14.—The R1 wave in health. This small upright wave immediately follows the S wave in leads I, CR4, and CR7.

FIG. 15.—Low T wave in cardiac pain. A low T in CR4 is the only suspicious sign in the resting cardiogram; it became inverted after exercise (A).

may then go undetected (Fig. 19 and 20). Care should be taken not to mistake as terminal dipping of the T an appearance where through tachycardia an early incidence of the P wave may prevent the completion of the descending limb of the U wave so that the natural dip between the T and U waves is no longer matched by a similar bay between the U and P waves (Fig. 21).

FIG. 16.—Low T wave in cardiac pain. A low T in leads I and CR7 is the only noticeable change in the tracing from a patient who died suddenly three months later from an extension of cardiac infarction.

FIG. 17.—Blunt T wave in cardiac pain. This deformity in lead CR4 is the main cardiographic change in a patient who died six months later from further cardiac infarction.

INVERSION OF THE U WAVE

The sixth wave has attracted attention since the early days of electrocardiography and Einthoven (1906) at first thought it was abnormal, but later he realized that it could be found in health (Einthoven, 1912). Others (Lewis and Gilder, 1912) have described a peaked variety of short duration and a long shallow kind, and Maekawa (1931) considered that the second variety was pathological. There is general agreement now that an upright U wave is a normal finding irrespective of contour, but such unanimity of opinion is not found concerning inversion of the U wave. Katz (1946)

FIG. 18.—Blunt T wave in cardiac pain. This deformity of T in CR4 in (B) is the only residual sign of cardiac infarction nine months before (A).

FIG. 19.—Terminal dipping of T wave in cardiac pain. This change in CR4 (A) preceded by four months the more obvious signs of cardiac infarction shown in (B).

stated that little clinical weight should be allocated to a cardiogram in which the only deviation from the normal was in the U wave, and Solarz and Elek (1943) said that a study of U wave patterns was of little value in diagnosis. Others, however, have been at variance with these views. Thus, Nahum and Hoff (1939) found an inverted U wave only when heart disease was present and at times it was the only cardiographic evidence of myocardial damage, while Palmer (1948) concluded that inversion of U in one or more leads should always be regarded as pathological.

Inversion of the U wave was not met with once in our healthy series. Other workers have had identical experience, among them Stewart and Manning (1944) using limb leads only, and Myers *et al.* (1947) using a full range of chest leads in a smaller series. Graybiel *et al.* (1944) using limb, CR4, and CF4 leads in 1000 cases found inversion of U twice, and in both the change was confined to lead III; they did not state whether the change was associated with inversion of the T wave as well, nor did they test the effects of deep inspiration on the inversion. The infrequency with which inversion of the U wave has been reported in healthy subjects and its presence with other cardiographic changes in patients with heart disease (Nahum and Hoff, 1939; Papp, 1940; Solarz and Elek, 1947; Palmer, 1948) makes it very likely that the finding is pathological. Although it has usually been found in coronary artery disease, it has also been reported in hypertensive, valvular, and pulmonary heart disease. Palmer found inversion of the U wave in two cases of hypertension where coronary artery disease appeared to be absent. We found it three times in patients with

FIG. 20.—Terminal dipping of T wave in cardiac pain. This deformity in CR4 is the most noticeable change in a patient who later showed more obvious signs of cardiac infarction.

FIG. 21.—Apparent dipping of T wave caused by tachycardia. Early incidence of the P wave has interrupted the descending limb of the U wave giving a false impression of terminal dipping of the T.

left ventricular preponderance from hypertension in whom cardiac pain was absent, and we have found it once in patent ductus arteriosus where the cardiogram showed early left ventricular preponderance. Our experience leads us to emphasize caution in the diagnosis of an inverted U wave where the dip is not obvious, but we believe that true inversion of the U wave, either in the first two limb leads or in CR4 in the absence of other heart disease, is evidence of myocardial damage from coronary atheroma; proof of this will evolve from the natural progress of the case or from the test of cardiac anoxæmia (Fig. 22 and 23).

SUMMARY AND CONCLUSIONS

Experience has shown how unreliable is the patient's description of his pain as a guide to the diagnosis of coronary artery disease. It is known too that physical signs are sparse, and the electrocardiogram alone can provide the information that may save a patient from unwarranted invalidism created by a faulty interpretation of the chest pain in the absence of this test.

It was for this reason that we undertook an investigation of the lesser cardiographic signs found in cardiac-like pain, which hitherto have not been acknowledged as evidence of coronary artery disease. We first sought these changes in tracings from 156 healthy subjects and afterwards in those from patients where myocardial damage had been confirmed. Before accepting a particular cardiographic sign as proof of such heart disease we required, in addition to its absence from the healthy series, that the tracing in patients with cardiac pain should demonstrate undisputed cardiac infarction on another occasion, or that a one-time physiological cardiogram, recorded fortuitously when cardiac pain was absent, should subsequently show the lesser changes following the onset of pain; or that the less obvious changes should be joined by those typifying increased cardiac ischæmia in the course of the exercise test; or that cardiac infarction should have been discovered at necropsy in a patient who had only shown the less obvious signs at a recent cardiographic examination.

The investigation has allotted to these lesser changes in the electrocardiogram a significance no less important than the presence of Q waves and/or inverted T waves. Actually, the mortality rate,

FIG. 23.—The U wave in cardiac pain. Plane depression of S-T in I and CR7 and inversion of U in CR4 are the only changes in the resting cardiogram. After exercise (A) the T as well as the U is inverted in CR4, and U wave inversion becomes a conspicuous change in CR7.

FIG. 22.—The U wave in cardiac pain. Inversion of U in CR4 (B) is the only residual sign of cardiac infarction three years before (A).

for patients showing the more obvious changes is no greater than for those showing the lesser changes, and since both types of cardiogram indicate myocardial damage from coronary artery disease, they hold equal place in its diagnosis and prognosis.

A wave in the P-Q period or an R1 wave sometimes preceded or appeared alongside the characteristic changes of cardiac infarction; their frequent occurrence in health, however, excluded them as specific signs of myocardial injury. The lesser cardiographic changes that did signify such injury from coronary artery disease included notching of the QRS complex, depression of the S-T segment, a low or blunt T wave, dipping of the terminal limb of the T wave, and an inverted U wave.

Slight notching of the QRS, particularly in lead III and in chest leads over the right ventricle, is common in health. Moderate or greater notching in chest leads over the left ventricle which show a qR pattern is evidence of a myocardial injury.

Depression of the S-T segment was a common change in patients with cardiac pain and it proved to be the most significant of the lesser cardiographic signs. Frequently, such depression involved only a portion of the S-T segment and did not over-reach the isoelectric level. Depression of its early portion produced either a *claw* or a *sickle* deformity and was less common than a *trough* depression involving the middle portion of the S-T segment; this deformity was a particular feature of lead III R (lead III during deep inspiration). Depression of the distal portion of the S-T segment produced either a *plane* or a *wing* deformity. Plane depression of the S-T segment proved

to be a common sign and we apportion to it exceptional value in the diagnosis of cardiac pain. Wing depression was a less common abnormal finding in the resting cardiogram, but it frequently appeared as part of the deformity characterizing the positive exercise test.

Although a low T wave may be found in lead I and/or in CR4 in healthy subjects, it should be viewed with suspicion in a patient with chest pain, and another cardiogram recorded following exercise. A low T wave in CR7 is seldom met with in health and when the deformity signifies myocardial injury it is accompanied by a low T in lead I and depression of the S-T segment in III R.

A blunted T wave in CR4 or CR7 with a slight loss of height is a pathological change, as also is dipping of its terminal limb below the isoelectric level.

Inversion of the U wave appearing in the first two limb leads and especially in CR4, in the absence of other heart disease, is evidence of myocardial injury from coronary artery disease.

The recognition of these lesser cardiographic signs contributes to a readier diagnosis of cardiac pain and reduces significantly the number of such patients regarded in the meantime as having a normal electrocardiogram.

Of those patients attending the Cardiac Department, some were under the care of Dr. Wallace Brigden. Many of the electrocardiograms in healthy subjects were recorded by Dr. Aubrey Leatham when he was Sherbrook Research Fellow, and Dr. Geoffrey Storey helped with the exercise tests when he was Registrar to the Department.

REFERENCES

- Bayley, R. H. (1946). *Amer. Heart J.*, **31**, 677.
 Carter, E. P. (1914). *Arch. intern. Med.*, **13**, 803.
 — (1918). *Arch. intern. Med.*, **22**, 331.
 Dressler, W. (1943). *Amer. Heart J.*, **26**, 313.
 —, Roesler, H., and Lackner, H. (1951). *Brit. Heart J.*, **13**, 496.
 Einthoven, W. (1906). *Arch. Internat. Physiol.*, **4**, 132.
 —, (1912). *Lancet*, **1**, 853.
 Evans, W. (1951). *Brit. Heart J.*, **13**, 457.
 Goldberger, E. (1947). *Amer. Heart J.*, **34**, 395.
 Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A. (1944). *Amer. Heart J.*, **27**, 524.
 Hall, G. E., Stewart, C. B., and Manning, G. W. (1942). *Can. med. Assoc. J.*, **46**, 226.
 Herrick, J. B. (1919). *J. Amer. med. Assoc.*, **72**, 387.
 Katz, L. N. (1946). *Electrocardiography*. 2nd ed., Kimpton, Lond.
 Leatham, A. (1950). *Brit. Heart J.*, **12**, 213.
 Lewis, T., and Gilder, M. D. D. (1912). *Phil. Trans. Roy. Soc.*, **202**, 351.
 Maekawa, M. (1931). *Acta. Sch. Med. Univ. Kyoto*, **14**, 16.
 Master, A. M., Friedman, R., and Dack, S. (1942). *Amer. Heart J.*, **24**, 777.
 Myers, G. B., Klein, H. A., Stofor, B. E., and Hiratzka, T. (1947). *Amer. Heart J.*, **34**, 785.
 Nahum, L. H., and Hoff, H. E. (1939). *Amer. Heart J.*, **17**, 585.
 New York Heart Association. *Crit. Com.* (1940). New York.
 Palmer, J. H. (1948). *Brit. Heart J.*, **10**, 247.
 Papp, C. (1940). *Brit. Heart J.*, **2**, 9.
 Pardee, H. E. B. (1920). *Arch. intern. Med.*, **26**, 244.
 Parkinson, J., and Bedford, D. E. (1928). *Heart.*, **14**, 195.
 Rosenman, R. H., and Reynolds, R. W. (1950). *Amer. Heart J.*, **40**, 867.
 Solarz, S. D., and Elek, S. R. (1943). *J. Lab. Clin. Med.*, **28**, 936.
 Stewart, C. D., and Manning, G. W. (1944). *Amer. Heart J.*, **27**, 502.
 Wedd, A. M., (1919). *Arch. intern. Med.*, **23**, 515.
 Weinberg, S. L., Reynolds, R. W., Rosenman, R. H., and Katz, L. N. (1950). *Amer. Heart J.*, **40**, 745.
 Wilson, F. N., and Hermann, G. R. (1920). *Arch. intern. Med.*, **26**, 153.
 —, Johnston, F. D., Rosenbaum, F. F., Erlanger, H., Kossmann, C. E., Hecht, H., Cotrim, M., De Oliveira, R. M., Scarzi, R., and Barker, P. S. (1944). *Amer. Heart J.*, **27**, 19.
 Wolferth, C. C., and Wood, F. C. (1932). *Amer. J. med. Sci.*, **183**, 30.
 Wood, P., McGregor, M., Madgison, D., and Whittaker, W. (1950). *Brit. Heart J.*, **12**, 363.