ON T WAVES NORMAL IN SIZE AND DIRECTION BUT ABNORMAL IN CONTOUR

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MANY different abnormalities of the QRS complexes have been described, and a large number of these are easily recognized and have been shown to have a more or less precise significance. On the other hand, the different abnormalities of the T complex that have come to be widely recognized as distinctive and as having a definite connotation are few. Abnormalities in the direction of the T wave have received a great deal of attention and several different varieties of inverted T waves are distinguished. Abnormalities of the size of upright T waves have also been described, although these are recognized with greater difficulty than abnormalities in the direction, and their meaning is less clearly understood. Comparatively little has been done toward the analysis of abnormalities of the shape of upright T waves, although it is known, for example, that potassium retention in some cases of uremia may give rise to a tall, pointed T wave of more or less distinctive outline.[1,2]

The purpose of this article is to call attention to certain types of upright T waves which differ in shape from normal T waves and become inverted under a variety of circumstances, alike in certain particulars.

MATERIAL AND METHODS

We have examined 100 normal electrocardiograms collected by Bryant.[3] The ages of the subjects studied by him ranged from 19 years to 32 years. He recorded the standard and unipolar limb leads and precordial Leads V_2 and V_4. We have taken in Lima and in Ann Arbor 100 additional electrocardiograms on normal subjects 20 to 75 years of age. This series includes seven precordial leads (V_1, V_2, V_3, V_4, V_5, V_6, and V_E), as well as the six limb leads. In 80 of these 100 cases the effect of carotid sinus massage was investigated. In forty instances, all of the thirteen leads mentioned were taken before, and at least Leads I, II, V_R, V_F, V_3, V_4, V_5, and V_6 were taken during this procedure. In the remaining forty cases it was studied in a smaller number of leads. The age of the subjects upon which this test was performed ranged from 20 to 75 years, with an average age of 37 years. The effect of carotid sinus massage upon the form of the T wave

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Many of the observations upon which this article is based were made with the assistance of a fellowship from the W. K. Kellogg Foundation and with the aid of grants to Doctor Frank N. Wilson from the Horace H. Rackham School of Graduate Studies and the S. S. Kresge Foundation.

was also studied in a series of 600 patients with various types of heart disease. In this group, tracings were taken only when inspection of movements of the galvanometer string shadow indicated that changes were taking place. Blood pressure readings were taken during the period of carotid sinus stimulation in many instances. When this procedure produced changes in form of the T wave the patient was kept under observation and additional tests were made during a period which varied from a few months to one and one-half years. All electrocardiograms were taken with the patient in the recumbent position. The precordial points from which leads were taken were carefully marked. All records were taken with an electrocardiograph of the string galvanometer type.

OBSERVATIONS

The T Waves of Normal Subjects.—In describing the T waves of the normal records, we may ignore the variations in the polarity and contour of this deflection in Lead III and in Leads V_L and V_1, which are well known. We may also pass over variations of like kind in Leads V_F, V_2, and V_E. The form of the T wave in the remaining leads (I, II, V_R, V_3, V_4, V_5, and V_6) was much more constant.

In all these leads, except V_R, the normal T wave is upright and has a more or less characteristic shape. The slope of its ascending limb is much more gradual than that of its descending limb (Fig. 1,*A* to *E*). The normal T wave of Lead V_R would have the same shape if its direction were reversed (Fig. 1,*F*). The difference between the two limbs of the T wave is well brought out by dropping a perpendicular upon the base line from the apex of this deflection. The angle made with this perpendicular by the tangent of the first limb is much larger than that made by the tangent of the final limb (Fig. 1,*A* to *F*). These angles were measured in approximately forty cases. Their absolute and relative magnitudes vary greatly with the height and the shape of the apex of the T wave. For that reason we shall not present a detailed analysis of these measurements.

In the vast majority of instances both limbs of the T wave were smooth and the changes in their slopes from point to point were gradual. The final slope was often followed by a U wave of small size. In ten normal subjects, 5 per cent, the T waves in one or more leads did not correspond to this description. In some cases the two angles mentioned were approximately equal, the peak of the T wave was unusually sharp or unusually blunt, notching was present, or some other peculiarity in form occurred. Unusual oscillations at the very end of the T wave, possible U waves, sometimes were observed. Because T waves showing these features occurred in only a small percentage of cases, we consider them atypical (Fig. 1,*G* to *X*).

Atypical T Waves in the Electrocardiograms of Patients With Various Diseases.—In the tracings of patients with heart disease, atypical T waves were much more frequent than in the records of normal subjects, and the atypical features were much more pronounced. In many electrocardiograms of such patients the atypical T waves were frequently the only peculiarity suggesting that the heart was abnormal. We have, however, no reliable statistical data upon which an

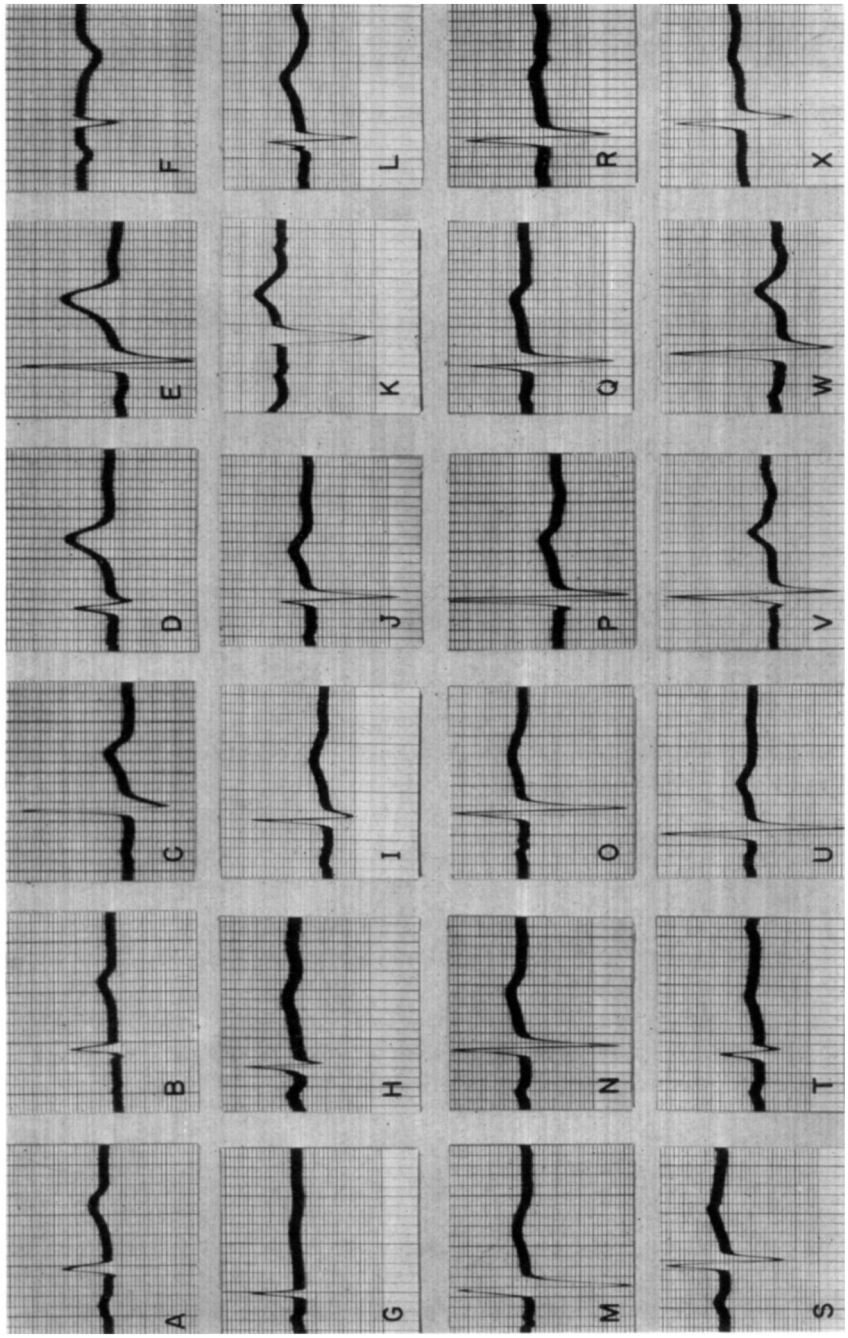

Fig. 1.—The T waves of the complexes shown in Strips *A* to *F*, inclusive, are regarded as normal in shape. The inverted T wave of Strip *F* illustrates the normal appearance of this deflection in Lead V_R. The T waves of the remaining strips, *G* to *X*, inclusive, are regarded as atypical in shape. In some instances the two angles made by the tangents of the ascending and the descending limbs of the T deflection with a perpendicular to the isoelectric level drawn through the summit of this deflection are approximately equal (*G*, *H*, *I*, *J*, *M*, *S*, *T*, *U*, *V*, and *W*), or the angle made with this perpendicular by the tangent to the descending limb is larger than that made by the tangent to the ascending limb (*K* and *L*). In other instances the apex of the T wave is usually sharp (*V*) or unusually blunt (*G* and *M*). In still other cases there is an irregularity or notch on the descending limb of the T deflection (*O*). In such cases carotid sinus pressure may cause inversion beginning in the vicinity of the notch or irregularity. Sometimes there are oscillations at the very end of the T wave (*P*, *Q*, and *R*) which differ in character from the normal T wave. Carotid sinus stimulation often produces inversion beginning near the very end of the T wave under these circumstances.

accurate estimate of the frequency of atypical T waves in tracings of patients with heart disease can be based. They were encountered under a variety of conditions. They occurred in precordial leads taken from points near those which yielded inverted T waves, and were found in tracings taken during attacks of angina pectoris[4] and in tracings taken after exercise tests.[5] Similar deflections were found in the tracings of patients who were convalescing from acute illness or who were suffering from a severe metabolic disorder, frequently when the heart was known to be involved. Atypical T waves often seemed to be associated with a prolongation of the Q-T interval, but in many such instances the end of the T waves could not be determined with sufficient accuracy to make it possible to measure this interval satisfactorily.

Carotid Sinus Stimulation.—In normal subjects carotid sinus stimulation produced slight variations in the amplitudes and contour of the T wave. In no instance did it alter the polarity of this deflection. The maximal change in amplitude was 1.1 mm. and the average change was 0.2 millimeter. In 52 per cent of the cases no modifications of the T wave were observed. Naturally, bradycardia occurred in practically all instances.

In the electrocardiograms of patients with cardiac abnormalities inverted T waves often became more inverted during carotid sinus massage. In only two cases did such T waves become upright. In Lead V_R, in which the T wave is normally inverted, any change which occurred was opposite in character to those which took place in the leads from the left side of the precordium. Atypical upright T waves of the kind described in the preceding paragraph often became inverted during carotid sinus stimulation. Sometimes the inversion was confined to the terminal part of the deflection and was of the V-shaped type. We may leave out of consideration here such changes in the T waves as accompanied the development of an independent ventricular rhythm. This phenomenon seldom occurred.

Carotid sinus massage induced, of course, both bradycardia and a fall in the blood pressure. It is impossible to say whether the T-wave changes observed were due directly to the change in the heart rate, to the change in the blood pressure, or to these combined with other factors.[6,7] In many cases of chronic heart disease in which T-wave changes were observed following carotid sinus stimulation, serial electrocardiograms were taken over a period of several months. In some of the instances in which carotid sinus stimulation produced temporary inversion of previously upright T waves, persistent inversion of these deflections was observed days or weeks later. In other cases, T waves which were persistently inverted when the patient was first seen became upright later, and in many such instances temporary inversion could then be induced by carotid sinus stimulation. We may, therefore, say that carotid sinus stimulation frequently produces temporary changes in the T waves of the kind that will become persistent subsequently, or temporary T-wave changes of a kind that were persistent in the past.[6]

The leads in which the T waves became inverted on carotid sinus stimulation were rather constant for the same individual, but varied from one patient to another. This suggests that the phenomenon under consideration is determined

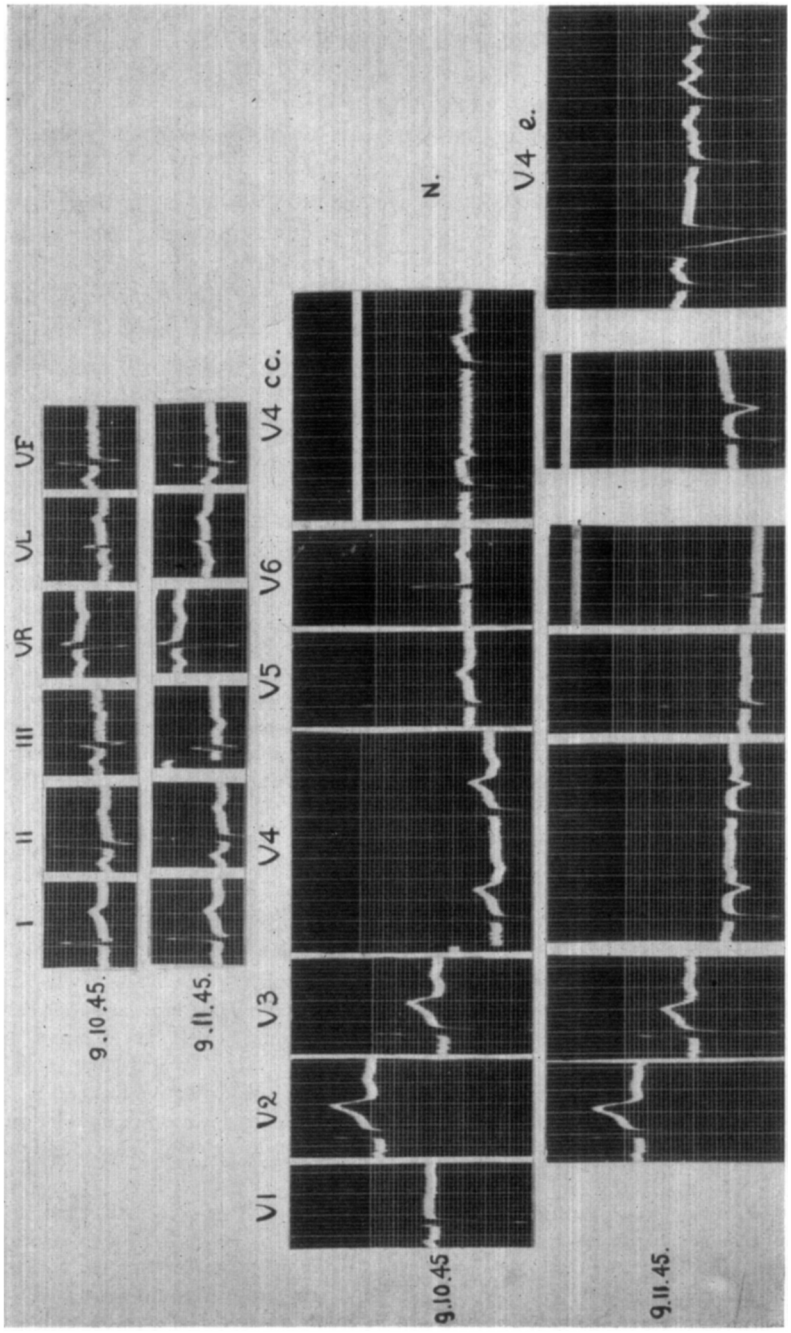

Fig. 2.—Electrocardiograms in Case 1. See text for explanation.

by the underlying condition of the heart, and even by conditions existing locally in the myocardium. In some cases in which the T waves were extremely labile, it was possible to change the polarity of this deflection at pleasure.[7]

During carotid sinus massage the T waves of some hypertensive individuals became inverted when the heart rate was relatively fast and the blood pressure was still above the normal range. In such cases the T-wave changes cannot be ascribed to abnormal variations in the heart rate or in the blood pressure. That carotid sinus stimulation can exert direct effects upon the ventricular myocardium seems improbable because all attempts hitherto have failed to demonstrate any direct action of the vagus nerves on the ventricular muscle. Sometimes the inversion of the T waves induced by massage of the carotid sinus region persisted for a considerable period and only became upright when the heart rate and the blood pressure had returned approximately to their original levels.

Frequently the inversion of the T waves produced by carotid sinus stimulation was the sole electrocardiographic abnormality detectable, but the suspicion that the heart was abnormal which it aroused was supported by the clinical data. The most striking changes in the T waves were recorded in patients with arteriosclerotic and hypertensive heart disease, but the same phenomena were occasionally observed in syphilitic, rheumatic, and congenital heart disease.[6,7]

ILLUSTRATIVE CASES

CASE 1.—The patient was a man, aged 55 years, whose blood pressure had been elevated for several years. The electrocardiograms taken on Sept. 10, 1945, were not definitely abnormal, but the contour of the T waves as atypical, particularly in Lead V_4 (Fig. 2). On carotid sinus stimulation, these atypical T waves became inverted (V_4 *c c*). On the following day the patient's clinical condition and the deflections of the limb leads were unchanged, but the T wave was inverted in Lead V_4 and flat in Leads V_5 and V_6. Carotid sinus stimulation increased the depth of the inverted T wave in Lead V_4 (V_4, *c c*), but produced no changes in the other leads. After exercise the patient complained of retrosternal oppression and the T waves in Lead V_4 became upright (V_4, *e*); at this time, however, the T waves of the complexes which followed compensatory pauses due to extrasystoles were flat or showed slight terminal inversion. All of the T waves were inverted when the effect of the exercise had worn off. Serial electrocardiograms taken at intervals over a period of one year showed striking changes in the shape of the T waves not associated with changes in the patient's clinical status.

CASE 2.—The electrocardiograms reproduced in Fig. 3 are those of a man, aged 53 years, who had hypertensive heart disease. In the records taken on Sept. 25, 1947, the T waves are flat in Lead II and slightly inverted in Lead V_F. The Q-T interval is somewhat prolonged and, following long postextrasystolic diastoles, there is flattening of this deflection in Lead I. In the tracings of the following day the T waves of precordial Leads V_5 and V_6 were somewhat atypical before and definitely inverted during carotid sinus stimulation (V_5 *c c* and V_6 *c c*); stimulation also induced moderate changes in the T deflection in Lead V_4 (V_4 *c c*).

CASE 3.—The electrocardiograms reproduced in Fig. 4 are those of a man, aged 51 years, with hypertensive heart disease. The tracing of May 5, 1946, shows left axis deviation and flat T waves in Lead II. On Jan. 14, 1947, one month after splanchnicectomy, the T waves were taller and the heart rate was faster. On Oct. 25, 1947, electrocardiograms were taken before and after carotid sinus stimulation. The classical limb leads labelled *a* were taken before and the tracings *b* during carotid sinus stimulation. The precordial leads were apparently normal, but the T waves in Leads V_4 and V_5 were atypical, and of the type that very often become inverted during carotid sinus stimulation. During carotid sinus massage the heart rate decreased,

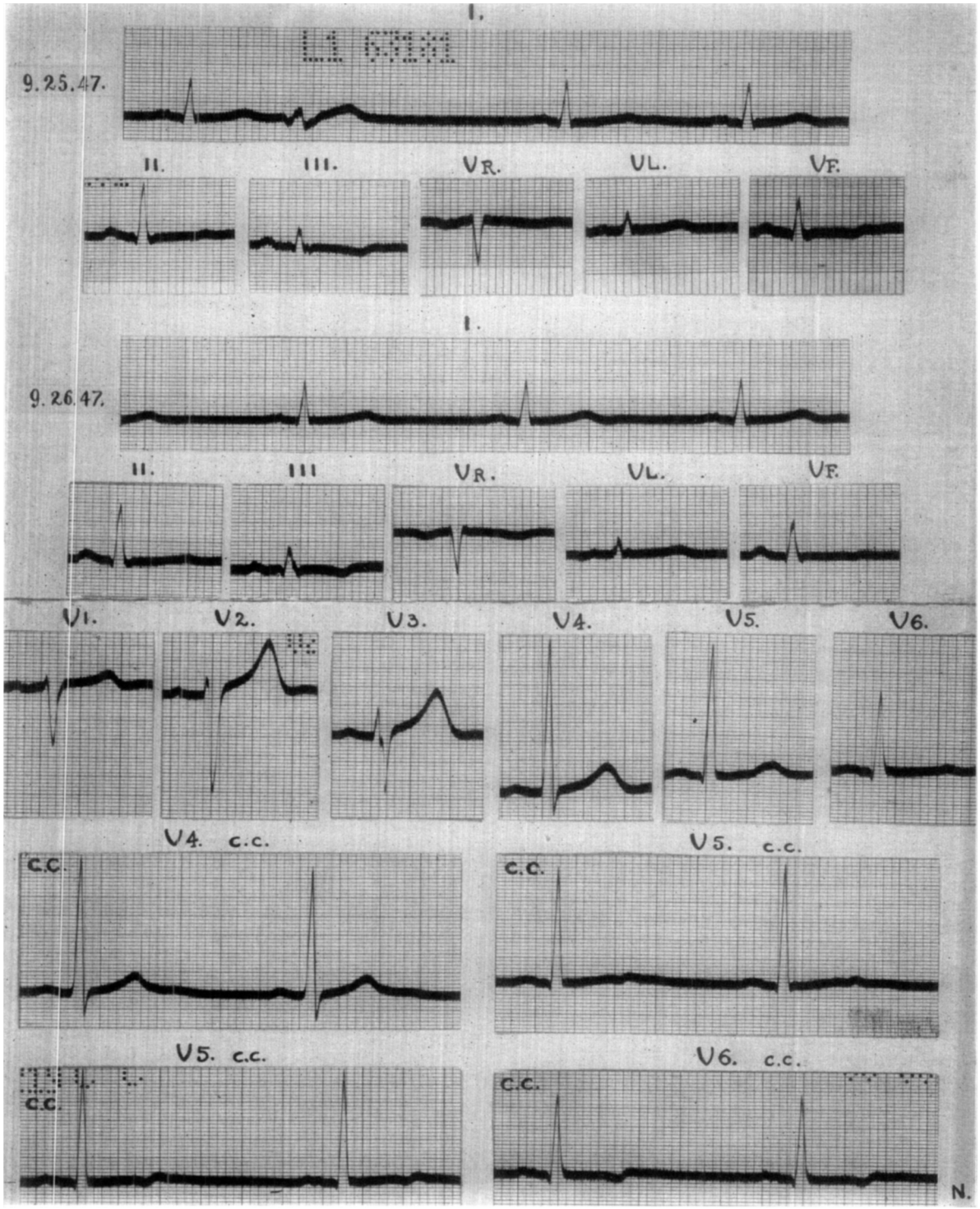

Fig. 3.—Electrocardiograms in Case 2. See text for explanation.

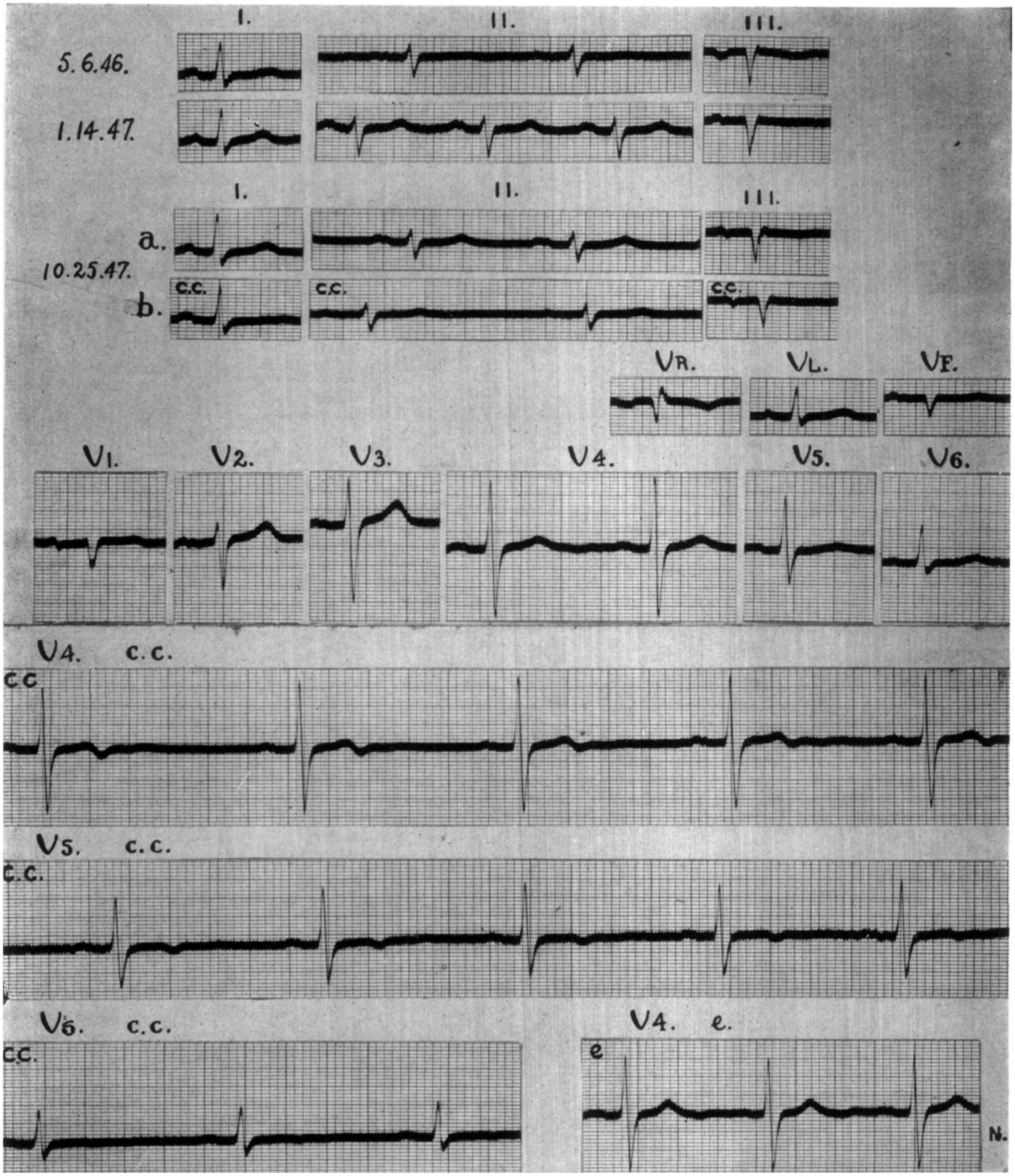

Fig. 4.—Electrocardiograms in Case 3. See text for explanation.

the blood pressure fell from 160/110 to 122/88, and the T waves became inverted in these leads ($V_4\,c\,c$ and $V_5\,c\,c$). In Lead V_6 the T deflections became nearly isoelectric ($V_6\,c\,c$). The tracing marked $V_4\,e$ was recorded after moderate exercise. Here the T waves are upright but atypical. In the electrocardiograms taken a few hours later the T waves were flat in Leads II, V_4, V_5, V_6, and V_R.

CASE 4.—The patient was a man, aged 62 years, who was having severe attacks of angina pectoris. The electrocardiograms showed inversion of the T waves which became more pronounced during carotid sinus stimulation. The complexes reproduced in Fig. 5 were selected from a continuous tracing taken during an attack of angina pectoris. During the attack the T waves became upright and there was slight downward displacement of the RS-T junction. The Q-T interval was considerably prolonged while the T wave was upright, and this deflection was definitely atypical in form. Note that it is distinctly notched and that the final limb falls very slowly. In other electrocardiograms taken on the same patient during attacks of angina pectoris, reversal of polarity of the T waves was observed with simultaneous pronounced RS-T displacement. In Lead V_R the displacement was upward and was accompanied by terminal inversion of the T wave; in Leads V_3, V_4, V_5, and V_6 the displacement was downward and the terminal part of the T wave was upright.

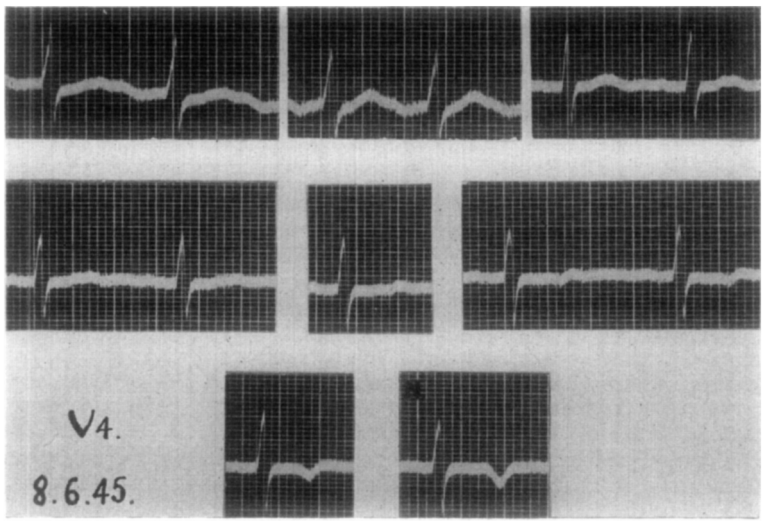

Fig. 5.—Electrocardiograms in Case 4. The complexes reproduced were selected from a continuous tracing taken during an attack of angina pectoris. During the attack the T waves became upright and these deflections were definitely atypical in form. The Q-T interval was considerably prolonged while the T deflection was upright.

CASE 5.—A 56-year-old man complained of pain in the left upper quadrant of the abdomen. The blood pressure was 190/130 and there was a history of mild angina pectoris. Electrocardiograms taken on Aug. 20 and Sept. 1, 1946, were similar to those taken on Sept. 3, 1946, which are reproduced in Fig. 6, *a*. The T waves are flat in all the limb leads and inverted in precordial Leads V_3, V_4, V_5, and V_6. Prolonged carotid sinus massage produced moderate bradycardia, and a fall in the blood pressure to 120/80 for a period of at least one-half hour. During this time the T waves became progressively more inverted in the precordial leads, the previously isoelectric deflections in Leads I, II, and III became inverted, and those of Lead V_R became upright (Fig. 6, *b*). Moderate exertion produced tachycardia, with a rise in the blood pressure to 240/140. The T waves became upright. Tracing V_3, *e* shows a series of complexes from a continuous tracing. The first strip is the control and the other strips show the progression of the changes following the exercise test. On Sept. 18, 1946, the patient felt stabbing pain in

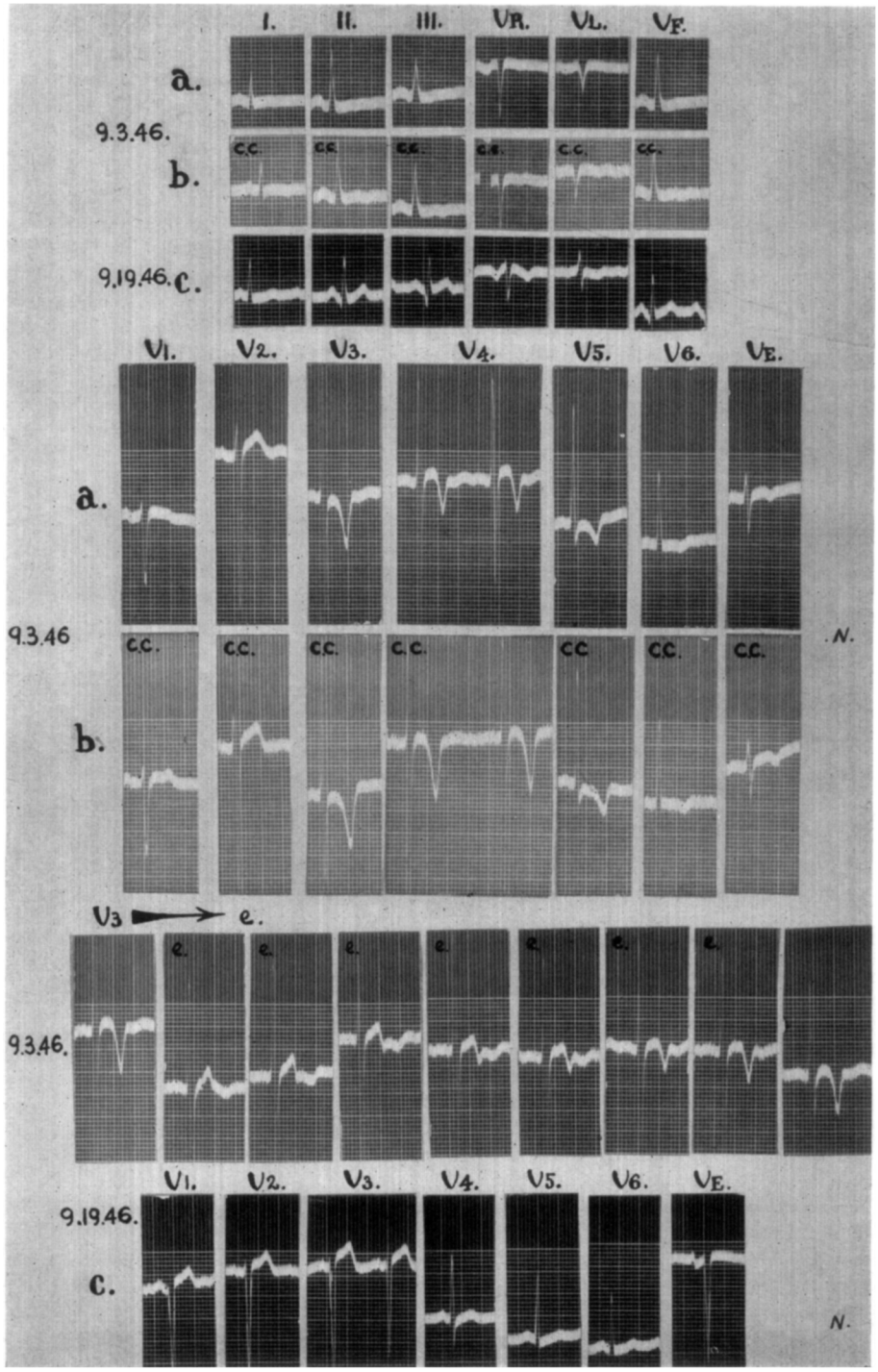

Fig. 6.—Electrocardiograms in Case 5. The electrocardiograms labelled *a* were taken before, and the tracings *b* during prolonged carotid sinus massage. Tracing V_3, *e shows the progression of* the changes following an exercise test. The electrocardiograms labelled *c* were taken several hours after an episode suggesting myocardial infarction.

the left upper quadrant with radiation to the left arm and precordium. On the following day his condition was precarious; shock was severe and the blood pressure fell to 120/75. The electrocardiograms taken at this time showed a pronounced reduction in the size of the R waves in Leads V_2 and V_3, slight upward displacement of the RS-T segment in Lead V_3, and deep Q waves in Leads II, III, and V_F. The most striking change, however, was the reversal of polarity of the T waves in all leads (Fig. 6, *c*). This pnenomenon was observed in a series of electrocardiograms taken during a one-half hour period. A fresh anteroseptal myocardial infarction was suspected. The patient died eighteen hours later. The autopsy showed general arteriosclerosis and fresh myocardial infarction. The lower part of the septum and a small area in the anterior ventricular wall were affected. The subendocardial layers and papillary muscles were predominantly involved. The necrotic process was found to be more advanced in some places than in others. The coronary arteries, chiefly the anterior descending branch of the left coronary, were partially occluded. There was, however, no complete coronary occlusion. Nearly two liters of blood were found in the left pleural cavity, the hemothorax being due to the rupture of an aneurysm of the descending aorta.

DISCUSSION

In the electrocardiograms of normal persons the contour of the T waves varies within rather narrow limits. In the tracings of patients with obvious or suspected heart disease, however, T waves of atypical shape are fairly common. Many of these atypical T deflections become inverted during carotid sinus pressure, after long postextrasystolic diastoles, or in the course of time even when there has been no definite change in the patient's clinical status. On the other hand, inverted T waves, particularly those in which the inversion is sharp and terminal, are often converted into atypical upright T waves by exertion, or other circumstances which increase the load upon the heart. The same reversal in polarity may occur during an attack of angina pectoris or even in the course of myocardial infarction. The changes in the T waves under consideration may be accompanied by changes in the level of the RS-T junction. They are not secondary to changes in the form of the QRS complex. Consequently, these T-wave changes are of the kind called primary by Wilson and Finch[8]; they depend upon changes in the ventricular gradient,[9,10] or, in other words, upon variations in the repolarization process, presumably dependent upon factors which affect the ventricular myocardium locally.

In 1931, Wilson, Macleod, and Barker[9] pointed out that in normal subjects the ventricular gradient points from the base toward the apex of the heart and suggested that this means that the average length of systole is greater in the base of the heart than at the apex, or greater on the inner than on the outer aspects of the ventricular walls, or both.

Recently a number of investigators have published electrocardiograms obtained by introducing an exploratory electrode into the cavity of the human right ventricle.[11,12,13] When the heart is normal the T waves inscribed in such leads are sharply and deeply inverted. In leads from the right side of the precordium, the QRS deflections are similar in general outline to those of the internal leads, but the T waves are strongly positive. It is evident, therefore, that although the subendocardial muscle of the right ventricle passes into the active state in advance of the subepicardial muscle, it passes out of this state later. In other words, the duration of the excited state is longer on the inner than on the outer aspect of

this wall. Whether or not this is also true of the left ventricular wall is uncertain. The T wave is normally sharply inverted in Lead V_R, which presumably reflects the potential variations of both of the ventricular cavities, and this strongly suggests that the cavity of the left ventricle, like that of the right, is negative during the inscription of this wave. It is true that Wilson and Herrmann,[14] in experiments in dogs, found that the deeper layers of the left ventricular wall pass out of the refractory period earlier than the superficial layers. In their experiments, however, the heart was exposed, and cooling and drying of the epicardial surface may have increased the duration of the excited state of the outermost layers of the muscle. There are, furthermore, no data bearing upon the form of the T deflection in direct epicardial leads or in leads from the ventricular cavities in these experiments. Byer, Ashman, and Toth[15] in experiments in dogs in which the chest was not opened have shown that cooling the endocardial surface of the heart prolongs the Q-T interval and greatly increases the size of the normally upright T waves in leads from the precordium.

We may conclude, tentatively, that the duration of the excited state is greater on the inner than on the outer surface of the ventricles, and, consequently, that the cavities of both ventricles are negative and the epicardial surfaces of both ventricles are positive during the inscription of the T deflection. If this is the case, reversal in polarity of the T wave is the result of a change in the relative length of systole on the two surfaces of the ventricular walls. No detailed explanation of this phenomenon is possible until we know what factors determine the normally greater duration of systole on the inner aspect of the ventricular walls, and how they are modified by various procedures. Some of the factors that come to mind are: blood supply, temperature, the influence of the vegetative nervous system, differences in tension in the various parts of the ventricular walls during ventricular contraction, and inherent differences in the muscle of different parts of the myocardium. There are at the present time few data bearing upon the influence or lack of influence of these factors or of others that may be involved. It has, however, been shown that local ischemia of the ventricular wall, produced by compression of a coronary artery, induces sharp inversion of the T waves in direct leads from the epicardial surface.[16] It has not been shown that the T waves of unipolar cavity leads are affected in the opposite manner, although it seems probable that this is the case. A more severe grade of ischemia leads to elevation of the RS-T segment in the epicardial leads and presumably to downward RS-T displacement in the cavity leads.

In a case of syphilitic aortitis reported elsewhere, the coronary blood flow was evidently greatly decreased. During life the patient had very frequent attacks of anginal pain. The autopsy showed that the coronary ostia were almost completely obliterated although the coronary arteries themselves were not involved. There was extensive necrosis of the subendocardial muscle of the left ventricle including that of the septum and the papillary muscles. In the right ventricular wall, patchy necrosis was present.[17] The subendocardial arteriolar plexus, described by Gross,[18] and the Thebesian vessels did not prevent necrosis, and only the Purkinje network remained undamaged.[17] These findings suggest that when the coronary circulation is uniformly impaired, lesions are likely to

occur in those places where systole is thought to be longest in normal subjects. It is probable that the physiologic, mechanical, and circulatory conditions are not entirely uniform throughout the left ventricular walls.[17,19,20] During the attacks of angina pectoris in this case, the electrocardiographic abnormalities consisted of downward displacement of the RS-T segment in the leads from the left side of the precordium and upward RS-T displacement in Lead V_R. Similar electrocardiographic changes have often been recorded during attacks of angina pectoris,[21-25] and there is some evidence that when anginal seizures are severe and persistent, necrosis of the subendocardial layers of the left ventricle is not infrequent. In 1932 Buchner[26] demonstrated that in the hearts of patients with angina pectoris necrosis was confined chiefly to the inner layers and papillary muscles of the left ventricle. Other studies have confirmed these observations, and RS-T displacement of the type described has been related to subendocardial injuries.[17,19,25,27-30]

The reversal of polarity of inverted T waves in the precordial leads during attacks of angina pectoris and the downward displacement of the RS-T segment accompanying it strongly suggest ischemia of the subendocardial muscle of the left ventricle.[19] In one case in which this phenomenon was present, necrosis affecting chiefly the subendocardial and papillary muscles of the left ventricle was observed.[19] Sodi-Pallares, Vizcaíno, Soberón, and Cabrera[12] have reported a case in which the inversion of the T wave in a lead from the right ventricular cavity became more pronounced during a spontaneous attack of angina pectoris. They ascribed this phenomenon to subendocardial ischemia.

We have shown that exertion and carotid sinus stimulation may reverse the polarity of the T waves under certain circumstances. Both have pronounced effects upon the heart, mediated at least to a large extent, through the vegetative nervous system. It is, therefore, difficult to say whether the changes in the T waves which they induce depend directly upon the changes in the heart rate and its effects upon coronary blood flow, upon the mechanics of cardiac contraction, tension in ventricular walls, and so forth, or whether these effects are primarily due to alterations in sympathetic and vagal tone. Segers[31] has shown that mechanical factors such as the tension in the cardiac walls, and chemical factors such as the concentration of adrenalin, acetylcholine, potassium, and calcium have important effects upon the monophasic electrogram of the frog's heart and particularly upon the cardiac afterpotentials.[31] We have no data which justify any conclusion as to the exact mechanism by which exertion or carotid sinus stimulation produce the electrocardiographic effects described.

SUMMARY

In normal subjects the polarity and contour of the T waves are relatively constant. Atypical T waves of normal polarity occur only occasionally in the tracings of normal subjects, but are common in the electrocardiograms of patients with heart disease.

In many instances the behavior of these atypical T waves and the previous or subsequent clinical status of the patient indicate that the myocardium is abnormal.

During carotid sinus stimulation and after long postextrasystolic diastoles, many of these atypical upright T waves become inverted. On the other hand, many inverted T waves are converted into atypical upright T waves by exertion or angina pectoris.

In some cases atypical upright T waves furnish the only objective evidence that the heart is not normal.

We are deeply indebted to Dr. Frank N. Wilson for his helpful suggestions. We also desire to express our thanks to Dr. F. D. Johnston and Dr. J. M. Bryant for their kind interest in the preparation of this paper.

REFERENCES

1. Wilson, F. N., and Herrmann, G. R.: Some Unusual Disturbances of the Mechanism of the Heart Beat, Arch. Int. Med. **31**:923, 1923.
2. Keith, N. M., Burchell, H. B., and Baggenstoss, A. H.: Electrocardiographic Changes in Uremia Associated With High Concentration of Serum Potassium: Report of Three Cases, AM. HEART J. **27**:817, 1944.
3. Bryant, J. M.: The Distribution of Myocardial Action Currents in Relation to the Cardiac Silhouette, Master of Science Thesis, University of Virginia, 1946.
4. Alzamora-Castro, V.: Contribución al Conocimiento del Significado de las Modificaciones del Segmento S-T y de las Ondas T, Gac. méd. Lima **2**:285, 1946.
5. Alzamora-Castro, V.: I. Significado de Algunas Alteraciones de las Ondas T, Arch. Inst. Cardiol. México **16**:541, 1946.
6. Alzamora-Castro, V., and Rubio, C.: II. Significado de Algunas Alteraciones de las Ondas T, Arch. Inst. Cardiol. México **17**:98, 1947.
7. Alzamora-Castro, V., Rubio, C., Battilana, G., and Subiría, R.: I. Estudio y Significado de Algunas Ondas T Anormales. Los Efectos de la Compresión Seno-Carotídea, Rev. argent. de cardiol. **14**:163, 1947.
8. Wilson, F. N., and Finch, R.: The Effect of Drinking Iced-Water Upon the Form of the T-Deflection of the Electrocardiogram, Heart **10**:275, 1923.
9. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The T-Deflection of the Electrocardiogram, Tr. A. Am. Physicians **46**:29, 1931
10. Wilson, F. N., Macleod, A. G., Barker, P. S., and Johnston, F. D.: The Determination and the Significance of the Areas of the Ventricular Deflections of the Electrocardiogram, AM. HEART J. **10**:46, 1934.
11. Hecht, H. H.: Potential Variations of the Right Auricular and Ventricular Cavities in Man, AM. HEART J. **32**:39, 1946.
12. Sodi-Pallares, D., Vizcaíno, M., Soberón, J., and Cabrera, E.: Estudio Comparativo del Potencial Intracavitario en el Hombre y en el Perro, Arch. Inst. Cardiol. México **17**:630, 1947.
13. Battro, A., and Bidoggia, H.: Endocardiac Electrocardiogram Obtained by Heart Catheterization in the Man, AM. HEART J. **33**:604, 1947.
14. Wilson, F. N., and Herrmann, G. R.: An Experimental Study of Incomplete Bundle Branch Block, and of the Refractory Period of the Heart of a Dog, Heart **8**:229, 1921.
15. Byer, E., Ashman, R., and Toth, L. A.: Electrocardiograms With Large Upright T Waves and Long Q-T Intervals, AM. HEART J. **33**:796, 1947.
16. Bayley, R. H., La Due, J. S., and York, D. J.: Further Observations on the Ischemia-Injury Pattern Produced In the Dog by Temporary Occlusion of a Coronary Artery. Incomplete T Diversion Patterns, Theophylline T Reversion, and Theophylline Conversion of the Negative T Pattern, AM. HEART J. **27**:657, 1944.
17. Alzamora-Castro, V.: IV. Contribución al Conocimiento del Significado de las Modificaciones del Segmento S-T. Angina de Pecho e Infartos Subendocardicos, Arch. Inst. Cardiol. México **17**:870, 1947.
18. Gross, L.: The Blood Supply of the Heart, New York, 1921, Paul B. Hoeber, Inc.
19. Alzamora-Castro, V., Rubio, C., Battilana, G., and Subiría, R.: II. Estudio y Significado de Algunas Ondas T Anormales. Su Vinculación con la Isquemia Subendocardica, Rev. argent. de cardiol. **14**:178, 1947.
20. Johnson, J. R., and Di Palma, J. R.: Intramyocardial Pressure and Its Relation to Aortic Blood Pressure, Am. J. Physiol. **125**:234, 1939.
21. Parkinson, J., and Bedford, D. E.: Electrocardiographic Changes During Attacks of Angina Pectoris, Their Bearing on the Origin of Anginal Pain, Lancet **1**:15, 1931.
22. Feil, H., and Siegel, M. L.: Electrocardiographic Changes During Attacks of Angina Pectoris, Am. J. M. Sc. **175**:255, 1928.

23. Wilson, F. N., and Johnston, F. D.: The Occurrence in Angina Pectoris of Electrocardiographic Changes Similar in Magnitude and in Kind to Those Produced by Myocardial Infarction, Tr. A. Am. Physicians **54**:210, 1939.
24. Wood, F. C., Wolferth, C. C., and Livezey, M.: Angina Pectoris. The Clinical and Electrocardiographic Phenomena of the Attack and Their Comparison With the Effects of Experimental Temporary Coronary Occlusion, Arch. Int. Med. **47**:339, 1931.
25. Bryant, J. M., and Wood, J. E.: Tobacco Angina, AM. HEART J. **34**:20, 1947.
26. Buchner, F.: Die Rolle des Herzmuskels bei der Angina Pectoris, Beitr. z. path. Anat. u. z. allg. Path. **89**:644, 1932.
27. Boyd, L. J., and Scherf, D.: The Electrocardiogram After Mechanical Injury of the Inner Surface of the Heart, Bull. New York M. Coll. **3**:1, 1940.
28. Master, A. M., Gubner, R., Dack, S., and Jaffe, H. L.: Differentiation of Acute Coronary Insufficiency With Myocardial Infarction From Coronary Occlusion, Arch. Int. Med. **67**:647, 1941.
29. Bayley, R. H.: The Electrocardiographic Effects of Injury at the Endocardial Surface of the Left Ventricle, AM. HEART J. **31**:677, 1946.
30. Wolferth, C. C., Bellet, S., Livezey, M. M., and Murphy, F. D.: Negative Displacement of the RS-T Segment in the Electrocardiogram and Its Relationships to Positive Displacement; An Experimental Study, AM. HEART J. **29**:220, 1945.
31. Segers, M.: Le Rôle des Potentiels Tardifs du Coeur, Mem. Acad. roy. de méd. de Belgique **1**:7, 1941.