THE OCCURRENCE IN ANGINA PECTORIS OF ELECTROCARDIOGRAPHIC CHANGES SIMILAR IN MAGNITUDE AND IN KIND TO THOSE PRODUCED BY MYOCARDIAL INFARCTION*

FRANK N. WILSON, M.D., AND FRANKLIN D. JOHNSTON, M.D.
ANN ARBOR, MICH.

DURING the last few years we have observed a number of cases of angina pectoris in which electrocardiograms obtained during paroxysms of substernal distress, either spontaneous or induced, have shown striking changes in the form of the ventricular complex comparable in magnitude and similar in kind to those which occur during the first few hours following the sudden occlusion of a large coronary artery. We would like to present here our clinical and electrocardiographic observations in five such cases, and to make a few comments with reference to their interpretation.

CASE 1.—Mr. L. E. T., an American office worker, aged 35 years, was first seen on Feb. 2, 1935. Early in the summer of 1934, while playing golf, he noticed pain on the ulnar side of the left arm which disappeared promptly when he rested. In subsequent attacks the pain began under the middle and upper sternum and radiated to the left arm as its intensity increased. The distress invariably disappeared within two minutes when he stopped exercising. Walking, climbing stairs, and lifting were the types of effort which most frequently caused pain. The symptoms were most likely to develop on exertion in the cold, or on exertion soon after a meal. The pain was severe but dull, and was accompanied by a sensation of fullness. There was no history of rheumatic fever or diphtheria. When he was 17 or 18 years of age, the patient had a penile lesion which was not followed by a skin rash. No antisyphilitic treatment was administered. In 1917 he had had intestinal obstruction, but recovered without an operation.

On examination there was no enlargement of the heart, but a very faint aortic diastolic murmur was audible along the left border of the sternum when the breath was held after forced expiration. There was slight accentuation of the aortic second sound. The blood pressure was 125/70. The remainder of the physical examination was entirely negative. The Kahn test was strongly positive, and a diagnosis of angina pectoris due to syphilitic aortitis, with obstruction of the coronary orifices, was made.

The electrocardiogram (Fig. 1*A*) showed slight left axis deviation, not outside normal limits, and a heart rate of 88 per minute. After exertion sufficiently severe to induce mild anginal pain (Fig. 1*B*) the heart rate was approximately 130 per minute, and the electrocardiogram showed pronounced downward displacement of the RS-T junction in Lead II and less marked RS-T displacement in the same direction in Leads I and III. In Lead I, in which the T wave was originally sharply upright,

From the Department of Internal Medicine, University of Michigan Medical School.
*This study was aided by a grant made to F. N. Wilson by the Horace H. Rackham School of Graduate Studies. It was reported at a meeting of the Association of American Physicians, held on May 31, 1939. See Trans. Assoc. Amer. Phys. 54: 210, 1939.
Received for publication Sept. 1, 1940.

this deflection was flat or slightly inverted. In addition to these changes in the final ventricular deflection, there was a striking increase in the size of the S deflection in Leads II and III.

Nitroglycerine and aminophyllin were prescribed, and the patient reported on Feb. 25 that he was still having attacks, but that they were much less frequent and of shorter duration than formerly. Between Feb. 16, 1935, and April 10, 1935, eight intramuscular injections of bismuth salicylate (2 gr.) were given, and soon after these injections were begun, the patient stated that there was a slight increase in the severity of his symptoms.

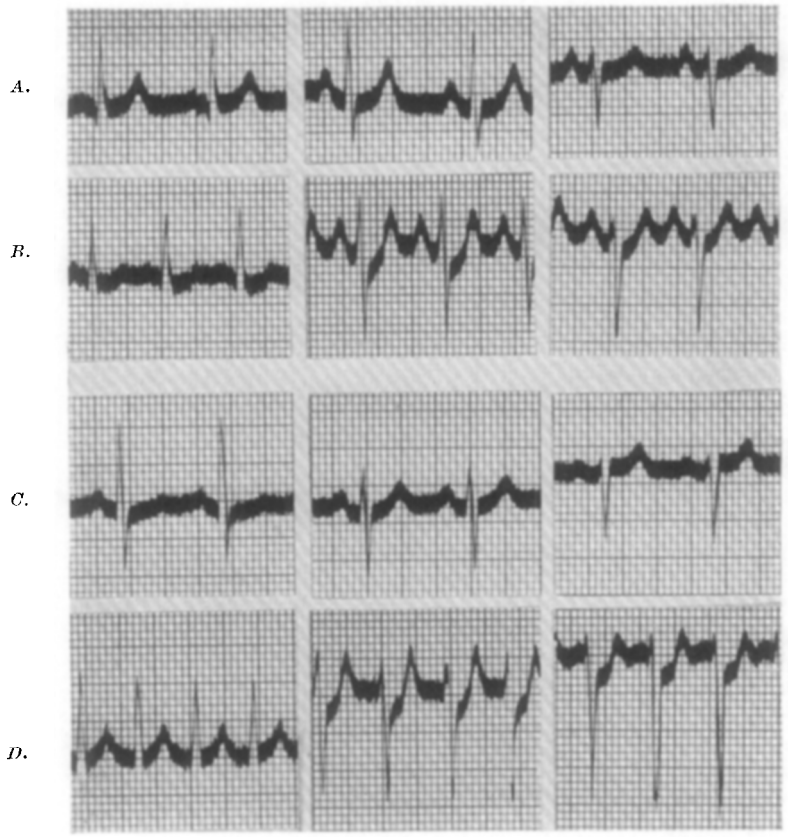

Fig. 1.—*A*, Case 1. Control electrocardiogram. *B*, Case 1. Electrocardiogram taken after exertion sufficiently severe to induce mild substernal distress. *C*, Case 2. Control electrocardiogram. *D*, Case 2. Electrocardiogram taken during a mild paroxysm of anginal pain induced by exertion. (Reproduced by courtesy of the Macmillan Company.)

On May 14, 1935, the patient's wife reported that he had died on May 4. On this date he went to the assistance of a woman who had fallen and broken her leg. The effort and excitement were promptly followed by a severe attack of chest pain, accompanied by dyspnea. These symptoms persisted until death a few hours later.

CASE 2.—Mr. O. S. G., a gas station attendant, 46 years old, was first seen on Feb. 27, 1928. At that time he was complaining of headache, shortness of breath, giddiness, and a sense of oppression beneath the sternum. He had been having headaches in the occipital region on the right side for eight or nine years, but they had been severe for only two years. The dyspnea, which occurred only on exertion,

and giddiness had been troublesome for one year. The substernal discomfort had occurred on two occasions only and consisted in a burning sensation beneath the surface of the chest. There was a history of gonorrhea many years before, and of three attacks of pneumonia within the preceding nine years.

On examination the heart was borderline in size, and no murmurs were heard. The aortic second sound was somewhat accentuated. The blood pressure was 216/146. The electrocardiogram showed slight left axis deviation. The patient was seen again on Aug. 26, 1929. At this time he stated that after his first visit he did not improve. The basal metabolic rate was measured elsewhere and was reported to have been plus 38 per cent. The administration of iodine and x-ray irradiation of the thyroid gland were followed by improvement, and he was able to return to work. Two weeks before this second visit he began to have attacks of rapid heart action. A tentative diagnosis of paroxysmal tachycardia was made at this time. The blood pressure was 140/80. The basal metabolic rate was -1 per cent.

The patient was seen for the third time on Jan. 15, 1935. He stated that for approximately two years he had felt fairly well, except that during the preceding four or five weeks he had begun to have a burning sensation beneath the upper sternum. This was associated with eructations of gas, but he did not feel distended. This sensation frequently occurred at night. He thought it was often brought on by exertion, but it sometimes lasted for fifteen minutes even if he remained quiet. He could sometimes walk a considerable distance without trouble, but one severe attack was brought on by shoveling snow, and he remained in bed for two days after this. The burning sensation was accompanied by constriction, and radiated to the ulnar side of the left arm.

The heart was not enlarged at this time, either to physical or roentgenologic examination. The aortic second sound was markedly accentuated. No murmurs were heard. The blood pressure was 210/125.

The electrocardiogram (Fig. 1*C*) which was taken while the patient was at rest showed slight left axis deviation, but was not outside normal limits. The heart rate was approximately 94 per minute. Following exertion sufficiently severe to induce mild substernal distress (Fig. 1*D*), the heart rate was 180 per minute, and there was pronounced downward displacement of the RS-T junction and segment in Leads II and III of the electrocardiogram. In addition, there were a very conspicuous change in the form of the T deflection in Lead I and a great increase in the size of the S deflections in Leads II and III. The P wave was not distinctly visible, but was apparently superimposed upon the end of the T deflection of the previous cycle because of the rapid heart rate and a slight prolongation of the P-R interval. Extrasystoles were noted after the exercise test, but were not recorded. About one-half hour later, shortly after an orthodiagraphic examination of the heart, the patient was found lying on the floor in one of the dressing rooms of the Department of Roentgenology. He was unconscious, pulseless, and extremely cyanotic, and was making gasping respiratory movements. He could not be revived.

CASE 3.—F. L. K., a physician, aged 66 years, was seen on Dec. 9, 1937. He stated that he had been well until the latter part of October of that year, when he began to have anginal pain. The first attack occurred while he was driving his car, and lasted about fifteen minutes. After this, exertion and excitement frequently induced attacks, and nocturnal attacks often awakened him from sleep. Cold increased the tendency to paroxysms. The pain was felt first in the muscles below and above the elbow joint, was bilateral, and was perhaps more severe on the right side. After it began in the arms, it was felt in the mid-chest on both sides of the sternum, but more on the left. It spread to the neck, jaws, and teeth, and even to the top of the head and as low as the waistline. It consisted in severe aching, with pressure and constriction. The longest attack lasted twenty minutes.

Nitroglycerine gave prompt relief. The first effect of this drug was to cut down the peaks of the waves of pain. The patient did not use tobacco. He said that his blood pressure had been a little high since his student days.

He was distinctly overweight. The heart was not enlarged. There was a faint, late, systolic murmur at the apex. The blood pressure was 160/98. The remainder of the examination was negative.

An electrocardiogram was taken, and this procedure was immediately followed by a spontaneous attack of angina. The first curve (Fig. 2*A*) shows slight left axis deviation, with partially inverted T deflections in Lead I and slight flattening of the RS-T segment in Leads II and III. The electrocardiogram which was taken at the height of the attack (Fig. 2*B*) shows pronounced downward displacement of the RS-T junction and segment and definite changes in the QRS complex, consisting in the development of a prominent S deflection in Lead II and a pronounced increase in the size of the S deflection in Lead III. The heart rate rose from approximately 70 per minute before the attack to approximately 100 per minute when the distress was at its height. In a later and similar spontaneous attack there was a rise in the systolic blood pressure from 160 mm. Hg to 180 mm. Hg. After nitroglycerine the pain subsided promptly (Fig. 2*C*), and the electrocardiogram regained its original form within fifteen minutes (Fig. 2*D*).

The patient died suddenly about one week after these observations were made.

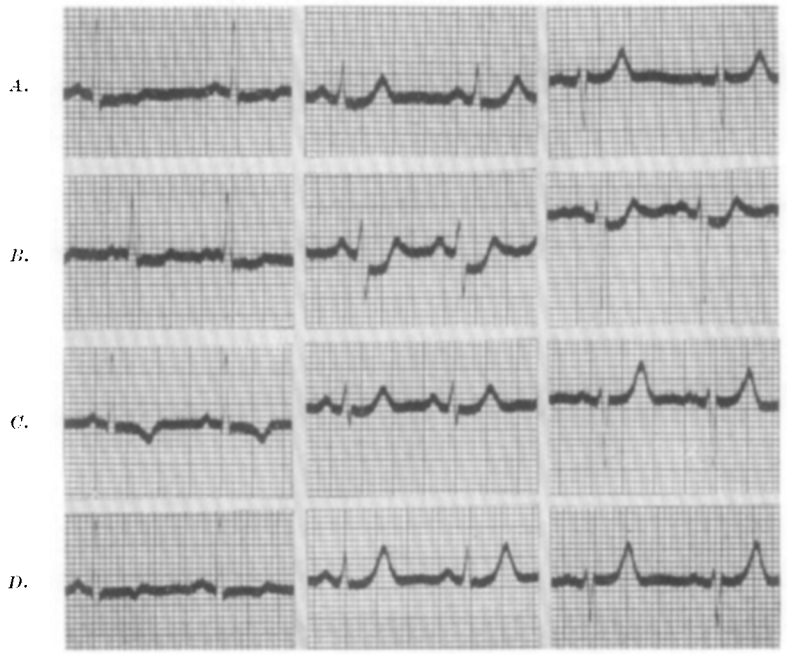

Fig. 2.—Case 3. *A*, Control electrocardiogram. *B*, Taken during a spontaneous attack of anginal pain. *C*, Taken five minutes after *B*, following the administration of nitroglycerine ($\frac{1}{100}$ gr.). *D*, Taken ten minutes after *C*.

Case 4.—Mr. T. McL., an electrical engineer, was first seen at the University Hospital on June 1, 1927. At this time he was complaining of epigastric pain which occurred two to three hours after meals. This distress was of three years' duration. A clinical diagnosis of peptic ulcer was made, and evidence of duodenal ulcer was discovered on roentgenologic examination. A suitable diet and alkaline powders were prescribed. The patient was not seen again until Dec. 5, 1938, when

he was admitted to the hospital. At this time he was 42 years of age. He stated that the gastric symptoms had eventually disappeared, and that he had been well until November, 1937, when he began to experience sharp, excruciating pain in the precordial region, with radiation to the left arm. These attacks of pain were precipitated at first by heavy lifting, and later by walking. They became more frequent and more severe until May, 1938, when the patient consulted a physician and was put to bed and told to reduce his consumption of tobacco to six or seven cigarettes per day. He had been in the habit of smoking twenty or more cigarettes daily. After he had been in bed for two days, the attacks ceased. He remained in bed for six weeks. After this he gradually increased his activity, and finally returned to work in September, 1938. After he had been working for several weeks, the anginal attacks suddenly returned in severe form, and since that time he had been unable to do anything involving exertion or excitement without distress. He stated that his blood pressure had been elevated for at least three years, but that the systolic pressure had never been above 190.

When he entered the hospital, the patient was having a great many attacks of pain each day, and many attacks at night, as well. Nitroglycerine gave prompt relief, and he was taking 40 to 50 tablets of the drug daily. The pain never lasted more than four or five minutes. It was followed by transient weakness of the left hand.

On examination the patient was slightly obese. The heart was borderline in size. The cardiac rhythm was normal, and no significant murmurs were heard. The heart sounds were loud, and there was slight reduplication of the first sound at the apex. The remainder of the physical examination was entirely negative.

The blood pressure was 165/110. Examination of the blood and urine, kidney function tests, and the Kahn test disclosed nothing abnormal. An orthodiagraphic examination of the heart showed questionable, slight enlargement.

A number of electrocardiograms were taken. All of those which were made when the patient was free of pain showed inversion of the T deflection in Leads II and III and slight downward displacement of the RS-T junction in all three limb leads (Fig. 3).

On the morning of Dec. 10, 1938, the patient was told by his physician to stop smoking. He did so, and had no more attacks of pain until Dec. 14, 1938. On this day he was brought to the Heart Station and was asked to smoke a cigarette. A single chest lead (Lead V_5) was taken before he began to smoke and was repeated at intervals thereafter. The control curve showed flat, inverted T waves and very slight downward RS-T displacement. A typical and severe anginal attack began within a few minutes after the patient started to smoke. It was accompanied by downward RS-T displacement of as much as 0.3 millivolt. The electrocardiographic changes began before the pain and outlasted it. Their exact duration was not ascertained. The heart rate rose during the attack from 77 per minute to 115 per minute. The blood pressure was not taken at this time. Another mild attack of pain occurred without obvious cause in the evening of the day of this experiment. On the following day the patient left the hospital. Further information as to the subsequent course of his illness is rather meager.

On Jan. 16, 1939, the patient's home physician wrote that he was still taking about 20 nitroglycerine tablets per day. He was able to walk from seven to fourteen blocks without pain, but had frequent, severe attacks in the early morning hours, some of which had lasted as long as thirty minutes in spite of nitroglycerin at five-minute intervals.

On Jan. 27, 1939, the patient himself stated that he had continued to have attacks, but that these were less frequent and milder than before, and that he had been able to shovel snow without symptoms.

CASE 5.—V. L., an American attorney, aged 50 years, was admitted to the University Hospital on Feb. 11, 1939. He stated that for the preceding 25 years he had frequently experienced a heavy feeling in the epigastrium, accompanied by pyrosis. These symptoms usually developed in the late afternoon, but sometimes came on fifteen to twenty minutes after a meal. They were promptly relieved by soda, which he was taking 3 or 4 times each day. In May, 1938, he began to have very brief attacks of substernal pressure, accompanied by pain high in the back and in both shoulders, with radiation down the outside of the arms as far as the elbows. These attacks were mild, and he paid little attention to them until late in November, 1938, when he had a severe attack in the evening while he was sitting quietly at home. A second attack occurred the same evening, and thereafter he had had attacks daily. In some attacks the pain in the arms was quite severe. Occasionally, there was pain in the left arm only. The distress never lasted more than three or four minutes, and it was never precipitated by excitement or by exertion. Most of the attacks occurred at night and awakened the patient from sleep. For relief he sat up or got out of bed and walked about his room. Nitroglycerine ($\frac{1}{150}$ gr.) relieved the discomfort at once. There was no dyspnea during attacks, but there was a transient feeling of weakness after they subsided. The intensity of the substernal distress and the intensity of the pain in the back and arms seemed to vary independently, and one sensation sometimes occurred without the other.

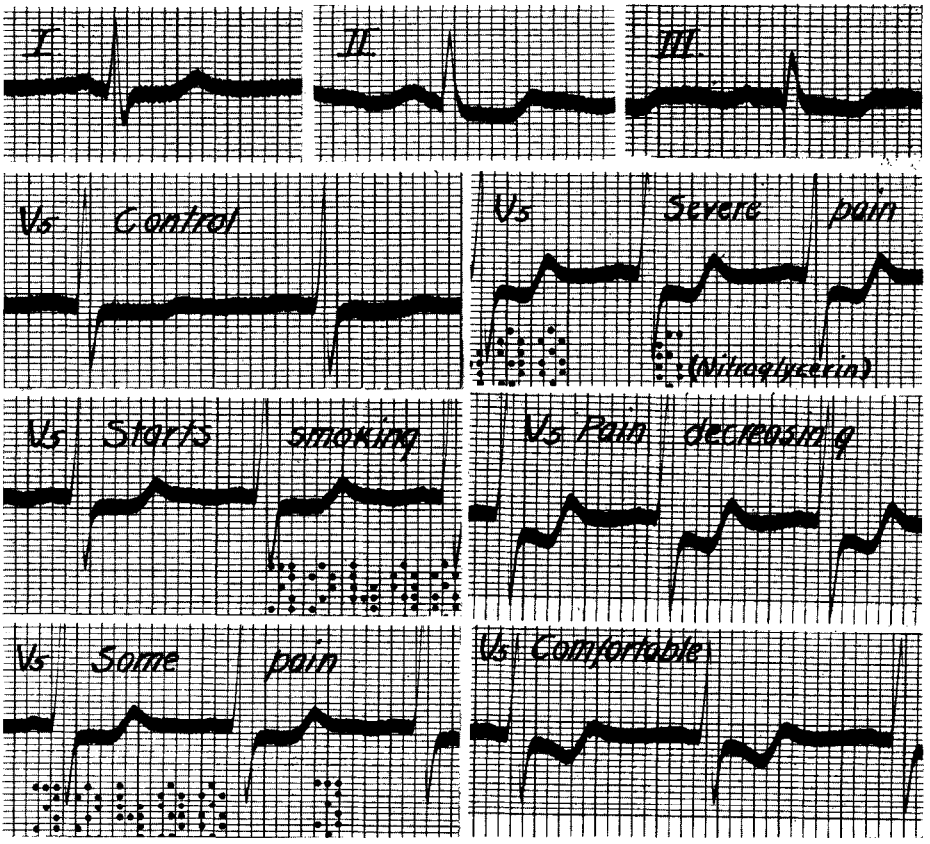

Fig. 3.—Case 4. Top row. Standard electrocardiogram. The remaining curves represent a single precordial lead (V_5), taken before, during, and after an attack of anginal pain induced by smoking and relieved by nitroglycerine.

The past history was negative except for whooping cough, measles, mumps, and chicken pox during childhood, and estivo-autumnal malaria in 1908, and again in

1910. The patient was in the habit of consuming about twenty cigarettes and two or three cups of coffee daily. He used alcohol in moderation.

On physical examination he was moderately obese, but no definite abnormality of any kind was discovered. The blood pressure was 160/100. The Kahn test, gastric analysis, the routine blood cell count, the estimation of the basal metabolic rate, and roentgenologic examination of the cervical spine, the heart and aorta, the gastrointestinal tract, and the gall bladder disclosed no abnormalities. The urine contained a very faint trace of albumin, and a few finely granular casts were found in the sediment. A number of electrocardiograms were taken while the patient was at rest, and one after mild exertion. All of these curves were considered well within normal limits. One of these electrocardiograms, taken on Feb. 11, 1939, is reproduced in Figure 4*A*.

While in the hospital the patient continued to have two or three of his attacks daily. Most of these attacks occurred between 7 P.M. and 8 A.M. Aminophyllin (0.2 Gm. q.i.d.) was given without noticeable effect upon the frequency of the attacks. On one occasion the blood pressure was taken during an attack and was found to be 200/120. There was a daily rise in temperature to 99° or 99.5° F.

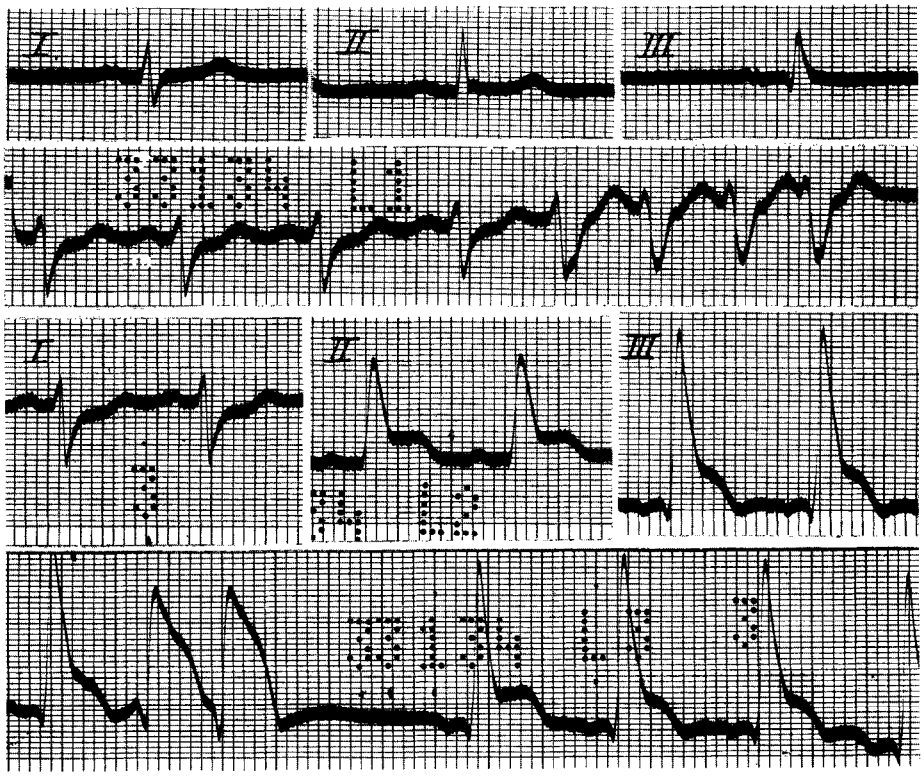

Fig. 4.—Case 5. *A*, Standard electrocardiogram. *B*, Lead I taken during the early stages of a spontaneous attack of anginal pain. *C*, Standard electrocardiogram taken at the height of the attack. *D*, Lead III, showing ventricular extrasystoles of monophasic outline.

On Feb. 18, 1939, a series of electrocardiograms was taken during a severe attack. The earliest curves of this series (Fig. 4*B*) show single ventricular extrasystoles and runs of ventricular extrasystoles constituting very brief paroxysms of ventricular tachycardia. In all of the electrocardiograms which were taken at the height of the distress (Fig. 4*C* and 4*D*), the QRS interval measures approximately 0.16 second, the chief initial deflection (R) is greatly increased in height in Leads II

and III, and the RS-T junction is greatly displaced from the isoelectric level. The RS-T displacement is downward in Lead I and upward in Leads II and III and resembles that seen in the early stages of infarction of the diaphragmatic wall of the heart. The extrasystolic ventricular complexes became practically monophasic as a result of the magnitude of this displacement of the junction of the initial and final deflections.

Following the administration of nitroglycerine the patient's distress disappeared promptly, and within a few minutes the electrocardiogram had practically regained its normal outline.

The patient was discharged from the hospital on Feb. 18, 1939, but was asked to return on Feb. 21, 1939, in order that further electrocardiographic studies might be carried out. A control electrocardiogram taken at 11:42 A.M. on this date shows no abnormalities (Fig. 5*A*). The heart rate at this time was 86 per minute, and the blood pressure was 138/88. The patient was then asked to smoke two cigarettes. When he had finished smoking, at 12:10 P.M., he complained of a slight burning sensation on the lateral aspect of the left upper arm; the heart rate was 94 per minute and the blood pressure was 140/88. The electrocardiogram (Fig. 5*B*) showed slight downward displacement of the RS-T junction in Lead I and pronounced upward displacement of this junction in Leads II and III. At 12:20 P.M. the electrocardiogram had returned to normal; the heart rate was 90 per minute and the blood pressure was exactly the same as at 12:10 P.M.

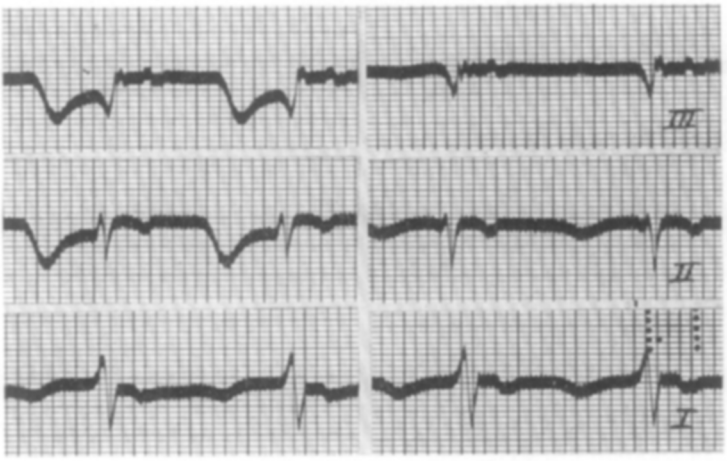

Fig. 5.—Case 5. *A*, Standard electrocardiogram before smoking. *B*, Standard electrocardiogram a few minutes after smoking two cigarettes.

A heavy luncheon had no effect upon the form of the electrocardiogram which was taken at 1:03 P.M., when the heart rate was 100 and the blood pressure 183/80. In the course of the afternoon it was observed that the electrocardiograms which were taken shortly after the patient had finished a cigarette did not always show RS-T displacement. He was then connected to a cathode-ray electrocardiograph so that the electrocardiogram might be observed continuously as a standing wave on the screen of the cathode-ray tube. It was then found that, while he was smoking, pronounced RS-T displacement came and went at frequent intervals. On each occasion it lasted fifteen to twenty seconds only. When he had finished smoking it soon disappeared permanently.

Because of these observations the patient was asked to discontinue smoking. Aminophyllin, erythrol tetranitrate, and quinidine, and a reduction diet were prescribed. He has continued to have attacks, but they have been less frequent and less severe. A study of his sensitivity to foreign proteins is being carried out.

DISCUSSION

The electrocardiographic changes which occurred during a spontaneous attack of anginal pain in Case 3 and those which occurred following exertion sufficiently severe to induce mild pain in Cases 1 and 2 are strikingly similar. In all three instances there was a pronounced increase in the size of the S deflection in Leads II and III and pronounced downward RS-T displacement in Leads II and III. Changes of this kind in the QRS complex have not, so far as we know, been observed in coronary occlusion. The RS-T displacement, however, is similar in magnitude and in kind to that seen immediately following sudden occlusion of the anterior descending branch of the left coronary artery. There is one difference. Following occlusion of the artery mentioned the RS-T displacement is usually definitely discordant, i.e., it is upward in Lead I and downward in Lead III. In the three cases of angina pectoris described, however, the RS-T displacement is either concordant (downward in all three leads) or so inconspicuous in Lead I as to make its classification as concordant or discordant difficult.

Electrocardiographic changes of the same kind, but even greater in magnitude, so far as the RS-T displacement is concerned, were recorded under similar circumstances by Scherf.[1]

The electrocardiographic changes which occurred during a spontaneous attack of anginal pain in Case 5 are of a different kind. Here the RS-T displacement is definitely discordant (downward in Lead I and upward in Leads II and III) and not distinguishable in type or in magnitude from that frequently seen immediately following infarction of the posterior or diaphragmatic wall of the heart. The changes in the QRS complex are striking, but difficult to classify. The great increase in the QRS interval indicates that a pronounced disturbance in intraventricular conduction occurred, but whether this was dependent upon the development of block in the right branch of the His bundle or upon a widespread depression of the specialized ventricular tissues is not clear. It should be noted that prominent Q deflections in Leads II and III, such as are commonly seen following infarction of the posterior wall of the heart, did not appear. Brow and Holman[2] recorded transient RS-T displacement of large magnitude and of this same type during a spontaneous attack of anginal pain. In that instance, however, there was a deepening of the Q deflection in Lead II immediately after the attack, and an electrocardiogram which was taken four months later strongly suggests that there was an infarct in the posterior wall of the heart at that time. As to when this infarction occurred the history gave no unequivocal clue.

The electrocardiographic changes which occurred in Case 4 during an anginal attack induced by smoking were recorded in Lead V_5 only. The

RS-T displacement is similar in magnitude and in kind to that seen in the early stages of infarction of the posterior wall of the heart, but no QRS changes are present.

The electrocardiographic changes described are similar in magnitude and in duration to those produced in animal experiments by temporary occlusion of one of the large coronary arteries. They indicate that the disturbances in the coronary circulation which occur during paroxysms of anginal pain are sometimes of very great magnitude. It is not surprising that sudden death, presumably from ventricular fibrillation, is a not uncommon event in angina pectoris, even when this condition is not complicated by coronary thrombosis.

It seems to be the prevailing opinion that the substernal discomfort and transient electrocardiographic changes which occur in anginal paroxysms are dependent upon myocardial ischemia brought about by an increase in the work of the heart, rather than by a change in the caliber of the coronary arteries affected. When, as in Case 5, pronounced electrocardiographic changes of the kind produced by temporary occlusion of a large coronary artery appear, disappear, and reappear without any material increase in heart rate or in blood pressure, this view is clearly untenable. It must, we think, be admitted that in some instances anginal paroxysms are precipitated by contraction of the coronary arteries involved. Whether contraction of the larger coronary arteries takes place, or whether it is the caliber of the arterioles that changes, it is not possible to say. The character of the electrocardiographic changes suggests that the change in arterial or arteriolar caliber is local, not general. The electrocardiographic changes which are attributed to myocardial ischemia and the subjective changes seem to vary independently both in magnitude and duration. This circumstance, together with the evidence that coronary spasm does sometimes cause anginal paroxysms, makes it seem possible that the stimuli which gave rise to the subjective sensations are of arterial rather than of myocardial origin.

The lack of parallelism between the magnitude of the electrocardiographic changes, and hence we may assume of the disturbances of the coronary circulation, and the work of the heart, as represented by the increase in heart rate, in blood pressure, or in both, was not, in our judgment, confined to Case 5, but also existed in Case 3 and probably in Case 4.

It has been clearly demonstrated that cigarette smoking produces constriction of the peripheral arterioles, both in healthy subjects[3] and in patients with angina pectoris.[4] Healthy young subjects may also display electrocardiographic changes immediately after smoking.[5, 6] Cases have frequently been reported in which precordial pain, not definitely anginal in character, was induced by smoking and disappeared promptly when this habit was given up. A few instances of this kind have come

to the personal attention of the authors. Instances have also been reported in which a patient subject to typical anginal paroxysms could induce an attack by smoking.[4] Cases 4 and 5 apparently belong in this category. It is our opinion that in both of these cases some part of the coronary arterial system was the seat of a disease process, presumably atherosclerotic in nature, and that the affected vessels were abnormally sensitive to nicotine or to some other constituent of cigarette smoke. The observations made in Case 5 strongly support this view. They do not, perhaps, establish it as correct beyond question.

SUMMARY

The pronounced electrocardiographic changes which sometimes occur during a paroxysm of angina pectoris indicate that the disturbance of the coronary circulation which occurs in this condition is at times as great as that produced by the sudden occlusion of a large coronary artery.

Attacks of anginal pain may occur which are accompanied by profound alterations of the electrocardiogram under circumstances which make it necessary to assume that the attendant myocardial ischemia is due to a change in the caliber of the coronary arteries affected, rather than to an increase in the work of the heart alone.

Nicotine or some other constituent of cigarette smoke sometimes induces coronary "spasm" in patients who are subject to angina pectoris.

REFERENCES

1. Scherf, D.: Koronarerkrankungen, Ergeb. d. ges. Med. **20**: 237, 1935.
2. Brow, G. R., and Holman, D. V.: Electrocardiographic Study During a Paroxysm of Angina Pectoris, AM. HEART J. **9**: 259, 1933.
3. Maddock, W. G., and Coller, F. A.: Peripheral Vasoconstriction by Tobacco and Its Relation to Thrombo-Angiitis Obliterans, Ann. Surg. **98**: 70, 1933.
4. Ralli, E. P., and Oppenheimer, B. S.: Changes in the Peripheral Circulation Accompanying "Tobacco Angina," Proc. Soc. Exper. Biol. and Med. **26**: 9, 1928.
5. Segal, H. L.: Cigarette Smoking: I. As a Cause of Fatigue. II. Effect on the Electrocardiogram With and Without the Use of Filters, Am. J. M. Sc. **196**: 851, 1938.
6. Graybiel, A., Starr, R. S., and White, P. D.: Electrocardiographic Changes Following the Inhalation of Tobacco Smoke, AM. HEART J. **15**: 89, 1938.