

which toxins and bacteria find their way into the system and through which other bacteria of the mouth may readily pass.

Concerning the cementum, it makes no difference whether the suppuration occurs at the gingival attachment or at the apex of the root, in which case, following the death of the pulp, the infection passes through the apical foramen or foramina into the tissues immediately surrounding the apex. Thus we see that these two chronic infections, namely pyorrhea and chronic alveolar abscess, both propagated by dead cementum, are the results of two factors, the first being a gingivitis, the second, following the death of the tooth pulp.

The first requisite in the conservation of healthy teeth and their investing structures is the avoidance of gingivitis, which is caused by numerous faulty conditions, among which are (1) salivary calculus and (2) serumal calculus, and which is due to (a) lack of contact of the teeth; (b) improper contact; (c) deviation from the normal, smooth contour of the teeth; (d) lack of cleanliness; (e) misuse of the toothpick, rubber-band, floss-silk or tooth-brush; (f) overhanging margins of fillings; (g) ill-fitting crowns, etc.

In a study made by Dr. Arthur D. Black of 500 mouths of young adults of an average age of 26 years, none of whom as yet had disease of the periodontal membrane, only 5 per cent. of them were free from gingivitis; in the remaining 475 there were 4,253 areas of gingivitis, an average of 8.53 for each person of the 500 examined.

Ninety-five per cent. of all persons examined had one or more areas of gingivitis, 55.1 per cent. of these being due to trauma, 37 per cent. of which were due to imperfect dental operations, less than one third due to salivary calculus and less than one sixth to serumal calculus.

Chronic suppurative pericementitis (pyorrhea) has its beginning in a gingivitis which may result from many causes; and, while serumal calculus beginning in the subgingival space may be the primary etiologic factor, it is usually a result rather than the cause, as within the pus pocket the calculus is deposited on the denuded root from the serum which constantly bathes the chronically infected tissues. Thus, we consider serumal calculus a cause if on the enamel, and as a result if on the cementum.

Pyorrhea is probably not due to any specific organism. The greatest achievements in medicine have been in the prevention of disease rather than in its cure, and so must the greatest dental service be attained by prevention rather than by restoration. This prevention must begin with the eruption of the temporary teeth and continue at intervals, depending on the necessity of each individual, through life.

It has been shown that gingivitis is the primary factor in pyorrhea, loss of gum tissue, loss of the alveolar process and finally loss of the teeth; consequently, it is the primary gingivitis which should not be allowed to continue and become chronic.

Physicians should be able to recognize inflammation of the gums as they recognize inflammation in other parts of the body. They can be easily taught to detect ill-fitting crowns, overhanging margins of fillings, salivary calculus and even serumal calculus in pus pockets, and should refer cases to the dentist who can remedy these defects.

Because of the insidious onset and chronic course, gingivitis is frequently neglected by the dentist and is not recognized by the physician until, from a toxemia

or development of an infection in some remote part of the body, the patient seeks relief, of his physician, at which time irreparable local damage has been done and distressing disease may have developed from the primary focus within the mouth.

Routine physical examination should include a more thorough oral examination, and the treatment of oral lesions should be based on a working knowledge of pathology.

In the vast majority of cases, neglected gingivitis becomes chronic, and its duration, over a period of months or years, not only leads to a destruction of normal tissues both soft and bony, but is a menace to health in two ways: (1) by the constant suppuration with absorption and swallowing of bacterial by-products, and (2) by the entrance of bacteria themselves into the lymph and blood streams, with their arrest in distant parts where they set up other foci of infection.

ABSTRACT OF DISCUSSION

DR. BYRON C. DARLING, New York: What Dr. Potts has said bears out what we are about to learn from the roentgen-ray examination of the teeth. If one will observe the alveolar margins carefully, one will see that the little entering wedge, the swelling of the pericementum, is present in many of the teeth. In fact, it seems almost universal. What Dr. Potts has said gives me an opportunity to know that the increase in width of the black line is a pathologic condition and is so recognized by the periodontist.

DR. F. B. MOOREHEAD, Chicago: The fibers of the periodontal membrane are attached through the agency of the cementoblasts and, whenever the cementoblasts are destroyed in an appreciable area, there is no hope of any reattachment of the periodontal membrane; the surface thus denuded becomes, in substance, foreign matter. Any repair that takes place under these circumstances is scar tissue and is not safe.

DR. HERBERT A. POTTS, Chicago: I wish to emphasize the importance of taking care of these infections and irritations which do become chronic. They cause little or no pain or inconvenience to the patient and, in the course of months or years, result in serious losses.

ANGINA PECTORIS

SOME CLINICAL CONSIDERATIONS*

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From time to time it is with some profit that we make an inventory of whatever information we can bring to bear on the study of a disease to see what progress has been made. In the study of heart disease it is unnecessary to dilate on the great aid in understanding the arrhythmias that has resulted from modern cardiography. The latter has thrown some light on diseases of the heart muscle, although there still is considerable vagueness and speculation concerning the relation of heart muscle disturbances to changes in the electrocardiogram. Likewise, the study of the vital capacity of the lungs and dyspnea of cardiac patients has afforded us some aid in following the course of our patients, in cataloguing them into various groups according to the degree of compensation, and to some extent in prognosticating as to the future course of

* From the Medical Clinic of the Peter Bent Brigham Hospital.

* Read before the American Climatological Association, Washington, D. C., May 2, 1922.

events. It is my personal judgment, although this point is disputed by some observers, that the introduction of the Wassermann test has taught us that syphilis of the heart is comparatively rare in clinical medicine except when the disease has involved the aorta or aortic cusps; i. e., in the absence of aortic insufficiency and aneurysm of the aorta. Turning our attention to angina pectoris, the question arises as to what progress has been made in the management of this extraordinary disease.

It is not the purpose in this study to discuss the theories as to the cause of angina pectoris or to give a complete clinical picture of the disease. Some points of interest that developed in the course of a study of 103 cases among patients admitted to the Peter Bent Brigham Hospital and seen in private practice will be taken up, with emphasis on those features that apparently were helpful in the clinical management of the problems that presented themselves.

TERMINOLOGY

There is a great deal of confusion in the literature and in the minds of many of us as to where to draw the line between angina pectoris and other cases of heart disease with precordial pain or even functional and vasomotor disturbances. It would be well if we could do away with such terms as false angina, pseudo-angina and juvenile angina. Unfortunately, at times it is the only recourse we have; but to use such vague terms only conceals our ignorance or uncertainty of the true state of affairs. It would be well for physicians to take the position that the diagnosis of angina pectoris is to be used in heart disease when it is meant that the patient has attacks of oppression or constriction or pain in the chest, generally in the region of the sternum (not particularly in the precordium or apical region), having a more or less characteristic distribution and radiation, especially related to effort and associated with some other symptoms, such as mental apprehension, fear of death, temporary dyspnea, immobility and vasomotor disturbances such as pallor and perspiration. It must also mean that the patient whose condition is so diagnosed is likely to die suddenly, either with little warning or after only a short illness. It is important to keep the latter connotation in mind, for other types of heart disease have repeated periods when the patient is confined to bed with increasing degrees of cardiac failure and the accompanying signs of decompensation. But the latter patients are likely to die in bed, not unexpectedly but after a lingering illness. Occasionally angina pectoris accompanies valvular disease or chronic myocarditis, and the clinical picture is thus complicated. If this attitude is accepted, there will be less confusion in our terminology. The expression, "This patient has a little angina" should mean that the diagnosis is angina pectoris, but that the pain, not the disease, is slight; for the patient with only a slight amount of pain may be dead of angina in a few days; on the contrary, another patient who has severe precordial pain may not have angina at all and may live for a great many years.

ETIOLOGIC FACTORS

The average age of this group of patients was 54.1 years; that of the males was 54.3, and of the females, 53.6. It is significant that patients with angina pectoris are comparatively more often encountered in general practice than in hospital wards, for many of them are

ambulatory and do not consider themselves sick and, what is more important, are regarded rather lightly by the attending physician. To include none but hospital cases might give wrong impressions, because it seems that while women with "minor complaints" frequently reach the public wards of hospitals, men do so only reluctantly, as they try to continue at their work. Of these 103 patients there were sixty-eight males and thirty-five females, i. e., almost twice as many of the former as of the latter. This sex discrepancy is striking and deserves some explanation. It is especially significant when we consider that vascular hypertension does not affect males as often as females; in point of fact, it is much more common in women. Dr. J. P. O'Hare,¹ who has studied this disease in a large number of patients, finds that of 153 cases of vascular hypertension, 64.7 per cent. were in females and 35.3 per cent. in males. Appreciating that some hypertension is commonly associated with angina pectoris, there must be some other factor that turns the proportion in favor of males. A point of great importance in this connection, I believe, consists in physical work and muscular strength. Men are physically stronger than women and, in general, work harder. It is unusual to find angina pectoris in weak, feeble, underdeveloped or undernourished persons. Many of them are somewhat overweight and, what is more important, most of them are well set, sturdy and of the muscular type. I have been struck by the good physical development of most of the patients with angina, and with the excellent history as to general health and freedom from minor ailments. This has been so striking that it has seemed to warrant a place among etiologic factors. The weak do not have angina; it is rather the well and sturdy.

What specific diseases stand in any etiologic relation to angina pectoris? Syphilis has generally been given the place of first importance. I believe that this point is overemphasized. To be sure, it is often difficult to determine whether or not syphilis is the cause of any chronic condition. The history obtained from the patient is unreliable. The postmortem findings are often either uncertain or disputable, even when the disease may be present. At times outspoken evidence of syphilis is found, particularly when there are typical changes in the aorta or in the central nervous system. In general, however, the Wassermann reaction of the blood will come as near to the truth as any other means we have, and can fairly be used for purposes of this study. Of the eighty-one patients on whom this test was made, the reaction was positive in only six, i. e., 7.4 per cent. Thus, it follows that in about 7 per cent. of these cases syphilis was the cause of angina pectoris, and therefore the disease plays a relatively unimportant rôle as an etiologic factor. However, it is imperative to watch for this condition, for, if discovered in its early stages before the aorta and aortic valves have become extensively involved and distorted, anti-syphilitic treatment may promise more than if there was no specific condition to treat. The presence of a diastolic murmur, heard best at the base of the heart, should always be carefully watched for and, if present, make one investigate more carefully for syphilis.

Among other conditions that stand in some etiologic relation to angina pectoris is diabetes. Its frequency is probably greater than is generally appreciated. Of

1. O'Hare, J. P.: Personal communication to the author.

the 103 patients in this series, seven had diabetes. Gout has long been regarded as a possible precursor of angina, and this was found to be so in five patients. There were two with hypertrophic prostate, but this probably is no more than the accidental incidence of two diseases in the same individual. The frequency of organic valvular disease associated with angina is not very striking. There were two instances of typical mitral stenosis, one of which came to postmortem examination and was there verified. This patient died very suddenly. In addition, there were two patients with rheumatic aortic insufficiency, one having typical signs of aortic stenosis as well. Both of these patients were young, 19 and 29 years old, respectively, although they suffered attacks that were typical of angina pectoris. A third patient was a woman of 48 who had typical anginal attacks and showed signs of aortic insufficiency. The Wassermann reaction was negative, and there was no history of rheumatic fever. This may have been an instance of syphilis, notwithstanding the negative serum reaction.

By far the most common and frequently the only important accompanying condition is arteriosclerosis. Unfortunately, no careful tabulation as to the comparison of the frequency of arteriosclerotic changes in the various parts of the body is possible from this study. It would be instructive to know whether angina occurs in those patients with large blood vessel sclerosis or with changes in the finer vessels. O'Hare² has recently been studying this question with relation to vascular hypertension, and has found that simple hypertension is most commonly associated with sclerosis of the retinal arteries rather than with the large vessels such as the radial or brachial, and that the patients with the lower blood pressure had more sclerosis of the large vessels and less of the finer ones. It is my opinion that, since angina pectoris generally occurs with a moderate elevation of pressure, sclerosis of the large blood vessels is associated with it rather than sclerosis of the finer ones. Certainly most patients that I have examined with angina have shown few changes in the retinal vessels as compared to those with vascular hypertension. The majority of the patients in this series had definite sclerosis of the radial arteries, although there was a smaller group in whom the peripheral blood vessels did not seem to be very striking.

BLOOD PRESSURE OBSERVATIONS

A study of the blood pressure in this series has brought out some interesting figures. The average systolic pressure of ninety-nine patients was 160.6 mm., and the diastolic was 95.0 mm. The lowest systolic was 80 mm., and the highest, 260 mm. Most of them were between 140 mm. and 200 mm. There were sixty-four of such patients in this series. Eleven had a pressure over 200 mm., and twenty-four under 140 mm. Several of those who had a low pressure died in the attacks while under observation or shortly after it, and may well have had a higher blood pressure before the fatal attack. Some years ago, with Tranter,³ I reported two fatal cases of infarction of the heart with coronary thrombosis, in both of which there was a strikingly low blood pressure. In some others of this series with low

blood pressure undoubtedly the same pathologic condition was present. There were several instances, however, of normal blood pressure, i. e., from 120 to 140 mm., in patients suffering from typical angina who gave no evidence of such a serious complication as infarction of the heart.

Some observations on the blood pressure were made in relation to the attacks of angina pectoris and the administration of nitroglycerin. One patient just before an attack had a systolic blood pressure of 140 mm. and a diastolic pressure of 90 mm. During the attack it rose to 180 mm. systolic and 120 mm. diastolic, and then at one minute intervals after the administration of nitroglycerin by mouth the readings were 184/110, 158/90, 150/90, 140/90, 140/90 and 140/90. Another patient, who had a blood pressure of 110 mm. systolic and 70 mm. diastolic, showed an increase to 164 mm. systolic and 94 mm. diastolic during an attack; 0.9 mg. of nitroglycerin was then given by mouth, and at two minute intervals the readings were 155/90, 140/88 and 132/88. In a third patient, just before the attack the blood pressure was 162 mm. systolic and 88 mm. diastolic; during the attack at 7:09 p. m. before any medication was given it was 154 systolic and 78 diastolic. Then 0.0003 gm. of nitroglycerin was given at 7:11 p. m. The blood pressure was 148 systolic and 76 diastolic, and the pulse 100. Another tablet was given, and at 7:15 p. m. it was 140 systolic and 82 diastolic, and at 7:18 p. m. it was 138 systolic and 84 diastolic and the pulse was 100; at 7:20 p. m. the blood pressure was 144 systolic and 82 diastolic; the third tablet was given, and the blood pressure was 142 systolic and 78 diastolic; this was at 7:23. Finally, a fourth tablet was given, and the blood pressure was 140 systolic and 78 diastolic at 7:25 p. m. By this time the pain had disappeared. A fourth patient who had a blood pressure of about 200 mm. for some years was suddenly taken with a severe attack of angina pectoris. When seen shortly after the attack had begun, the systolic pressure was 160 mm., and during that day the pressure gradually fell to 120 systolic and 80 diastolic. On the following day it was 110 mm. He died twelve days after the onset of this attack. The observations on the first two patients disclosed a distinct increase of the blood pressure, with the development of an attack of angina and a fall in the pressure as nitroglycerin made the attack subside. The third case showed no appreciable change with the onset of the attack, but the pressure diminished somewhat as the attack subsided after nitroglycerin had been given. It is difficult to say from these observations whether the subsiding of the attack caused a fall in pressure or the reverse. The latter view seems the more likely, and probably any other factor that might reduce the blood pressure could end the attacks.

What general conclusions can be drawn from this study of the blood pressure in angina pectoris? It is evident that any pressure from subnormal to a marked hypertension may exist in patients suffering from angina. The extremely high pressure of from 200 to 250 mm., although fairly common in adult age, occurs in only about 10 per cent. of the patients with angina. Some of these patients subsequently develop a lower blood pressure, and therefore a good deal will depend on the stage of the illness during which observations are made. Furthermore, the low blood pressure needs to be interpreted carefully. Some of the patients turn out to be young persons who have rheumatic aortic dis-

2. O'Hare, J. P.: Paper read before the Interurban Club meeting, Boston, April 7, 1922.

3. Levine, S. A., and Tranter, C. L.: Infarction of the Heart Simulating Acute Surgical Abdominal Conditions, *Am. J. M. Sc.* 155:57 (Jan.) 1918.

ease as the underlying cause of the angina pectoris, while another group contains those patients who are seen in an attack of closure of a coronary vessel. In the latter, the pressure may be very low, 90 mm. or less, while the patient appears to be in a fair general condition.

EXAMINATION OF THE HEART

On examining the heart, one is generally impressed by the absence of significant findings in most instances of angina pectoris, and yet it is this negative state of affairs that is very important and makes us be on our guard. Frequently the differential diagnosis is between a simple minor complaint such as indigestion, constipation or neurosis, and this most deadly disease. The heart is generally enlarged, slightly or moderately, rarely markedly. Not infrequently, on percussion, no evidence of enlargement could be made out. In some of these cases the roentgen ray or the electrocardiograms disclosed hypertrophy that was previously overlooked. In rare instances all methods failed to reveal any appreciable enlargement of the heart.

On auscultation, frequently no murmurs were heard. In this series of 103 cases, forty-seven showed no murmurs. There were forty-nine in which a systolic murmur was heard at the apex or the base of the heart or at both points. The systolic murmur heard in the forty-nine cases just referred to did not indicate a real endocarditis. Seven patients had diastolic murmurs. Two of these were definitely, and one probably syphilitic, and four had rheumatic valvular disease. Of the latter group three had rheumatic endocarditis of the aortic valve, and one of the mitral and tricuspid. It follows from these figures that valve disease in angina pectoris is unlikely unless there is in addition a rheumatic history of a syphilitic process.

The rhythm of the heart remains for the most part undisturbed in cases of angina both during attacks and during the intervals between attacks. In a small number of cases there may be heard an occasional extrasystole; but the dominant rhythm is maintained. During the more serious cases of coronary infarction, heart block may result, as was described in a previous publication,³ and at times paroxysms of tachycardia.⁴ It was quite surprising that out of 103 patients in this series only one had persistent auricular fibrillation, and yet these two types of heart disease affect individuals at the same average age. There was one patient who had transient auricular fibrillation, and in another it was a terminal event. During the same period covered in this study, 423 heart cases of persistent auricular fibrillation were observed, and about 200 of these were observed in elderly persons and were associated with chronic myocarditis, i. e., not of the rheumatic valvular type.

It seems most extraordinary that of these 200 persons suffering from chronic myocarditis with auricular fibrillation, not one had angina pectoris. The single instance of auricular fibrillation and angina that did occur was in a patient with rheumatic mitral stenosis. This incompatibility between the two conditions is of distinct practical aid. If a patient has chronic heart disease and auricular fibrillation, he is extremely unlikely to develop angina pectoris; and likewise, those with angina pectoris virtually always have a dominant regular heart rhythm.

The quality of the heart sounds is of some interest to us, particularly with regard to the first sound at the apex. In many instances this was distinctly diminished in intensity, and occasionally it was absent. In interpreting the loudness of heart sounds, due consideration must be given to the thickness of the chest wall and the amount of lung tissue in front of the heart. When sounds are distant as a result of obesity or overlying breasts, both sounds are affected; but when the second heart sound is distinct and the first muffled or barely audible, some intrinsic myocardial change is most likely the cause. In forty-nine patients, the first heart sound was considered to be diminished in intensity, while in the remaining fifty-four it was not diminished. In the group of cases with cardiac infarction to be discussed below, this feature of the physical examination was rather important. In four instances a distinct gallop rhythm was heard.

The signs of edema in various parts of the body were striking by their absence. In only thirteen cases was there any pitting edema of the legs, and in most of them it was very slight. The liver was palpable in seventeen cases, but with a very few exceptions there was no tenderness, and the edge descended only from 1 to 2 cm. below the right costal border. Râles were often found at the bases of the lungs. There were thirty-one instances in which râles were present. It is evident that not all of the patients actually had congestion from an impaired circulation, as in some there was an associated bronchitis.

ELECTROCARDIOGRAPHIC OBSERVATIONS

Recently there appeared a lengthy study of the electrocardiographic changes accompanying angina pectoris, by Willius.⁵ In general, he showed that various abnormalities in the form of the electrical complex might occur. From the point of view of aiding in the diagnosis of angina pectoris, most of the changes that were observed do not help, as they occur in other persons who do not have angina. Much more study concerning the significance of changes in the Q-R-S complex (the initial ventricular complex) and the form and direction of the T wave (the final ventricular deflection) will be needed before any conclusions can be made that are based on secure evidence. There are, however, several points that at present seem suggestive and helpful. A distinct spreading of the Q-R-S complex was found in eleven patients of this series. It is generally accepted that this indicates a defective conduction of impulses through the musculature of the ventricles, and is commonly found in chronic myocarditis. A second type of change in the electrocardiograms is a marked diminution in the height of the curves in all leads. This occurred in two instances, and both patients died several days after the tracings were taken. The diagnosis of both of these patients was infarction of the heart from coronary occlusion. One of the two was examined postmortem, and the diagnosis was confirmed. A large number of the patients showed electrocardiographic preponderant hypertrophy of the left ventricle, and many showed decided inversion of the T wave in one or more leads. It is premature to attach too great importance to inversion of the T wave, as it is observed in other types of patients, and yet some of the curves make one strongly suspect myocardial or coronary disease. Finally, there are a number of patients who show

4. Robinson, G. C., and Herrmann, G. R.: Paroxysmal Tachycardia of Ventricular Origin and Its Relation to Coronary Occlusion, *Heart* 8: 59 (Feb.) 1921.

5. Willius, F. A.: Angina Pectoris: An Electrocardiographic Study, *Arch. Int. Med.* 27: 192 (Feb.) 1921.

essentially normal tracings or who show only minor changes. It is evident that conclusions from electrocardiograms must be made with caution. Those abnormalities which indicate myocardial disturbances may throw enough weight in the balance to decide a puzzling diagnosis, and it is quite likely that in the near future more accurate data will be available for clinical purposes.

OBSERVATIONS ON THE VITAL CAPACITY OF THE LUNGS

In recent years the vital capacity of the lungs (the greatest amount of air that can be expired after a full inspiration) has been used as a guide to the progress of patients with heart disease. Peabody and Wentworth⁶ have found this to be more or less diminished in heart disease, and also that in the same patient with an improvement in the circulation, as a result of treatment, the vital capacity of the lungs is increased. Similar determinations were made in fifteen of the patients in this series, but the standards used in Peabody's work could not be used for comparative purposes. The vital capacity diminishes even in normal persons with advancing years; and since the patients

VITAL CAPACITY OF THE LUNGS IN ANGINA PECTORIS *

| Case | Sex† | Age, Yrs. | Ht., Cm. | Wt., Kg. | Surface Area, Square Meters | Normal Vital Capacity, C.c. | Actual Vital Capacity | Percentage Normal |
|-------------------|------|-----------|----------|----------|-----------------------------|-----------------------------|-------------------------|-------------------|
| 1. S. C. | ♂ | 64 | 164 | 65.7 | 1.73 | 4,330 | 3,200 | 74 |
| 2. B. B. | ♂ | 63 | 162 | 70.3 | 1.77 | 4,430 | 3,150 | 71 |
| 3. A. R. | ♂ | 63 | 160 | 56.8 | 1.59 | 3,980 | 3,200 | 81 |
| 4. C. H. F. | ♂ | 61 | 170 | 91.6 | 2.03 | 5,040 | 3,500 | 69 |
| 5. M. G. | ♂ | 56 | 165 | 63.5 | 1.70 | 4,250 | 3,000 | 71 |
| 6. M. S. | ♂ | 55 | 164 | 74.0 | 1.82 | 4,550 | 1,750 | 38 |
| 7. C. J. S. | ♂ | 54 | 152 | 65.3 | 1.57 | 3,930 | 1,900 | 48 |
| 8. M. U. | ♂ | 52 | 160 | 71.6 | 1.75 | 4,375 | 2,900 | 61 |
| 9. S. K. G. | ♂ | 45 | 170 | 81.6 | 1.93 | 4,825 | 3,375 | 70 |
| 10. H. A. B. | ♂ | 23 | 187 | 64.0 | 1.87 | 4,675 | 1,550 2,150 3,100 | 33 46 66 |
| 11. E. I. S. | ♀ | 62 | 168 | 71.6 | 1.80 | 3,600 | 2,000 | 56 |
| 12. E. S. | ♀ | 57 | 168 | 77.2 | 1.86 | 3,720 | 2,500 | 67 |
| 13. M. F. S. | ♀ | 57 | 156 | 81.0 | 1.80 | 3,600 | 1,300 | 36 |
| 14. B. B. | ♀ | 52 | 158 | 68.0 | 1.72 | 3,440 | 2,200 | 64 |
| 15. B. E. B. | ♀ | 45 | 170 | 83.6 | 1.95 | 3,900 | 1,450 | 37 |

* Some of these observations were made by Dr. J. H. Pratt of Boston.

† In this column, ♂ denotes male, and ♀ female.

we are considering are a great deal older than those used in previous standards, no accurate figures could be deduced. This will be impossible until a large series of normal elderly persons are studied and their vital capacity determined. The accompanying table gives the figures of fifteen cases in which observations were made. The percentage of normal was figured according to West's⁷ method, the surface area first being determined, and the result being compared with his standards, which were made on younger persons. In general, the vital capacity was diminished, but probably not as much as the figures would indicate if we could bring to bear the age factor. An interesting feature is that the vital capacity did not increase much, if at all, under treatment in the few that were followed. The only patient who showed a decided rise in the vital capacity had rheumatic valvular disease. This would indicate that only when there is the congestive type of cardiac failure accompanying angina pectoris is much increase in the vital capacity to be expected, even when the patient improves.

6. Peabody, F. W., and Wentworth, J. A.: Clinical Studies on the Respiration, IV, The Vital Capacity of the Lungs and Its Relation to Dyspnea, Arch. Int. Med. 20: 443 (Sept.) 1917.

7. West, H. F.: Clinical Studies on the Respiration, VI, A Comparison of Various Standards for the Normal Vital Capacity of the Lungs, Arch. Int. Med. 25: 306 (March) 1920.

INFARCTION OF THE HEART IN RELATION TO ANGINA PECTORIS

One feature of this study remains to be considered, and this concerns itself with the importance of infarction of the heart in relation to angina pectoris. Sir Clifford Allbutt has for a long time taught that angina is always due to an aortitis and has nothing to do with the heart itself. The other more common view is that it is due to coronary disease. The outstanding fact that impresses one who studies the heart in the necropsy room is that almost every patient with angina has more or less recent infarction of the heart due to coronary closure. It seems to me that although an aortitis may well be the underlying cause of the attacks of pain, the prognosis depends on the condition of the coronary arteries. In other words, patients with angina in the great majority of instances live until a coronary artery becomes occluded. Occasionally, even after such an accident occurs, recovery takes place and the patient subsequently succumbs to another infarction. It therefore becomes imperative in any given attack of angina to try to decide whether there has been a closure of a coronary vessel.

Recently J. T. Wearn⁸ has studied all the cases of cardiac infarction that came to necropsy at the Peter Bent Brigham Hospital, many of which I had the opportunity of seeing during life. His observations contain many points of practical importance. The attacks of constriction and pain that are associated with cardiac infarction are likely to last much longer than the ordinary attacks of angina. They may continue for many hours, and even a week or two. The picture is that of a patient in collapse. The pain is not likely to be relieved by nitrites, and even morphin may give only little relief. The pulse becomes small and thready, and the blood pressure falls quite markedly. Frequently the liver enlarges and may become tender. There are almost always râles at the bases of both lungs. Very commonly the temperature becomes elevated to 100 or 102, and an appreciable leukocytosis even as high as from 20,000 to 30,000 may develop. The patient can lie flat in bed and yet complain of difficulty of breathing. On rare occasions there is an icteric tint to the sclerae. Death is not likely to occur at the instant of coronary closure but rather a variable time afterward—from a few days to a week or two. In some cases the infarcted area of heart muscle eventually ruptures and the end comes suddenly, even when the patient may seem to be doing favorably. In other instances there is no rupture of the heart, and death comes more gradually, with signs of marked asthenia. Rarely the infarct heals and recovery takes place.

There is a group of cases of angina that includes some of those with cardiac infarction, in which the symptoms are practically all abdominal. Some years ago Tranter and I³ called attention to two such cases that resembled acute surgical abdominal conditions. There is little doubt that some patients are operated on for gallstones, gastric ulcer, acute pancreatitis, etc., who are really suffering from coronary disease. The difficulty in diagnosis becomes great when the findings are severe abdominal pain, spasm, tenderness in the upper abdomen, vomiting, collapse, fever and leukocytosis. If a patient is known to have had an elevated blood pressure and is found to have a normal or sub-

8. Wearn, J. T.: Coronary Thrombosis with Infarction of the Heart, Am. J. M. Sc., to be published.

normal blood pressure, cardiac infarction must be seriously considered. The quality of the heart sounds at the apex may help in differentiating the cardiac from the abdominal condition, for in attacks of coronary disease, the heart sounds are likely to become quite faint, particularly the first heart sound. Occasionally a pericardial friction sound develops. I have seen several patients in whom confusion between acute surgical conditions and angina pectoris arose. One had recently been operated on for gallstones, and none were found; a surgeon was called for another patient to operate for an acute intestinal obstruction. The surgeon hesitated and the patient recovered his customary health, but died suddenly a few months later. This condition will probably explain some of the negative laparotomies performed on middle aged or elderly persons.

SUMMARY

A clinical study of 103 cases of angina pectoris was made, on which were based the following observations:

The disease is much more common in males than in females, while vascular hypertension, which plays an important rôle in the etiology of the disease is, on the contrary, more common in women. I believe that physical work may account for this discrepancy. The typical patient with angina is a strong, muscular and well set person. The disease seems to be comparatively rare in the underdeveloped, poorly nourished and weak.

In this series, syphilis was an etiologic factor in only six patients. This would indicate that the importance of syphilis as a cause of angina pectoris has been over-emphasized. There were seven who had diabetes mellitus, and five who had gout. Organic valvular disease was rarely found associated with angina pectoris. Two patients had typical mitral stenosis, and there were two young patients with rheumatic disease of the aortic valves.

The average systolic blood pressure was 160.6 mm., and the diastolic, 95.0 mm. The variations from the mean systolic pressure extended from 80 to 260 mm. About two thirds were between 140 and 200 mm. Observations on the blood pressure in a few cases were made in relation to the onset of the attack of angina and the administration of nitroglycerin. Two patients showed a marked increase with the onset of the attack, and then a fall as the attack subsided after the nitroglycerin had been given. In a third the change was not significant either as the attack developed or as it subsided under treatment.

Examination of the heart in many cases was negative. Of these 103 patients, forty-seven showed no murmurs, forty-nine had a systolic murmur at the apex or base, and seven had a diastolic murmur. Of the latter group, two had definite and one probable syphilitic aortitis, three had rheumatic aortic insufficiency, and one had mitral and tricuspid stenosis. The heart was generally found hypertrophied on ordinary examination or by orthodiagraphy or electrocardiography. An occasional patient showed no hypertrophy even after the most careful search. The dominant rhythm of the heart in angina pectoris was regular, although there were occasional extrasystoles. Only one out of 103 showed persistent auricular fibrillation. This is particularly striking, because during the same period of time persistent auricular fibrillation was observed in about 200 other patients suffering from chronic myo-

carditis, who in general were of the same age as those with angina. There therefore seems to be a distinct incompatibility between angina pectoris and auricular fibrillation. Patients with auricular fibrillation are not likely to have or to develop angina pectoris; and, vice versa, those suffering from angina do not develop auricular fibrillation.

The quality and loudness of the heart sounds are of practical importance in angina, especially the first heart sound at the apex. The diminution of the intensity of the apical first sound may be of aid in diagnosis and prognosis. Edema of the limbs and swelling of the liver are rare, but congestive râles at the bases of the lungs are common, especially in those patients with cardiac infarction.

The electrocardiograms were occasionally normal in appearance, and frequently showed only slight alterations. There was evidence of left ventricular preponderant hypertrophy in the great majority of cases, and many had inverted T waves in one or more leads. Eleven showed a distinct spreading of the Q-R-S complex, and in two fatal cases the amplitude of all curves was greatly diminished.

The vital capacity of the lungs was determined in fifteen patients. As there are no satisfactory standards of elderly persons with which to compare, no final conclusions could be made. Using the customary standards that apply to younger patients, the figures obtained in this study showed a decrease of varying extent in all instances. They did not show an increase in the vital capacity when they improved under treatment as did other cardiac patients, except in the one instance in which there was an added factor of cardiac failure with congestion.

It is important to distinguish attacks of cardiac infarction from those of ordinary angina pectoris, for where the prognosis of the former is generally only days, that of the latter may be years. The patient having cardiac infarction shows the picture of shock and is cyanosed; the pain may last many days, and is not relieved by nitrites, or even at times by morphin; the pulse is likely to be a little rapid and small; the systolic pressure falls sometimes as low as 90 or 80 mm.; râles develop at the bases of the lungs, and the liver may become engorged and tender. A leukocytosis of 20,000 or more frequently develops. Occasionally the violent pain is localized in the upper abdomen, and there may be nausea, vomiting and marked rigidity of the muscles in the epigastrium. There are therefore some patients with cardiac infarction who present the typical picture of an acute surgical abdominal condition.

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An Accidental Death Every Six Minutes.—Seventy-six thousand accidental deaths—a life every six minutes—is the toll paid by Careless America during 1920, according to the report of the National Safety Council presented at the Eleventh Annual Safety Congress, which opened in Detroit, August 28. The 1920 toll of accidents represents the total population of the state of Nevada, and while it is a decrease of 3,300 over 1911, it is only 400 less than the 1919 total. The automobile fatality frequency for 1920 was 30 deaths a day, a total of 11,000 for the year. People died from falling accidents of all kinds at the rate of thirty-four a day. Burns claimed twenty-two lives a day, a total for the year of 8,088. Railroad accidents showed no decline as there were 7,769 such deaths. Over twice as many men died accidentally as did women.