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ON

ANGINA MINOR.

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BY

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ANGINA MINOR is a condition of some practical importance, and I trust that its consideration will help to crystallize our ideas; for there is still, in spite of much writing and talking, a certain haziness as to the significance of some of the pains and discomforts located in the precordial and substernal regions.

Up to December 31st, 1925, I have careful notes of 472 patients suffering from angina (309 up to July 31st, 1925, and 163 to December, 1925), mostly private patients; and it is on a careful scrutiny of these records that I base my remarks.*

One outstanding feature of the study of medicine to-day is the much greater attention paid to symptoms than to the physical signs of disease. In the latter part of the nineteenth century it was the physical signs of heart disease which occupied the minds of the profession, and the aim was to correlate the physical signs with the gross pathological changes present. This attitude dominated clinical investigation. Now with the swing of the pendulum the cross-examination of a patient is often considered even more important than the physical examination, and both are made with the object of obtaining a clear conception of the functioning power of the organ at fault, whether that organ be the heart, the kidneys, the pancreas, the liver, or any other viscus. For an organ may be gravely inadequate and yet present few, if any, objective signs of disease. It is here, in the assessing of the functioning power of the kidneys and pancreas, to give but two instances, that biochemistry has come into its own.

EARLY SYMPTOMS.

Symptoms are the earliest manifestations of disease more often than physical signs. There is some subjective deviation from the normal, such as discomfort, pain, exhaustion, or lassitude. The elucidation of the meaning of such deviations from the normal and their prognostic significance was the aim underlying all the work of the late Sir James Mackenzie. He, more than any other man, focused the attention of the world of medicine on the subjective aspect of disease. Of all subjective sensations pain is the most arresting, and is of the nature of a protective reflex. We are saved from destruction by the pain which we deplore. This is true even if we may have to admit that on occasions the anguish proves too great for the human mechanism, and becomes, in fact, the lethal factor precipitating the end which it is its function to avert. Peculiarly is this the case in patients suffering from angina pectoris, and it is essential that we should be familiar with the earliest manifestations of this condition or symptom complex, in order that our patients may have the benefit of a correct and early diagnosis, so that their lives may be ordered aright and their days lengthened.

"By angina pectoris I have in mind an arresting form of cardiac pain, substernal rather than submammary, in which the patient is conscious of a sense of oppression, or constriction, which may reach a high grade of intensity, even a condition of intolerable anguish. This pain has certain well recognized lines of radiation and is often induced by effort or emotion. There are in addition characteristic associated symptoms not invariably present—notably the sense of impending death, and indescribable anguish of mind accompanied by varying vasomotor disorders, such as cold sweats and deathly pallor." (Bradshaw Lecture, BRITISH MEDICAL JOURNAL, November 24th, 1923.)

With few exceptions, the full-blown stark anginal seizure cannot fail of recognition. It is otherwise with the earlier manifestations when such are present, and it is these less

* I gladly acknowledge the great courtesy of the medical men in charge of these patients, a courtesy which has enabled me to keep in touch with the after-histories.

obtrusive indications which I wish to discuss—symptoms which are often not recognized as anginal in nature, and in consequence their ominous significance is overlooked.

In 1905 Sir James Goodhart, in the Cavendish Lecture, said, "Angina is common among us if we are on the look-out for the milder forms," and with this opinion I entirely agree. Angina minor has been labelled the "petit mal" of angina, and Sir James Goodhart referred to it as "larval." Both terms indicate that there is an essential relationship between the mild and the grave forms. It is merely a question of degree, for both are true angina.

Minor angina has also been called "fractional," because the patient may only manifest "one, or at any rate only a fraction, of the essential characteristics of the major anginas; that is, it may lack some of those characteristics always associated in our minds, and in those of the general public, with angina."

Minor angina may be, and often is, a premonitory symptom, but one full of significance to the understanding clinician. Most patients who suffer from major angina will, when carefully questioned, give a history indicating that for some years previous to the first major attack there were larval or minor manifestations, anginal in nature.

Again, both major and minor may alternate irregularly in the same patient, or the minor may come as an after-math or echo of a previous major attack. In such a case, the diagnosis being then beyond question, an opportunity is given of studying the more elusive symptomatology of the milder disease fortified by the knowledge of its real significance.

CAUSE AND CHARACTER OF THE SYMPTOMS.

In angina minor, as in angina major, the symptoms result from cardio-vascular inadequacy, for the heart is in difficulties. Of these symptoms the commonest is substernal discomfort or pain on exertion or exposure to cold. This varies in degree and also in character. It is described by the patient as "a sense of oppression," "tightness," "constriction," a "feeling of weight," sometimes merely as "emptiness."

The site of the sensation is substernal, often in the middle or upper third, but it may be most marked at the lower third of the sternum, and sometimes even in the epigastrium, in "the pit of the stomach." This peculiar feeling is most commonly referred to by the patient as "indigestion," and not seldom he has been treated as a "dyspeptic" for years before the nature of the discomfort is revealed by an attack of angina major. These patients not uncommonly develop the habit of air swallowing, and an attack of angina minor simulating indigestion is accompanied by repeated attempts to "bring up the wind." When a sufficiency has been swallowed it is belched up, with undoubted relief to the patient. Such a sequence confirms the patient, and at times even his doctor, in the view that flatulence is the correct interpretation of the symptoms. And this view is still further corroborated by the fact that any exercise following immediately upon a meal—more particularly breakfast—is extremely likely to precipitate an attack.

The pains of angina, major or minor, are neither stabbing nor intermittent; they are continuous and persistent and have a crescendo and diminuendo quality, increasing in intensity if the effort which induced them is continued, and tending to cease when the patient rests.

The statement that anginal pain is an indication of myocardial exhaustion or evidence of myocardial failure seems to me to require modification, for cardiac failure may advance to an extreme degree without anginal pain, and definite anginal pain will sometimes disappear as cardiac failure becomes more pronounced.

The first manifestation of cardiac inadequacy may be pain, followed by dyspnoea if the effort is continued; yet at a later date in the same patient dyspnoea is the earliest sign of cardiac embarrassment, pain only manifesting itself if the patient persists in the effort. This may, of course, be due partly to variations in the threshold of pain, but I do not believe that that is the full explanation.

The causes which induce anginal pain have this characteristic—they are such as to bring about a rapid strain on the arterial side of the circulation and especially on the left ventricle. The two common exciting factors are effort and exposure to cold. In a large proportion of anginal patients there is either a lesion of the coronary arteries or persistent hyperpiesis; in the first, the supply of blood to the myocardium ceases to be adequate when the heart is called upon for an increased output; in the second, the left ventricle and the aorta are carrying the heavy load of a raised blood pressure.

Effort and exposure to cold, and at times excitement, result in a sudden strain, and the response to this strain is manifested in terms of pain. After a short rest and a more gradual resumption of the effort the myocardium sometimes accommodates itself to the altered conditions, and may even tolerate the effort with comfort. It is, in fact, a matter of readjustment.

During an attack of angina major, in addition to the terrible crushing agony substernal in site there are accompanying pains or unpleasant sensations which may be felt in the back between the scapulae, in the shoulders, arms, neck (suprasternal notch), jaw, even in the face and eyes. So in milder angina these radiated pains are to be noted, and they may prove of the greatest help in recognizing and assessing the significance of indefinite substernal discomfort.

The difficulty in diagnosis is increased when there is an entire absence of complaint of pain or tightness in the chest. In most instances, however, cross-examination will elicit the presence of some such substernal discomfort if the effort inducing the attack is unwisely persisted in. As an example of this the following case may be quoted.

CASE I.

In December, 1923, a man, aged 69, complained of severe neuralgic pain in the neck. The pain was crushing in character and situated in the suprasternal notch. It had occurred on several occasions in the two months before his visit. The teeth had been suspected and one extracted, but the pain recurred. It was brought on chiefly by exposure to cold, and also (although not so noticeably) by walking or effort. It was relieved by a hot drink or by going into a warm room. It did not radiate either into the back, or shoulder, or arms, but if he persisted in walking, or in whatever effort he was undertaking at the time, he developed an aching pain behind the manubrium. This important fact was only extracted by persistent cross-examination. Pain was also brought on by getting out of a warm bath and during the unavoidable exposure while drying himself.

He was firmly convinced that the trouble was in his throat, and he had seen several throat men, who had been unable to give him any relief. His throat was normal. There was some arterio-sclerosis, such as one would expect in a man of his age, and the blood pressure was normal.

I class this man as an angina minor of an eccentric type. Two other rather similar cases will further illustrate this point.

CASE II.

A man, aged 65, was first seen by me on March 13th, 1925. He complained that he had to stop when walking because of a peculiar feeling in the epigastrium. Persistence in walking caused salivation. Occasionally he experienced a sense of pressure in the chest behind the sternum, as if something within was pressing outwards. These symptoms were always more easily induced after a meal. He was a spare man, and was never seriously ill. There was a moderate degree of arterio-sclerosis. Blood pressure 185/110 mm. Hg. The symptoms were first noticed six years before he consulted me, but had been much worse during the last nine months.

On April 27th, 1926 (a year later), his condition was satisfactory, but he was still liable to the above symptoms.

The following is an interesting example of eccentric angina of a minor grade.

CASE III.

A male, aged 59, first seen on March 17th, 1924. In this patient walking induced an aching neuralgic pain in both gums, first the lower jaw and then the upper. The pain was more liable to come on when he went out into the cold air or "of an evening with the little dawg." Pain, first felt in the jaws, was next experienced in the epigastrium—"like unto indigestion"—and was associated with a feeling of complete exhaustion and collapse. All these symptoms vanished in a minute or two if he stood still.

He first noticed pain in the jaws three years previously, and on the advice of his doctor every tooth in his head was removed. This was followed by considerable improvement, although the pain in the jaw was still occasionally felt after effort. Six months ago, however, it was as severe as ever.

On examination there was no evidence of valvular disease; there was very marked emphysema, and the voluminous lungs made it impossible to determine the size of the heart. He had definite arterio-sclerosis; a blood pressure of 160/85 mm. Hg; cardiac rhythm normal.

These three cases are exceptional, but they serve as examples of erratic manifestations of angina minor.

A more common form of related or erratic pain is a dull ache or cramp in the shoulder or arm. This is usually described by the sufferer as neuritis or rheumatism, and when it occurs—as it not infrequently does—without much, if any, pain in the chest, it is misleading and its significance tends to be overlooked.

The most important feature in all these erratic or eccentric anginas is that the pain, wherever it may be localized—neck, face, wrist, shoulder—is brought on by effort or exposure to cold, and is relieved by rest and warmth. The "indigestion" and the "rheumatism" in the arm or shoulder are both dissipated by cessation of effort.

It is to be remembered that accompanying these substernal sensations of angina minor there is sometimes a peculiar psychical disturbance—an uncanny intangible sense of foreboding, a vague dread, a recognition that there is something fundamentally wrong, an apprehensiveness which may persist long after the symptoms have disappeared. This peculiar anxiety, which does not indeed attain to the *angor animi*, the sense of impending death, accompanying the major seizure, bears no relation to the degree of pain or discomfort, but is most suggestive and characteristic; Sir Clifford Allbutt regarded it as adding definitely to the gravity of the prognosis.

EXAMPLES OF ANGINA MINOR.

As fairly typical examples of angina minor I will relate briefly the following cases.

CASE IV.

A man, aged 64, complained of gripping pain of moderate intensity and a sensation of pressure, unaccompanied by any dyspnoea. The pain was substernal, at the level of the third space, and covered an area the size of a crown. It was induced by walking and ceased at once when he stood still. If he persisted, pain and numbness appeared in the left forearm and hand. He had suffered in this way for two years, and had been under continuous treatment for "indigestion."

CASE V.

In another patient, a man aged 56, the first indication of trouble dated back to December, 1920, and consisted in pain across the chest at the level of the nipples, brought on by walking up a slight incline. If he stood still it subsided. It ceased to trouble him, but recurred in March, 1921, when it was looked upon as "indigestion," but, as his doctor said, a diagnosis made "with mental reservations." A fortnight later he had five attacks of severe angina arising in the same area, and accompanied by pain extending from the tip of each shoulder down the arms to the thumbs. He thought he was dying during the acme of the attack, and was left emotional and depressed.

CASE VI.

In April, 1923, a male, aged 52, complained of dull aching pain in the upper chest. The pain was situated just below each clavicle in the nipple line. When the pain was more severe it was accompanied by an uncomfortable feeling down the inner side of each upper arm and in the front of the forearm. It sometimes attacked him in the bathroom when dressing, and was described by him as an intense heartburn, a "hot pain." He often suffered on the way to the station in the morning, and the pain would last about ten minutes. It was relieved by rest, by the eructation of wind, and at times by stretching his chest. This pain was never agonizing. He had first noticed it in 1921 after an operation for rupture. He soon recovered and led an active vigorous life without discomfort.

The pain recurred two weeks before he consulted me in 1923, and I then found a heart moderately enlarged; blood pressure 180/100 mm. Hg; no valvular disease; no dilatation of the aorta; aortic second sound accentuated; only a moderate degree of arterio-sclerosis. Heart rate 72 and regular. He was a thin, spare, active man, and experienced no dyspnoea on exertion. I classed him as a case of angina minor. He improved considerably under treatment, but was always liable to pain if he hurried, especially after breakfast.

In October, 1924, he was awakened at 5 a.m. by very severe substernal pain radiating into the armpits. The pain extended the whole length of the sternum and radiated into the shoulder and into the arms. The blood pressure had fallen to 150/85 mm. Hg, and a soft mitral systolic murmur had developed.

I did not see him again. He died suddenly in March, 1926, in a cab, five years after the first appearance of the minor angina. With the exception of the severe attack of pain which awakened him in October, 1924, he gave no history of anything which could be called a major attack.

CASE VII.

A heavily built, florid man, aged 60, first noticed slight sub-sternal pain six years ago at the age of 54. It was mid-sternal and was brought on by walking, especially if the day was cold. Although a medical man he did not attach any importance to this suggestive symptom until 1924—that is, two years ago. Then the pain became more severe and for the first time extended into both arms and forearms.

In November, 1925, he suffered from a frank agonizing attack of anginal pain which lasted some hours and mainly affected the lower third of the sternum.

On examining him, shortly after his attack, there were no objective signs of disease, except slight enlargement of the heart which extended 4½ inches to the left of the mid-line. The sounds were normal and the blood pressure 140/90 mm. Hg. Heart rate 72, regular. There was little, if any, dyspnoea on effort. The attacks were relieved at once by rest or a draught of whisky.

This patient died in a major attack in February of this year. Minor angina was present for four years before the true significance of the symptoms became apparent.

CLASSIFICATION.

Following Sir James Mackenzie, I think the most satisfactory classification is that into two groups; (1) anginal pain due to some essential disease of the cardio-vascular system, which he has termed primary angina; and (2) the second group in which all evidence of such cardio-vascular disease is absent, but where there is some factor outside the circulation which has caused a lowering of the threshold to pain, or which has depreciated the efficiency of the myocardium. The neurasthenic group, I think, might be viewed as belonging to the secondary anginas.

The chief pathological changes in the primary anginas are diseases of the myocardium, with or without frank coronary narrowing; arterio-sclerosis, with or without high blood pressure; and lastly, valvular disease, more particularly of the aortic orifice, often associated with aortitis. In this group the myocardium is frankly handicapped, and it is clear that, with the exception of the syphilitic cases and those with high blood pressure, treatment cannot be expected to produce much change in the pathological condition, which is progressive in character.

In the secondary group of anginas the possible adverse factors are numerous, including subinfections or other causes of toxæmia. The nervous instability of the climacteric is associated with the lowering of the threshold to pain. Prolonged worry, anxiety, overwork, or any factor causing neurasthenia, may bring about a similar sensitiveness to painful stimuli.

It is well to remember that in a patient with definite cardio-vascular disease correctly classed as primary angina there may be present in addition conditions which would in themselves cause secondary angina, and in prognosis such possibilities must be carefully reviewed.

INVESTIGATION OF THE PATIENT.

After careful cross-examination of the patient in regard to the pain, and after having decided that it is cardiac in type and entitled to the designation of angina minor, he must then be investigated in order to determine the presence or absence of cardio-vascular disease.

The whole cardio-vascular system must be passed under review in order to determine first whether a diagnosis of primary angina is justifiable, and the extent of the damage. Any enlargement of the left ventricle is significant. This is most marked in patients with unduly high blood pressures, although it is found also in those suffering from free aortic regurgitation, and again sometimes in the absence both of raised blood pressure and aortic regurgitation. The fair inference in the latter case is that there is depreciation of the myocardium, probably due to coronary disease.

When there is valvular disease it is practically always the aortic orifice that is affected. In an analysis of 309 cases of angina pectoris made in 1923, only 35 showed disease of the valves, and, with one exception, the aortic valves were damaged. There was definite aortic regurgitation in 26, and some degree of aortic obstruction in 10 patients. Only one suffered from frank uncomplicated mitral stenosis. If there is an aortic leak it is often slight and the patient does not present the typical clinical picture of free regurgitation. The symptoms in such patients are not those of valvular disease with cardiac failure, they are the symptoms of angina, and not to be distinguished from

the symptoms found in those other anginal patients in whom the aortic valves are competent.

Arterio-sclerosis is a common feature, as is to be expected when the age incidence of angina is remembered, and the condition of the arteries should be noted, especially those of the retina; and when possible the aorta should be x-rayed.

It is important to note the height of the blood pressure. It is unduly high in about half the cases. In 40 per cent. I have found it at or over 180 mm. Hg, and some amelioration of the symptoms is to be expected when the hypertension is modified. A marked rise in the diastolic pressure is of especial significance. Any drastic reduction of a raised blood pressure is, however, unwise in the interests of the patient, even if such a reduction can be brought about and maintained.

It is rare to find any change in the fundamental rhythm of the heart. Premature beats are not uncommon. Auricular fibrillation or the disorderly rhythm when it occurs may diminish the intensity of the pain, or even abolish it, but this is not invariably the case. In the following patient, on the contrary, the advent of fibrillation was accompanied by an increase in the liability to pain.

CASE VIII.

A man, aged 71, complained of gripping pain in the chest, brought on by walking. He had first noticed it five weeks before consulting me in January, 1925. It extended from the second rib to the xiphisternum and out as far as the nipples. On one occasion it was accompanied by "rheumatic pain" in the front of the left upper arm from the shoulder to the elbow. The pain came on four or five times a day; it was always induced by effort, and there was no dyspnoea. The heart was normal in size and free from valvular disease. The blood pressure was 140 mm. Hg, systolic. The rhythm was regular with an occasional premature beat.

He did well under treatment until May, 1925, and then the pain recurred and was more easily induced than in 1923. It also spread down to the finger-tips. In July, 1925, the only difference to be found on physical examination was that the auricles were fibrillating, and that the ventricular rate had increased to 120 per minute.

In this man the appearance of fibrillation was accompanied by an accentuation of the pain and not, as is more commonly the case, by an amelioration of the symptoms. He died in November, 1925, in a typical attack of agonizing angina.

If space permitted I could quote other similar cases, but these will suffice.

In the examination it is of importance to determine the factors which induce the pain, more particularly the nature and severity of the effort—that is, to estimate the amount of cardiac reserve; not only the cardiac response to effort should be noted, but the constancy of that response. It is satisfactory to find in any patient that there are days when he can do with ease that which on another day causes pain, for such patients prove more amenable to treatment.

There is something sinister about an anginal pain which invariably appears at the same fixed point on ascending an incline—at the same lamp-post, so to speak—and yet it is even more ominous when there is a steady diminution of the distance that can be walked without pain or discomfort.

When there are apparently no clear evidences of cardio-vascular disease, a careful survey should be made of the patient to discover, if possible, anything which may be lowering the threshold to pain. First, any toxic cause, such as a grumbling appendix, oral sepsis, sinus disease, or some pulmonary, renal, or alimentary disorder; further, worry, anxiety, overwork, or insufficient sleep, are quite likely factors. When some such cause is responsible for lowering the threshold to pain, it persists between the attacks of pain, and in consequence the patient is not normal even when quite free from angina. He is found to be toxic, or depressed and irritable, and shows the usual indications of nervous exhaustion.

When possible, as a final feature in the examination, it is well worth while to take an electro-cardiogram. This may reveal evidence of myocardial damage which might otherwise escape recognition. I will refer later to its value in prognosis.

DIAGNOSIS.

In the absence of syphilis or valvular disease of the heart, primary angina is rare under the age of 45, and it is

unwise to make such a diagnosis in the young if neither aortic disease nor an abnormal blood pressure is present. In fact, the diagnosis must be made on the character of the pain and not on the physical findings. Too often a correct diagnosis of angina is refused because on physical examination either nothing abnormal is found or there is only a moderate degree of enlargement. It must be remembered, before passing a heart as normal, that emphysema masks an increase in the size of the heart, and also makes it difficult to recognize any abnormality in the heart sounds.

Pain in the region of the heart is a common complaint in most patients with valvular disease, and in many others, especially in young males presenting the symptoms of the "effort syndrome" (the D.A.H. of army days). But this pain, though cardiac in origin, is not entitled to the label "angina." In this group the pain is submammary and often felt directly over the cardiac thrust. It may be stabbing, or an ache, and the skin and muscles of the adjacent areas are often tender on palpation, especially directly over the apex beat. Hyperaesthesia is much more noticeable in these patients than in the anginas.

Although usually induced or aggravated by exercise, the pain is often quite independent of effort and frequently occurs when the patient is in bed; and it may be accompanied by troublesome palpitation. If the pain does extend into the arm it is nearly always diffuse, involving the whole arm, and often both arms. It is less likely to have the segmental distribution or the characteristics of anginal pain. Occasionally it extends from head to foot, limited to the left side, and when the pain is bad there may be great nervous excitement—sometimes even nervous rigors—followed by a condition of prolonged nervous prostration. This is in great contrast to a primary angina, where cessation of an attack as a rule leaves the patient comfortable and free from physical distress.

The following is an example of cardiac pain which presents the characters of a secondary angina and which had been looked upon as major.

CASE IX.

A nurse, aged 30, was examined on April 9th, 1926. She was tall, with red hair, and a healthy complexion, and was alarmed and anxious about her heart. She complained of pain just to the left of the sternum in the fourth intercostal space. It was as if something was pressing hard on the chest. It caused her breathing to stop, and radiated into the left arm, involving the whole arm from shoulder to wrist in a cramp-like pain. The pain was induced by effort or annoyance. Examination showed a heart normal in size and sounds, free from any indication of disease. Blood pressure 125/75 mm. Hg. Heart rate regular. The heart sometimes raced at 100 to 120.

This patient's symptoms date from a sharp attack of influenza two and a half years before. Ever since she has been liable to vasomotor blotching, and her hands have been blue and cold in the winter. In addition to dyspnoea, she becomes dizzy and faint on climbing the stairs; she wakes in the night with a sensation of shortness of breath, which sometimes lasts for ten to thirty minutes. The attacks are always worse after she has been nursing a "bad case," and also after a period of excitement, but not while excited.

There are certain features in this patient which are very suggestive, and indicating that there are factors outside the heart; the vasomotor system is unstable, and the effect of any nervous exhaustion is noticeable.

THE OUTLOOK IN ANGINA MINOR.

The prognosis is admittedly difficult when the patient is afflicted by major angina; it is doubly so when the symptoms are of the slight, transient, mild degree present in angina minor. The great danger is that of a wrong diagnosis. The anginal element in the complaint is wrongly interpreted, until the "grand mal" stage arrives, and the diagnosis is "writ plain for all the world to see."

In angina minor the prognosis must be based on a consideration of the same facts as in angina major. It is the integrity of the myocardium and the soundness of the arterial tree that matters, and, of course, primary angina of the minor type is more serious than secondary angina minor.

The more reasons there are for believing that the threshold for pain is below normal, the better the outlook, and when the causes for the lowered threshold to pain can be removed there is still further reason for a tempered optimism. Subinfections, mental worry and strain, insuffi-

cient sleep, and factors of this kind can be treated, removed, or neutralized. The prospect brightens in so far as this can be carried out satisfactorily.

It is not wise to venture on any prognosis when the pain dates from a recent pyrexial attack such as influenza. The myocardium may take some months to recover from the transient toxæmia, and during the convalescence the symptoms may be misleading. Persistence in the liability to pain and discomfort in spite of treatment is an unsatisfactory feature, especially if combined with it there is clear evidence of cardiac embarrassment accompanied by enlargement of the heart. A high blood pressure, peculiarly enough, does not appear to add to the gravity of the prognosis. My figures for all anginas gave the average duration of life from the first appearance of pain as four and a quarter years in the high pressure cases (180 mm. Hg and over), and three and a half years in the group with pressures under 180 mm. Hg. As in many of these patients there was a year or two when the symptoms were of the mild minor type, before major angina developed, I feel inclined to view the minor angina with low blood pressure as probably less satisfactory than when the pressure is high.

When there is a clear history of syphilis the prognosis should be guarded until a full course of antisyphilitic treatment has been carried out, for occasionally the improvement is marked. Dr. Cotton has proved that the liability to attacks was diminished and the duration of life prolonged by vigorous antisyphilitic treatment in anginas of syphilitic origin. My experience definitely corroborates this. Potassium iodide in massive doses is not enough; the patient should have two courses of intravenous novarsenobillon and prolonged mercurial treatment.

The family history is always worth considering when reviewing the prognosis. The tendency to hyperpiesia is sometimes a family characteristic, and in such families death is common from angina, cerebral hæmorrhage, or cardiac defeat. With such a family history the expectation of life is poor over 50, and the mere grumbling of angina minor is exceptionally ominous. At times the electro-cardiograph findings indicate a speedy excitus, especially when they reveal the presence of a branch bundle lesion. Even when there is no evidence of a bundle lesion other well recognized deviations from the normal may be met with which have a sinister significance. The following case is of interest in this connexion.

CASE X.

In May, 1925, a medical practitioner, aged 60, complained of a "raw feeling" across the upper sternum, described by himself as pleurodynia, which he did not view with any apprehension. This sensation was noticed on going out of the house. Exposure to cold or damp would bring it on, but in a warm room he felt no discomfort. It did not interfere with his breathing. He had never been ill in his life, and this was the first time he had ever consulted a doctor. He had only noticed this queer sensation for four weeks.

On examination the pulse was 84, regular, not perceptibly collapsing. Arteries just palpable. Blood pressure 170/55 mm. Hg. The heart was 4 inches to the left of the mid-line; there were no thrills. Over the aortic area I heard a to-and-fro bruit, and the aortic second sound was not audible. A soft diastolic murmur conducted towards the apex could just be heard. The urine was normal.

On May 22nd an electro-cardiogram demonstrated a right branch bundle lesion. The blood pressure had dropped to 135/55 mm. Hg. The heart rate was 84, regular and normal.

On June 7th he had an attack of major angina which killed him in a few minutes. The total duration of life from the first indications of minor angina was only eight weeks.

TREATMENT.

My remarks on treatment must be brief. In the *Lancet* of November 8th, 1924, I discussed this fully, and the general principles indicated there apply with equal force to the minor form of angina, and, it might be added, with a fairer prospect of satisfactory response.

A damaged heart necessitates a sheltered life, and the reasons for the restrictions advised should be frankly explained to the patient. His full co-operation is imperative, and he must face his limitations and live within them. The efficiency of the myocardium must be maintained by adequate rest, a regulated life, and such medicinal measures as hæmatinics. When syphilis is an etiological factor, appropriate and efficient antisyphilitic treatment is, of course, imperative.

Since the work of the heart bears a relation to the body weight, an attempt should be made to bring the bulk of the body, when it is excessive, within normal limits. Careful dieting to obtain this result should be carried out and the results noted. An abnormally high blood pressure can sometimes be modified with definite relief to the overloaded myocardium.

Much can be done in the secondary anginas when by meticulous cross-examination the cause of the lowered threshold to pain has been discovered. All possible sources of persistent low-grade infections should be investigated and radically treated—dental sepsis, sinus disease, abnormal intestinal conditions, pyelitis, chronic appendicitis, etc.

An attempt must be made to eliminate worry and make sure of sleep. For this a mixture of ammonium bromide and phenazonum is especially helpful. In the readjustment of the patient's life, factors giving rise to mental strain and irritations should be obviated as far as possible.

As my last word, may I lay stress again on the very great importance of an early diagnosis when the symptoms, though but slight and elusive, are both ominous and sinister. An accurate diagnosis *then*, followed by wise treatment, offers some chance of arresting, or at any rate of retarding, the development of the pathological changes responsible for angina.

THE ACTUAL PLACE AND FUNCTION OF THE TUBERCULOSIS DISPENSARY IN THE TUBERCULOSIS SCHEME.*

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WHEN the council honoured me with the request to open this discussion I accepted with alacrity. The fascination of tuberculosis has held me a bondsman throughout my professional life. The range of interests is always widening. In offering a few thoughts for your consideration it will be my endeavour to do so briefly and simply, in the hope that it may stimulate members of the Conference to give us freely of their experience.

Forty Years Ago.

It is difficult to-day for the man in middle life—and still more for the younger man—to realize the attitude of mind towards tuberculosis which prevailed forty years ago and previously among both doctors and laymen. What was *then* accepted as care and treatment is *now* hardly imaginable.

The victim of tuberculosis—so-called consumption—was for the most part recognized too late. He was generally expected to die. The treatment meted out to him was empirical, unphysiological, and usually futile. The vain attempts at protection tended to hasten the end. With the death of the patient the interest that had been kindled by his illness was extinguished.

Speaking of the expectancy of life of the consumptive patient, Dr. C. J. B. Williams, in his treatise on *Pulmonary Consumption* (1871), says, with reference to the first ten years of his practice, 1830-40: "My general recollection of the histories of the developed disease at that time is that of distressing tragedies, in which no means used seemed to have any power to arrest the malady; and life was rarely prolonged beyond the limit of two years, assigned by Laënnec and Louis as the average duration of the life of a consumptive."

Conception of Tuberculosis Dispensary (1887).

Let me try to recall some of the circumstances and events which led to the remarkable alteration of outlook and action which now prevails.

It is some forty years since, following on the discovery of the essential cause of tuberculosis, its infective nature came to be admitted. While engaged in laboratory

work on tubercle, and attached to a general medical dispensary, it was borne in on me that acceptance of the infective nature of the disease carried with it far-reaching consequences. It seemed clear that, the infective nature of tuberculosis being granted, the principles which had been effectively applied in relation to other infective conditions were, with appropriate modifications, applicable to tuberculosis likewise.

Apart from a few special hospitals devoted to diseases of the chest, the chief avenue to care and treatment open to the poorer citizen suffering from tuberculosis forty years ago was by way of the out-patient department of a general hospital or similar medical institution. What happened daily in out-patient departments amounted to this—that the patient, suspected to be suffering from what was then spoken of as consumption, was examined for the presence of certain recognized signs of chest disease; when such were ascertained to be present, the sick man was accorded a prescription for his cough or other chief symptom, and commonly for cod-liver oil, and was invited to report himself from time to time for similar consideration and treatment. Occasionally he was passed on for admission to hospital, when such facilities were available. The number of special beds at that date was extremely scanty, and he was not very warmly welcomed in the general wards. The demand on beds was out of all proportion to the number of tuberculous applicants, with the result that even the small percentage which attained admission enjoyed the available benefit for a relatively short period. There was little serious effort to ensure anything more than amelioration or arrest of existing symptoms. The infective nature of the disease hardly came under review; nor were the facts of the illness linked in any close fashion with the conditions of the patient's home and household.

All this was radically wrong. It involved a disregard of the natural history of the disease, its infective origin, its widespread distribution, its lengthy course, its protean manifestations, its dependence on environmental conditions, its tractability when efficiently handled for a sufficient period, and many other facts which we now regard as commonplaces.

Reflection on such lines led me to press for a wider conception and fresh line of procedure. The proposal culminated in the establishment of the tuberculosis dispensary in 1887.

Purpose and Programme.

The essential purpose was the creation of a centre towards which patients suffering from this infective disease, and the friends of patients, and the public, could readily turn for every sort of direction and help, and from which there would radiate information of many kinds for the guidance of individual patients, households, and the community as a whole.

The original aims of the tuberculosis dispensary have been often detailed. They are so well known that on the present occasion there is little need to recapitulate them in detail. They constituted the first attempt to hunt down the infection, to discover the haunts and methods of the invader, and to raid him on his own territory. The principles and methods were incorporated in the well known circular of the Local Government Board of Scotland, dated March 10th, 1906, which reproduced *in extenso* a paper given by me to the International Congress at Paris in 1905. The principles and methods were frankly accepted by the Departmental Committee on Tuberculosis (1912), which adopted the programme of the Edinburgh Tuberculosis Dispensary as a model for the tuberculosis dispensaries of the country.

The Departmental Committee, in describing the functions of the tuberculosis dispensary, affirmed that it should serve as (1) receiving house and centre for diagnosis; (2) clearing house and centre for observation; (3) centre for curative treatment and supervision of ambulant cases; (4) centre for the methodical examination of contacts and the investigation of environmental conditions; (5) centre for continuous care of patients—so-called "after-care"; (6) information bureau and educational centre. The Departmental Committee placed the tuberculosis dispensary

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