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THE SYMPTOMATOLOGY OF CARDIAC PAIN*

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The loose term "angina pectoris" has long been used to cover indiscriminately the various types of cardiac pain. The fairly recent differentiation of coronary thrombosis as a separate clinical entity should serve to stimulate further precise classification. The purpose of the present communication is to record the differences in symptomatology observed in a number of cases of cardiac pain followed up and observed over a period of seven years. During that period it has become clear to the writer that there are several different types of "angina," and that accurate classification of these cases is helpful in prognosis and in treatment, and is essential for the intelligent performance of any clinical investigation of the subject. It is felt that the clinical characteristics of the various types are sufficiently constant to make it desirable that they should be placed on record, with a view to stimulating a wider interest and a more accurate terminology.

There are two chief methods for the making of a diagnosis—history and physical examination. In certain conditions the history is of especial value. Diseases of the stomach have always been remarkable for the paucity of physical signs, and in earlier days diagnosis depended upon an exact elucidation of the relation and response of the gastric sensation to food, alkalis, and posture, and to the presence or absence of water-brash, vomiting, and appetite. Special means of investigation, the radiograph, the fractional test-meal, and the chemical examination of stools for occult blood have confirmed scientifically the separate identities of the syndromes present clinically in hyperchlorhydria, gastric and duodenal ulcer, carcinoma, and gastritis. Gastralgia is not used as a label to cover all of these separate conditions. There is as little excuse for the term "angina" as a label to embrace the different types of cardiac pain. In the case of the heart, however, biochemistry and radiology are not helpful as differentiating methods, and the use of the electrocardiograph for this purpose is as yet limited. An exact history is therefore of the greatest importance in this group of diseases.

So far as the history is concerned cardiac pain can be classified under the following heads:

- Quantitative pain (angina of effort).
- Spasmodic pain (spasmodic angina).
- Continued pain (coronary occlusion).
- Pain having no specific relationships (pseudo-angina or angina innocens).

These types of pain may in any one case be found quite defined and separate, or it may be possible to recognize two or more of them as present in combination.

* Some of the material used in this paper formed the basis of two B.M.A. Lectures, to the Southampton and to the Hampstead Divisions.

Descriptions of the symptomatology of the different types of pain will at first be given; these will be followed by descriptions of certain other clinical syndromes with which cardiac pain is significantly associated.

Quantitative Pain (Angina of Effort)

"Angina of effort" is a condition in which the pain is cardiac in character, arising substernally and centrally in most cases, and eventually radiating down one or both arms, generally the left. In some cases the order of origin and radiation is reversed, the "pain" arising in the fingers, wrist, or lower arm, and eventually radiating back to the chest. This spread of pain is of two kinds, the spread occurring either as the disease progresses or as the individual attack develops. For example, the first symptom may be substernal pain, and for weeks or months the pain is localized to that situation, though varying in intensity. Later the pain radiates to the arm whenever it arises under the sternum. In other cases the pain at the commencement of the attack always begins under the sternum, and inevitably radiates to the arm.

As cardiac pain becomes less severe during treatment, the radiation frequently no longer occurs, and the actual area of the chest wall over which pain is felt becomes less, so that sometimes only one or more isolated spots are present at which quantitative pain still occurs. The patient may describe this localized pain as similar to that produced by a thumb being pressed hard against the chest.

The absolute rule in angina of effort is that *the symptom is completely absent during rest, and is evoked by exercise*. The presence of "pain" is strictly proportional in every case to the amount of exercise undertaken, and the more intelligent the patient the more accurately will his account of the symptoms bring out this relationship. One such patient stated without questioning that he could walk for about 300 yards on the flat at four miles an hour, at which point the pain began; he could walk for an indefinite period at two miles an hour, but hills pulled him up immediately.

From this quantitative relation of the pain it is clear that patients soon learn to walk slowly, for they recognize that by doing so pain is absent or slight. Thus pain in angina of effort is very rarely severe, unless emergency or stress forces the patient to disregard the warning sensation in the chest. It is generally described as an "oppression," a "tightness," a "gripping feeling," a "lump," a "bursting feeling," a sensation of "distress." These sensations will only become pain if exercise is continued. It occasionally happens that in individuals with a sensitive nervous system true pain is complained

of from the first onset of the sensation. This is, however, a less common occurrence.

In some instances the pain is, in its first stage, only noticed after a meal. The exact quantitative relation to exercise still persists, but the pain in these cases only occurs if such exercise is taken on a full stomach. The increased cardiac output following a meal, or the splanchnic congestion, with presumably some systemic depletion of blood, may be causative factors producing this peculiarity. Another factor which not infrequently accentuates angina of effort is external cold. When the temperature is lowered, and particularly in the presence of a cold wind, the pain is apt to come on with less exercise than usual.

Angina of effort is found characteristically in arteriosclerosis, the heart muscle being affected in a variable degree. The degree of involvement of the myocardium is proportional to the amount of dyspnoea associated with the pain. The pain itself, although symptomatic of organic disease of the heart muscle, is also a measure of the sensitivity of the central nervous system. It is therefore most important to obtain a clear answer to the question, "Which is it that pulls you up, pain or shortness of breath?" Patients whose nervous thresholds are low complain of pain and of no dyspnoea; patients whose myocardium is more severely affected complain chiefly of dyspnoea, and secondarily of pain.

The writer believes that a second factor is involved in this relation between the severity of the pain and the amount of dyspnoea. This factor is the rate of development of the arterial obstruction. If a lesion is slow in development it is more insidious, and allows of some degree of physiological or nervous adaptation, and it can cause a physical disturbance with less pain, whereas a sudden lesion is more acutely painful. The history of coronary thrombosis bears this out, for here the abruptness of the lesion and the severity of the pain are both maximal. In contrast with this much fibrosis is found post mortem in many hearts, associated with, and presumably resulting from, slowly advancing coronary atheroma, yet in such patients cardiac pain has been absent. In such cases it would seem that the heart muscle fibres have been killed off painlessly one by one by the very slowly advancing coronary obstruction.

Pathological Causes of Angina of Effort

The type of pain suffered from is so analogous to that found in intermittent claudication that it is difficult to avoid the conclusion that the cause is partial obstruction to coronary flow. Such a partial obstruction occurs most commonly in atheroma of the coronary vessels, and this lesion is probably responsible for the vast majority of all such cases. It is also found in syphilitic aortitis, in which condition the coronary walls are normal, but the coronary orifices, where they pierce the aortic wall, become involved in syphilitic oedema, inflammatory swelling, or fibrosis. Severe anaemia, by producing a general anoxaemia, may apparently produce the angina of effort in patients who otherwise would have been free from it, or who previously had no such symptoms. Carey Coombs¹ has described "angina" in pernicious anaemia, and the same symptom has been seen by the writer in simple anaemia following a haematemesis in two cases.

Relation to Other Types of Cardiac Pain

There is a close aetiological connexion between angina of effort and coronary thrombosis. Sometimes angina of effort precedes the latter condition. In this case the symptoms of partial coronary occlusion due to the atheromatous change are manifest in the form of the quantitative cardiac pain. Where coronary thrombosis occurs with no previous history of angina of effort, either the

coronary lumen was adequate, in spite of the presence of atheroma, or the patient's nervous system was relatively insensitive.

Coronary thrombosis is frequently the starting-point for a subsequent period of angina of effort in a previously healthy individual. It is easy to understand that the coronary block, while killing some heart muscle fibres, renders others, which are partially served by collateral circulation, only relatively poor in blood supply. When these fibres are at rest they are adequately nourished; when they are working above a certain level of effort their blood supply is inadequate and pain occurs.

There is also a definite connexion between angina of effort and spasmodic angina. As with coronary thrombosis a period of quantitative pain may precede, for months or years, the onset of attacks of spasmodic angina. Since the mechanism of the latter condition is still obscure the functional connexion is less obvious than is the clinical. Spasmodic angina is commoner in nervous, highly strung sufferers from angina of effort than in those of a more phlegmatic disposition. The underlying coronary disease is present in both classes, and in most, in addition to the spasmodic attacks of severe pain, there is present the quantitative dull pain and sensation of constriction characteristic of angina of effort.

Spasmodic Angina

In spasmodic angina the patient is seized by a paroxysm of severe pain. This has the same distribution and radiation as that described as occurring in angina of effort. It varies from the latter condition in certain important clinical particulars. The onset is sudden, the degree of pain is maximal from the first minute or so of the attack, and there is frequently a sensation of impending death; no fear of death is felt, for such is the agony of the pain that any release would be gladly sought. The pain is agonizing, strangling, constricting, or tearing, but is less severe than that felt in some cases of coronary thrombosis. The onset of the attack is determined by such things as emotion, a cold wind, or previous exercise. Exercise has a different relation to the pain in spasmodic angina from that found in angina of effort. Over-exertion may, so to speak, pull the trigger, or a period of over-exertion during the day may produce an attack, or a series of attacks, in the evening. There is no direct quantitative relationship. The attacks not infrequently arise at night, but whether they then result from slowing of the circulation rate during sleep, from the flat position in bed, or from some abdominal cause, either digestive, reflex, or mechanical, is still undetermined.

During the attack the patient is pale and anxious, and prefers to remain quite still. Sweating is frequent. The appearance suggests a slight degree of cyanosis. An outstanding feature which is of clinical help in distinguishing spasmodic angina from coronary thrombosis is the relief produced in the former condition by the inhalation of amyl nitrite. This drug has no analgesic action in coronary thrombosis.

The mechanism of production of the attack of spasmodic angina is obscure. It is true that sufferers from the condition are nearly always of a highly reactive nervous temperament. It is rare to find a really phlegmatic patient who suffers from it. The sudden onset, the sensitive nervous system of the patient, the fairly frequent association between spasmodic angina and Raynaud-like spasm of peripheral blood vessels, and, finally, the dramatic effect of the antispasmodic drugs of the nitrite group, are all evidence in favour of spasm as the mechanical cause of the attack.

Presumably this spasm would interfere with the blood supply to the myocardium, and there are three ways in which ischaemia of the heart muscle could be brought

about as a result of it. It is possible that a diseased coronary branch, not yet rigid, could go into spasm and produce ischaemia. In this case the spasm would occur in the main branch and near the area of disease. A second theory would be that the terminal arterioles of a diseased coronary branch would go into spasm, there being a state of irritation resulting from anoxaemia, or possibly from some secondary trophic change in the local nerve endings. Thirdly, if an area of heart muscle is relatively ischaemic from disease of its nutrient artery, it is possible that the arterioles entering the area as anastomotic branches from the adjacent normal coronary branches might go into spasm as a result of some essential chemical or physical difference between the normal area of muscle and the area less well supplied with blood.

There are thus two ways in which amyl nitrite might produce relief: either it could relax the spasm of a diseased but not rigid vessel, or it could increase the lumen of the anastomosing collateral arterioles. In the latter case the ischaemia produced by the diseased vessel would be removed by the increased blood flow obtained from the relaxation of these anastomosing vessels. Clinically speaking, true spasmodic angina is a symptom of importance. If it becomes established as a fresh event in a case of angina of effort the prognosis appears to be materially worsened. If it occurs in syphilitic aortitis or in aortic incompetence the prognosis is also worse.

Since this condition has, until the last few years, been confused by all observers with either coronary thrombosis or with angina of effort, no exact knowledge as to its prognosis or its mechanism has been accumulated. The statements in textbooks regarding its immediate fatality must therefore be accepted with reservation until further information is available.

Coronary Thrombosis

The pain of coronary thrombosis has already been referred to, for comparison, in the description of the other types of cardiac pain. The pain is due to the abrupt blocking of a branch of the coronary system, with the resulting anoxaemia, degeneration, and fibrosis of the corresponding muscle area. The onset is therefore abrupt, and the general tendency is for the pain to be severe at first and slowly to subside over a period of days. The pain may be continuous or may occur in waves, with periods of remission. In either case the average period during which pain is suffered is from four to eight days. The type and the severity of the pain vary greatly.

It is generally true that when the pain is very acute the lesion is extensive; the converse, however, does not hold, for a fatal thrombosis may be preceded by a comparatively mild attack of pain. In severe cases the pain is tearing, rending, or compressing, is unbearably acute, and is accompanied by a sensation of impending death. It is generally severe, but sometimes is so mild as to be mistaken for a somewhat obstinate attack of flatulence. In rare cases the thrombosis may occur entirely without pain. The manner of onset, duration, and diminution of the pain are features of greater diagnostic value than is the severity.

The situation of the pain varies considerably. In characteristic cases it is substernal or precordial, and radiates to one or both arms, generally the left. It may, however, remain central in position, or radiate through to the back. In other cases it is felt in the upper epigastric region, and may then simulate gall-stone or gastric colic. A relation to respiration is not very infrequent; generally it is only a deep breath which evokes the twinge of pain, but rarely the respiratory relationship is so definite as to suggest a true pleurisy or a lobar pneumonia. Dyspnoea and cyanosis, fever and leucocytosis are generally to be found if looked for.

The pain is not relieved by amyl nitrite, morphine being necessary, often in repeated doses. Coronary thrombosis may occur in an individual who has had no previous symptoms, or may supervene during the course of angina of effort. In the latter case the patient recognizes the two types of pain as being perfectly distinct.

"Pseudo-angina" (Angina Innocens)

This label is unsatisfactory, on philological grounds, for the pain is anything but false. The writer suggests the term "angina innocens," by analogy with "diabetes innocens." The syndrome referred to occurs in nervous individuals, and is characterized by precordial pain, nervous fatigue, and lassitude, there being no evidence of organic cardiac disease.

The pain is of two types, persistent and spasmodic. Either or both types may be present in any one patient. The persistent pain is often nothing more than a dull ache, which is precordial in position and which may radiate into the arms. The pain has no quantitative relation to exertion, and may not occur at all during exercise. The individual is of the type who "lives on his (or her) nerves," being often capable of considerable exertion, provided it is undertaken for some exciting or pleasurable reason. Afterwards the patient collapses and the precordial pain is greatly increased. The pain is persistent, to a greater or lesser degree, over quite long periods of time—days, weeks, or months—but is accentuated by periods of overwork or by shorter spells of over-exertion. It may not amount to more than a dull ache. An accentuation of pain is felt after the exertion is over; there is no warning tightness or pain immediately the level of exercise rises above a certain measure, as is the case in angina of effort.

Sometimes a second very characteristic type of pain is also complained of. It is alarming both to the patient and to the physician. The patient is generally standing at the time, and may be engaged in some such exertion as washing the face. Suddenly an acute attack of precordial pain occurs which may or may not radiate to the arms, or indeed to the back and legs in addition. Within a few seconds the patient begins to feel faint, and then loses consciousness. This part of the syndrome is like that suffered from in an ordinary syncopal attack. There is no sudden abrupt loss of consciousness, as in epilepsy, and no convulsive movements or other epileptic phenomena are observable. Such attacks do not occur with great frequency. Three or four may have been suffered from in the course of a year or two. They are followed generally by an accentuation of the symptoms of lassitude and general fatigue complained of by the patient.

Other signs suggestive of a neurasthenic condition of the cardiovascular system as a whole are present in these patients. Palpitation and consciousness of the heart-beat are common, precordial tenderness invariable. It may be said here that precordial tenderness is also common in spasmodic angina, but is very unusual in simple angina of effort. Its absence, then, is evidence of an organic cardiac lesion. Fatigue is complained of rather than dyspnoea in angina innocens; standing is more trying than walking. The condition is frequently the result of a true nervous fatigue, which, in its turn, may or may not be the result of some long-continued physical abnormality such as chronically infected tonsils. Psychological stresses are frequent, and in a number of cases a history of a "nervous breakdown" is found; in some cases a more serious mental lapse has previously occurred.

In many cases physical signs of a labile vasomotor mechanism are demonstrable, the pulse rate being considerably raised and the blood pressure lowered in the standing posture, as compared with the readings taken when the patient is lying flat.

Special Conditions**SYPHILITIC CARDIAC PAIN**

Syphilis is a disease of the aorta rather than of the heart; but the oedema and fibrosis produced in the aorta does effectually interfere with the efficiency of the coronary circulation by decreasing the lumen of the vessels where they pierce the aortic wall. A further effect of syphilis is the production of an aortic incompetence. Patients with aortic syphilis suffer from three types of pain, excluding that produced by a definite aneurysm—these are: angina of effort, spasmodic angina, and nocturnal pain.

The angina of effort is identical in every particular with that already described, but it is found typically in a patient who has no signs of hyperpiesis or arteriosclerosis, and in whom, if there is no aortic incompetence, the heart is not enlarged. The typically organic nature of the pain in an individual who has apparently no other evidence of organic cardiovascular disease has led the writer not infrequently to suspect aortic syphilis, the diagnosis being ultimately confirmed. If the disease is discovered at this stage full anti-syphilitic treatment will remove the anginal pain, render the Wassermann negative, and restore the patient to apparently normal health. The syndrome is thus one with which medical men should be familiar, for its recognition will enable treatment to be begun at a stage where a good result is still possible.

In patients with a syphilitic aortic regurgitation, in addition to angina of effort, a spasmodic angina is sometimes found. This is symptomatically identical with spasmodic angina in non-syphilitic cases. A nocturnal pain, recognized sometimes by the patients as being of a cardiac nature, is not uncommon in syphilitic aortitis. It is generally central, and may be felt in front under the sternum, or may be referred more to the back. Occasionally it is affected by posture. It is somewhat indefinite in its characteristics, and is more remarkable for what it is not than for what it is. It is not quantitative to exercise, as in angina of effort; it is not severe and radiating, as is spasmodic angina; it is not persistent, as is coronary thrombosis. It is, however, an intrathoracic pain, noticed chiefly at night, and sometimes referred to the heart. The writer regards it as a manifestation of the aortitis. Its presence in an individual with angina of effort is always suggestive of syphilis of the aorta.

AURICULAR FIBRILLATION AND CARDIAC PAIN

It is a curious fact that cardiac pain is generally not present in patients whose auricles are fibrillating. It has been remarked by many observers that, when auricular fibrillation supervenes in a patient who has hitherto suffered from anginal pain, the latter symptom disappears. The reason for this is not understood.

A case of the writer's bore out this same fact in a very striking manner. The patient was a particularly intelligent individual, and a lawyer by profession, so that reliance can be placed upon his evidence. He was seized by a severe and painful attack of coronary thrombosis, proved electrocardiographically. The rhythm was regular. On the second day of his illness he was conscious of two facts. The first was that the heart was thumping and fluttering irregularly, and the second was that simultaneously the cardiac pain had disappeared. He remarked on the coincidence himself.

It is difficult to understand why auricular fibrillation and cardiac pain so rarely coexist. Very considerable further observation is necessary on the point.

CARDIAC PAIN IN MITRAL STENOSIS

Sudden severe attacks of pulmonary congestion, associated with paroxysmal dyspnoea, are not infrequent in advanced cases of mitral stenosis. These attacks are sometimes associated with very severe pain of an anginal

nature. They occur in children as well as in adults. The onset is generally while the patient is at rest, for such patients are generally bed-ridden. Dr. Lees of Great Ormond Street found that full doses of atropine sulphate 1/50 grain, or even 1/25 grain, would relieve the pain.² Some degree of cardiac pain is occasionally found in ambulatory cases of mitral stenosis. It is vaguely increased by exertion, but there is no exact relationship to this.

Conclusions

An attempt has been made to state clearly and concisely the clinical characteristics of the various types of cardiac pain as observed by the writer over a period of seven years. A number of such cases have been followed up throughout this period.

Stress is laid upon the necessity in all such cases of carefully taking the history and analysing the symptoms. Unless such a differentiation is made, research work upon the prognosis, or upon the value of various therapeutic measures in cases of cardiac pain, is founded upon an insecure basis, and is likely to be misleading or fruitless.

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PAROXYSMAL NEURALGIC TIC AS A SEQUEL OF TRIGEMINAL NEURITIS*

BY

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The term "trigeminal neuralgia" has been used mostly to designate a particular form of paroxysmal facial pain limited to the distribution of the fifth cranial nerve, and unassociated with any clinical sign of damage to the nerve, such as anaesthesia or motor palsy. Various other names have been given to this complaint, such as trifacial neuralgia, tic douloureux, and major trigeminal or epileptiform neuralgia; but tic douloureux, though expressive, is also used by many persons to designate the pains of migraine and other periodic headaches. Moreover, now that paroxysmal glossopharyngeal neuralgia is becoming better known, the term tic douloureux is equally applicable to that complaint, in which the pain, though somewhat different in distribution, is identical in character with that of trigeminal tic.

In order to distinguish sharply these paroxysmal neuralgias, which are not due to any gross lesion or irritation of these nerves, I propose the names trigeminal tic and glossopharyngeal tic. By the use of these terms it will be impossible to confound these characteristic neuralgias with the minor or atypical neuralgias, though it is true that gross irritation of the fifth or ninth nerve by tumours at the base of the brain have occasionally set up paroxysmal neuralgia not to be distinguished from trigeminal or glossopharyngeal tic, as in the cases described by Weisenburg¹ and by Parker.² Typical trigeminal tic is also met with as an occasional complication of disseminated sclerosis. Chronic facial neuralgia, often with severe paroxysmal exacerbations and intense hyperaesthesia of the skin, is a common sequel of trigeminal zoster in old people. Lastly, severe paroxysmal facial neuralgia is a rare sequel of medullary thrombosis involving the spinal root of the fifth nerve, as in cases of thrombosis of the posterior inferior cerebellar artery.

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