PRECORDIAL PAIN

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During the 1939-45 war, the incidence of precordial pain increased, organic disease accounting, in my experience, for rather less than half the cases. Often the diagnosis is difficult because of misleading symptoms and a paucity of clinical signs. Sometimes the symptoms alone make the diagnosis easy; and in between these extremes are cases of such a curious mixture that it is fair to conclude that in the past the combination of both functional and organic factors has not been sufficiently emphasized.

Common Causes of Chest Pain

The commonest cause of pain in the chest is the functional disorder usually termed benign cardiac pain or angina innocens, but better described as *left-sided infra-mammary pain*. It is due to an anxiety state and is discussed below.

Pectoral fibrositis is very common and is diagnosed not only by the presence of tender areas elicited by deep palpation but also by the patient's statement that the pain arises on movement of the thorax and on muscular contraction in the affected area. When the upper part of the chest is involved, it is possible to produce referred pain in the arm by pressing on the painful site. In many instances there has been strain of the pectoral tissues and a toxic state provokes persistence of the symptoms and signs. Introspection following strain of the pectoral muscles and giving rise to chronic tenderness and aching of the left breast is regarded by Mendlowitz¹ as the second most common cause of pectoral pain in American soldiers and recruits to military service.

Arthritis of the shoulder-joint is not infrequently associated with fibrositis and should always be sought in suspicious cases with pains in the upper part of the chest.

True angina and *pain from coronary thrombosis* are dealt with below.

Paroxysmal tachycardia (with its coronary insufficiency), *pericarditis*, and *pleurisy* are not difficult to exclude by their special characteristics. Here one may refer to those cases of coronary infarction where deep breathing also causes pain in the precordial area as the result of pericardial involvement.

Mitral stenosis is sometimes accompanied by

left-sided infra-mammary pain. The statement that pain in this area is never due to heart disease is incorrect. Though it is usually a symptom of neurosis, when it accompanies mitral disease pain is probably the result of both mental and physical fatigue.

Apart from invasion of the spine, pleura and adnexa by neoplasm, other causes of pectoral pain are not common. A *pneumothorax* causing pain and dyspnoea, especially on exertion, is easily confused with true angina particularly when there is a partially collapsed left lung difficult to diagnose on clinical examination. A sudden onset in particular simulates a thrombotic attack.

Left-sided Infra-Mammary Pain

Nomenclature. This syndrome was first described by Da Costa (1871) during the American Civil War and was often associated with dysentery. During the 1914-18 war, it came into prominence once more under the heading of D.A.H. (disorderly action of the heart), irritable heart, effort syndrome, soldier's heart, and neuro-circulatory asthenia; but these descriptions do not always fit the bill. There may be no disorderly action, such as tachycardia or irregularity of the heart-beat, and occasionally no complaint of dyspnoea. The syndrome occurred in civilians in the 1939-45 war more than in soldiers,² for the obvious reasons that past experience had taught us to weed out recruits undergoing medical examinations who showed any likelihood of this disorder, and because the civilian population were subjected not only to real warfare but also to general anxiety.

Aetiology. Left-sided infra-mammary pain is commonly encountered in overworked and highly strung people. In peace-time, females with hypertension form the majority of such patients, but during 1939-45 a large proportion has consisted of males aged seventeen or more. Cardiologists consider that the condition is due to mental fatigue and not to coronary insufficiency. A strained pectoral muscle can initiate fear, with psychosomatic disturbance, and infection lowers the resistance and morale. Certain it is that reassurance can effect a cure, provided the mental stress at the same time is removed.

Infection (focal sepsis) appears to play a part.

In contradistinction to the association with dysentery noted by Da Costa, I have found pharyngeal infection to be the most constant toxic factor.

Experiments point to the hypothalamus, with its sympathetic centres, as the probable site of disorder. The auriculo-ventricular conduction time can be lengthened by stimulation of the hypothalamus in dogs; and sweating and even ulceration of the duodenum can be produced by traumatic lesions of the hypothalamus in man.

Symptoms

The patient usually complains that pain arises in or under the left breast, coming on at any time, day or night, and sometimes waking him in the night, though he is more likely to wake first. The pain is described as stabbing, throbbing, burning, and lasting hours, sometimes all day. It may arise with, but usually after, effort; but in so doing it tends to persist and is not relieved by cessation of activity. It often spreads to the left arm, usually the inner border, and this limb may feel numb and useless; sometimes the pain extends to the left side of the neck. I have only twice known the pain to spread to the right arm; it has been stated that such a spread excludes the psychosomatic illness and indicates true coronary occlusion, but this is incorrect.

Occasionally the symptoms overlie true angina, and then the patient's story is of real service; his attitude and reaction will probably give the clue to the assessment. It is in such cases that we tend to make a wrong diagnosis of pure neurosis, the symptoms of true angina being overshadowed.

Signs

There is not infrequently *tenderness to pressure* over the area of pain. Thomas Lewis³ pointed out during the 1914-18 war that pinching the sternomastoids, trapezii, and pectorals provoked more discomfort on the left side.

Hyperaesthesia has often been described. I have found the left mammary tissue, in both male and female, tender to the grasp, and believe that the hyperaesthesia of the left chest described by various authorities probably refers to tenderness on deeper palpation. True hypersensitiveness of the skin is rare.

Electrocardiography and *radioscopy* reveal no signs of disease. The *exercise-tolerance test* may be faulty, the systolic *blood-pressure* infrequently raised, *tachycardia* present, and a soft *systolic murmur* detected at the apex of the heart, none of these features being contingent upon organic defect.

Treatment

It is a pity that the word 'angina' is used so often in connection with what is described as benign cardiac pain; it is better to discard a name which to the layman is associated with the dread of sudden death. In fact the terms 'angina,' 'tired heart,' and 'flabby heart-muscle' should never be mentioned to the patient, for they may haunt him for the rest of his life. Herein lies the first principle in the treatment—reassurance. To the intelligent inquiring patient it must be explained that there is no organic disease, and that the symptoms, though real to him, are the result of fatigue and anxiety. This is often a difficult pill for the patient to swallow. Likening him to a sensitive wireless set, tuned in to distant stations and reacting accordingly, compared with the inferior models, does much to satisfy his doubts. It is explained that if the mind is tuned in to discomforts they become so much more apparent and must be faded out and in this way eventually obliterated. I have known an understanding talk with a patient abolish the precordial pain at one consultation. Moreover, once the diagnosis has been established, further cardiac examinations are redundant and harmful, provoking introspection by raising doubts as to the state of the myocardium.

Some patients need physical rest, but rest in bed is bad for those with nothing but an anxiety state. It is a different story where the neurosis is superimposed on organic pathological conditions such as hypertension, valvulitis, and coronary dysfunction.

In any case sedation is necessary. Bromides are a good standby though used far too indiscriminately; if they are given for too long they may cause depression, lack of concentration, and even incoordination with nystagmus. Restlessness and insomnia may require barbiturates, particularly in a hypertensive patient. It is wise to substitute a non-barbiturate from time to time, such as carbromal, or bromo-iso-valeryl urea (B.V.U.); isobrom is a happy combination of these latter drugs with enhanced action, and can be used during the day with little narcotic effect. For those who cannot sleep on retiring a quick-acting sedative is best, such as Seconal or Carbrital. Hexobarbitone is rapid in action but doses larger than 4 gr. may upset the digestion and cause vomiting. Evidorm, a combination of quick and slow acting barbiturates, has a rapid and more lasting effect. Digitalis is often useless in cardiac neurosis except for its vagotonic effect in damping down the pacemaker at the sino-auricular node in patients with tachycardia and overaction of the sympathetic supply. Small doses of belladonna also stimulate the vagal nerve endings and slow the heart-rate; large doses paralyse and provoke tachycardia.

True Angina

Aetiology. True angina, coronary pain, effort angina, or angina pectoris occurs usually in middle age, mainly in men, but has been observed in the fourth decade, and I have described it in a child aged fourteen with progeria (premature senility).⁴

Angina pectoris should be regarded more as a syndrome than a disease, for it may accompany spasmodic conditions of the arteries further afield, as in intermittent claudication ; it may occur in people without evidence of arterial disease ; and it is a symptom found not only in atheroma of the coronary vessels but also in thrombosis and infarction of those vessels, in syphilitic endarteritis, and in rheumatic aortic lesions. It also occurs in advanced anaemia and in untreated myxoedema, from myocardial anoxaemia, when the pain is caused by the toxic effects of accumulating metabolites.

The precordial discomfort is nearly always caused by effort ; but emotion, cold, or dyspepsia may also bring it on. The pain is quantitatively related to effort and is increased by fatigue or anxiety and lessened by a carefree holiday. A cardiovascular lesion is surely not responsible for these changes ; it is the overlabile autonomic system and the exaggerated response to stimuli with increased vasoconstriction or failure of a vasodilator response of the coronary arteries which causes the ischaemia. There is therefore an additional qualitative factor. This overactive autonomic system explains why the effect of overwork and fatigue, with inadequate leisure, invokes angina ; and why the business man during the week develops pain on walking to the railway station, yet can play a game of golf without discomfort and be free from attacks while on holiday.

Only 15 per cent. of patients with calcification of the coronary arteries have been known to report an anginal syndrome. Few persons with this complaint experience pain on going upstairs or walking about their business premises ; some can even run upstairs without discomfort, but it is the slow incline that catches them out. The fact that belching seems to relieve the pain suggests associated gall-bladder disease, but belching probably denotes the cessation of the attack. Oesophageal spasm may also be present ; it can be regarded as a counterpart of anginal pain, for it can be prevented or relieved by nitroglycerine. The statement that this eructation is due in most cases to air swallowing is erroneous. Some patients, thinking their symptoms are due to dyspepsia, swallow air while attempting to dislodge the wind, but they are in the minority. It is also a fact that many patients with angina of effort do not complain of associated flatulence. On the other hand, true angina is often associated with

gall-bladder disease, which then excites the angina and can be likened to the trigger of a loaded gun. Cases are on record where cholecystectomy has abolished angina for a long time. Similarly, in some cases the provoking factors are a distended colon or stomach, the result of overfeeding or of indigestion.

Anginal pain can be incited by exposure to cold, as in coming out of a warm theatre or walking from a warm room to a cold one, and then it is not the effort but the alteration in temperature which induces the attack, the result, no doubt, of a rise in the blood pressure which increases the work of the heart. Getting into bed with cold sheets has a like effect. This pain must be regarded as of spasmodic reflex character and associated with coronary spasm.

Tobacco is a rare primary cause of angina, yet the symptoms at times are relieved by abstinence, especially from cigarette inhaling. It seems feasible that some people may become more sensitive to tobacco with advancing age. Coronary restriction can be produced experimentally in animals with nicotine. Repeated arterial spasm is likely to lead eventually to arteritis and thrombosis.

A special type of angina develops during anaemia and is then due to the smaller supply of oxygen to the muscle when an extra call is made on the heart. This can be cured by restoration of the haemoglobin level. Any anaemia may cause this symptom, whether from haemorrhage, pernicious or hypochromic anaemia, or leukaemia ; but young people and children with severe anaemia do not get angina.

Campbell⁵ holds the view that people with this type of angina will eventually develop true angina of effort, believing that the anaemia alone cannot cause the angina unless associated with coronary sclerosis. Other cardiologists incline to the belief that the lack of oxygen suffices to explain the pain, in the absence of coronary disease.

Angina has also been described in association with thyrotoxicosis and with myxoedema ; it has been alleviated by thyroidectomy in thyrotoxicosis and by thyroid administration in myxoedema. It must be remembered that, when thyroid extract is administered in overdosage, a state of overaction of the sympathetic is induced, with the consequent tendency in some people to coronary spasm.

Symptoms

True angina used to be regarded as a sudden severe pain associated with a sensation of impending dissolution. We know now that the pain may be mild, even a dull ache, and rarely associated with the fear of death. It is in the so-called vasovagal syncopal attacks and in sensory epilepsy that this dread is more commonly experienced.

There is always a positive history of the symptoms produced by effort and relieved in a short space of time by rest or by nitroglycerine; frequently aggravated by exposure to cold or effort after a meal. Cold raises the blood pressure and throws greater strain on the heart; while a full stomach with increased blood supply reduces the coronary flow.

Some patients get pain first thing on making an effort after breakfast, and then are relieved for the rest of the day. There is a story of a doctor who preferred to wind up his car and get the trouble over rather than have the symptoms tending to persist over a longer period. Other patients complain mainly at the end of the day when they are tired; in this instance the sympathetic nervous system comes strongly into play.

Some patients lose their angina for a time. This fact does not indicate an error in diagnosis. The threshold for pain has been lowered by the removal of anxiety and consequent overaction of the sympathetic nervous system. On the other hand, patients cease to experience effort angina after sustaining a myocardial infarct, presumably because the pain-producing area has necrosed.

An objection to the term 'precordial' is that, whereas true anginal discomfort is often sternal in site, it is usually felt across the upper chest, or arises in areas to which the pain may spread. I have known anginal pain arise in the left wrist or both wrists; in the left arm or both arms; in the back or nape of neck; though in almost every case it was also experienced across the upper chest. The pain may spread to the throat, especially to the left jaw, and even the left side of the face and to the left eye and behind the ear. When this pain spreads from the upper sternum to the right arm it is almost proof of coronary occlusion. Very rarely is pain felt in the right arm in cardiac neurosis, but it must be remembered that anginal pain may on rare occasions be described only in the right upper chest and right arm.

When angina is due to syphilitic occlusion of the mouths of the coronary arteries (in contradistinction to the usual atheroma extending along the coronary vessels), the pain on effort tends to last longer, is not soon relieved by rest, is often more spasmodic, and is associated with nocturnal paroxysmal dyspnoea and angina decubitus. These nocturnal symptoms are the outcome of the mechanical effect on the coronary flow, relative to pressure from a dilated aorta in recumbency. It must be remembered, however, that syphilis does not always cause dilation of the aorta. Incidentally, in syphilitic angina it is much more important to treat the heart before the syphilis, for syphilitic tissue is better than none.

Diagnosis

The diagnosis can frequently be made from the description of the symptoms alone. Clinical examination may not reveal any significant sign in the heart, arteries or blood pressure. There may be no evidence of cardiac failure. Radioscopy has frequently revealed a normal size and contour of heart, and aorta, in undoubted cases. Electrocardiography may not reveal any sign of a pathological state of the heart muscle until permanent occlusive changes arise. In doubtful cases it is advisable to repeat electrocardiography immediately after exercise or after inhalation of 10 per cent. oxygen, thereby provoking precordial pain. By these tests, which should be accomplished within three minutes of the onset of pain, one may observe an alteration of the complexes in the electrocardiogram, revealing signs of temporary muscle defect. Injections of adrenalin will provoke angina but the concomitant risk of ventricular fibrillation forbids its use as a test. Confusion sometimes occurs in the interpretation of an inverted T-wave in lead I in hypertensive patients. Intravenous injections of potassium salts will cause the T-wave to become upright in pure hypertension, whereas they will accentuate the inversion in coronary occlusion. Unfortunately this procedure is not without risk, since potassium salts given intravenously slow the conduction mechanism of the heart.

I would refer briefly to those unfortunate people with para-oesophageal hernia. This hiatus hernia provokes three types of symptoms: chiefly gastric, occasionally respiratory, and not infrequently precordial pain simulating angina. The common occurrence of pain with flatulence after meals, the persistence of the pain or discomfort at rest, and the daily recurrence over long periods will suggest this abnormality. To add to the confusion I have found that nitroglycerine will give relief by its antispasmodic effect on oesophageal tissue, in the same way as inhalation of octyl-nitrite.

Prognosis

I believe that patients without true pain, especially where there is no spread to the neck and jaw, or where the discomfort is mainly one-sided, and particularly those who can walk off their symptoms and have no sense of constriction or suffocation, have a more favourable prognosis.

Spasmodic angina, though little understood, sometimes has a favourable prognosis, especially in the emotional type where effort does not play so important a part. Vasodilators are not always successful in its treatment, and then the symptoms run their course.

Angina of old age does not appear to alter the

course of the natural span of life, possibly because of the development of multiple anastomoses in the circulation of the myocardium.

A patient with angina very rarely dies in the first attack ; he may live, with recurring attacks, for 15 years or more. Death is usually associated with coronary thrombosis, not necessarily occurring at precisely the same time as the vascular clotting, for other factors come into play, such as ventricular standstill and fibrillation, contingent upon involvement of the conducting mechanism associated with coronary spasm. Such spasm no doubt at times may be widespread involving coronary arteries without marked atheroma. The prognosis is influenced to a large degree by the mental reactions of the patient to his complaint, by the care taken in regard to habits and diet, and by the history of thrombotic attacks.

Treatment

There are few conditions where advice is so strongly needed about general hygienic measures and regulation of the patient's life. For prophylaxis he must take fresh stock of himself and, according to the severity of his symptoms, limit his activities, especially those associated with overstrain.

At the outset it is best to explain to the patient that, with age, there is a lessening of cardiac reserve, and this takes the form of warnings that he is overstepping the boundaries of his heart's capacity for work ; that if he limits his physical and mental effort he will be freer from the pain or discomfort, and he will need to live within the limits of his tolerance. 'Regular habits' and 'going slow' should be his watchwords. He should take longer over his dressing in the morning, not hurry over his breakfast ; it may even be necessary for him to get up half an hour earlier. Then he should rest before and after meals. We often hear the patient's story of pain after breakfast when effort is needed to catch the train or bus, and how much more distress there is if he has to walk against a cold wind. He must stop when pain arises, and must curtail his future efforts to a point short of the production of this pain. It is wise to try and break the habit of this pain-producing effort, for by walking slowly the patient may be able to live for years without requiring vasodilators ; and, when he finds freedom in this way, there may appear in time less tendency to pain.

If any special effort is required, or if he knows that a certain effort must be made which usually brings on his pain, a tablet of nitroglycerine should be chewed a few times and allowed to be absorbed from under the tongue just before such an effort. The nitroglycerine is more rapidly effective taken

in this way than when swallowed. For a longer action which need not start so quickly it is better to swallow the nitroglycerine. The tablets should be fresh. Patients are sometimes afraid of taking these tablets, either because they produce throbbing in the head and slight giddiness or headaches in those with so-called hypotension, or from a fear of drug taking. As a rule by reducing the dose one can discover the requisite amount to relieve the symptoms without causing unpleasant effects. It is wise to start with a small dose, especially for patients with the minor manifestations ; even 1/200 gr. may be found efficacious. Larger doses, say 1/150 gr. or 1/100 gr., may be necessary later.

Food must be carefully chewed ; the stomach cannot take over the duty of the teeth. Indigestible food must be avoided. By the age a man develops angina he should know what disagrees with him. Small meals at short intervals should be taken rather than larger meals at longer intervals.

Avoidance of gastric and colonic distension is very important. Much has been written about the harmful effects of alcohol and tobacco. If alcohol suits the patient, a moderate amount of it, particularly in the evenings, does no harm. Cassidy⁶ declares that tobacco has little if any effect on coronary disease, but he admits that cigar smoking has been known to induce intermittent claudication. It is therefore wisest to prescribe moderation in smoking and avoidance of inhalation. Vasoconstriction can be caused by nicotine, and some anginal subjects do get pain after cigarette smoking. We cannot altogether exclude the psychological effect where the patient has been forbidden this luxury and fears the consequence of indulging it.

I am not convinced of the efficacy of nicotinic acid. Experimentally, a high dosage is required to promote vasodilation of the coronary vessels, and then tingling and pricking sensations become very unpleasant. Nicotinamide produces less side-effects but probably also less vasodilation.

The same may be said of testosterone therapy ; great claims were made in the United States of America, but they have not been confirmed in this country. Vitamin E has also recently been in vogue but without convincing testimony.

We should also resort to psychotherapy, which can do much for a hypersensitive patient ; he may even lose his angina for a time.

Cardio-omentopexy has gone out of favour, though some good results were recorded, presumably by supplying fresh channels from the vascular tissues of the omentum to the surface of the heart.

Thyroidectomy is now little heard of in the treatment of angina. A series of cases published

some years ago gave promising results, presumably by lowering the basal metabolism. In a proportion of the cases thyroid feeding became necessary owing to the development of myxoedema, which has also been known to provoke angina.

I am investigating the effects of propyl-thiouracil but have not yet given it an extensive enough trial.

Cervical sympathectomy has given good results. It was once believed that abolition of the pain in angina might place the patient in a more serious state, since he would make an effort, possibly with a fatal result, if he did not have the warning pain; this theory is no more accepted. Smithwick's operation, by which the lumbar and dorsal sympathetic ganglia are removed, is of chief value for the hypertensive patient with vasomotor angina.

Khellin,⁷ an Egyptian drug produced by extraction of the seeds of a plant (*ammi visnaga*) which grows on the shores of the Mediterranean, has been used for centuries as an antispasmodic in the treatment of renal and ureteral colic. Chemical analysis has shown it to be a di-methoxymethyl-furano-chromone belonging to the same group as the coumarines. Khellin causes a prolonged relaxation of all the visceral smooth muscles, especially the ureters, but it was not until 1945 that it was discovered that khellin acts as a coronary vasodilator without affecting the blood pressure. The drug can be obtained in this country from British Drug Houses as a tincture, and 1 dr. should be given three times a day; up to 3 dr. can be given as a single dose. Khellin also has a beneficial effect on bronchial asthma by inducing relaxation of the bronchial muscles.

Since infection appears to play a part in coronary disease, eradication of a toxic focus, if possible, should always be advised. Gall-bladder lesions are commonly associated with angina; both heart and gall-bladder are innervated by the vagus; and Rae Gilchrist⁸ believes that cholecystectomy is justified in many cases with obvious gall-bladder dyspepsia. A patient with angina usually undergoes such an operation well if a skilled anaesthetist is employed.

Coronary Thrombosis

Etiology. Coronary thrombosis develops usually after the age of fifty, but 11 cases have been recorded between the ages of twenty and thirty, ten of which were in males. There was no syphilitic infection in these; a raised blood pressure was found only in one, and signs of arterial disease were absent, apart from coronary atheroma. Miller and Woods⁹ reported one additional case in this country, in a patient aged twenty-two.

Symptoms

The pain of coronary thrombosis is not always

precordial, for it arises not infrequently in the epigastrium, suggesting acute dyspepsia, gall-bladder trouble, or a perforated peptic ulcer. In many cases, however, it is definitely felt over the heart, in the left mammary area, or in the mid-line, and it tends to spread to the shoulders, jaws, arms, back and nape of neck, just as in angina pectoris. Shock and circulatory collapse may be the only symptoms and signs of infarction, in which case severe pain may come on at a later stage.

There are at times prodromal symptoms of indefinite pains under the sternum or of aching in the left arm; these may exist for as long as 14 days before the attack, and are probably due to a small subendothelial haemorrhage in a coronary vessel leading later to thrombosis.

Frequently the first attack has been unrecognized and called dyspepsia or pleurodynia, and has been so slight that the patient has not even sought medical advice. Careful questioning may elicit the previous history of precordial discomfort on effort.

In contradistinction to angina of effort, the patient with thrombosis tends to be restless in an attack. He may move about, trying to secure a comfortable position. I have known a patient to get out of bed and, on her knees, bend forward on the floor, in a partially successful attempt to obtain some measure of comfort. In the early stages there are commonly nausea and dyspnoea.

It has been stated that even where there is no pain there is always dyspnoea. This is not quite true. I have certainly encountered severe pain without respiratory distress.

Signs

There is evidence usually of the various degrees of shock, many very severe with pallor, sweating and vomiting.

The pulse varies according to the pathological upset in the myocardium and conducting mechanism. One expects to find a weak pulse and lowered blood pressure; yet occasionally the pulse may be normal, and the condition of the patient fairly good. The pulse, which is usually of poor volume, may become slow by damage to the auriculo-ventricular bundle or to the sinus node, producing a wandering pace-maker, or it may be rapid from auricular or ventricular tachycardia. The abnormalities in auricular rhythm arise especially when the auricular branches of the coronary vessels are involved. Premature beats are commonly encountered.

It has often been stated that the blood pressure falls rapidly in coronary thrombosis; this is true in many cases, but sometimes it takes 12-24 hours before it drops. I have known a very dyspnoeic sweating patient with chest pain have a blood

pressure of 170 systolic, and be in consequence not recognized as a case of coronary thrombosis. The blood pressure in this case dropped to 120 and 115 mm. Hg. in 24 hours.

Electrocardiography in difficult cases can give the clue to the diagnosis usually in the three common leads, but occasionally only in the fourth lead. The electrocardiogram can be more easily interpreted a few days after the initial attack.

Pulmonary congestion must be looked for, and inquiry made about nocturnal wheezing, as this portends left ventricular failure. Attention should be paid to the development of an evanescent pericarditis and slight pyrexia with leucocytosis, especially on the third or fourth day, accompanied by a raised blood sedimentation rate.

Recurrent precordial pains following a coronary thrombosis suggest weakening of the cardiac muscle and the development of an aneurysm. This can be seen by radioscopy, sometimes in an oblique view, as a prominent immobile area.

Prognosis

Patients occasionally die suddenly in a first attack, but usually they either succumb during the first few weeks or make a good recovery for a while.

Untreated cases seem to develop angina of effort more readily than those adequately treated.

The amount of shock is a guide to prognosis.

Though the pulse may be good and the condition of the patient fairly good, death may ensue in a short time from further occlusion.

Clots forming on the endocardium over the infarcted area are responsible for emboli causing occlusion both in systemic and pulmonary circuits, for both ventricles can become involved with one infarct.

Fibrillation carries with it a graver prognosis.

Treatment

The most essential treatment of coronary thrombosis is rest in bed and alleviation of the pain and shock. For rapid relief of pain there is nothing so good as an intravenous injection of $\frac{1}{6}$ gr. of morphine or omnopon. This is supplemented by hypodermic injections, and as much as $\frac{1}{2}$ gr. of morphine may be found necessary, and morphine may be repeated every few hours in smaller doses. Omnopon also contains papaverine, which relieves spasm of unstriated muscle, but the amount present is too small to be significantly effective; 100 mg. of papaverine is a useful dose. Dilaudid has ten times the analgesic action of morphine, but the duration of freedom from pain is not so long. This drug should be used where there is idiosyncrasy to morphine. Sometimes the dilaudid provokes nausea and vomiting, and

then codeine phosphate, 1 or 2 gr., should be tried. If 2 gr. does not relieve the pain sufficiently, no success will be attained with larger doses. *Hyperdoric* morphine is also useful for prolonged action. Physeptone is under trial. This new drug (dl. 2 di-methyl-amino 4; 4 diphenyl-heptone-5 one hydrochloride) has spasmolytic and analgesic properties, making it particularly suitable for relief of pain of visceral origin. It may be administered by mouth or by subcutaneous, intramuscular, or intravenous injection. Weight for weight it is equal to morphine in its analgesic action; 10 mg. of physeptone produces an effect about equal to that of $\frac{1}{6}$ gr. of morphine, with relief of pain for three or four hours; 30 mg. of physeptone may be required for severe pain. The hypnotic effect and side-effects are less than those of the opiates. The relief of pain also combats shock.

Inhalation of oxygen may be necessary. The funnel method is wasteful. The B.L.B. mask is effective but can be trying to a restless dyspnoeic patient; in which case the nasal catheters on a spectacle frame are best. The oxygen tent is somewhat alarming to the patient and presents difficulty in nursing; it takes time to install and needs careful maintenance to prevent sudden lowering of oxygen concentrations.

Of the anticoagulants, heparin has the disadvantage of having to be given intravenously and requires laboratory control by the blood coagulation time. Dicoumarol can be administered by mouth, but again needs control by estimation of the prothrombin level. It is slower in action than heparin and probably safer. Both may, from overdosage, suddenly cause haemorrhage in various parts of the body as a result of severe hypoprothrombinaemia. The risk is particularly great with a patient suffering from coronary artery degeneration, where there is liability to subintimal haemorrhages. I consider the use of this drug is contraindicated where there is concomitant hypertension. From experiments carried out by Peters *et al.* (1946)¹⁰ it was estimated that the embolism or mortality rates were considerably reduced.

One should aim at giving dicoumarol on alternate weeks, in sufficient doses to lower the prothrombin level below 50 per cent. to 60 per cent. On an average the dosage employed is approximately 100 mg. a day; patients naturally vary with their reactions and some require as much as 150 mg. a day. I have known the prothrombin level to return to normal in seven days following the cessation of the administration of the drug. It must be borne in mind that the effects are cumulative with sudden development of severe hypoprothrombinaemia; hence the advocacy of using alternate weeks for the administration

of dicoumarol, as a safeguard in prolonged therapy.

Of coronary vasodilators the nitrites lower the general blood pressure; this must be avoided when dealing with coronary thrombosis. Papaverine hydrochloride has little toxic effect and apparently is not habit-forming. I have used it with apparent success but have not yet been able to obtain the synthetic compound, eupaverine, which is said to have a greater antispasmodic effect. The dose of papaverine is usually 100 mg. two or four times a day. It has practically no narcotic or depressing effect on the circulation.

Aminophyllin is one of the xanthine groups; it has popularity as a coronary vasodilator, and McMichael¹¹ has urged its use as an augmentor of cardiac output in left ventricular failure; he has proved this effect by catheterization of the right auricle. This drug is very effective in reducing venous pressure. Certainly patients with left ventricular failure improve after administration of aminophyllin when given either by mouth or parenterally, preferably the latter. There is evidence to show, however, that it should not be used in coronary thrombosis during the period of shock, since it may produce a fall in blood pressure and tachycardia. Intravenous administration should particularly be avoided where a lowering of blood pressure may produce ill effects.

Ventricular tachycardia, strangely enough, appears to be promoted by digitalis therapy. Unless there are signs sufficiently significant of cardiac failure, digitalis should not be prescribed. Some cardiologists say it should always be avoided, especially since recent investigations have provided evidence that there is increased coagulability of the blood after digitalis therapy.

Quinidine is the best drug to restore normal rhythm when auricular fibrillation develops, and it should be combined with digitalis when accompanied by congestive failure. It should not be used if a conduction defect is present; it retards impulse formation at the sino-auricular node,

slows conduction, and lengthens the refractory period, but it prolongs the prothrombin time.

Extra-systoles can usually be abolished by giving quinidine, 3 gr. a day, or papaverine, 100 mg. three times a day.

Mercurial diuretics may be found more beneficial than digitalis in congestive failure, provided the kidneys are not diseased. The drug should be administered cautiously; because sudden death has taken place after larger doses, especially where the kidney tubules are degenerated.

The patient should be kept in bed for a month in mild cases, or six weeks in more serious cases. Bed-pans are usually essential. Occasionally the patient cannot manage a bed-pan, and then the best procedure is to bring the level of a commode in line with the bed. The patient slips his legs outside the bed and is easily lifted on to the commode. Returning to bed offers no great difficulty.

After the patient is allowed up, he should stay three months away from his usual work.

Summary

The common causes of precordial pain are discussed with special reference to left-sided infarct-mammary pain, true angina and coronary thrombosis.

The aetiology, diagnosis, prognosis, and treatment are dealt with in respect of each disease.

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