

Observations ON ANGINA PECTORIS

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Angina pectoris is not a specific disease of the heart, but merely a single symptom of varied origin. If there is anything certain about it, it is its uncertainty. The initial seizure may prove fatal, or recurring attacks may continue indefinitely without gravely interfering with the business of life; but sooner or later it causes death. The striking feature of most diseases of the heart, even mortal disease, is the absence of pain. Anguish may be great from shortness of breath, a feeling of suffocation or consciousness of the heart beat, but actual pain is rare. In angina pain is the dominating symptom—"a suffering as sharp as any that can be conceived in the nature of pain"—and often accompanied by something beyond the nature of pain, a sense of impending death. The pain is as a rule related to the chest; it comes suddenly and goes suddenly. This may be the whole feature of an attack.

NATURE AND DISTRIBUTION OF THE PAIN

Angina varies greatly in its severity in different patients. Slight forms are common, the grave ones relatively rare. In the milder degrees it is only a slight uneasiness about the chest, with no particular localization or characteristics. There may be sensations of numbness, deadness, oppression, constriction, or tightness. It has been compared to a constricting band, or the action of pincers on the chest, and is usually described as dull. Whatever its character, in severe cases the agony may be exquisite.

The site of the pain is generally behind the centre of the sternum; but it may be referred to one side of the chest, usually the left. The pain most commonly radiates into the left shoulder and down the ulnar border of the arm as far as the elbow or fingers; less commonly into both arms; and rarely, into the right arm alone. It may pass upwards into the neck or backwards between the shoulder-blades. In exceptional cases it is referred to the epigastrium or the xiphoid cartilage, and consequently ascribed by the patient to some gastric disorder. For a short time after the attacks the affected arm may be weak; it may be so tender that blood pressure examinations are impossible; or it may be insensitive, though this is rare. Attacks of herpes in the painful areas, atrophy of muscles, and vasomotor phenomena such as pallor or cyanosis have been recorded. As a rule the character and the distribution of the pain in successive attacks remain constant.

Distribution of the Pain

	Cases
Substernal	95
Substernal radiating into the left arm...	36
Substernal radiating into the right arm	2
Substernal radiating into both arms	24
Substernal radiating into the neck	19
Substernal radiating into the back	8
Chiefly epigastric	11
Chiefly about the shoulder...	5
	200

Coinciding with the pain there is, not infrequently, a feeling of impending death, which may be more overwhelming than the pain itself. This *angor animi*, as Ryle¹ points out, is not a fear of death, but a sense of impending dissolution, often accompanied by a sensation of constriction of the chest and the feeling that any movement, thoracic or systemic, would prove fatal. Usually associated with pain, it may, however, occur

independently—the *angina sine dolore* of Gairdner. In one attack which we witnessed this *angor animi* was the prominent symptom, the patient being quite definite that he did not suffer pain. He died three years later from an infarct of the heart. In the typical attack the patient stops and holds on to some support, unable to speak, breathe, or move from the intensity of his distress; his face pale and covered with sweat, or unaltered save for the expression of his agony. The initial attack generally follows physical exertion. Several of our patients suffered for the first time when hurrying to catch a train, walking uphill or against a strong wind, or lifting an unusual weight. But it may ensue on ordinary exertion, particularly shortly after a meal. Emotional disturbance—joy, fear, excitement, fits of temper (as in John Hunter's case)—may also be the exciting cause. Cessation of the exertion is rapidly followed by cessation of the pain, but as time passes slight degrees of exertion or disturbance may bring on an attack.

This angina of exertion must be clearly distinguished from comparable attacks occurring during rest, the angina of decubitus (infarct of the heart). These may arise when the patient is quietly reading; or he may be roused from sleep by the pain. In most instances they succeed attacks of the angina of exertion, but occasionally they come without warning. In a series of 84 cases of infarct this unforeseen catastrophe occurred in 7 cases.

Duration of Attack

The duration of attacks varies. In the angina of exertion the onset and termination are usually abrupt, the seizure lasting from a few seconds to a minute or two. It rarely lasts for more than ten minutes, but we have seen one persist for forty-five. In the angina of decubitus the attacks may recur in quick succession for several hours (*status anginosus*) or may last for a day or more, notwithstanding liberal doses of opium. The termination of attacks is sometimes accompanied by the passage of large quantities of urine, or eructations; but this is unusual. Following attacks the patient as a rule feels exhausted for a time, and then quite well save for the remembrance of his agony.

Such are the usual symptoms in the initial attacks. Eventually death occurs—instantaneously, after a few minutes of pain, or following a prolonged paroxysm. Rarely, a paroxysm is followed by cardiac failure of the congestive type, unaccompanied, as a rule, by pain.

ETIOLOGY

Angina is more common in men than in women, occurring most frequently in the fifth and sixth decades. Our youngest patient was aged 23; but cases have been recorded at the age of 7 (Stewart) and 12 (Allbutt, Heberden, Wild). It is most common among brain workers, as contrasted with the labouring classes, and in occupations which entail nervous strain. Only 13 of our 200 cases were of the hospital class.

We have failed to trace any special relationship between angina and the infections. Apart from the exanthemata, rheumatic fever was the most common antecedent in this series, but its incidence was small. The association of angina and syphilis is, however, closer. In this series 17 patients were known to have suffered from syphilis and 4 from gonorrhoea, the incidence of venereal disease thus being 10.5 per cent. Only one of these patients was in hospital, where the incidence of syphilis in our wards, as shown by routine Wassermann tests, was 8.6 per cent. in the men and 7.2 per cent. in the women. But several writers have found a higher incidence of syphilis in angina, Huchard reporting its presence in 33 out of 130 cases of angina, and Josué in 30 out of 100 cases.

Syphilis is the common cause of angina before 40 years of age, and is generally associated with aortic valvular disease, the valves and the orifices of the coronary arteries both being affected. In this series the three men aged 30 were all syphilitic. After 40 it is less important. One must remember, however, that angina may occur in rheumatic aortic disease, which was the cause in our patient aged 23.

The recognition by Jenner of the common association of angina and disease of the coronary arteries led to the theory that it was due to this arterial lesion; but it was soon found that the coronary arteries might be grossly diseased without the occurrence of angina, and, conversely, that they might be normal in typical cases. It was then suggested, on theoretical grounds, that angina was due to spasm of the coronary arteries. The discovery that the first part of the aorta was frequently diseased in these cases changed the venue to this part, the pain being ascribed to stresses upon it. It was found, however, that these lesions were not invariable. Attention was then paid to the muscle of the heart; but again it was shown that myocardial lesions were inconstant. It was now apparent that angina could not be due to any specific anatomical lesion, but must be the result of some physiological disturbance of the cardiac muscle. Angina, it was suggested, might be due to distension of the heart, with consequent strain upon the muscle, or to muscular exhaustion, or to strain upon a damaged heart or aorta.

Muscular Anoxaemia

The current theory is that angina, however caused, results from muscular anoxaemia. This theory was first formulated by Allan Burns in 1809,² and has been elaborated experimentally by J. A. MacWilliam and W. J. Webster³ in 1923, and clinically by C. S. Keefer and W. H. Resnik⁴ in 1928.

Allan Burns's statement may be quoted:

"In health when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action the heart and every other part has its power augmented. If, however, we call into vigorous action a limb round which we have, with a moderate amount of tightness, applied a ligature, we find that then the member can only support its action for a very short time; for now its supply of energy and its expenditure do not balance each other; consequently it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence. A heart, the coronary arteries of which are cartilaginous or ossified, is in nearly a similar condition. It can, like the limb begirt with a moderately tight ligature, discharge its function so long as its action is moderate and equal. Increase, however, the action of the whole body, and along with the rest that of the heart, and you will soon see exemplified the truth of what has been said; with this difference, that as there is no interruption to the action of the cardiac nerves the heart will be able to hold out a little longer than the limb."

MacWilliam applied a tourniquet to the arm and noted the results: (a) so long as the forearm muscles remained inactive, and (b) when these muscles were thrown into action. As he found that the symptoms might, in part, be due to turgescence of the limb if the constriction was suddenly applied, he made the limb ischaemic by means of an elastic bandage, applied first to the fingers and the hand, and only removed after the application of the tourniquet. In a limb thus treated and kept immobile no notable result followed for the next twenty minutes, save for some coldness in the part, an inclination to shift the position of the arm, and a certain amount of discomfort at the site of the constriction. Muscular activity of the forearm muscles, however, rapidly produced marked discomfort. Fatigue, as measured by the number and force of contractions executed within a given period, was readily produced, the fatigue point being reached in one-half or one-third of the time required in the normal limb. The forearm, too, rapidly became painful, and, as the

fatigue point was approached, so acutely painful that the desire for relief became urgent. Cessation of the exercise did not relieve the pain, which ceased only after relaxation of the tourniquet and the return of blood to the ischaemic muscles. Though relief of pain was thus readily afforded, fatigue was still apparent, and passed off only after some interval of time.

"It need hardly be pointed out that the foregoing observations have a close bearing upon the problems associated with the production of pain in angina pectoris, showing as they do how readily acute pain can be excited in skeletal muscles working with lack of blood supply, the pain developing while the contractile power, though to some extent weakened, is still sufficient to execute movements of considerable force and energy—that is, long before complete fatigue."

This experimental work is supported by clinical observations. In intermittent claudication severe pain and weakness in the muscles of the legs may ensue on exertion and cease when the exertion ceases. In senile gangrene from obliteration of an artery pain is constant; and in cases of embolic arterial block—for example, in mitral valvular disease—pain is always present if a limb is involved. We have notes of seven cases of this kind where gangrene followed; and in a case where gangrene resulted from thrombosis of a popliteal aneurysm acute pain in the leg occurred suddenly, and persisted. Intermittent claudication is exactly comparable to angina of exertion; senile gangrene and embolic block to angina of decubitus (infarct).

Keefer and Resnik⁴ analysed a series of 399 cases of angina in which post-mortem examination had been made. In 380 cases the coronary arteries were diseased, and in the remaining 19 the aortic valves were incompetent. It may readily be assumed that, in certain circumstances, all these cases suffered from anoxaemia of the myocardium. Disease of the coronary arteries must necessarily limit the flow of blood to the heart, which may be sufficient for the muscle working quietly, but be inadequate to meet any additional exertion. In aortic incompetence the diastolic blood pressure is low, and, as the muscle of the heart is supplied with blood during diastole, its blood supply even at rest must be limited, and may become insufficient if extra exertion demands a more ample supply. It is in the syphilitic cases that angina is most common, for here aortitis is the rule, and the coronary orifices are frequently involved in the lesion. The resulting ischaemia produces pain. There is no essential difference between the angina of exertion and that of decubitus. In the first, the ischaemia is relative and intermittent; in the second, absolute and permanent. If the circulation is merely impaired, symptoms will only ensue when exertion renders the parts relatively ischaemic, while if the block is complete the symptoms will continue for an indefinite period.

A similar line of reasoning explains the occurrence of angina in all the other conditions with which it may be associated. It occurs, rarely, in severe anaemias in which ischaemia might readily occur. But the ischaemia in these cases is general and affects all the tissues of the body, and the general debility as a rule prevents exertion capable of throwing undue strain upon the heart. In mitral valvular disease and congenital heart disease venous congestion, if present, is general, and the whole heart fails, dyspnoea checking the exertion before the painful stage of ischaemia develops. In coronary disease only a part of the heart is concerned, the greater bulk being unaffected. The heart as a whole can thus respond effectively to a demand for increased activity, but the part whose blood supply is limited now becomes relatively ischaemic, and painful sensations arise. A liability to angina can coexist with the possession of considerable power by the heart as a whole. In one of our patients the blood pressure at rest measured 280 mm. Hg.

Post-mortem Appearances

In this series a post-mortem examination was obtained in 13 cases. Of these, 12 presented infarcts or fibrosis in the muscle, the coronary arteries being extensively diseased. The other patient, a man aged 47, who had been off work for a couple of months on account of shortness of breath and palpitation on exertion, had his first attack of angina when walking, and died two days later during another attack. There was well-marked syphilitic aortitis above the aortic cusps, which were thick and shortened, with a slight recent acute endocarditis superadded. The orifices of both coronary arteries were involved in the aortitis and greatly narrowed. The arteries in their course were normal and the muscle of the heart intact. In two cases there was rupture of the heart.

Vasomotor Disturbances

Vasomotor disturbances not infrequently accompany attacks of angina. In two of our patients the blood pressure during an attack measured 160 mm. Hg, and in the intervals 120 mm. in one case, and 130 to 140 mm. in the other. In another patient an "interval" blood pressure of 140 mm. Hg rose to 190 mm. during an attack. Sometimes attacks occur during a period of high blood pressure, and cease with its disappearance. A stoker, aged 50, caught cold, and a week later began to suffer from attacks of angina on exertion. After a few days he entered hospital and his attacks ceased. His blood pressure, on admission 215 mm., fell steadily, reaching 145 mm. on the fifth day, and 135 mm. before discharge. At the same time his left heart, which on admission had been slightly dilated, recovered its tone. He was in hospital three and six years later suffering from bronchitis and gastritis. The angina had not recurred. His systolic blood pressure was then between 135 and 150.

Some patients who suffer from angina tell us that, while they can in favourable circumstances undertake considerable exertion without distress, attacks ensue if they are exposed to cold, if exercise is taken immediately after food, or if they are subjected to nervous strain. The cause is to be found in vasomotor alterations in the distribution of the blood. Everyone knows that the increased supply of blood to the digestive organs after a meal produces mental languor as the result of the lessened supply available for the brain. Cold, food, emotion, are all factors which influence the blood pressure and the distribution of the blood throughout the body.

Professor MacWilliam points out another possible factor.

"So far as my observations go there is not necessarily such rise of blood pressure on exposure to cold as would throw a decidedly greater strain upon the heart. It seems to me that some transient attacks of pain, with no obvious and sufficient exciting cause, probably depend upon a central nervous factor, an increased susceptibility to pain from irritant influences arising in the heart. Exposure to cold, for instance, often tends to excite neuralgic pains, anywhere, in a susceptible subject."

Such an explanation seems feasible. Many patients are highly strung and intolerant of discomfort, and some are quite unable to bear a blood pressure examination of the left brachial artery. Several, too, have told us that their attacks cease as soon as they have swallowed a tablet of trinitrin, before any effects of the drug could have been manifested.

Intoxications

In rare instances angina seems directly related to intoxications; tobacco, tea, and coffee being the common offenders. One of our patients suffered from exophthalmic goitre and two from myxoedema; in these toxic causes might have been present. F. R. Fraser, however, informs us that in his experience angina is very rare in Graves's disease; so the association was probably accidental. We have seen one clear case due to tobacco.

The patient was a healthy, well-built man, aged 21. His first attacks had occurred six years previously, when serving in the Army, from which he was, in consequence, discharged. At that time he smoked heavily, but subsequently gave it up until shortly before his admission to hospital. The attacks at once recurred. The pain, he said, was never very severe, but sufficient to make him stop work. It was sub-sternal, and occurred only when he was at his work, which was arduous. For a week before admission the pain had been increasing in severity, and he had become short of breath on exertion. His organs seemed sound. His pulse rate on admission was only 40 per minute, but the heart's action otherwise was normal, and the blood pressure within normal limits. His fingers showed a fine, rather irregular, tremor. In a few days his pulse rate rose to 68, and all discomfort disappeared.

Nicotine has a powerful action upon nerve cells, producing an initial stimulation followed by paralysis. At first the pulse rate slows and the blood pressure rises; and this is followed by an increase in the pulse rate and a fall of blood pressure, due, in part at any rate, to direct action of the drug upon the myocardium (Cushny).

THE NERVE SUPPLY OF THE HEART

The exact details of the nervous supply of the heart are as yet undetermined. The pathway of the efferent nerve fibres is fairly clear; the inhibitory impulses travel

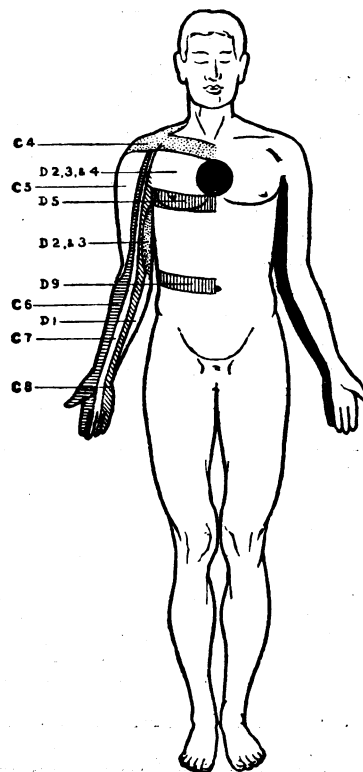


FIG. 1.—Showing the usual distribution of the pain in angina and the relative spinal distribution.

along the vagus nerve, while the accelerator impulses, issuing from the upper dorsal segments of the cord, pass through the upper cervical ganglion and the inferior cardiac nerve, and perhaps, in part, through the middle cardiac nerve. The pathway of the afferent fibres is less clear. The depressor impulses pass upwards along the vagus nerve, but the other afferent impulses pass through the sympathetic system, entering the cord through the upper five or six dorsal roots. There is no evidence of their passage through any cervical root, or the vertebral nerve (Langley).⁵

The reason for the characteristic distribution of the pain in angina is thus apparent. The cardiac sensations enter the upper dorsal segments of the spinal cord. The peripheral representation of these segments is shown in Fig. 1. Extreme stimulation of the dorsal segments leads

to an overflow of the stimuli to adjacent segments and so to radiation of the pain into the neck and epigastrium.

DIAGNOSIS

The diagnosis is established by the history of attacks of substernal pain occurring on exertion and relieved by rest, with or without the characteristic radiations and *angor animi*. The distribution of the pain varies in different patients, but it is never referred to the apex of the heart save as a part of a widespread distribution. Pain in this area alone, a common complaint in stout women, is due to causes other than cardiac. Some are gastric in origin, some due to hyperaesthesia of the breast, some to fibrositis or strain of the pectoral muscle.

The majority of our patients were at work, apparently in their usual health, at the onset of their symptoms, only 44 being under medical care at the time. Of these the largest group complained of cardiac symptoms, a lesser number suffering from symptoms due to arterial or renal disease.

Although the majority of our patients made no complaint prior to the onset of their anginous attacks, subsequent examination, as a rule, revealed pathological changes. In 90 cases the heart was enlarged; in 83 of 136 cases the blood pressure was over 150 mm. Hg, the extremes being 105 and 280 mm. In 14 cases the aortic valves were incompetent, and in 45 mitral murmurs were present though not necessarily due to valvular disease. One patient had mitral stenosis. Apart from murmurs the cardiac sounds were often abnormal, the first sound being weak, muffled, or distant, the second sound accentuated or intoned, suggesting myocardial or arterial disease. The electrocardiograms were frequently abnormal. Out of 122 cases the QRS complex was abnormal in 16; T-waves were normal in 46 cases, inverted in Lead I in 39, and in Lead III in 37 cases. Heart-block was present in 5 cases, bundle-branch block in 8, extrasystoles in 13, fibrillation in 4 cases, and irregular rhythm in one. In the exceptional case the most careful examination may fail to reveal any abnormality, though as a rule some deviation from the normal is revealed. It may seem difficult in such cases to relate the pain of angina to the heart, but a little consideration shows the reasons. The cause of the symptoms is insufficiency of the cardiac blood supply from narrowing of the arteries at their orifice or in their course, and neither of these lesions has distinctive physical signs. Syphilitic aortitis is often a post-mortem discovery, as it fails to show itself unless "something happens"—incompetence of the aortic valves, narrowing of the coronary orifices, or aneurysmal dilatation. A purely local lesion of the coronary arteries will only manifest itself by physiological disturbances. In one of our patients who died of angina, due to coronary stenosis at the orifice from syphilitic aortitis, the heart itself was quite sound. Arterial disease is usually associated with high blood pressure, renal disease, syphilis, etc., which present their own characteristic signs. The combination of aortic valvular disease and angina always strongly suggests a syphilitic origin, especially in men under 40 years of age, though it may occur in cases of rheumatic or other origin.

PROGNOSIS

Most patients with angina die more or less suddenly. In experimental ligation of the coronary arteries in animals, the cardiac action becomes irregular from extrasystoles, which are followed by tachycardia, ventricular fibrillation, and death. In some cases of angina the cardiac action rapidly weakens—the pulse rate usually increasing—and failure ensues. Life to the anginous patient is uncertain, but may be prolonged for years. Dr. Arnold died in his first attack; John Hunter had angina

twenty years before he died. Two of our patients died at the age of 76—one twenty years, the other fourteen years, after the first attack. Another died at the age of 70, thirty years after the occurrence of Stokes-Adams attacks, due to heart-block which persisted unchanged. Several patients are working, even playing golf, after five and ten years. Even those who have had definite infarcts may make a good recovery. One of our patients worked hard for five of the seven years which elapsed between his infarct and his death, and one of A. G. Gibson's patients lived an active life for five years after his attack, dying at the age of 72.

The best outlook is that of the toxic and anaemic group if the cause of their symptoms can be removed. Of the other groups, those with a high blood pressure, those with aortic valvular disease, those with a low blood pressure, in order. Patients with high blood pressure can, as a rule, be relieved by appropriate treatment. Syphilitic valvular disease can be helped by specific treatment. Those whose hearts are weak, without obvious cause and so in all probability from coronary stenosis, are unlikely to obtain satisfactory remission of the underlying pathological cause.

Two other factors are important: the exciting cause of the original attack, and the mental attitude of the patient towards his difficulties. It is obvious that if an attack supervenes on trivial provocation it is more likely to be due to a gross lesion than one which follows severe physical or mental strain. The mental attitude is equally important. Can the individual react to his limitations successfully, adapting his life to his altered equilibrium, or will he fail to do so, and continue to run his usual course? An attack of angina gives warning that the pace in future must be lessened.

TREATMENT

The general lines of treatment are well defined. The treatment of the toxic and anaemic cases is obvious; and that of aortic valvular disease, with angina, is similar to that of the cases without anginous symptoms. In the cases due to coronary disease every endeavour should be made to *prevent undue strain upon the heart until satisfactory anastomoses have had time to form* and to supply the myocardium with sufficient blood. The coronary circulation is ample, and free anastomoses exist between the main arteries, and between their branches. An attack of angina indicates that some arteries are narrowed, and that closure may occur. Functional overactivity of the heart will lead to disaster. In angina confinement to bed is not always helpful, though sometimes useful from the psychological standpoint; but activities must be kept at a low level for a prolonged period. All other treatment is subsidiary. In patients with high blood pressure increased elimination must be secured by the usual methods. In syphilitic cases specific treatment must be instituted. In patients with weak hearts stimulant treatment is indicated.

The treatment of a paroxysm is necessary but of less importance than its prevention. Angina is a danger signal, a sign of some harmful activity, and if we make the patient insensitive to his discomforts he will soon kill himself. If the blood pressure is raised during attacks the nitrites are useful, but they are useless if the blood pressure is not raised. Sedatives should be given without hesitation, morphine being the most effective. Chloroform is sometimes necessary.

Surgical Treatment

The surgical treatment of angina is still in the experimental stage, and we doubt if it will ever prove of much value. All surgical treatment aims at the relief of cardiac pain; but pain is a useful danger signal, an

indication to the patient that his current activities are harmful and must be stopped at once, or death will ensue. But there remain a few patients whose sufferings are so severe that relief at any cost seems desirable.

Numerous operations on the cervical nerves and ganglia have been performed with the object of severing the sensory communications between the heart and the brain; but so far as our knowledge goes these operations are based upon erroneous theories. The sensory tracts run into the upper dorsal segments of the spinal cord, and not along the neck. It is known that left-sided pain is referred from the heart to the upper five or six segments, the chief pathway being along the inferior cardiac nerves, the stellate ganglion, the vertebral sympathetic ganglia, and the white rami communicantes to the spinal cord. Subsidiary pathways may exist along the middle cardiac nerves and the lower cervical sympathetic trunk to the stellate ganglion; and directly from the heart to the upper dorsal vertebral ganglia* (Fig. 2).

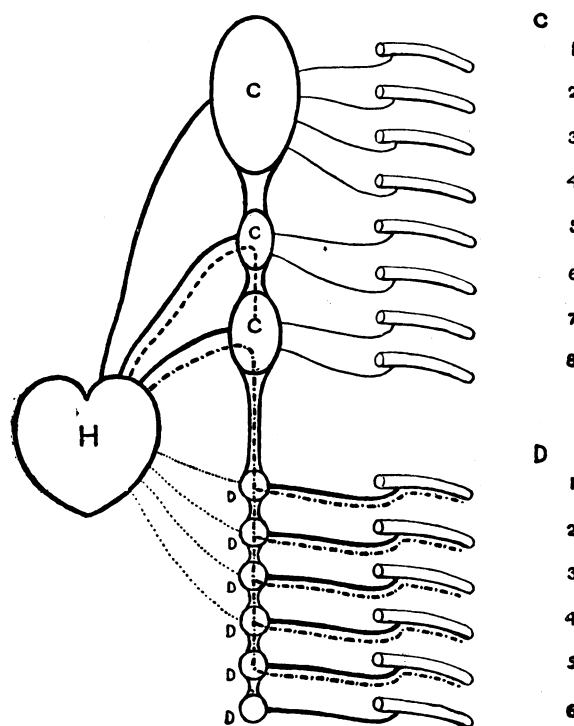


FIG. 2.—Diagrammatic scheme of the sensory nervous connexions of the heart. C, Cervical sympathetic ganglia; D, Dorsal sympathetic ganglia; D, Dorsal nerve roots; ———, Main route of sensory impressions; - - - - - , Secondary route; Direct route.

The pathways might be blocked by: (1) removal of the stellate and the upper dorsal vertebral ganglia; (2) section or block of the rami communicantes; (3) section or block of the posterior nerve roots.

The least dangerous procedure seems to be alcohol injection of the rami communicantes, of which favourable results have been reported.⁷ James C. White has now operated upon 17 patients who were subject to severe and frequent attacks of angina which resisted medical treatment. All recovered from the operation without serious trouble, the only unfavourable sequels being attacks of pleurisy in three cases, which rapidly subsided. In 7 cases complete relief from the left-sided pain followed, and in 5 the relief, while not complete, was considerable. In 3 cases improvement was slight, and in 2 cases no improvement resulted. Five patients have been able to return to work. He states that shortness of breath, peculiar sensations, without pain, in the region of the heart, or mild attacks of right-sided angina, have always afforded sufficient warning to the patient that

the danger zone of exertion was being approached. The upper dorsal ganglia were removed in 3 patients whose general condition permitted an open operation by Henry's route. All these patients made good recoveries, and the pain was completely checked.

Eight of these patients have died, seven of them within a year of the operation, one from an empyema, the others from cardiac causes. One patient is alive three years after operation, and two patients two years after operation. Such results must be expected, for the operation is merely palliative and in no way curative. But relief from severe recurrent pain, even if of short duration, may be an inestimable boon to some patients.

We have great pleasure in acknowledging the kindly assistance of many friends; in particular, G. B. Fleming, F. R. Fraser, D. Dale Logan, G. D. Logan, J. A. MacWilliam, J. K. Rennie, James C. White, and Farquhar Macrae.

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THE DEVELOPMENT OF THE SCIENCE OF NUTRITION IN RELATION TO DISEASE*

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In the last twenty years there have been rapid developments in our knowledge of animal nutrition. This knowledge continues to accumulate at an increasing rate; it has such a direct and important bearing on the prevention or cure of disease that nutrition may now be regarded as a new branch of medical science.

HISTORY

It is interesting to contrast the outlook of last century with that of the present day. At the beginning of the period our ideas were little in advance of those held by the Greeks. Writers still believed that there was only one nutrient substance common to all foodstuffs. Early in the century it was established that heat and muscular energy had their origin in a process of oxidation, and calorimetric studies had shown that the human body obeyed the law of conservation of energy. By the middle of the century, chemists had differentiated food constituents into three "proximate principles"—proteins, fats, and carbohydrates—all yielding energy. At the end of the century, the energy values of different foodstuffs, and the energy requirements of the human body under different conditions, were fairly well known, and the position of the calorie as the symbol of nutrition was well established. Proteins were recognized to be of special value, being necessary for construction in growth and repair of "tear and wear" of the tissues. Dietaries were calculated in terms of calories and protein, and studies in the physiology of nutrition were concerned mainly with metabolic processes involving exchanges of energy, and with the metabolism and chemistry of the proteins and their derivatives, and to a lesser extent of

* Summary of a British Medical Association Lecture delivered to the Dundee Branch on March 11th.