ANGINA PECTORIS

A

THESIS

BY

WILLIAM ALLAN, MB.CM.

Medicine is a strange mixture of speculation & action
(Latham.)

DUMBARTON.

1895.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Preface.

All that is claimed for this 'Thesis' is that it is 'Angina Pectoris' as 'refracted' through the mind of the writer.

The 'sad occasion' of the more particular study of Angina was by reason of my father falling a sufferer of the disease.

And now, in the following pages is set forth — and indeed, "as through a glass darkly" — the image of these strange phenomena grouped under the name of Angina Pectoris

William Allan

Angina Pectoris = Pain or anguish of the breast. From the Greek 'ἀγχω', I strangle. 'Angina' is pronounced with the "i" short.

Angina Pectoris.

– History –

Heberden

The attention of the medical Profession in England was first drawn to Angina Pectoris by Dr. Heberden.

But away far back at the very dawn of the Christian Era the first authentic case of Angina was described by the Roman Philosopher Seneca. He himself was the sufferer and he tells us his physicians called the disease "meditatio mortis". Undoubtedly Seneca suffered from Angina 'sine dolore'. Thence through the long flight of ages to the solitary but genuine case reported by Morgagni of a lady who died at Venice in 1707.

In 1768, a French physician, Roregnon, in a letter sent to a colleague gave a clinical description of a true angina paroxysm, which he had witnessed in one of his patients who subsequently died very suddenly. Roregnon made a post-mortem examination but he had given no satisfactory pronouncement as to the cause of death. Later in this same year, 1768, Heberden* of London read a paper on a group of symptoms to which he afterwards gave the name 'Angina Pectoris'. And these symptoms he connected with disease of the heart. To Heberden then may be justly given the honour of the first and exact recognition of this symptom group. And it is of exceeding interest to learn that associated with Heberden in the early discussions as to the exact nature of Angina, were John Hunter and Edward Jenner.

Latham* (1845) followed Heberden closely in argument and warmly supported his views. Parry* (1799) and Stokes* (1854) however were sceptical and doubted the

Note "Unknown" in his letter to Heberden spoke of this separation. (1769)

individuality of Angina. Latham, though, was the first who (professionally) separated in the symptom of 'pain', the pain of the body and the pain of the mind.

In 1876, Gairdner in the article on Angina in Reynold's System of Medicine amply vindicates the distinct and definite character of the Angina syndrome.

Then in the early seventies the clinical experience of Lauder Brunton in the exhibition of amyl nitrite marked an era of great advance and success in the treatment of the angina paroxysm.

Dr Heberden associated Angina with disease of the heart and subsequent study has shewn that Angina may be associated with various pathological lesions of the heart and arterial system. Amongst these many changes, Angina is always associated with one — a heart the myocardium of which is weak and degenerate.

And further, in this degeneration the general arterial system more or less participates.

Definition.

Angina Pectoris may be defined as a paroxysmal pain – often intolerable, overwhelming and anguishing – of a weakened heart ; a pain referred to the region of the precordium, often extending into the left arm, and associated with a sense of impending dissolution, a dread fact often realized with most startling suddenness.

Classification

Angina Pectoris has been classified into two broad groups, Angina Pectoris Vera and A.P. Notha The only advantage to be gained by this classification has an important bearing on the prognosis and treatment Herein, consideration of A.P. Notha is deferred till the treatment of A.P. Vera has been discussed.

As the symptoms are mostly subjective their description varies with the individual sufferers. The symptoms may be exaggerated or particaley or wholly concealed. And so it is extremely difficult to get a true and exact account of the phenomena. Further there can be no doubt that language fails to give a satisfying expression of the sensations felt.

And more there occurs cases of sudden death which in the immediately preceding syndrome more or less resemble Angina Pectoris. These cases, and others where death is the fact alone, unwarned and unsuspected, are in every probability of 'pathological kindred' with angina.

Symptoms

A typical attack of Angina but rarely is seen. It is sudden of onset, swift of progress and abrupt of termination. Suddenly, and with severity, — often with no apparent determining cause the patient

Note* Dr. Madden, in the Practitioner (1872), described his own sufferings. He had a feeling of constriction, as if the chest were bulged and the abdomen retracted. Even although his hand felt that this was not so, the sensation was still as real.

Note† The distribution of the lesser internal cutaneous nerve (Nerve of Wrisberg) is over this aspect of the arm and its spinal origin is contiguous to that of the augmentor

Symptoms.

Pain

is seized with a feeling of distress somewhere in the precordial area. This distress, at first perhaps slight, rapidly deepens into agony which radiates to the base of the chest, producing there a sense of constriction*; ascending to the root of the neck and the left shoulder, the left arm is attacked, the pain being especially severe about the middle

its site

of the inner aspect of the arm+ The pain occasionally descends to the wrist giving a painful sensation as if the wrist were tightly gripped. Even the fingers, and especially those supplied by the ulnar nerve are sometimes visited by the pain. And it has been noted by Eichhorst that the muscles of the ulnar nerve distribution in the hand have

and radiation

become atrophied. Although this radiation of the pain is, in a way, highly characteristic of Angina it does not always range over so wide an area.

The left side of the thorax and the left arm are the usual seats of the pain. Sometimes it is the

Symptoms. right side and arm, and sometimes both arms at once. In descent the pain may travel from the loins into the thighs.

The pain also varies in intensity and character, from a slight uneasiness of oppression, or constriction, or fulness, up to the most excruciating agony as if the chest were gripped in a vice, or the heart transfixed by a redhot dagger, and the pain shooting in the fulness of its severity to the shoulder and arm, following the course of the cervical and brachial plexuses. It may be noted here (and it is further discussed in the Section of Pathology) that the augmentor (Sympathetic) nerve of the heart is connected through the last cervical and first dorsal sympathetic ganglia with the corresponding segment of the spinal cord. The "pain" travels thus to the cord and therein is transferred by irradiation to the spinal nerves above the origin of the augmentor.

Note* Those attacks are longest which come on during the night and seemingly without provocation If brought on by exertion they cease with the exertion

Symptoms

The cardiac terror

Associated with this physical pain there is a profound feeling of mental distress in the awful contemplation of approaching death. This distress might fitly be described as a psychical pain, a mortal agony of the mind. This pain is pathognomonic of Angina. Often it is unassociated with physical pain. and Latham observed "that the dying sensation more often surpasses the pain, than the pain the dying sensation".

Duration of attack

The paroxysm generally lasts but a few minutes; sometimes, even an hour or two; but it is more likely that the apparently lengthened paroxysms are either false or a rapid succession of attacks which will generally end in death. The affected parts are often left with the sensation of numbness and formication; and occasionally the arm for a time has been powerless. Further the affected area frequently shows vaso-motor disturbance At first pallid and with cold perspiration, afterwards

Note * In some cases of Angina, associated with aneurism of the aorta and with aortic regurgitation, voluntary movements, such as forced inspiration and violent jactitation of the arms, have been observed. and by these means the pain has been more or less relieved. (Balfour).

And Trousseau says, in speaking of Angina in a case associated with aneurism " The pain was such as to make the patient cry out"

John Hunter found no pulse in either of his radial arteries " and he produced the voluntary act of breathing (Home)

He would, at times, get ease by walking. (Johnson) (U.S.A)

Symptoms. becoming reddened and flushed. Sometimes, there is no pain in the arm and it has been recorded, of the pain beginning in the fingers, ascending the arm and thence to the precordium — in this, simulating the aura epileptica Again, a case has been reported where pain only in the arm preceded death for many years. There is reason, however, to suspect that many of these variants have occurred in cases confounded with true angina

Local tenderness on pressure (hyperaesthesia) over the precordium is often noticed Again, in other cases pressure has afforded a certain measure of relief.

The patient Aspect and attitude At the first indication of, and during the attack the patient [redacted] assumes a fixed attitude* He fears to make the slightest movement — even to speak — lest he precipitate the end. The face is expressive of great suffering and anxiety. Deadly pale, bedewed with sweat drops, and features pinched — the facies Hippocratica

Note* In one of my cases (No 2) I was very much struck by the rigid stoop of the shoulders – pointing to mischief within the chest. And I think such a stoop would strengthen a diagnosis of angina.

Symptoms

Often there is but a haggard and startled look.*

The breathing is generally quiet and regular

The respiration

or perhaps just slightly accelerated – that is as far as physical appearances go. The patient may complain of a sensation of choking or suffocation which he may call a difficulty of breathing. Of course in the presence of a heart lesion there may be associated orthopnoea or dyspnoea. And in fatty heart, too, the respiration is often of peculiar rhythm. Still this choking sensation is very probably due to diminished oxygenation of the blood and, as such, adds to the suffering of the patient.

The pulse.

The condition of the pulse has been greatly controverted. It varies according to the time of examination and the severity of the attack. H Eberden's statement was that "at least sometimes it is not disturbed or quickened" Later writers made various and conflicting statements

Note. Sphygmographic tracings very often indicate a fulness of the artery between the beats — virtual tension.

The upstroke is small and slanting, indicating a weak ventricle against a strong opposition.
The downstroke is also slanting and not decrotic, indicating a weak recoil and lessened outflow from the arteries via the capillaries to the veins

Symptoms

The truth is in all. The pulse may not indicate high arterial tension*. Generally, it is small and weak and somewhat rapid and perhaps irregular. During the paroxysm it gets more and more frequent till through rapidity and weakness it becomes well nigh imperceptible.

After an attack the frequency and weakness of the pulse remain. In some severe cases bradycardia has persisted for hours, even days afterwards.

As regards the heart it is difficult to get a complete examination of it during a paroxysm.

The mental faculties, for the most part, remain quite clear and the nervous system intact.

Giddiness, syncope, muscular twitchings and disorders of the special senses, may be attributed to an associated fattily degenerate heart with atheromatous vessels or to the gouty diathesis.

Symptoms

Sickness with salivation or vomiting or profuse discharge of flatulence very often accompanies or follows the paroxysm. These symptoms are not so much of the direct cause of the paroxysm as a part of the symptom group, being no doubt due to sympathetic irritation of the respiratory and gastric distribution of the vagus and of the phrenic nerves. when hiccough occurs.

Gastric.

At the end of the paroxysm there is often also an irresistible call to pass urine — even when the bladder is empty. The urine often is pale and abundant, just as in Hysteria, Epilepsy and Asthma.

Many of the attacks of Angina are very imperfect, still they are just as significant. The pain may disappear although the patient may still suffer from faintings Such cases are included in Angina sine dolore.

The attacks, if they recur, are of increasing and prolonged severity and at lessening intervals (again resembling Epilepsy).

Symptoms

The attacks may however vary both in intensity and character. Of course, treatment may so modify the cause that the attacks gradually lessen into milder and more imperfect paroxysms.

Illustrative of true Angina is the following case, the first of three herein quoted.

Case I.

Case I. A man, aet 54. Stout build: complexion rather pale. His father died suddenly "of heart-disease" History of mental worry and insomnia. Took little or no exercise. A heavy smoker.

One night after dinner he ran upstairs. Halfway up he "had to stop." A pain had seized him in the region of the heart and he felt as if he "were about to burst and die" The pain travelled right down to the little finger. The attack lasted a minute, which seemed a life-time, and departed as suddenly as it came. During

11/4.* The best position in bed is to rest with the body at an angle of 45° to the legs.

Case I.

the paroxysm he felt that "to move or to speak would be death." For about ten minutes after the attack he was afraid to make any movement although he spoke freely.

He was put to bed* without undressing.

Next morning he was undressed and examined.

The temporal, radial, and femoral arteries were slightly stiff and atheromatous. The radial pulse was 88, weak and soft but regular. The heart was feeble of impulse. The cardiac dulness was slightly increased The first sound was weak and distant. The aortic was accentuated.

Eyes - no indication of senile change.

lungs. liver and kidneys were normal.

The superficial veins of the thorax were rather distinct and deeply blue. The tongue was slightly furred.

Note *

3	out of	100	cases	noted by	Heberden	were	women.
8	" "	89	"	" "	Forbes	"	"
14	" "	98	"	" "	Balfour	"	"
42	" "	234	"	" "	Huchard	"	"
1	" "	40	"	" "	Osler.	"	"

— which gives that only 12½ % of the cases were women

Etiology. Angina rarely occurs before middle life. Quain gives it that 80 % of all cases occur in patients over forty years of age. The average is more

Age nearly fifty years. Still true cases of Angina, ending in sudden death, have been recorded in patients of tender age. If the age be under forty the following elements of predisposition may be noted. Syphilis – a potent factor in early arterial decay. Excessive indulgence in alcohol. Strain from overwork. Pre-existing heart- and lung disease – especially a pericarditis in childhood.

Sex As regards sex, men are far more frequently attacked than women *

Heredity Heredity, in a tendency to early decay plays an important part in the predisposing causes. Of this, a notable example is seen in the Arnold family, where members of three generations died suddenly of Angina

One could hardly say that the following illustrious men lived in ease and luxury, yet they were all victims of Angina.

	age.	Death
John Hunter	65.	After twenty years of suffering
Thomas Chalmers	68	In first attack
Thomas Arnold	47.	In first group of attacks (lasting two hours).
Matthew Arnold	66	after three years.
Charles Dickens	58	Five years after a railway collision
W. M. Thackeray	52	———

Etiology

And it may be here noted that Angina, as Sir John Forbes put it "is attendant on ease and luxury"* Sedentary habits, obesity, and especially the Gouty diathesis are potent in the predisposition to Angina. As regards this diathesis Dr George Balfour believes it to be present in nearly all persons beyond middle age.

Gouty diathesis

Any disease, such as a Specific fever may leave the heart exceedingly weak; and any undue strain before the heart has sufficiently recovered, may strongly predispose to Angina. This is more especially true of Influenza and to a less extent of Pneumonia and Enteric fever.

As regards heart disease it is remarkable that Angina is not attendant on valvular defect (vide Diagnosis).

Exciting causes

The immediate or exciting cause is often very slight. Physical exertion – going uphill or upstairs

Etiology. Walking or running against the wind (especially if it is cold) will suffice to excite a paroxysm. And these causes are more potent if the exertion be made shortly after meals – and more readily after breakfast. The mere exposure to cold very often incites an attack. Excess in eating and drinking are often the only apparent excitants. Emotional excitement, especially of anger, and even during sleep in dreams, is a frequent cause. More probably the attacks in bed are due to flatulent distension of the stomach and bowels, in combination with the recumbent posture, leading to actual interference with the action of the heart. (vide note on posture in bed).

Diagnosis

"Nemo mortalium omnibus horis sapit"

The diagnosis of Angina is not as a rule difficult but to be complete demands a searching overhaul of the whole organization.

In the first place one must not be misled by the words of the patient. People are too often "nervous" about the state of the heart. Hence objective phenomena are most to be relied upon and yet the only objective fact may be death. In questioning the patient valuable aid is obtained in ascertaining the bearing of Aetiology in Age, Sex, Heredity and the history of how the attack was brought on.

Pain

Pain about the region of the heart is often complained of by hysterical and nervous women and more so if the uterus or its appendages are diseased

Dyspepsia [with salivation, hiccough

Note *
Pericarditis, with associated myocarditis may have no physical pain

Diagnosis and vomiting (gastralgia and cardialgia)] is also a prolific source of cardiac pain. And in the majority of dyspeptics complaint is made of a dull aching pain in the left arm, such pain being liable to sudden exacerbation.

Spinal irritation (with intercostal neuralgia) with hyperaesthesia over and pain radiating into the cardiac area and perhaps to the arm, as is frequently seen in chlorotic patients who often are also greatly pained by the hard faecal masses of constipation — in such cases great alarm may be aroused.

Other pains as in pleurisy and especially (with reference to the pain) in pericarditis* show by the thermometer their inflammatory origin When there is no sensation of approaching death and the pain is more or less continuous

The sickening and excruciating pain.

Diagnosis of hepatic, renal and intestinal colic should easily be excluded.

In Rheumatism and Gout the heart is often seized with severe twinges of pain. All such pains affecting the heart or referred to the region of the heart may cause great anxiety. The patient, uneasily conscious of his heart, may say he "was greatly frightened," but never is the pain associated with the "meditatio mortis."

In speaking of Angina, Stokes said that many of the cases described as true Angina, were in reality cases of cardiac asthma or dyspnoea. Latham also called attention to this confusion and termed such cases "angina notha". It is highly advisable, however, not to use this term. In cardiac asthma – more properly, dyspnoea the patient is livid and with cold extremities. He often wakens out of sleep, gasping for breath

Diagnosis

He does all he can to get breath. His respiration is imperfect from a weak heart and the Dyspnoea is due to a congested venous and pulmonic system and hence imperfect oxygenation of the blood. Still cardiac Dyspnoea may be associated with Angina vera.

In the Diagnosis of true Angina it may be recognized as a dictum that there is no idiopathic Angina. There is always a cause to be found.

If the attacks be at all severe and frequent the patient has a haggard and anxious expression. In other and mild cases there may be every appearance of good health in the intervals between the paroxysms.

Examination of the heart and pulse often reveals a dilated and weak heart with an atheromatous condition of the arteries. The detection of a heart lesion more especially of aortic incompetency strengthens the probability of the attacks being true angina. For very

Diagnosis often aortic insufficiency is an incident in heightened arterial tension followed by arterial degeneration, resulting in a dilatable and inelastic condition of the aortic walls. Before development of the murmur this condition is recognized by the ringing accentuated aortic second sound.

Pain is often most distressing and persistent in valvular disease and adherent pericardium. If the lesion present be of the mitral valve the great probability is that the pain is not of Angina.

If an aneurism of the aorta be present it must be carefully remembered that pain from pressure of the tumour – even if ever so small – is often most severe and agonizing. But it is more or less constant nor is there ever realized the physical agony of dying to such an extent as in the "meditatio mortis".

Now although every weak and degenerate (fatty or fibrous) heart does not reveal itself in a paroxysm

Diagnosis

of angina, yet angina is always so constantly associated with degeneration of the myocardium, that even the suspicion of a fatty heart will strengthen the diagnosis of angina.

Further, in some cases of angina, the paroxysms cease as such, but the patient is subject to attacks such as are termed Adams' disease. Vertigo, syncope and bradycardia, and sometimes apoplectiform attacks complete the syndrome. And in one of these attacks death may occur.

The chief elements, then, in the diagnosis of angina are the age and sex of the patient; the existence of degeneration of the heart and vessels, with perhaps, a history of heredity; and the true description of the phenomena of the attack with its direct exciting causes.

Case II.

Case II. A man. aetat: 64. System generally showing signs of breaking down. Stockbroker.

Family history of Gout and Heart disease. One sister had Exophthalmic Goître. He himself had been conscious of an "occasional slop in the heart's beating" during the past 7 or 8 years.

Hurrying across the street one winter day he felt a "tremendous oppression on his chest": Three days after he was seized with an attack of Angina. There "was no great reason why the attack should have set in" He had just finished a busy day in the office and on leaving he was seized just as he entered the street from the close. The pain began "over the heart" and spread to the left shoulder and arm and down to the wrist. He could breathe "easily" but neither speak nor move. He felt as if he "would surely die". The attack "could not have lasted

Case II. "more than a minute".

The heart's impulse was feeble. There was I think, slight dilatation. First sound weak; second, weak but of heightened pitch. Pulse was small, feeble, regular and quick - 104. The feeling of weight over the chest had been troubling him for months. He often felt giddy on first rising in the morning. Lungs. Liver. Spleen. and Kidneys seemed normal. The arteries, if anything, felt slightly stiff.

He died about four weeks afterwards, quite suddenly. He had several attacks during the interval; about six during the first two weeks, and over 20 during the latter two weeks. After one of the more severe attacks the pulse was exceedingly slow (38) and feeble. This was verified by auscultation of the heart. This bradycardia pointed to complete exhaustion (for the time = paralysis of the Augmentor nerve. Exhaustion not so complete is revealed in Tachycardia.

Morbid Anatomy

Post-mortem examination on fatal cases of Angina reveals various pathological changes in the heart, aorta and general arterial system. And, it may be asked, which of all these is the primal lesion?

The answer — any single one, or any combination of such lesions in so far as is established the tendency to degeneration of the myocardium.

The cavity of the chest, as a whole, has been distorted and constricted by chronic diseases of the lungs and pleurae — such distortion ultimately telling on the heart.

Aorta

The aorta has been found dilated or aneurismal or atheromatous, and with calcareous deposition and once or twice the endothelium of the vessel has been found acutely inflamed (aortitis). These conditions interfere with the coronary blood supply and with the efficiency of the ventricle.

The heart has been found displaced.

Note* Edward Jenner was the first to attribute Angina to coronary disease.

Morbid Anatomy

Various valvular lesions especially of the aortic valves were present. The myocardium has been found hypertrophied, dilated and in a state of degeneration. And it may be, also laden with fat.

The Heart.

The coronary arteries* were exceedingly often found rigid from atheroma and calcification, and hence with the lumen more or less occluded. Sometimes the orifices of the vessels have been almost - been wholly - obliterated. This occurs especially in Syphilitic arteritis.

Coronary Arteries

The anterior branch of the descending ramus of the left coronary artery is the main supply to the walls of the left ventricle. And it is this artery which is chiefly affected by atheroma - calcification, thrombosis and embolism. Huchard in examining 145 cases of Angina found in all, the coronary arteries more or less atheromatous and the myocardium degenerate

The nerves of the heart have not as yet

Morbid Anatomy been properly investigated Neuritis of the vagus has sometimes been found.

The pericardium in some cases was thickened and adherent. And occasionally some of the adhesions affected the coronary vessels. Mediastinal and abdominal tumors have also been recorded.

But, always associated with whatever other change or lesion may be, is a weakened and degenerate myocardium. And almost as constantly the systemic arteries show extensive atheroma.

Pathology With advancing years great changes come, but gradually and insensibly, over the general vascular system. Decay, which is a truly physiological, if a terminal phase of development, first sets in throughout the arterial system. This retrograde change first shows in diminished arterial resilience which decidedly impairs the value of the vascular system as a nutritive agent, and marks a step on the downward path to the grave. (Foster)

Further changes, in capillary obsolescence, heighten the peripheral resistance and hence a gradually increasing arterial tension.

We know that the main facts of the circulation are (1) the systole of the ventricle, (2) elasticity of the arteries and (3) the peripheral resistance. With age and premature decay come alterations in all three. The heart may become

Pathology

weak in muscle or deranged in nerve. The arteries may suffer change in their elastic and muscular fibres and in vaso-motor mechanism. And thirdly, from say impurity of blood, there may be an obstructive alteration of the capillary environment.

Thus, the peripheral resistance being increased and the arterial elasticity lost in part, the heart hypertrophies in order to maintain the efficiency of the circulation. The blood within the arteries is thus kept at a high pressure and this pressure furthers the arterial change. Even in young and healthy arteries increased tension renders their walls more and more rigid. (Foster).

Now, these terminal phenomena are not of disease. ..... "Time has laid his hand

"Upon my heart, gently, not smiting it"

This loss of arterial elasticity is quite sufficient to

Pathology account for the hypertrophy of the heart, but of course if atheromatous changes of the vessel walls be present also, then such a condition will call for increased hypertrophy. The heart itself participates in the textural change of decay and loses in part its youthful vigour The heart's energy lies in its muscle fibre and the genesis of this energy is the result of heart metabolism and is only controlled by the cardiac nerves. The muscle fibre of the heart is not the same as skeletal muscle fibre, which is a mere instrument of a motor nerve. The histological unit, (of the heart) the muscle fibre cell, is incompletely differentiated protoplasm and so it retains in part the attributes of spontaneous movement and a form of growth beyond mere hypertrophy but not quite up to regeneration.

In health any permanent resistance

Pathology

to the onward flow of blood is met by hypertrophy of the heart muscle.. But in face of mal-assimilation, degeneration and dilatation may rapidly follow an imperfect and feeble hypertrophy, and the heart reveals in various symptoms, the failure of its myocardium.

Assimilation may fail the heart for many reasons. An imperfect blood supply — defective in quantity or quality. Lessened calibre of the coronary vessels from atheroma or congenital smallness; or the heart being in proportion too large: — all result in a blood supply defective in quantity.

And no matter the patency of the vessels, if the blood be poor in quality or laden with toxic by-products of a perverted metabolism, the result is the same. For want of proper nutriment the hypertrophy does not compensate, and the struggling and failing heart gradually suffers decay

Pathology. The degeneration affects the muscle fibre cell and from this arise many and varied symptoms. On this consideration Parry said that the occurrence in a defined manner of some of the symptoms of a weakened heart and degeneration is revealed in the complex phenomena known as Angina Pectoris

But more — added to such — or rather allied with such a heart condition is the neuropathic diathesis or state. And it is this state which determines the occurrence in this defined manner, an Angina paroxysm.

The weakening of the heart is of course mostly felt by the left ventricle. Gradually the tension in the aorta — often the reflected result of increased peripheral resistance — becomes higher than the ventricle can overcome. The ventricular output gets gradually less as this aortic tension

Note

The effect of heightened arterial tension may tend along different lines in different people.

Thus, in one the vessels of the brain are the first to give way — Apoplexy: in another, the aorta yields — aneurysm: and in still another the aortic valves yield — aortic incompetency.

Again, in advanced years, the increased interventricular tension may result in mitral incompetency. This senile change in the heart's structure is general. In angina — with obstruction and change chiefly over the area of distribution of the anterior branch of the left coronary artery the mitral valve escapes and retains its structural integrity.

Pathology increases and consequently the residual accumulation also increases. Something has to yield*. In some hearts the mitral valve gives way leading to regurgitation. In other cases the myocardium.

Above the increasing interventricular tension the ventricle walls yield. The embarrassment of the ventricle deepens and the climax is the paroxysm of Angina. Thus, Angina is the result of the effort of a weakened heart to overcome the actively or relatively increased arterial tension.

An inelastic aorta failing to pass the blood efficiently onwards, so allows the tension immediately in front to rise above the power of the ventricle. Such a condition of the aorta may lead to aortic insufficiency or to aortic aneurysm and hence Angina may be associated with such conditions

That increased tension is the immediate

Vide. Note, page (11).

Pathology exciting cause of a paroxysm has been disputed by some because the radial pulse does not always indicate high tension. A normal tension is a relatively increased tension to a weakened heart. It does not matter what height the tension is, so be it that, for the time, it is beyond the powers of the myocardium. And tension in the aorta from immediate or remote causes is not necessarily revealed at the radial pulse.

Broadbent says that if at any time the artery be full between the beats then vicious tension exists, that is, there would be tension were there adequate vis a tergo. And this condition is satisfied in some unwonted exercise which is so common an excitant of the paroxysm. Further, the exhibition of amyl nitrite during a paroxysm is successful, because by paralyzing the constrictor nerves of the whole arterial system it causes a marked dilatation

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and, as it were, by suction power relieves the engorged aorta and ventricle. These arguments, then, are decidedly in favour of an increased tension — relative or absolute.

So long as the circulation is tranquil all goes well but any cause which weakens the ventricular systole or increases the tension it has to overcome, at once precipitates an attack of Angina.

In a normal heart an increase of arterial tension is met (1) by slowing of the heart through vagus inhibition or (2) by Dilatation of the peripheral arterioles following on impulses from the heart via the Depressor nerve to the vaso-motor centre. But if the blood accumulates in the aorta through its inability to pass the blood onwards the action of the Depressor nerve is of no avail

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and further often the condition of the blood – laden with impurities, – causes by irritation contraction of the arterioles and obstructive change in the capillary environment:

Then the slowing of the heart by vagus inhibition only worsens the plight of the ventricle. The vagus by its action weakens the systole of the auricle; the stimulus to the ventricle is thereby weakened and so the beat of the ventricle is actually diminished in force and the pause between each cardiac cycle is lengthened.

The vagus action then does not help the heart; and so the aid of the augmentor nerve is called for. Now the augmentor influence is dangerous to the heart's integrity. Through its influence the innate energy – the vis insita – of the heart's muscle is set free and the heart's contraction is greatly augmented, but at great expense. If the augmentor be successful the ventricle gets rid of its accumulated blood and

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the paroxysm ceases. But it may be that the whole energy of the heart has gone forth in vain and death occurs suddenly with the heart in diastole. If the augmentor influence be successful the myocardium is left greatly weakened and hence the recurrence of paroxysms with increasing frequency and severity, or even a fatal result after one or two attacks. In a weakened and degenerate heart the augmentor influence is prejudicial to the worst degree, for the increased genesis of energy called forth saps the strength of the myocardium which cannot replace it by reason of defective blood supply.

But the augmentor is not always and ever successful. If the heart become extremely dilated it ceases to beat, and as the period of possible recovery is in seconds relief must come

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speedily or — death.

That this undue influence of the augmentor on the struggling heart is very hurtful is very probable from the following considerations. A man seemingly in good health and not complaining goes to bed. Next morning he is found dead in bed. On post-mortem examination, the heart does not show any great or profound change — macroscopically at least. And the cause of death — from due consideration — has been acknowledged as Angina

When another patient is examined after a paroxysm. His heart is beating very slowly — bradycardia. This points to augmentor exhaustion.. During the attack he may complain of hyperaesthesia over the precordium. Such a symptom is also present with bradycardia following fracture of the cervical spine

Again it is noted that the physical pain of

Pathology

angina radiates only over the area of distribution of such nerves which find their spinal origin on the same plane and above the origin of the augmentor.

Looking now at certain cases of persistent tachycardia, e.g. as in Exophthalmic goitre. After many years the tachycardia may change to bradycardia. Again take the physiological action of digitalis in increasing doses: (1) beat is slowed and strengthened – vagus being stimulated and also the heart muscle. (2) beat is raised through paralysis of vagus, and (3) great irregularity, through the ventricles having taken on their own idio-rhythm, and this rhythm at times co-inciding, at other times, not, with the auricular rhythm

And so in the Angina paroxysm we can imagine a somewhat similar train of events. To begin with, a ventricle in distress. The vagus goes to help but only makes matters worse. The augmentor

Pathology influence now sets in, and to counter act this the vagus may be so incited that it speedily reaches the state of paralysis and the ventricle is left to its own rhythm and under the influence of the augmentor.

The profound cardiac pain which is so often, but still not a necessary, feature of angina has been attributed to various causes.

The heart in its histology is unique and hence its own peculiar pain. The organ itself is not sensitive to many impressions which are elsewhere often productive of severe pain, and hence extensive change and disease and little or no pain — as in pericarditis. (Vide antea).

But when cardiac pain is felt it has a profound effect on the patient. Even the consciousness of the heart's action must be called pain for when any part of the body obtrudes — however slightly on the sensorium then

Pathology such part is the seat of altered sensation — pain. But some people may be unduly appreciative of the normal action of the heart. We may however, call this consciousness of the heart's action, the first Degree of cardiac pain. It may be caused by indigestion, excess of alcohol or tobacco, emotion — such as grief, and hysteria and nervous exhaustion. The second degree of cardiac pain gradually merges from the first, and from similar causes. In this Degree the pain is akin to 'physical' pain but is also accompanied with great anxiety:

And this great anxiety, deepening into the awful cardiac terror of "mediatis mortis" which characterizes true angina.. Doubtless there is a continuity in kind between these three degrees yet they differ just as the stages of inflammation do, and their individuality and their right to specified nomenclature must be recognized.

Pathology

Looking now to the actual paroxysm of angina we know the ventricle is dilated and often extremely so. Now, all hollow organs of involuntary muscle fibre are extremely sensitive to increased internal pressures. The excruciating pain of a distended bladder or stomach or colon is well known. Increasing pressure and exhaustion of the muscle fibre is followed by dilatation and in this ventricular distension the nerves and bloodvessels are implicated. The vessels are more or less occluded and hence the blood supply is diminished. Further, the rise of pressure within the auricle, following the increase within the ventricle retards the coronary venous circulation, and so further impedes the coronary circulation as a whole (Porter).

Thus the weakened and struggling heart is deprived of its blood supply. Romberg defined pain as the "prayer of the nerve for more blood" and it

Pathology

is well known that ischaemia in any part of the body is followed by excruciating pain.

Traube holds that the pain of true angina is due to tension and stretching of the nerves of the heart during extreme dilatation. And it has also been suggested that the nerves as they closely follow the distribution of the coronary arteries may suffer more severely from tension if the arterial walls are hard and rigid from atheroma.

But, as already noted there are cases of angina sine dolore. Going back to the consideration of the distension of other hollow muscular organs, say the bladder, it is found that if the distension is not relieved, the pain at first so excruciating, gradually dies away. Now in angina the myocardium is profoundly degenerated. The heart in these cases, in embarrassment, rapidly dilates and the intensity of the stimulus is at

Pathology

nerves beyond the perception of the nerves which are also degenerate with the muscle fibre. This form of pain then, may not be present in Angina and the patient may be only conscious of the third and extreme degree of cardiac pain, the "mediastis nervosis". And only very very rarely – if indeed at all – is this form absent. Pain in or over the heart we know as ever a source of anxiety and fear. But in angina there is more. There is the feeling of the actual presence of the last dread enemy at the innermost gates of life. And this feeling – as I take it – is due to the great call on the Kat-abolic nerve. In the symptom "Tremor Cordis" there occur suddenly in the rhythm of a regularly beating heart three or four successively small and incomplete systoles and ending as suddenly as they began with a violent beat or throb And the violence of the throb is in proportion to the number

Pathology of the imperfect beats The explanation is, the vagus is itself inhibited. The ventricles beat in their own idio: rhythm, rapidly and imperfectly – and hence increasing ventricular residual accumulation. This condition requires the aid of the augmentor to expel the abnormal ventricular contents and so allow the heart to get back to properly regulated action (Balfour)

And in precisely similar manner an attack of Angina may be developed. Vagus inhibition – as from anger. Increasing residual accumulation. Ventricular idio: : rhythm. Augmentor aid, and cessation of paroxysm

The sensation felt in tremor cordis is totally different from mere physical pain, and, at the moment of cessation – coincident with the throb – is felt a lightning flash of the true 'meditatio mortis' and sine dolore. The augmentor is a purely sympathetic nerve and hence its expression of unwonted stimulation or exhaustion

Pathology is in this peculiar sensation, the real essential of angina.

As the processes of decay, which culminate at some time in an angina paroxysm, are slow and gradual, symptoms, at least suggestive, may at times be manifest from operation of one or more of the causes cited under Etiology.

Reviewing, we may put in columns the various conditions present in the heart during the paroxysm.

(α) Muscle fibre	(β) Nerves	(γ) Vessels
Increased expenditure. Diminished income. Stretching.	Increased stimulus through Ischaemia. and Stretching.	Obstruction in circuit by Stretching. at exit by increased pressure.

And within the ventricle is an ever increasing tension: and then, also, in many cases the abnormal sensations thus excited may be magnified by a hyperaesthetic consciousness which is part of the neuropathic state.

Pathology

The view held by Heberden, and supported by Latham, was that Angina Pectoris was a neurosis culminating in spasm. The effort of a weakened heart endeavouring to overcome the increasing tension may be compared to spasm but complete spasm is not possible in a weakened heart nor is spasm possible in a muscle deprived of blood.

And supposing complete spasm did occur, the result would necessarily be death from the break in the circulation. Again, post-mortem examination has never found the heart in systole but always in diastole.

Laennec and Hope held that angina was a pure neurosis. The pain was periodic and paroxysmal, obeying the law of all neuroses. But a heart which is morbidly so profoundly sensitive to irritation must always have a pathological basis for this hyperaesthesia.. And the pain of angina has

Neurosis may be defined as a functional disturbance of a nerve or a group or system of nerves, manifesting itself in "hyperaesthesia"; and further the neurotic state is generally traceable to heredity.

Pathology

been too often compounded with adjacent neuralgiae

A fatal case of angina is never the result of a pure neurosis. vaso-motor spasm and reflex inhibitory influence will not of themselves precipitate a paroxysm of true angina. The heart must at the same time be in a degenerate state. Very often arterial decay and vaso-motor disturbance are associated with the neurotic diathesis, and if with this there be an inherited or acquired predisposition to cardiac disease and degeneration such a combination may be manifested in an attack of angina. Angina has been defined as "a mere neurosal incident of cardio: vascular disease" Now this is to be remembered that in the consideration of angina the heart and the vessels must be looked on as a single and indivisible system. And in angina the vascular part of this cardio-vascular system often shows such changes which are apparently truly neurosal

Pathology

and no doubt this element is part of angina

Inhibition of the vagus if powerful enough may cause the heart to miss a beat and the result is syncope; while paralysis of the augmentor enfeebles but does not stop the heart. And in less grave forms of heart trouble, say persistent irregularity, there is always a permanent change in the heart muscle.

Trousseau considered angina an epileptiform neuralgia. Richardson held a somewhat similar view. The pain sometimes beginning in the hand or fingers and travelling upwards and thus simulating the aura epileptica. The face at first pallid and bedewed with sweat and afterwards getting flushed and red. These symptoms associated with the paroxysmal and periodic pain and no apparent lesion of the heart were the clinical evidence which led Trousseau to this conclusion. Angina is however extremely rare

Pathology in epilepties. Still the question might be raised, may not some neuroses have but one and that an exclusive manifestation? Cases of traumatic epilepsy with associated attacks - very suggestive of Angina have been reported. Here it might be noted that Van Gieson — in the relation of auto-intoxication to the pathogenesis of neuroses holds that Epilepsy is the manifestation of auto-intoxication through the brain-cells.

In like manner the manifestation may be through a weakened heart. And looking to the relationship of gout and angina this theory of auto-intoxication appears a very likely one.

Parry called angina pectoris, Anginosa Syncope or Syncope Anginosa and considered it was a syncope associated with anxiety and unusually severe pain. But in syncope the patient falls limp and unconscious. The pulse is imperceptible and the breathing faint.

Note* It is remarkable how consciousness — although not always complete — is retained in most cases of Angina as is known, the carotid arteries have a special mechanism whereby their internal pressure is always maintained at a high level. In syncope these arteries partake in the general relaxation whereas in Angina there is no such relaxation and hence the cerebral circulation is efficiently maintained.

Pathology In angina paroxysms the patient in the majority of cases is conscious to the last*. His pulse is never quite imperceptible and his respiration is often quite unaffected or only slightly increased in frequency.

Death in angina is from failure of the heart to expel its abnormal contents. This failure (asystole) may be quite sudden or ingravescent. In syncope the heart beat is inhibited. Recovery may take place within a few seconds even after complete inhibition.

Syncope is, however, often associated with a fatally degenerate heart, and hence the angina paroxysm may occasionally partake of a syncopal type. And in some cases where angina attacks have ceased the patient is yet troubled, and from syncopal attacks.

And so it is not surprising to find, at least sometimes, patients suffering from angina lapsing into unconsciousness, even into death, in apparently attacks

Pathology of syncope, but which are, in all probability, really Angina sine Dolore. From these considerations, then, we would say that Angina is totally distinct from Syncope.

Prognosis. The prognosis in true Angina is always grave and as uncertain as grave, depending chiefly on the textural soundness of the heart muscle. Pronounced fatty degeneration means extensive and irremediable textural change and hence fatal Angina is more to be dreaded. Habitual high tension (as indicated by pulse) points to some obstruction in the arterio-capillary area. This condition, however, can be remedied – at least mitigated. It also points to some degree of vigour in the heart. If the impulse of the heart be at all fairly well marked, one can rest assured that the ventricle yet retains in good measure its pristine vigour.

Attacks coming on with increasing severity and at lessening intervals point to a speedy end. Still treatment may post-pone such a calamity and so modify a provisional prognoses.

Prognosis

Recovery from even severe Angina is quite possible, in so far as the patient may live for 5, ~~perhaps~~ 20 years after the first attack.

If the heart be more dilated and in a state of asthenia treatment may do great good. But if failure follows a considerable hypertrophy the results from treatment are never great or lasting.

The severity of the pain, the frequency and the facility of exciting the attacks, and a degenerate condition of the myocardium, with persistent arrhythmia and more so if the patient be not much over middle age (pointing to premature decay — Balfour. G. W.). and with a history of heredity — such a combination is of very unfavourable prognosis. The end is generally sudden.

It may be noted here that John Hunter lived for twenty years after his first attack and

Prognosis

from that time till his death he accomplished much good and laborious work.

Death may take place (1) Suddenly and instantaneously in the first attack, with or without a cry — as is often seen in elderly people, after some undue or unwonted exertion. At the autopsy coronary Disease is invariably found.

(2) In the first paroxysm or group of paroxysms, which may extend over a varying period, from a minute to a day. (3) Or the end may come after many years and suddenly as in the above two modes.

Case III. Man. Act. 53 Good bodily frame.

Of Sedentary habits. A heavy smoker, and drinker of malted liquors. Had suffered business losses and consequent mental worry and insomnia.

For about 10 years he had suffered mobile rheumatic pains and especially over the left shoulder.

During the last three or four years he was conscious of occasional cardiac intermissions. His pulse, he said was always over 80.

And just of late he had complained of attacks of pain about the heart even when walking gently. On these occasions he would stop and look into the shop windows till the pain died away. One night having been aroused from sleep and suffered exposure to cold he was seized with a violent paroxysm of Angina Next day any attempt to rise brought on threatenings of the attack. During the attack

Case III his chest felt "as if gripped in a vice" and the "meditatio mortis was expressed in the words "I felt as if I were just without the judgment."

The heart was weak in impulse, and slightly dilated. The sounds were weak. Pulse 104. but regular. Liver was (slightly) enlarged. Kidneys and spleen normal. Lungs right. And there was no evidence of arterial degeneration.

Treatment. The treatment of Angina Pectoris is directed, firstly, towards immediate alleviation of the paroxysm and secondly, towards the strengthening of the myocardium directly and also indirectly through influencing for good the general bodily metabolism and in this way removing any fault within the arterio-capillary area. As regards prophylaxis the patient will in his own bitter experience have learnt in full the principles of submission and limitation in the daily walks of life and duty.

In 1872, Lauder Brunton found on examination of a patient suffering from a paroxysm of angina that the radial pulse indicated high arterial tension. Knowing that amyl nitrite had a speedy and powerful influence to lower arterial tension he exhibited the Drug and with the happiest of results.

Treatment

Amyl nitrite is best given by inhalation because by mouth it is not always so successful. Its full effect is produced in from half to one minute, the dose required being from one to three minims. Larger doses can quite safely be given for amyl is not nearly so dangerous a remedy as is generally thought.

Amyl nitrite

Amyl nitrite paralyses the sympathetic ganglia and the motor nerves of the vaso-constrictor apparatus and great dilatation of the vessels ensues.

Beginning with a small dose tolerance is soon established and without doubt the use of amyl in cutting short the paroxysm conduces to permanent relief, and that, even with diminishing doses. This is the view that Ringer holds but of course the other lines of treatment must also conduce, and in no small part, to the subsequent and permanent success. Amyl is also analgesic but this

Treatment

property is reduced or lost if the Drug is not fresh.

Its use is almost always successful. The dilatation of the vessels (which persists for some good time after its other effects have gone) causes the blood, accumulated through whatever cause within the ventricle and aorta, to be sucked out into the peripheral area of diminished pressure. In cases where amyl has failed to relieve or the relief granted was but transient there has nearly always been found aneurysm of the aorta. In such cases a hypodermic of 1/4 grain of morphia will give the desired relief. Still amyl nitrite is the first and best remedy to subdue the paroxysm.

Nitro-glycerine

Nitro glycerine (Trinitrin: Glonoin) is a trinitrate of Glycerine but in the system it is changed into a nitrite and its action is similar to Amyl. It is slower in giving relief – 3 to 5 minutes.

Treatment

minutes, but its influence is more lasting – being extended over 3 or 4 hours. Nitro glycerine is therefore best used to sustain the beneficent effect of amyl nitrite. It has no effect at all, on the heart.

Headache and flushing usually follow the first administration of the Drug but latterly only a feeling of warmth is expressed. In truth when the Drug has been exhibited on true indications no unpleasant feeling results, not even after such large doses as ten minims every 4 hours. Like amyl and many another Drug it has been looked on with awe by the timid and the alarmist. Nitro glycerine may be given in the 1% solution of the B.P. – dose 1 min

But far better is the 'tabella' preparation. Each tabella contains 1/100 grain of the Drug and by nibbling a bit every 10 or 20 minutes the best effect of the Drug is obtained and free from all unpleasantness.

Note* In such cases the heart will be found beating with great rapidity and hence the non-success of a drug which increases the heart beat in rate, without lending to its strength.

Treatment

But, be it noted that in some cases of Angina the first attack has so weakened the heart that seizure after seizure follows. Amyl or nitro glycerine gives no relief and therefore they are credited with failure. In such cases* stimulants are indicated and to administer at the same time small doses of the nitrite.

Nitrite of Sodium (gr I - IV) acts also like amyl. It is however much less useful, and is further an unstable salt. If the two previous nitrites be not tolerated this nitrite may be tried.

Other nitrites have a similar action to Amyl. Their action though rapid is transient. Thus Propyl, isobutyl and ethyl nitrites may be used. The last is contained in the B.P. Spt. Aetheris Nitrosi, hence in an emergency this drug may be of genuine service if given in large enough dose.

Treatment

A Drug more rarely used from its great expense is Erythrol tetranitrate. It very much resembles nitro: glycerine but its influence, if more lasting, is less potent. It is a difficult drug to work with and has to be handled very carefully. Dose grs I - VI.

All the Drugs above discussed should be Dispensed alone — free from combination.

Previous to the use and unquestioned superiority of the Nitrite group in the immediate and initial treatment of Angina many remedies had been tried. The leading principles were stimulants to the heart, and opiates to relieve pain.

Alcohol, ether, and ammonia were found the most useful; combined with heat, which indeed is the most powerful of cardiac stimulants.

The opium was given by mouth or as morphia

Note If flatulence be troublesome a draught
of Spt. Ammon Aromat. 3ß, Spt. Chlorof. min XX,
& Spt. Aeth Co 3ß with or without
Chlorodyne min X, may be administered.

Treatment

hypodermically. Large doses of morphia can be given – half a grain every four hours, with 1/100 gr of atropine, in each dose. To get effect as rapidly as possible plunge the needle deeply.

Chloral hydrate was given and in large doses with success, for chloral lowers blood pressure (1) by the heart (2) by the vessels. But its depressant action on the heart is greatly against its use.

Chloroform was often given by inhalation and in emergency may be used with effect.

Venesection was of no use except perhaps in those very few cases where there was great venous congestion. In such cases amyl was of no use, indeed its administration seemed to worsen the plight of the patient. The withdrawal of blood – even in small quantities 4 to 6 oz – in such cases gave great relief. As regards venesection in other conditions, it has only to be noted that blood-letting

Treatment

unless in such quantity as to be Dangerous, does not affect arterial tension.

Cardiocentesis. — plunging a needle into the muscle of the heart has often stimulated its flagging energy

The immediate good effect of a hypodermic of Strychnine over the heart is due to the prick of the needle.

Oxygen is another admirable cardiac tonic, and in the state of profound fatigue following a paroxysm of angina a few whiffs of the gas every hour has a wonderfully restorative and stimulating effect.

In the second part of the treatment; medicine, diet and exercise, all are of great importance

The careful avoidance of any exciting cause is to be followed up by an attempt to remove the more remote or predisposing causes. A careful diagnosis therefore will point out most truly the lines of treatment.

Treatment

And to be at all successful, the treatment must be carried over many months till long after complete disappearance of angina.

Our first consideration will be the heart, and its medicines. And of all medicines, Digitalis is easily the first of heart tonics. It is a true heart-food, and so improves the nutrition of the enfeebled myocardium. For this purpose small doses must be given and the beneficial effect of the Drug is only obtained through prolonged administration. The heart is to be coaxed — not goaded.

Digitalis

As regards the action of the Drug, the heart through inhibitory stimulation is slowed — bradycardia, and the pause in the cardiac cycle being prolonged. Hence the myocardium will get an increased supply of blood. But the chief effect of Digitalis is local and direct on the contractile muscular fibres. To increase the

Note on Strophanthus. This drug has never come into great favour. Perhaps the chief reason for this is that the preparations used were unstable.

For now, when given in tabloid form it undoubtedly has a good effect on the heart muscle.

Treatment

muscular tonicity and contraction – the result being a heart beat of greater vigour and a more firmly contracted myocardium. The fibres of the muscle hypertrophy – there is no hyperplasia and this result is best obtained from intermittent use of the drug. A dose of 4 minims twice a day – missing the middle day of the week may be continued for many months. The cumulative effect of digitalis is but an expression of its physiological action in larger doses.

Digitalis contains four glucosides – digitalin, digitalein, digitoxin and digitonin. The first three contract, the last dilate the arterioles. Digitoxin is the most potent and being insoluble in water is absent from the infusion. Hence the Tincture is the better preparation to use. Digitalin is often used but is neither so good nor reliable as the tincture.

If the heart be exceeding weak from extreme

Treatment

fatty degeneration digitalis must be most sparingly used. But there will always be some intact fibre which will respond to the treatment. And any response, no matter how feeble, is a decided advantage.

Then again, the effect of Digitalis in contracting the arterioles and so raising blood pressure is not very evident after 4 minim doses. Further the increased blood-pressure following administration is in part due to increased and more efficient output from the left ventricle. Still in any case it is advisable to exhibit some vascular stimulant – a vaso-dilator.

This desired effect is in most cases kept up by the continuing use of nitro-glycerine. Another property of these small doses of digitalis is the beneficial tonic and better effect on the stomach.

Reverting to the necessity of counteracting the arteriole effect of Digitalis, and if the use of

Treatment

of nitroglycerine be suspended, Potassium Iodide is more than a mere substitute, and is often of remarkable service — if well borne. It keeps the blood pressure low and the circulation tranquil. Excitement of the heart is allayed and the arterioles dilated. But the real benefit of Potassium Iodide to the arterial circulation is very probably due to its influence on the environment of the capillary circulation. The blood is so influenced that the flow into the veins is easier and fuller and hence the fall of arterial tension. For this purpose small doses gr $\overline{I}$, t.i.d. are sufficient. In very much larger doses gr $\overline{XXX}$ – $\overline{LX}$ t.i.d, the iodide has a remarkably powerful and permanent influence for the good on atheroma and therefore the drug should be then exhibited if there be any evidence or even suspicion of such arterial change. And its exhibition should be pushed with confidence and vigour.

Potassium Iodide

Note
Both Pot Iod and Mercury give the best results when arterial tension runs high.

Treatment

The Diatheses of Gout and Rheumatism will also benefit through the administration of the iodide.

As large doses of the Potassium depress the heart Sodium Iodide may be used wholly or partially instead.

Mercury

The use and action of Mercury are in part very similar to Potassium Iodide. Both have the beneficial 'alterative' effect. This effect is due to its influence on the capillary environment. It promotes venous return and stimulates elimination of waste material. When within the bowel Mercury acts as an antiseptic; and also by its influence over the liver determines a greatly increased biliary output. Therefore in all cases of Angina, save those where the heart is in a state of profound degeneration, with vessels of greatly diminished tension Mercury in the form of Blue pill gr I. t.i.d is of invaluable good. . The patient is in

Treatment

bed, and kept at absolute rest for Days after the paroxysm and during this time — before the administration of digitalis is begun — Mercury acts with charming success and especially so if the pulse indicate high tension Later on, the Drug is given only occasionally.

Next in value comes Arsenic.

Arsenic

Arsenic acts (1) on the stomach which is so often disordered (2) on the nervous system giving tone and vigour to the whole body. It has also a directly beneficial effect on the heart, and its effect in Angina is sometimes magical. Like Potas Iodide and Mercury it influences the capillary environment — where the business of the blood is done — and promotes healthy metabolism.

It is given in small Doses — Liq Arsenici hydrochlor min II. t.i.d p.c.J. and over a lengthened period.

If intolerance, persistent and intractable supervene Arsenic may be given in small enemata — 3ii to iv.

Treatment

containing min VI to XII of the Liq Arsenici.

Like Potassium Iodide, Arsenic is beneficial in gout. And it may be stated that there is no danger in leaving off the Drug.

Strychnine

Strychnine is a powerful adjuvant to digitalis and like this Drug it is cumulative only in large doses. Small doses can with safety be given for a long period.— Liq Strych hydrochlor, min II - IV. t. i. d.

It acts as a bitter tonic to the stomach and imparts a good tone to the nervous system. Further, it is a direct tonic to the heart and may be given hypodermically in the precordial region. The capillary circulation is also promoted as is seen in the use of the Drug in cold hands and feet.

Phosphorus is allied, and similar in many respects, to Arsenic, but its influence is not so powerful for good. The dose must be exceedingly small.

Treatment

small – Pil Phosphori, gr I (equal to 1/80 gr of Phosphorus)

The hypophosphites prove a useful tonic, as a combination of many valuable drugs may in this way be exhibited. Broadbent (W) speaks very highly of Phosphorus.

The salts of Calcium, have, of late, attracted much attention in cardiac therapeutics. Experiment shows that both calcium and barium increase greatly the force of the cardiac contractions.

Barium

The use of the Barium waters at Llangammarach has been followed in cases of heart failure by great and lasting good.

A mixture of the hypophosphites (as prepared by Fellows) containing also Arsenic and Barium with perhaps a little pepsin has yielded excellent results (in Case III).

In cases where there is marked anaemia Iron in some mild preparation should be given. Iron mixes well

Treatment with digitalis if these be added to the mixture dilute Phosphoric acid. Or, the Pil aloe et Fer (B.P) may be ordered nightly. With this pill mercury may be combined and half of the aloes deleted.

In anaemic and neurotic patients, it may be recalled that intolerance of Strychnine and arsenic is easily excited and digitalis is more apt to sicken.

With a weak heart are always associated a weak stomach, torpid liver and atony of the intestines with constipation. If it be necessary to prepare the stomach for the exhibition of the remedies just discussed, the best response will be elicited by an alkaline tonic and a skimmed milk diet. The liver and bowels will quickly improve under the Pil Hydrarg. given at night, followed by a saline draught in the morning

Treatment

With the skimmed milk diet and the Saline Draught the kidneys are efficiently flushed, and in a few days the system is prepared to receive the full benefit from the medicinal remedies.

Alcohol is not required, except as indicated in the prostration following a paroxysm. Elsewhere it is neither stimulant nor food. Its quasi-stimulant action is hurtful to the heart, and directly it may aggravate an irritable stomach.

The sipping slowly of a little hot water, three times a day, half an hour before meals, is infinitely better than alcohol in any form. The addition of a little of some alkaline salt — ordinary soda or cream of tartar, lends to the good of the hot water. By this (1) the heart is stimulated (2) thirst relieved (3) stomach cleared of mucus (4) the gastric juice stimulated in flow.

Treatment

Diet. The rules as to diet must be strictly enforced, of course with consideration of individual taste. The meals are to be light and bland. The value – the curative effect, indeed, – of milk treatment in persistent disorder of the stomach, is without doubt.

At first, let the diet be chiefly milk. There is no fear of the patient being starved, for the digestive and assimilative powers are feeble and slow. Afterwards

Diet

'stoppy' meals must be strictly avoided. Excess of liquid, diluting the gastric juice and distending the stomach itself, interferes with the muscular movements of the organ and so hinders digestion and therefore tends to flatulence.

Again in cases where the circulation is weak liquids are slowly absorbed from the stomach. An interval of 4½ to 5 hrs should be between each meal; and after each, rest for about 3/4ths of an hour should be enjoined.

The last meal should be not later than 7-30 pm. and sld be very light.

Treatment.

Regarding Rest and Exercise, each case must be given individual consideration. Sleep is all important, and should at least extend over a period of eight hours Insomnia often disappears under the general systemic and cardiac treatment. But if it persist, Sulphonal gr XXV to XXXV, taken in hot milk three hours before retiring is the best hypnotic. Its effect may be strengthened by a XX grain dose of Potassium Bromide, two hours after taking the Sulphonal. Paraldehyde and Chloralamid may be also used.

Sleep

In the beginning of the treatment the patient must be kept absolutely at rest. He is to make little or no voluntary movement. This is to be insisted on.

Rest

He should even be fed. After Days of such quiet rest, gentle massage may be commenced and the patient allowed to sit up in bed for a few minutes

But one must ever remember that capacity for

Treatment

for even such limited movement will vary from day to day.

Gradually he is allowed on to his feet. But the most of the day is to be still spent "at rest" Massage is now more vigorous. And so, day by day the sphere of movement is widened. Soon he is walking outside in the sunshine and when fit he goes off for a change and then to live as far as possible a life in the open air. When walking for some time is not followed by breathlessness or praecordial pain or anxiety, Oertel's hill climbing exercises may be with care commenced.

The Nauheim system of baths are quite unsuitable to most cases who have suffered angina.

Electricity

Last but not the least comes Electricity. The therapeutic use and good of Electricity have never become at all properly appreciated by the profession and yet when rationally employed it is conducive of great and permanent benefit.

Note Trousseau in his clinical lecture on angina cites two remarkable successes of Electricity.

Treatment

In every form of derangement of the cardiac nervous government — such as often follows influenza, and strain of a heart labouring under valvular defect I have seen surprisingly good results follow the use of electricity. The general effect of Electricity — if given in proper doses, — is both sedative and stimulating. Thus, the cardiac nerves are in their over fatigue too wearied to rest. The stimulating property of Electricity rouses them to rest and the result is sedative and grateful.

Perhaps the chief reason Electricity is not so generally employed is the seeming difficulty of regulating the Dose in respect of the strength of the current and the frequency and duration of its application.

Without Doubt the continuous current is best; such as may be obtained from a small battery of from 4 to 6 № 3 Leclanché cells.

Treatment

Now the application must not be made in any haphazard way. Begin with small doses – one milli ampère (1.M.A), of short application (one to two minutes) once or twice daily. The patient himself will prove the best judge of the strength of the current. Gradually the applications may be made more frequently but neither the dose nor the duration should be augmented. The stabile anode should be over the course of the vagus nerve in the neck, while the kathode is slowly moved up and down the cervical spine. Another very important point is the superficial area of the electrodes. The kathode should be three times larger than the anode – the anode being about 2½ inches long, and 3/4ths inch broad.

False Angina

False Angina. Pseudo angina. Angina Vera. Angina Vaso-motoria (Nothnagel).

So numerous and diverse symptom groups, resembling more or less imperfectly true Angina, the above terms have been given. Such cases are exceedingly common. Some authorities say they are merely imitative, while others maintain that they are really gradations towards, and may latterly evolve into true Angina — even in a few days. Huchard says "Il n'y en a qu'une seule" Powell says "there is a continuity in the phenomena" of the "true" and the "so called" false angina, and the difference is but of degree — not of kind.

However one must remember that many cases have been recorded as angina which undoubtedly were not. And again, the manifestation of true

Angina vaso-motoria.

angina may be so slight as to be mis-apprehended or overlooked. And no doubt many cases of false angina ultimately end in true. Still we would not call "congestion" inflammation, and yet the difference is only of Degree. Hence it is desirable that the term angina should not embrace the slighter degrees which lead up to the true angina paroxysm. Describing a case of angina vaso-motoria after the lapse of many years, Habershon writes, "The patient is a woman over 50 years of age, and the attacks have continued since the age of 19. It is interesting to note that in her case the heart has become weak and degenerate." And the symptoms of the latter end were of all the severity of the true angina.

The attacks of pseudo-angina are not

Angina Pseudo: Vaso-motoria.

so abrupt of onset as in the true, and are not so severe although they are of longer duration. Very often they are preceded or accompanied by profound and widespread vaso-motor phenomena, such as flushing, pallor, coldness of extremities, dermatographia and noises in the head. Emotion and Dyspepsia rather than exercise are the causative elements.

The respiration is often hurried and gasping and the patient is restless and tosses about. As a rule the patients are younger than those who are afflicted with true angina, and are obviously 'neurotic'. Women are just as often attacked as men — this in striking contrast to true angina. The pain may be acute but the sufferer has no "meditatio mortis" although he may declare "he was frightened he was going to die" And never do such cases end in death.

In the treatment of Pseudo-angina many lines

Angina vaso-motoria.

may be followed. Caution as to the use – or abuse of tea and alcohol; Over work – mentally; Excess in venery. Any cause of high tension – diabetes, Kidney Disease, gout or anaemia to be suitably ordered. The Diet and habits of life are to be restricted and regulated.. The immediate treatment will vary according to the severity of the attack.. Heat – in its varied application – will be the most efficacious, and bromides will prove a useful sedative.. Alcohol is better avoided.

Epidemic Angina such as Gelineau recorded in 1862 is hardly worth passing notice.

The 'Tobacco Heart'.

The chief effect of the active principle of tobacco – nicotine – is seen on the heart. and the symptoms are generally those of vagus paralysis

The heart becomes very irregular (1) as to rate, (2) rhythm, (3) force. The symptoms also vary with the blend and kind of tobacco used.

Thus the finer tobaccos oftenest reveal their toxic effect in an intermittent pulse and a liability to syncopal attacks. Whereas the coarser kinds cause great irregularity – amounting to delirium cordis – associated with more or less cardiac pain and anxiety. Habitual and heavy smokers, then, may be seized by such a combination of symptoms as may be highly suggestive of angina.

Thus – a sudden, and, as it were, a

Tobacco. spontaneous attack. Features, pallid and bedewed with sweat. Vertigo and Syncope. Cold extremities. Great cardiac pain and anxiety. Very feeble pulse.

The treatment is – absolute abstinence from tobacco.

There can be no doubt that a toxic agent – such as tobacco – by its selective action on the heart, determines a process of decay, and more especially so, in a subject of the neuropathic diathesis, and this process of decay is sometime revealed in the Angina paroxysm.

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