NOTES AND COMMENTS ON A CASE
OF ANGINA PECTORIS

HISTORY:—

During the last six years of his life my father was liable to paroxysmal attacks of intense pain over the region of the heart radiating down the left arm. Such paroxysms were invariably associated with the sense of impending death—angor animi—and were absolutely typical of angina pectoris.²

Already for years his pulse-tension⁴ was known to have been high and his prematurely aged appearance testified to involutionary changes associated with diffuse, vascular degeneration.⁵

He was a tall well-built man with powerful muscles but remarkably old looking for his years, so that at about thirty he would be taken for fifty.

From the age of fifteen years to thirty-five his life had been strenuously active. Six years of this time had been spent in the Australian bush, and for another five years he worked as mechanical engineer with a firm of locomotive builders, besides carrying on scientific studies in his spare time. Later his occupation as astronomer entailed irregular hours and exposure in the open air, with prolonged spells of observing, often for the greater part of the night, and kept up for many nights running. In after years
he remarked, that the irksome position which the astronomer
has often to take up for hours besides exposure to cold,
were not the least of professional hardships.

Enthusiasm and eagerness for his work would carry
him on regardless of fatigue, and would frequently
lead him to make extreme demands on his mental and
physical powers. 7

Long journeys on scientific investigation had been
taken into arctic and tropical wilds. Six months
spent in exceptionally high altitudes of the South
American Andes, were then already at the age of forty-
five felt to be a severe strain; the attacks from which
he suffered twenty years later recalled the symptoms of
circulatory embarrassment which he had then experienced
in a milder degree.

Finally the hill climbing which the situation
of the Edinburgh observatories necessitated was almost
from the first more than he could manage without great
effort.

He was a strictly temperate man, abstainer and non-
smoker; though he certainly did consider a somewhat
ample allowance of flesh-food essential. 8

While in the tropics he suffered from yellow fever
and also contracted malaria. With the exception of
some tendency to digestive disturbance of which heart-
burn was one of the usual symptoms, he enjoyed very
fair health until over fifty years of age.
In 1890 he incurred a very definite muscular strain causing severe pain in the left shoulder; this afterwards became chronic and was thought to be rheumatism. That same year he had influenza for the first time, and since then repeated attacks of influenza began to undermine his strength and apparently led up to the first attack of angina pectoris which occurred in 1899, six years before his death.

As regards his first attack: there was the history of somewhat unusual amount of physical exertion in connection with a scientific experiment; digestive disturbance and malaise supervened which were attributed to a mild attack of influenza. He had been in bed for two days when he was suddenly seized with intense cramp-like pain at the heart, and down the left arm, with a sense of constriction about the lower costal zone associated with an absolute conviction of imminent death. There was a feeling of great inflation of the stomach which seemed as if closed up by spasm, and sudden development of considerable abdominal distension. He was lying quietly in bed at the moment of seizure, which occurred about 1½ hours after supper. Relief of the flatulence coincided with subsidence of the attack, as has been emphasised by Sturge. From that time he was unable to carry on his ordinary duties without undue effort and fatigue, and began to complain of difficulty in going uphill. He had to guard against hurrying,
otherwise the same feeling of constriction and pain in the chest tended to recur. A month later another definite attack came on suddenly while out walking on a frosty winter's night. It was observed that he became very pale and his hand felt icy cold. Until the symptoms began to pass off he remained standing rigidly still, but was able to speak quietly and slowly in his usual voice."

In summer his health seemed to improve and for fully a year he remained comparatively free from distress and managed to carry on his work. But he made it a practice to carry Nitro-Glycerine about with him as without this drug he could not walk up-hill. He had to avoid undue exertion, or sudden exposure to cold and had to rest after meals.

The following year he again had influenza (?) This time with grave nervous symptoms, so that he had afterwards to take a prolonged holiday, during which time all the more severe symptoms of angina kept completely in abeyance. This illness is vividly recalled to my mind when I read in Reynold's System of Medicine the short account of the illness from which John Hunter suffered in 1785 and 1789, a few years before his death of angina pectoris. It began gradually with indigestion and depression, and with what was most unusual in him gloomy anticipations
as to the gravity of his ailment. He looked much more ill than the immediate symptoms at first would account for. Obstinate hiccough developed, and then intense right-sided earache with pain at the back of the ear and over the right parietal region. Wasting was rapid and extreme. Later there was mental and physical unrest and wakefulness with some disturbance of the higher faculties as well as transient bulbar symptoms. The right-sided hemianopia which was recognised later most likely occurred at this time.

Stengel's article in the British Medical Journal is suggestive that these various illnesses, whether due to influenza or not, will have coincided with further vascular and myocardial lesions; and that the decided downward step with each of these several indispositions marked a definite advance in cardio-vascular degeneration.

After another, and more typical attack of Influenza which left him greatly debilitated he was advised to go abroad. The exertion and excitement of the journey induced an intense attack, the first after complete remission of the actual paroxysm for twelve months — and for several weeks he was confined to bed suffering from frequent attacks of agonising severity.

Under treatment he temporarily improved. With avoidance of all exertion and rigid attention to diet &c. the attacks were kept somewhat under control; but
recurred from time to time in greater or less intensity. He became greatly aged in appearance and steadily weaker. In spite of precautions the attacks gradually became more frequent. At the time of examination here appended that is five years after the first occurrence of angina pectoris one finds him an invalid unable to exert himself either physically or mentally without risks of grave distress. Though not more than sixty-seven years of age he has the appearance of quite an old man.

He is usually able to be up and about, weather permitting he can walk a few hundred yards out-of-doors but dreads exposure to wind and cold. He is cheerful and bright and full of interest in everything connected with the Observatory, and in the many other subjects that had always appealed to him, while he keeps himself busily employed with light mechanical engineering work which he finds less tiring than mental exertion. He stoops slightly and his gait is somewhat hesitating owing to debility and impairment of sight. He is spare to emaciation and easily feels chilly; the face is comparatively plump, and when he feels well is of a healthy-looking ruddiness. The bald scalp is attenuated. There is well-marked arcus senilis. The hands are thin and pallid and often cold, with brittle nails showing longitudinal grooving; several of the phalanges show slight thickenings.
The radial pulse is visible but the line of the artery is not in evidence; the wave is variable. When the patient feels well it is of moderately large amplitude, and somewhat gradual rise and fall, frequency is usually between 60 and 72 per minute. There may be an occasional missed beat when as the tracing shows the pulse is not absolutely regular in time and force. The vessel feels like a tendon; it is not tortuous nor can any patchy thickening be felt; during manipulation the artery tends to harden under the finger, the pulse-wave becoming smaller and more sustained, while the patient complains of vague discomfort referred to the action of the heart, and may become appreciably pale in the face.

The superficial temporal arteries are very tortuous with thickened walls, and the slightest touch may make them become rigid and stand out prominently. Other superficial arteries could also be felt to have uniformly thickened walls; no modular arterial thickenings have been found anywhere.

The visible veins of the hands and forearms are full and prominent. There are no varicose veins and no distended venules about the thoracic arch. Visible venous pulsation in the neck has only latterly been noticed, and there is now some tendency to oedema of feet and ankles. As regards the capillary circulation the general appearance of the patient betokens a scant
capillary supply (Mackenzie).

Subjective symptoms referable to the circulatory system indicate great contraction of the field of cardiac response. Signs of cardiac embarrassment such as shortness of breath, disturbed pulse as to rate and character, with a sense of tightness at the heart amounting even to intense pain may be brought on by any sort of slight exertion; and will be discussed more fully in connection with the description of the attack of angina pectoris.

One should mention that these limitations of cardiac response were subject to considerable variation.

Proceeding to examination of the chest and praecordia; emaciation has accentuated the normal hollows, the bones and muscles are large, but the muscles of the left shoulder girdle show undue wasting being distinctly smaller than the corresponding muscles of the right side.

Otherwise there is no asymmetry, and no prominence of the praecordia. The apex beat is not visible, nor is there any visible pulsation over the praecordia or episternal notch. In the epigastrium slight heaving pulsation has latterly become noticeable. In the neck slight arterial pulsation and the diastolic collapse (Wenkebach) of the venous pulse are to be seen.

Until quite recently no venous pulsation has been noted in the neck. I believe though, that this was
due to lack of attention to the slighter manifestations of this symptom; but the well-marked movement of the venous wave which is now in evidence is certainly a new development.

On palpation the apex beat may be felt rarely in the 5th interspace 4½ inches to left of midsternum; the impulse is very feeble and limited in extent. There is no other palpable pulsation over the praecordia.

It is only within the last two years that enlargement of cardiac dulness has gradually developed. Latterly right-sided cardiac dullness has become more marked so that now the dulness extends from 4½ inches to the left, 2½ inches to the right of midsternum at the level of the 4th rib; and the upper limit corresponds to the upper border of the 3rd costal cartilage.

Only six months ago there was no dulness to the right of the sternum and the left border was at 4½ inches left of midsternum.

On auscultation the first sound is found to be very feeble and the second accentuated. Occasionally the first sound is represented merely by a very faint muffled sound having a double element. - (the radial pulse is of course missed then, though tracings may show the small wave). Over the left side of the heart the first sound is weaker than over the right side; it is short clear and sharp and most distinct over the tricuspid area. Over the pulmonary area it is slightly prolonged and harsh.
The pulmonary second is slightly louder than the aortic second; the accentuation of the latter being more relative as compared with the very gentle first sound.

At the time I have just referred to the heart-sounds were clear and abrupt in all areas; though the feeble first sound contrasted strongly with the accentuated second sound this discrepancy was more extreme over the left heart. The pulse was only rarely intermittent, and that temporarily after attacks.

Undue accentuation of the aortic second sound has been present for long. Latterly the pulmonary second sound has become the more pronounced.

Examination of the lungs gives evidence of emphysema: there is some bulging above the clavicles with coughing and forced aspiration; but there is no bulging of the interspaces, and the extraordinary muscles of respiration are not in action.

Percussion gives a hyperresonant note; the extent of lung resonance is increased and encroaches on the cardiac and liver dullness.

Expiration is prolonged, and sometimes a few scattered râles may be heard.

There is morning cough with slight tenacious mucoid expeditoration; the cough has the normal expulsive character and the voice is natural.
Alimentary System: The appetite is good.

A feeling of distension of the stomach very readily occurs after eating, usually within half-an-hour after a meal. This is liable to cause a feeling of "Pressure on the heart" and then pain in the left elbow comes on and ushers in the train of symptoms constituting an attack.

He is frequently troubled with eructations and regurgitation of sour-smelling glairy fluid tasting "like vinegar". This is usually at a later stage of digestion, though if milk-food be taken it may occur almost at once. Hiccup and vomiting has been associated with this sourness and profuse salivation supervenes.

This condition too tends to initiate an attack.

Subject to heart-burn especially at night, but not so much since the regulated light meals are being taken. For years already he has been prone to slight attacks of jaundice. During the last five years there has been sluggishness of the bowels, which has to be carefully guarded against as constipation, colic, flatulence, diarrhoea, or other abdominal disturbance are very liable to cause attacks.

The tongue readily becomes furred; the pharynx is slightly granular with distended vessels.

There is no abnormal fulness or retraction of the abdomen. The abdominal wall is thin and the level
of the lower border of the enlarged liver is in evidence. Liver dulness in the mammary line extending from the upper border of the 4th to three fingers' breadths below the costal margin. The liver border feels firm and rounded with no abnormal irregularities of its surface it extends across the middle line midway between umbilicus and ensiform.

During the last nine months the size of the liver has been a very variable factor: acute enlargement supervening on attacks of angina pectoris whereas during remissions the area of liver dulness would again decrease so that the lower border might be scarcely below the costal margin. Eventually however, hepatic enlargement became persistent, and pulsation of the liver can now be felt.

I have no tracing of this liver pulse; therefore cannot positively exclude the movement of systolic retraction conveyed by the enlarged right ventricle; but it felt like a true expansile liver pulse; and if so must have been ventricular in time corresponding with the late venous pulsation in the neck due to the reflux wave of increased pressure produced by ventricular systole in tricuspid regurgitation with the right auricle passively distended.

Splemic dulness is slightly enlarged.
The urine is somewhat scanty and concentrated and commonly deposits urates and uric acid on standing. Phosphates may be thrown down on heating. As yet there had never been any albumen present at any of the frequent examinations made. Unless large doses of alkalies have been taken the reaction is highly acid.

Compared with his former high standard of intellectual ability his mental faculties have greatly weakened during the last five years. Memory for recent events is distinctly impaired whereas he dwells with minuteness on events of his youth and childhood.

Sustained mental effort is a strain and readily induces an attack. Irritability and restlessness are often the precursors of an attack.

He is strangely optimistic as regards his present state of health; this is especially remarkable after an attack, when in spite of great physical weakness this sense of well-being soon reasserts itself, and mental activity may cause him to exert himself with reckless unconcern.

He is liable to phases of mental hyperactivity with great restlessness and wakefulness when there may be transient confusion of ideas. Thick slurring speech and dysphagia, also partial aphasia with transposition of nouns have been temporarily present and may super-vene on an attack.
There is homonymous restriction of the right field of vision; this impairment of sight as well as mental asthenia cause him to dislike reading. Visual acuity is excellent. Acuity of hearing is distinctly variable; defective ordinarily but becomes sharpened during the periods of unrest.

Unless under the stimulus of excitement his movements are slow and give evidence of effort which makes him appear older than his years, and he soon becomes fatigued.

There is considerable wasting of the muscles of the left arm and shoulder with corresponding loss of power, but not the slightest trace of nerve lesion. That this is associated with defective blood supply is shown by the left pulse being smaller, with tendency to coldness of the limb and pallor of the skin surface.

Muscular sense including stereognostic sense are excellent, but equilibration is slightly defective because of defective eyesight.

Tendon responses are normal and equal on the two sides, except diminished in the left arm. Superficial reflexes are brisk. Organic reflexes are unimpaired except transient dysphagia as above noted with concomitant transient dysuria.

The pupils react normally. Bilateral myosis with staring of the eyes owing to retraction of the upper eyelid are well-marked during the periods of restlessness.
Sensory functions are unimpaired. No headaches or vertigo. Sleep is fitful and often disturbed by vivid dreams.
ANGINA PECTORIS. DESCRIPTION OF THE ATTACK

PRODROMATA & AFTER-EFFECTS.

A not infrequent warning of an approaching circulatory climax is a state of restlessness with mental activity and sense of well-being; an accentuation of this condition may give rise to mental aberrations as previously referred to. At such times arterial contracture would invariably be accentuated with hard pulse and heightened blood pressure.\(^2\,3\)

Apparently almost the reverse also predisposes to an attack. I mean when the heart has become temporarily depressed; though this would of course mean lowered systolic pressure.\(^2\,4\) But the attacks which then occurred might be called "asthenic" for the characteristics of angina would be less pronounced and there would be alarming collapse and early indication of cardiac failure. This type of attack of angina pectoris may apparently be induced by various depressing influences e.g. after free purgation or depressing drugs such as Calomel.

The actual attack of angina pectoris may come on with alarming suddenness and with no apparent exciting cause.

Usually however, a distinct disturbing factor can be determined, and the patient is fully conscious of impending distress and anxiously studies its advance, or with delight appreciates that it has been abortive.

16.
As regards the immediate disturbances inducing an attack - rightly named Angina - I cannot do better than quote from Dr. Gibson's book on "Densities of the Heart and Aorta":

"The exciting causes which induce attacks are for the most part influences capable of inducing conditions of stress. These are sometimes purely mechanical such as are often seen in connection with alimentary disturbances. Dilatation of the stomach or distension of the intestines may bring on an attack, while an unfavourable attitude unconsciously assumed during sleep is enough to precipitate an access. Physical exertion, especially after meals, is the most frequent determining agent. Mental efforts also induce attacks but disturbing emotions, whether of joy, grief or anger, are much more powerful. The influence of external agencies, such as cold and damp, must not be overlooked (especially in the production of one distinct variety, this does not apply here). "It seems also probable that certain disturbances of the system may reflexly precipitate an onset. Some toxic substances lastly, have an exciting as well as a predisposing tendency. Tobacco is the chief agent of this group, but tea, coffee, and alcohol also claim their victims.

After tobacco has been deleted from Dr. Gibson's list every item of the above quotation might apply word for word to this case. As regards tea, coffee
and alcoholic stimulants, even in very small quantities they have undoubtedly caused circulatory disturbance and attack.

When he was still able to attend to occupation and go about mental effort, especially long-continued mental effort, or physical exertion such as hill-climbing or stairs - the more so when there was the slightest appreciation of hurry, no matter whether any haste had actually been exerted or not - were the usual exciting causes of an attack.

Owing progressive debility, many of these risks have grown beyond reach. Latterly the attacks have usually been associated with alimentary disturbances such as gastric distension, active intestinal peristalsis, or the dyspeptic states to which he is liable.

Russell has drawn attention to an arterior-cardiac reflex having its origin in the abdomen which apparently may become hypersensitive and give rise to exaggerated response.

Gaskell’s and Head’s researches indicate that these various symptoms like the actual breast-pang itself - "empty focal stimulation of the spinal segments or roots causing reflected sensory impressions. The stimulus inducing referred sensations may be initiated by any of the viscera; but the response is exaggerated, when it emanates from a region of abnormally irritability by persistent efferent impulses from the embarrassed heart."
and which may extend widely beyond its original focus.

This carries one back to Trousseau who recognised that there was a parallel between angina pectoris and epilepsy.

Cold, especially a sudden chilling of the surface has for long been a dreaded enemy and has to be carefully guarded against. Here again Russell's suggestion of a hypersensitive vaso-motor centre comes in aptly.

In the small hours of the night the patient often wakes up with the apprehension of imminent attack. This seems to be due more to cardiac depression than to faulty posture, though such may have helped to increase cardiac embarrassment. Digestive disturbances may be at work then as well as in the day-time.
Description of the attack of angina pectoris may be given in three stages.

1st Stage, ADVANCE

From the first the patient is intensely conscious of heart-strain and sense of effort on the part of the heart with a feeling of praecordial oppression and tightness round the lower thoracic zone generally merging into cramp-like pain "Herzkrampf" which increases in severity. This pain extends over the whole praecordia and is most intense over the lower sternal region at the level of the 5th costo-sternal junction.

At the same time the left elbow begins to ache, and as the attack advances the pain here becomes excruciating - indeed it may be so intense as to overshadow the praecordial distress - and be the first actual pain complained of, though there would always be an initial warning referred to the heart.

In a lesser degree this pain extends down the left forearm and involves the whole of the left hand. Other regions are usually similarly affected especially the left shoulder corresponding to segment C. IV of Head's diagrams and the left scapular region. Often there is a broad band of pain across the lower part of the chest from the left axillary region to the right
mammary line. Pain in the interscapular region seems to be prominent in those attacks induced by a gastric reflex and may involve also the right side.

More rarely there is pain at a lower level in the left infrascapular and lumbar regions and seemed to be associated with intestinal disturbance - flatulence.

Occasionally the right hand, especially the little and ring fingers together with a strip along the ulnar aspect of the right forearm have been implicated, usually in combination with intense pain over the right side of the chest in the inframammary region; this was a later feature in the course of the disease, and might possibly have special bearing on right-sided cardiac embarrassment. Pain over the right scapular region was by no means unusual. Over all the regions affected the sufferer experiences the sensation of intense cold, painfully intense cold, until the parts would seem numb with cold; and indeed the skin surface is felt to be strikingly cold; even through thick flannels the cold patches could be definitely appreciated.

Owing to the condition of intense distress I have never been able to make an actual examination as to sensibility over these areas. My impression has invariably been that at first the painful regions are hypersensitive; for the least touch would be characterised as heavy pressure or rough handling. As the
sizure passes off, in the left hand at any rate, the painful tingling sensation is experienced that accompanies restoration of circulation. Sir B.C. Brodie was impressed by the similarity between these sensations in angina pectoris with those of gangrene.

With the first onset of praecordial distress the finger on the pulse can easily appreciate a hardening of the vessel wall so that the radial artery feels like a tube beneath the finger. This hardening of the artery at first gives place to periods of more or less complete relaxation, which may be only slight and very transient, the hardening becoming more intense and persistent as the attack advances until the arteries remain absolutely rigid.

Simultaneously with these changes in the pulse the patient's face becomes expressive of anxiety. During the periods of hardening of the pulse the features become drawn and tense so that the lines of the face are accentuated in an expression of intense agony; the superficial temporal arteries stand out prominently, at first turgid, but as the attack advances their calibre is seen to shrink and the bends appear exaggerated.

The patient remains silent and rigidly motionless during the periods of spasm, but as the arteries relax this general tension yields in temporary respite. But he is fully aware of imminent recurrence of the spasm and his state of restless apprehension during the
interval of relaxation is in striking contrast with the silence and immobility maintained during the phase of contracture.

The pulse is slow at first and of the protesting character as in the early stage of fever, the impact of the wave being forcible and the wave well-maintained giving a strong deliberate beat. During the periods of relaxation the wave is fuller and more bounding in character and the frequency increases. As the hardening of the artery recurs the wave amplitude becomes less and at the same time the pulse frequency increases.

At first the superficial veins are engorged, but as the periods of constriction become more intense and prolonged the veins no longer remain filled and the surface of the body becomes pale.

Respiration is gasping and quickens in rate pari passu with the pulse.

2nd Stage Fully developed Attack: When the attack is at its height the patient's condition is one of extreme distress. The features are drawn and expressive of anguish, the face is ashen-pale, and the eyes are wide open and staring.

The mind is usually active and consciousness unimpaired, as he keenly watches the course of the attack and (in the less severe ones) is able to ask for any remedies which he hopes may give relief, and
appreciates every detail of his symptoms.

Sometimes he gets into a state of frenzy with anxiety and anguish, or the faculties become blunted and he appears to give up the struggle. Such times he afterwards will have only imperfect recollection of the attack, often ignoring its occurrence absolutely; or again he may seem to faint away entirely.

The posture assumed is not unlike that of an asthmatic; the body is kept rigid usually in a sitting or semi-sitting position supported on the right elbow, the neck is extended and the chin thrust forward. The legs gradually become more and more drawn up. When the attack is at its height the diaphragm is drawn up and fixed so that the epigastrium is indrawn.

The surface of the body is pale and cold, over the painful areas the cold is intense. The left hand seems absolutely exsanguinated and is almost powerless.

The pulse at first forcible and of high tension becomes intermittent and somewhat irregular, then increases in frequency while the amplitude of the wave diminishes as the pulse becomes wiry and uncountable; for the rigid arteries are felt to shrink until the radial feels like a tense string while the diminished calibre of the temporal arteries is plainly visible. The impact of the wave gradually weakens the pulse becoming thready, and the wave if appreciable is now extremely rapid and flickering.
Respiration owing to immobility of the diaphragm is thoracic in type. The experience that relief is associated with full power of thoracic expansion induces the patient voluntarily to exert himself and take long breaths, which effort results in curious facial contortions and blowing noisy expirations.

As the attack persists moist sounds become audiable with expiratory dyspnoea similar to that of asthma, and as oxygenation becomes interfered with there is some cyanosis sometimes amounting to luridity when the breathing will become hyperpnoeic.

Gradually the state of tension yields in exhaustion and collapse. The patient sinks back against the pillows slipping down into the bed, and the legs become extended while the arms lie limp by the sides; the facies is hippocratic and cold sweat covers the skin surface. The patient looks dying and feels dying.

This though seems to be the climax, for the arterial spasm yields just as death seemed imminent. As the arteries relax it feels like a weight lifted off the heart and the patient experiences intense relief though utterly exhausted, perhaps barely conscious. The soft pulse-wave becomes larger and slower, the diaphragm is in action again and full deep breaths are taken.

The patient sinks into the sleep of exhaustion possibly semi-consciousness while Cheyne-Stokes breathing may persist for hours, and is associated with rhythmic
tightening up of the arteries but apparently insufficient or too transient to give rise to painful response.

3rd Stage DECLINE: The attack does not necessarily progress to this extreme:

(1) Frequently it is abortive at the outset. When the onset of spasm has been associated with some reflex which in itself is of transient nature or otherwise, has been overcome, there is improvement as soon as the temporary disturbance has ceased.

(2) Relief may be sudden, often spontaneous. Such sudden cessation seems to depend upon the removal of some disturbing element before the strain has fatigued the heart.

(3) What more often occurs is a gradual fading away of the spasm which may or may not recur after intervals of relaxation. Such improvement may be looked for when the pulse remains full during the periods of relaxation and is not increasing in frequency nor losing in force; remissions then become longer while the periods of spasm grow shorter and more slight.

(4) Exhaustion terminating in a climax of relaxation as described.

Recrudescence of the spasm is more likely to occur when the attack has yielded in response to antispasmodic drugs (Nitrites) and the true cause of disturbance is probably still in action.
Reaction and after-effects:

Should the arterial spasm yield while the heart's action is still good the relaxed arteries quickly become well-filled. The pulse-wave is large, at first of the collapsing type characterising lowered peripheral resistance, it soon becomes more sustained and less empty between the beats. Respiration is full corresponding in frequency to the quickened pulse. This frequency gradually decreases though for some days the pulse-rate may remain increased and usually there is some intermission and irregularity. The arteries are now relaxed and soft, it may be a long time - days, weeks even months, before the arterial hardening manifests itself again.

Together with this increased activity of the circulation after an attack of angina pectoris, there is a state of general stimulation, and excitement, as though all the organs had been flushed out (literally) with blood and were working now with double vigour.

The eyes are bright, the patient talks, laughs and will not rest. The mind is intensely active, trains of thought chase each other with annoying persistence. The skin is flushed and warm, tingling pain takes the place of cold and numbness. After pain may be very persistent over the praecordia and down the left arm as also in other regions.
Perspiration is profuse, there may be diarrhoea and desire to evacuate the bowels, there may be salivation.

A prominent feature is profuse serous or serosanguinous expectoration amounting even to slight haemoptysis, while course rales are heard all over the chest, and there may be feeble breath sounds and impairment of resonance over the bases for some days. This exudation seems to be an invariable sequel to prolonged spasm of the systemic arteries. The quantity and the amount of blood present seem to bear a direct ratio to the duration of the arterial contracture; but in the asthenic type of attack where heart failure (probably inhibition) sets in early this expectoration is less or absent. Dull patches suggestive of infarction have actually been determined. As already noted expectoration of blood for some days after is frequently seen.

The liver region is usually very painful after an attack and the organ becomes appreciably enlarged. There may be slight jaundice and often slight diarrhoea. The urine is increased in density and loaded with urates.

Every gradation between a condition of circulatory activity and stimulation and a state of extreme cardiac exhaustion with depression may be observed after an attack. According as this is the case there may be subsequent exhilaration, often the attack is followed by
mental and physical fatigue causing the patient to sink into a sound sleep, or again exhaustion may be profound with apathy verging on coma. The pulse and respirations are in accordance — thus, after an attack of moderate severity the pulse tends to be more or less accelerated often intermittent to a greater or less degree, down to the extremely soft tardy pulse of profound depression, the infrequent slow waves alternating with series of small beats following each other in rapid succession, while the breathing may show Cheyne-Stokes variation with unconsciousness and absence of pupil reaction and contracted pupils during the apnecic phase.
The morbid processes at work here seem to be Cardio-vascular and Nervous.

One has to consider a diffuse cardio-vascular degeneration with associated tendency to arterial hypertonus, and with sudden intense symptoms referable to heart-strain.

The hypertonus seems to be under control of the nervous system, which evidently is very responsive, and of a high degree of reflex susceptibility.

As the existing cardiac dilatation has developed subsequently to the angina pectoris it is more likely to stand in the relation of effect than to be in any way a cause. Even now there is no evidence of valvular heart disease.

On account of the prevailing hypertonus there is a tendency for the blood-pressure, in the systemic circuit at any rate, to be maintained at high level. A further rise in peripheral resistance or a decrease in cardiac force leads to the verge of a cardio-vascular crisis, which may finally be determined by a great variety of conditions. On investigation these however, are all found apparently either to induce widespread vaso-constriction or else to exert a depressing effect on the heart. Either way, this would result in diminishing the ratio of intraventricular pressure to residual aortic pressure and angina.
pectoris is evidently associated with disturbed cardio-vascular equilibrium. 

For when one reviews what has led up to such attacks a previous state of heightened blood-pressure or of cardiac weakening (I wish to avoid the word "depression" as that suggests cardio-inhibitory influence, I am referring rather to exhaustion of the heart as regards response towards sympathetic stimuli) can usually be recognised, which would entail encroachment on cardiac reserve force; some such reflex as enumerated by Dr. Gibson, might now cause its limits to be overstepped, the outcome of this is sudden acute cardiac embarrassment of the nature of intense effort.

Clinical examination of the superficial arteries would indicate them to conform with the hypermyotrophic type of arterio-sclerosis. This is a generalised affection, and coexists with changes in the visceral arterioles such as Jores has described. Russell further points out that this diffuse arterio-sclerosis is quite distinct from the localised internal change of atheroma, of the existence of which we have no clinical evidence here. There is good reason to believe that capillary sclerosis as Sir William Broadbent points out may have been present for years and have been the initial degenerative change.
Following Allbutt’s clinical classification this would be a case of hyperpictic arterio-sclerosis to which with advancing years involutionary changes have been added.

It is interesting to find that a diffuse arterio-sclerosis affecting primarily the muscle cells of the media has been experimentally produced by injection of high-pressure drugs such as adrenalin chloride and also by certain infective agents as diphtheria toxin, only a thinning of the media resulted instead of hyperplasia. It has been suggested that this may be the acute form of the same type of arterial degeneration.

In arterio-sclerosis owing to diminished arterial elasticity the wave of systolic pressure is badly propagated along the arterial system, probably other factors such as roughness of vascular channels etc, contribute to premature dissipation of initial force of cardiac output (i.e. systolic blood pressure is badly maintained); at any rate the left ventricle has to produce a high systolic pressure to counteract this premature dissipation of force in order to meet the exigencies of the systemic circuit. This gives what Mackenzie describes as the characteristics of the pulse of arterial degeneration which when the arteries are moderately relaxed is the same as the pulse of advanced life: "A pulse wave
forcible during ventricular systole with the artery compressible after the wave is passed. In sphygmograms the tracing has a high upstroke, the lever maintained high during the ventricular systole and a rapid and deep fall during diastole. Such are the characteristics of the pulse in this case during the periods of freedom from attack.

Even so with this large systolic wave general nutrition is only poorly maintained as evidenced by the general appearance of the patient. It is only reasonable to assume that the heart itself is not exempt from this malnutrition; indeed examination of the praecordia gives abundant evidence that such is the case. The occasional dropping out of the pulse-beat indicates faulty myocardial activity. From the accompanying tracings one sees that the pause corresponds with the occurrence of an imperfect systole of ventricular origin, owing to depression of contractility. With the stethoscope one can hear the feeble sound corresponding to this contraction. It indicates that the heart has begun to give way under the strain of long continued "working at high pressure", hence the field of cardiac response is bound to be restricted and any additional strain may make excessive demands on the enfeebled heart.
Interference with the blood supply to the left arm is strongly suggested, and might be due to partial blocking of the main artery by atheroma.

Arterial hypertonus is another cause of increased work for the left ventricle, for it raises the diastolic arterial pressure and probably is the main factor in the production of strained contractility; it may cause rapid and great variations in aortic pressure.

Apparently in those suffering from arterial degeneration with associated thickening of the vessel walls there tends to be a state of general vaso-constriction due to some toxin in the blood stimulating the muscular fibres of the arteries and arterioles to increased activity; this probably through the medium of the vaso-motor centre. "It is in the capillaries that the resistance is initiated and the arterioles and in case of need the entire vaso-motor apparatus are called upon to co-operate". (Sir William Broadbent)
The conditions controlling the circulation in the capillary zone remind one of what obtains in a high level gathering ground of water. When one bears in mind the vast lymphatic circuit besides the infinitude of spaces bathed by liquor sanguinis and the continual escape of fluid from the various body surfaces, so long as outflow by the veins is free the circulation in this outlying zone is virtually unconfined.

Clinical experience has shown that the presence of deleterious substances within this high level gathering ground is conducive to narrowing of the afferent channels, which should be accompanied by a proportionate increase of strength in the action of the heart and a rise in blood pressures. Provided the pressure in the efferent drains of the venous system has not risen, this practically raises the capillary ground to a higher pressure level and consequently elimination as well as drainage from the capillary field would be augmented.

With increased tonus of the left ventricle there must be a complementary rise of pressure in the left auricle and lesser circulation, and the right ventricle therefore likewise has more work to do.

But morbid changes in vessels and heart tend to develop. With rigidity &c of arterial channels there is premature dissipation of systolic force which as the toxin reaccumulates is met by still further tightening up of the arterioles.
When the force-pump fails, narrowing of arterioles instead of furthering the flow, would act as obstruction more especially there would be a tendency towards stagnation in the zone of ultimate arterioles and greater transudation of fluid into the tissues there which would become oedematous. Cardiac effort may temporarily readjust the flow. As the heart fails, mean arterial pressure would sink, and this again sinks the capillary field to a lower pressure level and impedes efferent flow restricting the capillary ground to more confined limits, increases resistance.

With weakening of the right heart and yielding of the ostial fibres, waves of pressure are propagated against the venous stream and hamper flow to the heart. Eventually positive pressure is established in the great veins and further interferes with return flow from the capillary zone. With development of the ventricular venous pulse all the potential reservoirs of the hyperphatic system become filled and then the capillary ground is water lagged.
Arterial hypertonus considered side by side with the premature falling away of blood-pressure, as seen in arterio-sclerosis per se, strikes one as complementary; at first beneficial but eventually tending towards the vicious circle so often met with in the study of disease. One must remember that hypertonus is functional and a variable factor, whereas the lesions of arterio-sclerosis cause organic and permanent circulatory modifications.

A highly responsive nervous system probably has much to do with the possibilities of such states of vaso-constriction.

A consideration of the immediate exciting causes of an attack of angina pectoris as observed in this case suggests the following.

Increased work is being done by some organ—say, skeletal muscle, brain, abdominal viscus. When an organ is functioning local vaso-constriction in other circulatory areas determines blood-supply to the
active region. With degenerated arteries one may assume that such variations are not easily regulated and controlled. So long as local wants are inadequately satisfied one may assume afferent impulses to the vaso-motor centre causing summation of stimuli, and leading to more and more widespread and intense vaso-constriction, thus making increased demands on the heart leading to strain and attack. Perhaps here-in lies the raison d'être of the hypertonus and its concurrence with cardio-vascular degeneration.

That arterial hypertonus might cause heart strain, quasi angina pectoris (Gibson), is obvious, for provided systolic output remains adequate this arterial contracture through increasing peripheral resistance would produce the true high tension pulse, where diastolic pressure is high as well as systolic pressure, and intraventricular pressure has to be raised proportionately to overcome the heightened residual pressure in the aorta at the presphygmic phase of the cardiac cycle. Probably this effort on the part of the heart - left ventricle - gives rise to the feeling of strain.

At the outset of an attack the pulse wave is large and not easily obliterated indicating strenuous cardiac action against a high resistance, but missed pulse beats praecordial oppression and discomfort show that it is not without difficulty that the heart is maintaining the circulation at such high tension.
For a time therefore, the circulation is worked at heightened pressure insuring increased supply to any area of vaso-dilatation. Assuming this hypothesis to be correct when the demand has been supplied the impulse to the vaso-motor centre would cease and with arterial relaxation peripheral resistance falls and the heart naturally finds relief.

Should the heart through weakness or exhaustion fail to meet this demand for increased work with failing force of cardiac output systolic blood-pressure in the arteries would fall and approach the level of residual pressure in the arteries (diastolic pressure), the amplitude of the pulse-wave would become less; peripheral resistance being still high the downstroke is tardy giving the wiry pulse with all but imperceptible wave.

All this time subjective symptoms of distress are on the ascendent, and the surface becomes blanched and cold owing to this blocking up of the systemic circuit.

The splanchnic area on the other hand very probably the lesser circulation are engorged (cf. the action of adrenalin).

As the impact becomes more and more feeble the pulse frequency accelerates. Finally when the auricle becomes over distended and fails to initiate the new cycle the ventricle takes on the rhythm, and
premature systoles follow each other in rapid succession and there is tachycardia. 

This phenomenon probably depends upon abnormal stimulation of the bundle of His and is seen in cases of cardiac exhaustion by excessive arterial resistance. It suggests response of the ventricle to direct stimulation, when through exhaustion of contractility following on augmented impulses, the normal stimulus from the auricle fails to reach the a. - v. fibres.

With cardiac exhaustion apparently the vascular spasm must yield, blood-pressure fails and the strain is removed.

This seems analogous to the sudden yielding of the spasm of asthma or of laryngismus.

Is this due to some vaso-inhibitory impulse called forth by the heart-strain? For the heart being essentially a modified blood vessel, when excessive resistance threatens to overcome its function of contractility and the depressor nerve mechanism comes into play, one might surmise a co-related order for relaxation to reach the rest of the vascular circuit. Possibly the overfilling of the right heart and pulmonary circulation and splanchnic engorgement may give rise to afferent stimuli producing vaso-inhibitory effect on the systemic circuit.
as excessive intracardiac tension apparently had induced vagus stimulation. Or is the arterial relaxation due to exhaustion of the vaso-motor centre for it is conceivable that terminal arterial supply such as that of the brain would be greatly hampered by persistent vaso-constriction, especially if combined with failing systolic output.

Furthermore the height of circulatory level depends upon:

(1) Force &c of inflow
(2) Rate of outflow
(3) Calibre of channels &c.

With cardiac exhaustion force of inflow is failing—one of the factors of high blood-pressure has been set aside.

Then again rate of outflow has been reduced.

One might assume that when heart strain has reached a certain pitch incompetence would supervene and give relief, through failure of tonicity.

(4) And this is exactly what did happen in the later stages of this particular case, when the attacks took on a much milder form; but that was not until there were definite signs of right-sided heart failure. Such were beginning to show themselves when the accompanying case-notes were made.

Thus the heart's safeguard lies in its weakness—a seeming paradox—but backward pressure whether
through functional or organic valvular insufficiency would prevent excessive intracardiac tension. The high degree of intensity, I think I may say fatality of early attacks, when presumably the heart ought to be stronger, might be thus accounted for: the strong heart responding to greater strain involving intense degree of stress.

Decrudesence of the attack therefore, might depend upon:

I. Relaxation of the systemic circulation.
   due to (1) Cessation of stimuli to the vaso-motor centre. Such would occur of the cause of the disturbance has been overcome.
   (2) Exhaustion of the vaso-motor centre. Such apparently may occur and seem to depend upon alterations in the blood-supply to the medullary centre (Mummery)
   (3) Vaso-inhibitory impulses or inhibition of the vasmotor centre.
   (4) Direct relaxation of the muscular spasm as by Che Nitritisis

II. Dilatation of the heart, i.e. Depression of myocardial tonicity.
   (1) Temporary - Relaxation due to vagus stimulation?

One may assume that functional tricuspid dilatation aptly called the "safety-valve action" would even right through the pulmonary circulation help to relieve
the left heart, and might have much to do with the subsidence of painful manifestations expressive of undue intracardiac tension.

(2) Permanent. Apparently free regurgitation is incompatible with the full development of angina pectoris.

Relaxation of the arteries may be transient — perhaps the original disturbing factor is still at work. Recurrence of the spasm is especially liable to occur when relaxation has been obtained by the use of vaso-dilator drugs e. g. Nitrites, perhaps all the more so because of their depressing effect on the heart.

Alternate phases of relaxation and spasm frequently persisted for several hours after an attack, as though the vaso-motor centre were responding to persistent efferent stimuli whenever inhibitory influence of the vagus subsided (Brunton) or whenever sufficiently recovered from exhaustion (Mummery).

It is curious that such intermittent arterial spasm should tend to be accompanied by Cheyne-Stokes breathing, which, I believe, is acknowledged to depend upon intermittent activity of the respiratory centre. This suggests the circulatory spasm of hyper-tonus, likewise to be under central control. Apparently similar factors are conducive to both these phenomena,
and both may be due to variations in activity of the medullary centres.

One found that the phases of shallow breathing and apnoea corresponded with systemic relaxation, whereas hyperpnoea accompanied arterial constriction and more frequent pulse with more or less pronounced recurrence of distress.

Luciani regards Cheyne-Stokes breathing as automatic oscillations in the excitability of the respiratory centre itself (intrinsic); this might be dependent upon loss of influence of the higher regulating centres or diminished vitality of the respiratory centre itself.

Probably automatism here expresses exhaustion of the respiratory and vaso-motor centres, their higher nerve elements being the last to recover. Such would seem to result after prolonged attacks. During sleep these variations would be more pronounced.

In a case of tubercular meningitis in which lumbar puncture was done, one once had the opportunity of recording variations in pressure of the cerebrospinal fluid, while Cheyne-Stokes breathing was going on (Dr. Eve's apparatus was used). The pressure was found to be on the decrease during crescendo and high during the diminuendo phase of respiration. This agrees with clinical features of progressive cerebral compression. It might be taken as an argument in favour
of the view that the cerebral vessels are well-filled during the period of peripheral vaso-motor spasm. One must bear in mind though that pressure in veins and lymph spaces may be on the ascendent while arterial pressure is on the decrease.

As regards the various painful symptoms connected with an attack of angina pectoris, referred pain expressive of heart strain will doubtless largely account for these. For Head's areas representing spinal segments which through the rami communicantes and vagus are connected with the innervation of the heart are those involved.

Apparently other areas of visceral representation may likewise be implicated expressive of embarrassment of other viscera, possibly more especially of that organ from which the reflex promoting vaso-motor spasm had originated.

Such focal irritation of the spinal cord by summation of stimuli from the embarrassed heart would further explain the after pain and persistent hyperaesthesia which especially prevailed over the region of the apex beat and left arm.

Peripheral ischaemia owing to the spasm might account for some of these symptoms, for are they not identical with those of intermittent claudication? and much relief may be obtained by local warmth, gentle massage &c. But why go out of one's way for further explanation when Head's and Ross's researches amplified 45.
by Mackenzie so fully interpret all this.

The effects of an attack on the lungs and abdominal viseira are referable to vascular engorgement, which gives rise also to the after-pain over representative areas corresponding with these viseira, especially the liver and lungs.

Experimental investigation has demonstrated the absence of vaso-motor nerves in the pulmonary vessels; and the consequences of a prolonged attack are quite in keeping with this. Granted the pulmonary circulation to remain in statu quo, general vaso-constriction of the systemic circulation with increased resistance to outflow from the left ventricle would determine blood to the lesser circulation causing pulmonary hyperaemia. This would account for the profuse serous transudation even extrusion of red blood corpuscles into the air cells which leads to acute pulmonary oedema, haemoptysis and infarction.

With early cardiac yielding such oedema was found to be less pronounced; naturally with right-sided cardiac insufficiency permitting of backward pressure there would be less influx into the pulmonary artery; but it was just after those attacks that acute hepatic enlargement was most marked and the regions referable to the liver would remain particularly painful for many days.
Amyl Nitrite on the other hand was considered by the patient to aggravate the haemorrhage. In Andrew's Harveian Oration one finds that this drug causes a rise in pulmonary blood pressure.

Unfortunately for the above line of argument by the same authority "the mammalian pulmonary vessels do receive vaso-motor nerves"; but it seems that there was some fallacy in the method of investigation adopted by Bradford. \(^{15}\) (Brodie & Dixon, op. cit.)

Similarly the splanchnic area becomes engorged. Here Brodie's and Dixon's results do not give such a ready answer to the clinical problems of this case. This would be more of the nature of a venous congestion; certainly the liver seems to bear the brunt of it.

Salivation almost invariably occurred after an attack had persisted for some time. \(^{16}\) Perhaps this was merely a chance feature of this particular case, and attributable to gastric hyperacidity (which was a constant source of trouble here) such as the salivation of paroxysmal pyrosis described by Sir William Roberts. \(^{16}\) But Gaskell's investigations point to this symptom again having its origin in afferent stimuli from the heart exciting sympathetic secretory fibres. \(^{17}\)

Mackenzie further refers hyperventilation and diuresis to such stimuli reaching the floor of the 4th ventricle. \(^{18}\) It seems that overfilling of the pulmonary circuit during
persistent systemic spasm in prolonged attacks induces hyperaemia which in time is followed by exhaustion of
the respiratory centre manifesting itself in Cheyne-
Stokes breathing. Thus Cheyne-Stokes breathing and inter-
mittent vaso-motor activity may both be referable to
automatism of the medullary centres while defective blood
supply or fatigue is blocking the higher control.

Mental stimulation after attacks was evidently an
outcome of cerebral hyperaemia, and supervening on freer
circulation, which one would like to call reactionary,
and consider the proper sequel to this cardio-vascular
upheaval. The cerebral manifestations after more pro-
longed attacks are doubtless referable to the extreme drop
in blood pressure interfering with the cerebral circu-
lution, the higher functions of the brain failing first.

One found that attacks of angina pectoris tended to
occur in bouts culminating in a reactionary phase with
relaxation of arterial hypertonus; during which there
would be comparative immunity from attack for a more or
less prolonged period; this resulted in something like
periodicity in their occurrence.

This reaction struck one so forcibly that one began
to wonder whether this whole system - complex might not
be the expression of an eliminative process, the climax
of an ascendent degree of hypertonus which had gradually
become essential to the proper working of metabolism.

Why not? diuresis, diaphoresis, diarrhoea all
tend to supervene: a general activity of eliminative
channels. Surely this ought to suffice to expel some
of the hypothetical toxin supposed to be productive of the hypertonus, until it reaccumulates the incitie to arterial contracture would be removed. There is another point about which the patient was most emphatic: he had strong subjective sensation of pulmonary engorgement, from which he argued that the blood became oxygenated to its fullest degree and threw off an extra amount of effete products. To this surcharging with oxygen he attributed the state of exhilaration which might supervene on an attack.

For all I know these surmises may be very far-fetched; but I have again and again found that when a marked reaction supervened with eliminative activity, there would surely follow a quiescent period during which arterial contracture kept in abeyance, and greater liberties might be taken without ill-effect.

Temporary valvular insufficiency of which in the later course of this case there would be evidence after a paroxysm, would likewise account for such periods of intermission. Still these remissions were defined at a much earlier stage of the illness.
"A matter of great interest is the undoubted tendency of paroxysms to become less frequent and less severe when dilatation of the heart supervenes in cases which have not previously been characterised by this condition". Gibson.

The further course of this case is in agreement with the above remark. For with development of tricuspid regurgitation the attacks diminished in intensity and finally ceased altogether.

The final history of the case therefore has no direct bearing on the subject of this paper. Suffice it to say, that there was the clinical picture of gradual heart failure. Progressive increase of cardiac dullness to the right, muffled tricuspid 1st sound, well-marked venous pulse, anasarca and albuminuria marked the downward course.

It seemed as though with back-ward pressure relieving intracardiac tension the essential factor in the production of angina pectoris were abolished.

Vaso-motor spasm would still be determined though no longer associated with the painful symptoms. Angina sine dolore seems to correspond with these later manifestations of circulatory disturbance, when myocardial tonicity has become depressed.

This suggests that in such cases there would probably always be some degree of valvular insufficiency perhaps merely a certain amount of functional dilatation permitting temporary regurgitation when the left ventricle
encountered excessive visa fronte.

Perhaps this accounts for the comparative rarity of fully-developed angina pectoris in women, where systemic degeneration does not often assume such proportions, and in whom functional yielding of the heart seems to occur more readily; cf. the venous pulse of pregnancy\(^3\) and of chlorosis\(^5\). The nervous susceptibilities too are different in women; the female organisation seems to yield more readily to stress than strain against it.

Closely allied to angina pectoris seem to be those cardio pulmonary neuroses where there is asthma accompanied by praecordial pain and disturbed pulse. Doubtless here again focal irritation of the spinal nervous system is set up by visceral strain. Some of the symptoms might be referable to high peripheral resistance in the pulmonary circuit - possibly in some measure connected with emphysema; (the emphysema of such cases may to some extent be secondary to vascular morbidity), where the right ventricle seems to be experiencing some similar strain, which finds expression in thoracic pain that is such a prominent feature in this type of asthma.

Towards the final termination there were profound disturbances evidently due to gross interference with circulation.
Alternating phases of arterial contraction and relaxation prevailed, while Cheyne-Stokes breathing was uninterrupted for fully two weeks before the end.

This state obviously was the outcome of failing systolic pressure with persistent tendency to high peripheral resistance; therefore a falling off in mean arterial pressure with a rise in the venous, causing embarrassment primarily in the capillary zone of the circuit: edematous infiltration of tissues and retention of effete products sum up the more prominent evils that this entails.

...
TREATMENT:

As has been shown treatment was absolutely unavailing in preventing the downward course.

At the outset avoidance of strain and careful regulation of diet &c. kept symptoms more or less in abeyance.

After recurring influenza with probably further myocardial and arterial lesions, we find the symptoms assuming alarming proportions.

Treatment in Wiesbaden was mainly directed towards strengthening the heart: liberal proteid diet and carefully regulated doses of Digitalis caused amelioration in remarkably short space of time and for several months there was absolute freedom from attacks.

Another respite was procured through enforced rest from all work, owing to such intercurrent, when avoidance of physical exertion and mental strain gave a prolonged period of immunity.

Rigid milk diet worked wonders at one time when gastro-intestinal reflexes were the most prominent initiative to spasm and attack.

Following up this line of treatment on the assumption that toxins were conducive to hypertonus an endeavour was made to continue with relatively purin-free diet; but the patient could not tolerate this, restriction of proteids and mixed diet of easily
digested food-stuffs given in small quantities at fairly frequent intervals gave the best results. As regards drugs; potassium iodide was given in the early stage of treatment. Later Fraser's Tincture of Strophanthus was taken steadily for many months together with sweet spirits of nitre, and apparently supplied a necessary aid to the heart.

To treat the attack: one's first endeavour was if possible to remove the cause which was initiating the spasm and so obviate the disturbing reflex.

Failing this cardiac stimulants especially the diffusable stimulants which act rapidly, were found most advantageous, usually these had to be associated with vaso-dilators and antispasmodics—thus the nitrites together with small doses of brandy and the ethereated tincture of valerian (G. Ph.) were the usual standby to control the attack. Morphia might have to be given. Treating symptoms by warmth to the surface carminatives & c., would help to ameliorate distress.

Guard against recurrence by avoiding all extraneous causes of disturbance as much as possible, and by aiding the eliminative processes and by keeping a careful watch on the pulse in order to endeavour to
maintain a satisfactory state of circulation. Here Calomel especially seemed to have warded off many a crisis for it apparently lowered arterial resistance by actually getting rid of some disturbing toxin.

Continued use of the stronger nitrites seemed to be harmful; the benefits of vaso-dilatation being more than counter-balanced by their deleterious effect on the heart and digestion. Cardiac tonics did their share towards maintaining circulatory equilibrium.

With the exception of a few doses of thyroid tabloids, which could not be called a fair test, organic extracts have not been given a trial; it may have been mere coincidence, that those few doses after the apparent immediate bad effects had worn off were followed by a remarkably prolonged interval of freedom from angina with general improvement.
In conclusion, angina pectoris here seems to have been the expression of undue amount of cardiac effort, incurred by disproportion between the arterial resistance and the strength of cardiac contractions. This tends towards defective circulation of the blood, and primarily causes embarrassment in the arteriole side of the capillary field.

The symptoms are referable to heart-strain inducing reflex nerve stimulation.

Relief follows on lessening of the resistance through arterial relaxation, or through cardiac regurgitation.

By cardiac inhibition and depression of tonicity, functional incompetence is caused, and determination of blood to the visceral area and lesser circulation, followed by readjustment of circulatory balance at a lower pressure level.

With failure of tonicity and cardiac dilatation, backward pressure is established, and removes the cause of the strain at the cost of circulatory failure.

In this case the strain is an incidental by-effect of arterial hypertonus, which is itself to be regarded as a corrective process, essential to the proper discharge of metabolism, but associated with, perhaps also conducive to cardiovascular degeneration and impairment of myocardial contractility.

As in similar cases investigated by Mackenzie & others there will have been extensive myocardial fibrosis with sclerosid coronary vessels together with diffuse generalised arterio-sclerosis.
These figures approximately show the distribution of the painful sensations and hyperalgesia - the degree of shading is intended to indicate the intensity.

The right arm was very rarely affected; the other areas were usually all involved in the more severe attacks and the whole left side of the thorax and left arm became hyperalgesic, but the degree of pain was variable and to some extent differed in situation as regards intensity in the different attacks.

The order of involvement was something like this:

(1) Vague discomfort referable to the praecordia such as oppression, feeling of weight; there might be such sensations as "Fluttering of the heart".

(2) Intense pain in left elbow.

(3) Intense pain over apical region of heart and over sternum at level of 5th costal cartilage which extended
into
(4) Sense of constriction round lower part of chest with the feeling of tightening up of the diaphragm and spasm of the gastric cardia, which is followed by sense of gastric and abdominal distension.
(5) Pain over scapular regions; left first. Meanwhile the pain from the elbow has extended down to the fingers and thumb and from the apical region to the base of the heart.
(6) Pain over top of left shoulder (acromion process) which extends up the neck, and downwards over the deltoid region; later this pain may extend to the nape of the neck.
(7) Cold painful patches over various parts of the back and axillary regions; the patient calls them "collections of wind" and gentle rubbing over such places seems to facilitate eructations.

More rarely: (1) A broad band of pain extends transversely across the front of the chest and round the left side.
(2) Pain in right inframammary region.
Exhaustive representation of spinal roots.

Taken from Head's diagram "Brain" Vol. XIX p. 393; Fig. 32.
A from Gibson's "Nervous affections of the Heart." p. 28 & 29.
Figs. 13 & Fig. 14.

The same as on p. 57:
For comparison with normal spinal sensory distribution.
These tracings are good examples of the sphygmogram ordinarily obtained in this case.

They indicate a small artery with diminished resiliency (contracted vessel) and moderately high peripheral resistance in proportion to the systolic pressure as shown by the flat top, slanting upstroke, and gradual convex downstroke with the aortic notch high above the base line and ill-marked dicrotic wave.

The tracing obtained from the left radial is smaller than that from the right. Tracings 1 & 2 most fairly represent this difference, but tracing 3 gives the clearest definition of the characters of the sphygmogram.

At the time the tracings were being taken the pulse missed 8 beats in 3½ minutes measurements show that the pause in the tracings accurately represents the period of a dropped beat; it is followed by a large systolic wave which is strongly suggestive of such pauses corresponding with the occurrence of an imperfect premature systole.
These tracings were taken during a bad period with daily occurrence of attacks. The pulse-wave is smaller than in the preceding tracings, because of decrease in difference between systolic and diastolic pressures. The pulse-pressure is very sustained and was rising while the tracings were being taken and one could feel the pulse grow small and hard and incompressible.

They indicate greater fulness of the artery or obstruction to outflow with progressive impairment of cardiac force and might be taken as illustrating arterial hypertonus.
This was taken one hour after a moderately severe attack when still under the influence of Nitroglycerine. It shows the flat top and badly sustained wave of forcible inflow into an empty irresponsible artery. These jerky pulsations were felt by the patient "like the lash of a whip"
This series of tracings was taken during a period of discomfort with feeling of cardiac oppression, after a meal, and for which soda-mint tabloids had been taken.

The symptoms were passing off as these tracings were being taken.

They show that the pulse is diminishing in frequency and gaining in systolic force, whereas diastolic pressure is at any rate relatively not increasing; the tracing is suggestive rather of decrease.
These tracings were taken during a slight attack of angina pectoris. Nitroglycerine gr. 1/50 had already been given. When the sphygmogram was adjusted to take tracing I, he was feeling well and the pulse felt large and soft. By the time tracing II was taken there was slight recurrence of distress and the pulse was hard and incompressible.

This difference is well brought out in the tracings; the gradual upstroke and blunt systolic wave show that ventricular systole does not readily overcome the resistance. Heart-strain is further demonstrated by the more frequent occurrence of premature systoles.
The 1st & 2nd tracings were taken during a prolonged attack. The recorded beats are all premature systoles and follow each other so rapidly that the diastolic period of tracing is cut short by the subsequent upstroke; the frequency was steadily increasing. This is evidently a paroxysm of tachycardia.

In my notes of that date I find; "though the pulse-wave had a sudden rise and fall, the artery felt hard and cord-like and the temporal arteries were prominent. Pain in the left elbow and praecordial distress were on the increase at the time of taking the tracings". Relief was only finally obtained after morphia.

The 3rd tracing was taken on awakening, when the symptoms had quite subsided. Excepting the occurrence of a dropped premature systole, the pulse is regular; and as usual after an attack, shows a relatively low arterial pressure.
This tracing again shows the ample wave which might be maintained for many days after an attack. Besides the premature systole and long pause followed by a stronger beat, the other beats show further irregularity by unevenness in length of diastole.

22 July 03. Attack of rhythmical degeneration associated with alternate phases of hardening and softening of the pulse, now took the place of the typical angina pectoris which never again occurred. Compare this tracing with the first two tracings on the next page taken immediately after. Note that there was distinct evidence of backward pressure and circulatory insufficiency.
My notes of July 7th state "no more attacks of angina (since June 15th), but is none troubled with these symptoms of cardiac insufficiency associated with backward pressure. There is a well-marked venous pulse in the neck, but owing to failure in obtaining relaxation of the sterno-mastoid the tracings have all been unsatisfactory."

The tops of the waves have failed to appear in the tracings which makes their interpretation very doubtful. The elevation evidently corresponds with a ventricular regurgitant wave, and the depression occurs during ventricular diastole. There is considerable interference by the carotid pulse.

In the 3rd tracing taken two months later three waves appear in the upper line; but I cannot interpret them at all.

Clinically there was by that time anasarca and a regurgitant wave in the veins of the neck.
The following tracings were taken during an attack of rhythmical dyspnoea (Cheyne-Stokes) of angina sine dolore. During the crescendo stage the superficial arteries become prominent and the radial could be felt to harden, the pulse becoming smaller with more forcible impact. During the periods of quiet breathing the arteries soften and the amplitude of the pulse-wave increases. The dyspnoeic phase is associated with distress and the sense of oppression. During the quiet phase everything seems restful, there is a tendency to fall asleep or break off in the middle of a sentence. At that time I ascribed these pulse variations to arterial contraction and relaxation; it has since been pointed out to me that alternate turgidity and emptiness of the arteries would give this impression.

See next page for tracings.
There is further evidence of myocardial failing here:

In the 1st tracing taken during the phase of augmented breathing three premature systoles follow each other, of these the 2nd has failed to pass the aortic valve; its occurrence is indicated by alterations in pressure transmitted to the arterial column. Because of the great fall in arterial pressure the 3rd wave can easily pass the valve and appears fairly large in the radial tracing. This contraction is followed by a long diastole during which the arterial pressure falls lower still. A full ventricular contraction now occurs but it takes some time before the aortic notch has regained its former level in the tracing. The small size of the second beat after the long pause is probably of the nature of Pulsus Alternans.

The variation in level of the base line of the subsequent full beats is due to movements of the arm with laboured breathing.

In the other tracing taken during the quiet phase of respiration the pulse is seen to be of lower tension; two imperfect systoles succeed each other, and here too the large beat is followed by a smaller beat.
The following tracings were taken after the breathing had permanently assumed the Cheyne-Stokes type. In the notes I have described the pulse as readily obliterated by fairly light pressure.

The tracings show great relative feebleness of systole; the variations in character of the pulse which conveyed the impression of phases of tightening up and relaxation of the artery are well marked, nor does the 1st tracing give any indication of fulness of the vessel, whilst there is the flat top of the systolic wave associated with a hard artery wall.

No note can be taken of the smaller irregularities in the upper tracing owing to the fallacy of respiratory movements of the arm during hyperpnoea.
(Additional note referring to the tracings on p.70)

In the other tracing taken during the phase of quiet breathing the Pulse Alternans can be recognised; and here the systolic wave is sharp indicating lessened resistance and a soft pulse, such as would occur with anastomotic relaxation.

In describing these pulse-tracings I have endeavoured to follow Dr. Mackenzie's method of terminology as used in his book on "the Study of the Pulse".
REFERENCES


2. Latham "Diseases of the Heart"

3. Gibson "Nervous Affections of the Heart"
   Morison Lectures 1902 & 3, Lecture 1
   Edin. & Lond. 1904.

4. Barr. "An Address on Arteria Sclerotic"
   "The arterial tension or pressure is the result of the force of the ventricular systole and the resistance at the periphery"


6. Gibson "Probably a combination of physical and mental exertion will bring about the highest increase of pulse pressure. "Brit. Med.
   Journ. 1906 Oct. 20th, p. 1001."
7. Weber. "American Journ. of Med. Sciences" 1894, p. 291, Vol. 108 as conducive to Arterio-Sclerosis. "Excess" due to strained manner of living - it may be in physical labour, it may be excess and irregularity in mental work including anxiety and worry; it may be the habitual taking of too much or too little food &c."


14. Mackenzie "The Study of the Pulse" Edin. and Lond. 1902 p. 57 par. 48 Diminution of the capillary field and of the quantity of blood in the old".
"The cardiac field of response".

"Recurrent high arterial pressure as a cause of angina pectoris".
In his later writings Mackenzie lays stress on the pressure being relatively high as regards the contractible power of the heart muscle.

17. Wenkebach. "Diastolic collapse of the veins of the neck may be seen in most cases of marked stasis in the veins with the heart acting not too quickly".

"Liver movements due to cardiac aspiration".

see also par. 211 p. 251.
"the presence of a venous pulse of the ventricular form or of a liver pulse of either form indicates an advanced stage of cardiac failure".

"cerebral disturbances have a tendency to make their appearance".

22. Gibson. "Diseases of the Heart & Aorta"
   Edin. & Lond. 1898 pp. 764 & 765.


   NOTE: When Thyroid Extract had once been
   tried by way of treatment a state of
   depression ensued - possibly referable
   to the influence of the thyroid - which culminated
   in a series of attacks of this character.
   Apparently myocardial Tonicity had been
   depressed.

25. Trousseau. "Diseases of the Heart & Aorta"
   Clinique Med. de l'Hôtel Dieu T. II
   p. 444 Paris 1865

26. Russell. "Relations of Angina Pectoris and
   Allied Conditions Arterio-cardiac Reflex
   having its origin in the abdomen."

27. Gibson. "The Diseases of the Heart & Aorta"
   loc. cit. p. 764.

74.
" XVII 1894 p.339

"On the Disturbances of Sensation
with special reference to visceral Disease"
Gaskell "Journ. of Physiol." 1886 Vol.VII p. 1
"on the structure distribution and function
of the nerves which innervate the visceral
and visceral and vascular systems".
Mackenzie "Brain", Vol. XVI 1893 p.283

29. Mackenzie "The Autonomic Reflex"

30. Brodie "Lectures of Pathology & Surgery" 1846

31. Mackenzie "The Mechanism by which the symptoms
in Angina Pectoris are produced" .
"The pain of angina is a Viscero-sensory
reflex." "Evidences of a Viscero-motor reflex"

"Journ. of Physiol." Vol. V p. 92


34. Eainthoven. (Plügeris Archiv. 1892)
"Broncho-motor action of the Vagus"
to peripheral stimulation, Brain Vol. XVI
1893. p. 306.


"Irritable foci in the Cord due. to visceral disease".
42. Mackenzie. "The Study of the Pulse"
          Edin. & Lond. 1902 P. 70 Par. 58.
          and p. 76 par. 61.

          p. 45.

44. Gibson. "Diseases of the Heart and Aorta Edin
          and Lond." p. 164

          "The Meaning & Mechanism of Visceral Pain",
          "the most violent pains of which we are
          conscious are associated with hollow
          muscular organs, and that by producing
          violent contraction of a hollow viscus
          pain can be elicited".

46. Russell. "Arterial Hypertonus & Arterio-
          Aclerosi; their Relations & Significance"
          p. 474.

          Brit. 1904. CLXXVII S. 372.


49. Broadbent. "Clinical Significance and Therapeutic
          Indications of Variations in the Blood
          p. 1001.
50. Allbutt. "Clinical Remarks on Arterio-Sclerosis"

51. Allbutt.
"The Rise of Blood pressure in Later Life

52. Klotz. "A Discussion of the Classification
and Experimental Production of Arterio-
Dec. 22nd. p. 1767.

53. Moenckeberg. "Ueber die reine Mediaverkalkung der
Extremitatenarterien u, ihr Verhalten
zur Arteriosklerose". Archiv. fur path. Anat.
Berl. 1903. CXXI S.141-167

Dec. 22nd p.772

Edin. & Lond. 1902 pp.158-161 also
p. 52 par. 42
" pp. 56. par 47

57. Mackenzie. "The Study of the Pulse" p. 19

58. Gaskell. Schäfer's Textbook of Physiology
Article "The Contraction of Cardiac Muscle"


62. Gibson. "The Nervous Affections of the Heart". Edin. & Lond. 1904 P. 16 "The Pulse is very often indeed found to be one in which high pressure has obviously been present but is failing"

63. Russell Arterial Hypertonus & Arterio-Sclerosis, Their Relations & Significance.

64. Russell "On Arterial Sclerosis and Hypertonus in their relation to diet and to the digestive system". Brit. Med. Journ. 1904 June 4th. pp 1297 to 1300


68. "loc. cit. p. 55 "capillary obstruction is directly as the length and inversely as the cross section".

69. Mackenzie. "The Study of the Pulse". p. 185 "The Greater the tricuspid incompetence the sooner will the auricle be filled, and the earlier in the cardiac cycle will the wave (v) appear in the veins".


"It has been proved that the general as well as the local blood pressure is subject to reflex influences arising in distant parts". And p. 1001 "Arterial spasm general or local has important influence in modifying blood pressure ... it may produce rapid changes in its level.

"Angina is associated with strain and stress".

"Angina Pectoris due to temporary increased effort by an impaired heart".

"An absolutely low general blood pressure may be one which is relatively too high for the energy of the heart".

"Recurrent high arterial tension as a cause of angina pectoris".

"Under the action of adrinalin the whole venous system becomes over filled and the cavities of the right heart diluted".

79. Mackenzie & Wänkebach.

"Archiv fur Anat. u. Physiol. Berlin 1885 S. 236

die fraglichen Extra systolen werden beim Menschen an der A. - V. Grenze ausgelost.

80. Mackenzie "The Study of the Pulse"

Chap. IX p. 87 et seq. p. 126

tachycardia .... a long continued series of premature systoles".


"this form of arrhythmia is due to the small beat not starting at the usual place, at the mouth of the great veins, but instead of that the ventricle contracts before the auricle.


"The Sympathetic first excites and then depresses the various functions of heart muscle."

84. Mills. Journ. of Physiol. 1886 Vol. VII p. 87. "In the Fish stimulation of the ventricle itself leads to rapid imperfect pulsations."


86. Gaskell. Journal of Physiol. 1884 Vol. V.

"Stimulation of the vagus within the canal cavity produces purely cardio-inhibitory effects."

87. Gaskell. loc cit.

"On the structure and distribution of the vaso-inhibitory (vaso-dilatory and cardio-inhibitory) nerves" p. 28 to 40 and "On the nature of the action of the cardiac vagus nerves". pp. 46 to 52.


"Any lessening of the afferent blood diminishes blood pressure; every increase in resistance raises it up to a certain level; but if the resistance is too high the overflow is reduced and pressure falls."


91. Brunton loc cit.

"Increase of tension in the blood vessels of the medulla stimulates the vagus roots and slows the heart. Diminished tension lessens the stimulus to the vagus"


95. "The Respiratory centre goes back to its less educated form and reproduces in an exaggerated way the rhythmical character of the respirations that is more or less natural in infancy".
96. Eve. "A Cerebro-spinal Manometer"
Lancet Lond. 1905 Vol. I. pp. 1067 - 1069

Oct. 1904, p. 495.
"Cerebral vessels have very scanty vaso-
constriction supply". "The Action of
adrenalin upon the systemic vessels is
in proportion to the innervation of these
vessels".

98. Head. "On Disturbances of Sensation with
Special Reference to the Pain of Visceral
Disease".

Brain Vol. XVI 1893 pp. 100, 126 & 127
 especially in the splanchnic veins. 341.

Thorburn " Vol XVI pp. 356 & 357
Ross " 1888 Vol. X p. 333
Mackenzie "The Meaning & Mechanism of Visceral
 & 1453
& " " " June 30 pp. 1523 to 1528
Klotz. Op cit. p. 1769
"acute oedema of the lungs immediately
after the inoculation of the adrenalin"
p. 495. "The pulmonary arterioles possess no vaso-motor supply".


"Directly blood pressure has fallen there will be a tendency for the blood to collect in the large venous trunks especially in the splanchnic veins".


"Other Reflexes occurring during an Attack of Angina Pectoris".


115. Gibson "Diseases of the Heart & Aorta" p. 765

"The myocardium is frequently the seat of chronic interstitial myocarditis, often associated with arterio-sclerosis which...may effect the coronary vessels in particular"

The results of Mackenzie's investigations are confirmatory of this.


118. Fraser "the real nature of the action of the
New Series Vol XIVp. 152

and Clin. Lecture on Diseases of the Heart

XXXVI p. 393.

121. Brunton. "Action of Medicine"
Lond. 1897 pp. 332 - 337.

122. Brunton. Op. cit. "Alcohol is one of the
most powerful stimulants of the cir-
culation.....tends to keep the heart
active....the vessels appear to dilate
under its influence.

"Mercurial Aperients have a definite
and constant effect on the blood pressure.
.....by diminishing the peripheral resistance

In describing these pulse tracings I have endeavoured to follow Mackenzie's method of interpretation as described in his book on the Pulse, and have used his terminology. P.D.C.

Ref. "The Study of the Pulse"
Edin. 4th S. 1802.