Angina Pectoris.

An inquiry chiefly into its Pathology.

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Note - In order to facilitate the reading of the manuscript, I have placed the authorities referred to, at the end of the paper in the form of an appendix.
The name "Angina Pectoris" has been applied by Heberden to a group of symptoms, of which the more prominent, are—pain in the region of the heart, of a paroxysmal character, radiating generally on the left side, & arm, & associated with a peculiar sense of insecurity, or a feeling of constriction in the thorax.

English authors of text-books on the practice of medicine, while in part at least, admitting its neurotic character, have as a rule placed this disorder in the same section as diseases of the heart (Reynolds, Addison), functional diseases of that organ (Roberts), or under disease of the vascular system (Bristow). Among Continental authorities, we find that Ironsone gives it a place among nervous diseases, regarding it under "hyperesthesia of the cardiac plexus," & Zimmer in his Cyclopaedia, places it with the vaso-motor & trophic remedies, & classes it with the visceral neuroses.

The French claim priority in describing this disorder, on the strength of a letter from M. Rouquayrol in 1768 describing the symptoms of "une maladie nouvelle." He described the paroxysms, in the case of a cavalry officer named Charles, & after the autopsy, gave it as his opinion, that death was due to ossification of the costal cartilages.

Dr. Heberden, in the same year, was the first to draw the attention of the profession to the subject, in his Commentaries, & communications, to the College of Physicians, on this subject, in which he terms the disorder "Le dolorad pectoris." He subsequently applied the name "Angina Pectoris" to this group of symptoms, a name which has been associated with the disorder
Since his time.

The derivation of the term angina, it is stated by Jumers, is from the Greek 'angina', to strangle, and is equivalent to the Latin verb 'angor', to press tight, especially the throat; that there is no true angina present in the majority of cases. During a paroxysm, he shall see, it cannot therefore, but be regarded as unfortunate, that some less misleading name was not generally fixed upon for the disease.

Since Heliodorus' time, others who have taken up the subject have, according to the views of the pathology of the disease entertained, applied a variety of names to the disorder; in the eyes of some, 'angina', in others, 'angina', and in many, 'angina', it may be found treated of under any of the following synonyms:—Asthma asthmaticum, Angina cardiacae, Diaphragmatic gout, Angina pulmonalis, Angina cerebrovisceralis, Angina pulmonalis, Sanguine, Angina pulmonalis, Angina thoracis, Angina cerebrovisceralis, Angina cerebrovisceralis.

Before entering into a description of the symptoms and causes of true angina pleuritis, it may be as well to consider shortly the diseases for which at least, cases of what some authors term 'Angina sine dolore' or 'Numb angina', or 'Pseudo.

Angina'.

The term 'Angina sine dolore' has been applied to the subjective abnormal sensations often indefinite and difficult to define, experienced by the subject of cardiac valvular disease, these sensations not being associated with any distinct pain. I have noted these paroxysms in the case of a fellow student suffering from rheumatic disease of the aortic valves.
permitting of repiration. Prior to the establishment of the compensatory hypertrophy of the left ventricle, the attacks were frequent at first, gradually diminishing in force. As compensation became established, the exciting causes of a paroxysm, were very similar to those which occasion an attack of the then disorder, i.e., symptoms from that disease. He described his sensations as a "great fear," with a feeling of the heart was about to cease beating. Later questions however, I found he fell no really definite local pain. Dr. Gainsford states, that these abnormal sensations are not with in connection with every form of cardiac lesion which terminates fatally. That acute incompetency is most frequently the cause of this kind of suffering. He also considers the "Cheyne-Stokes" form of respiration, as characteristic of this form of angina, a form of respiration indicating a weakened condition of the heart.

"Pseudo-angina" is a term applied by Dr. Balch to a less frequent form of neuralgic cardiac pain, which is paroxysmal, but which does not lead to grave results, i.e., death. It is met with chiefly in young subjects, and is accompanied at times with palpitation, and disturbances of the respiratory function: faintness, vertigo, with pallor of the face; facial and irritable pulse, may also be present. The attacks may appear serious, but rarely prove so. Cases of this kind are met with frequently in general practice, chiefly among dyspeptic, anemic or nervous subjects, and among women. Those given to the abuse of tea or tobacco. The paroxysm is confined to the cardiac region, i.e., chest, sharp, passing off quickly.
not recurring, this perhaps the cause exciting causes are again in action. Dr. Thomas Shapte, in his "Notes and Observations on Diseases of the Heart, and the Dypsia in Connection Therewith" (Churchill 1874), at Chapter II, on "Pain of the Heart," has some interesting remarks on this subject. The suffering in "pseudo-anxiety," is generally associated with an anamnestic condition of the heart or its nervous structures. For this reason, some authors consider such cases as merely modified forms of the true anxia paroxysmal, the purely nervous type. Let him return to this subject, however, when treating of the pathology of anxia pectoris, i.e., classification adopted by various authors.
Course and Symptoms of Angina Pectoris.

The disease is characterized by paroxysms, separated by variable intervals, which are generally free from symptoms.

The patient on some slight exertion, or usually without any preliminary symptoms, is suddenly seized with a feeling of constriction at the base of the chest, and severe pain amounting, generally, to agony, of a peculiarly "unbearable" character in the cardiac region. The pain radiates from thence, down one or both arms, generally the left alone, along the sides of the neck, and down the back or up. There is an inability to breathe associated with the pain, but no symptoms of cyanosis are developed. The patient feels instinctively that any movement of the chest will make the pain worse, and an almost intolerable agony: a deep breath taken and held, however, does sometimes relieve the pain. In most cases the pulse tends to become feeble, and even fluctuating and irregular, particularly if the attack is prolonged; in other cases, the beat is not appreciably affected, or may even become slower than usual.

Consciousness is generally retained, although syncope may result from the severe agony and consequent inhibition of the heart in continued attacks. Sometimes nervous symptoms are manifest, towards the close of a paroxysm. Spasmodic muscular twitches in the limbs, disorders of the special senses, and even general convulsions have been observed. During the paroxysm, the face is pale, the expression anxious, beads of perspiration standing out on the brow.
The whole attitude or bearing of the sufferer expressing his intense agony, and alarm, which when once seen is not easily forgotten.

An attack may be made up of several brief paroxysms, with intermissions, as in the case of many neuralgias, or there may be only a single paroxysm. The pain may cease suddenly, with a sense of great relief to the patient, or it may pass off gradually; in both cases, a feeling of exhaustion more or less prolonged in proportion to the severity of the paroxysm, is left behind. A marked characteristic of the disorder is its great tendency to recur at uncertain intervals, under the influence of very slight exciting causes.

Very rarely does a first attack prove fatal, death however may take place during a paroxysm as in the cases of John Hunter, Dr. Arnold of Rugby, and others, or by a gradual process of exhaustion, the form of the cardiac contractions undergoing a progressive diminution, death ensues from asphyxia in a painless interval.

It may be of advantage to hereupon present unnecessary repetitions as we proceed, if before we go further with the subject, we direct our attention a little more in detail to the more prominent symptoms, which we shall consider, them in the following order: the pain, variations in the cardiac rhythm, pulse, the respiratory changes observed during a paroxysm, and the nervous symptoms occasionally manifested at the close of a paroxysm.
The pain which forms such a prominent feature in the paroxysm of pain, has been variously described by different writers: some say that it is punctuating, and hardly to be borne. Some describe it as "burning," others as "freezing." In character, it is usually felt about the lower part of the chest, in the region of the heart, but its position may vary, as it varies also in intensity, in different cases. In all, it is paroxysmal, reaching a climax in a few seconds, or minutes, and gradually disappearing. On the other hand, an attack may be formed of a series of paroxysms, with short intervals, which may go on for two or three hours or may be continued through several days. Ercoli, with variations.

The radiating pains in the arm and other parts, follow generally in point of time the cardiac pain; this state of matters, however, is sometimes reversed. Thus, Trouseau mentions cases in which the painful sensations originated in the left arm, rapidly spreading to the chest. The pain is chiefly confined to the left side, the left arm being more frequently affected than the right, but the centres of both may be involved. In some cases, no distinct sensation of pain is experienced in these nerves, but a feeling of tingling, or numbness, passing down to the finger-tips, may take its place. So the pain in the cardiac region is sometimes superadded as a sensation as if the heart were standing chill, a subjective sensation not altogether without its equivalent in fact, as we shall see when we consider the ultimate pathology of the complaint.

We cannot leave this portion of the subject,
without drawing attention to the eruptive, or sense of impending trouble, experienced by the patient during a paroxysm, which Dr. Hawthorn was the first to note, and separate, as distinct from the pain of angina pectoris. This peculiar sensation is experienced during every paroxysm, and in a large measure, contributes to form the intolerable nature of the suffering. Hawthorn describes it as a "sense of approaching dissolution."

The action of the heart is disturbed more or less, in most cases, during the paroxysm. It is generally found to be diminished in power, the sounds on auscultation being feeble, and the pulse weak. In some cases, we meet with decided intermissions in the beats, or considerable pauses during the diastole; in others again, the contractions are increased in force, and may be very violent. At the climax of the agony, we generally find the contractions weakened, and if the pain is very severe, they may be temporarily arrested.

The pulse, shows similar variations, during the paroxysm. In some cases, it is small and irregular, often imperceptible; in others again, its action is found perfectly regular, and steady, throughout a strong, and full pulse.

As a result of these motor changes, we find the skin, especially of the extremities, cold, shrivelled, pale, a general sensation of chilliness, at the same time, pervading the system. Towards the termination of the paroxysm, the opposite con-
may be developed, or profuse perspiration, or diarrhoea, may accompany, or follow, the sense of being experienced by the patient with the cessation of the pain. This free operation of urine, is well-marked in a case which came under Trousselot's care; the patient found by experience, that the paroxysms were always protracted, if he did not obey a call to make urine, and that if he omitted his bladder early, the attacks were not so severe.

In some cases, a cord-like hardness, of the radial artery, has been met with, at the beginning of the paroxysms, giving place gradually, to the opposite condition, with complete loss of tone. A case in which this condition was well-marked, is recorded by Trousselot. In his Clinical Lectures, he states of the forearm, or hand, was observed at the beginning of the attack, to become almost bloodless, and soon afterwards, to change to a livid bluish colour.

Dr. Sander Brumton's spirographie drawings, taken during the paroxysms, in a case of Chorea acuta, under the care of Professor MacLagan, in the Edinburgh Infirmary, demonstrated these conditions to exist in some cases. This case, of Sander Brumton's, which is referred to by most of the writers on this subject, will be found fully described in the reference given in the appendix. I also, in a paper in the Lancet, July 27, 1867.

The paroxysms or attacks, occur at uncertain intervals, and may be suspended entirely, for long periods. The general tendency is for the
ntravals to shorten, as the disease approaches a fatal
termination, the attacks becoming more severe, more
easily excited, and the debility great, exhaustion, more
prolonged.

Among exciting causes of the paroxysms, may be
mentioned any of the following: muscular
exertion, especially moving up a hill, or climbing
stairs; coughing; sneezing, straining, prolonged
speaking. Mental disturbances of any kind - e.g., hurt, anger, fear, joy, etc.,
cess of eating, drinking, also tending to
excite attacks. As the disease progresses,
the attacks come on from the slightest causes,
at times, no cause can be discovered.
The patient perhaps perhaps being quietly seated in a chair, reading. The paroxysms may
then come on in bed, while the patient is quietly.

sleeping, or through the influence of
dreams.
Pathological Conditions associated with Angina Pectoris.

These may be conveniently classified under three heads:-

I. Pathological lesions in the heart & vessels.

II. Pathological changes in the nervous structures contained in the thorax.

III. Changes in the abdominal organs.

I. The diseases of the heart, & vessels, found after death, associated with Angina Pectoris, are very numerous, & may with truth, be said to comprise nearly every pathological condition noted with in these organs. Jourdain gives the following list:— "Occlusion of the Coronary arteries, aneurisms, hyper trophy & dilatation of the heart, occlusion of the auricles, ventricles, and aortic valves, purpura, fatty accumulation in heart & pericardium, compression of heart by tumours, or enlarged abdominal viscera, inflammation of aorta, myocardial atresia, ossification of the costo cart. ribs," to which may be added, the various degenerations of the heart, & a dilated condition of the coronary veins. It will be sufficient for my purpose however, in this paper, merely to take notice of those lesions, which by their frequent association with cases of angina, are brought into prominence, & to furnish a few statistics on the subject.

15. Parry 9: according to Jourdain 10, was the first to notice the association of ossification of the coronary arteries with angina pectoris. 15. Gairdner 415. mentions the fact that Edward Jenner communicated this intelligence to Parry.
who took up the subject, published his view. Parry states in his books that angina pectoris, depends in most cases on some obstructive disease of these vessels. Among the authors whose statistics on this disease are most frequently quoted, are Sir John Forbes, and Cussana. Forbes states that out of 45 cases in which a post mortem examination was made — in 24, the aorta was found diseased, and in 16, the coronary arteries. No doubt, among the aortic cases, there must be a larger proportion, have been present, causes leading to diminished flow in the coronary vessels during life. If not, positive obstructive closure of the orifices of these vessels. Cussana gives the proportion in which coronary disease was present, as 21, in 86 fatal cases. From the enormous cases reported by different writers, in the journals, &c. elsewhere, &c. The opinion, he comes nearer the truth in this matter, particularly, if among diseases of the coronary vessels, he includes everything tending to obstruct the circulation in the heart substance. In dealing with statistics however, particularly in obscure nervous disorders like the present, it must be remembered that they cannot obtain absolute accuracy. The more serious cases, & those proving suddenly fatal, forming the majority of the cases on which they are built up.

Degenerative changes in the aorta, either alone, or associated with atherosclerosis and —
dilatation, an frequently met with in cases of aneurysm. Irons believes his lecture on this subject, mentions several, which can, in which paroxysms of aneurysm were an early symptom. Gainon 47 also draws attention to this point. It states, that in his experience, small aneurysms close to the heart more frequently originate symptoms of aneurysm, than larger, t more remote lesions.

Before referring to the degenerative changes so frequently met with in these cases, in the muscular tissue of the heart, I should like to consider at some length, a cause which brings about these degenerations, by obstructing the orifices of the coronary arteries; the cause of obstruction I shall consider first, in one that is associated with aneurysm in young subjects — I allude to acute endocarditis of the first portion of the aorta. This disease is only imperfectly described in pathological works, and generally referred to with the atheromatous process, as in some cases a probable originating cause of the more chronic form of inflammation. The researches of Bizzet, referred to by Payne, 13, and the more recent work of M. M. Comit and Ramvix, 14 on this subject, have done much to clear up the character of the changes, which take place in the aorta when affected with acute endocarditis. They describe the results of acute inflammatory action, as giving rise
to an "albuminous exudation, of greater or less thickness, of the consistency of jelly, transparent, and smooth, covering the lining membrane." This swollen substance, it found on examination, to be composed of embryonic cell-tissue, resulting from the proliferation of the surface of the inner coat of the vessel.

I should not have gone so much at length with this subject, but that it is hardly referred to in our text-books, and besides, it is of his importance, to be so far, that it has been found that obstruction to the coronary circulation arising from this cause, has been found associated with angina pectoris, in young people, and has tended to make, a generally chronic disorder, run a more or less acute course.

A case of this kind came under the care of Sir Ephraim "in 1876," and the specimen was brought before the Pathological Society of London in June 1876. At first, the heart of a woman, 27 years of age, which weighed 8 lbs. Its valves were healthy, but the inner coat of the first portion of the aorta, immediately above the sinus of Valsalva, was thickened, for about half an inch, leading to obstruction of the coronary arteries, which would only admit a trickle. The calibre of the vessels themselves, was normal, and it is mentioned that there were a few patches of "atheroma." In these vessels -
probably, the result of the breaking down of the thickened tissue. A case in many points similar, came under the case of Dr. George Dalgarno, and has been recently published by him, in the Edinburgh Medical Journal, into which he enters pretty fully, at p. 772.

The case, was that of a man, 24 years of age, who suffered from angina pectoris, which originated from coronary obstruction, thought about by acute endarteritis of the first part of the aorta. The process, "translucent," abdominal, affection, which at the autopsy was called, "atrophic thickening," was clearly the result of the more acute inflammatory process. Death took place during a paroxysm.

Doddert-Clarke also publishes a case in the "St. George's Hospital Reports," for 1879.

In this case, the man was 47 years of age, but his illness lasted little more than five weeks. His health being good, up to the period of his first paroxysm. The return was found to be healthy, but a similar lesion to the two cases already described, was found in the aorta, obliterating the oriifices of the coronary arteries. The vessels themselves were healthy, and no trace of athmosclerosis was present. That the translucent condition (which has in Dr. Dalgarno's case caused the internal coat of the aorta to assume nearly three times its normal thickness) might, under fatty and calcaneous changes, is quite possible. But in young Subjects, suffering from acute endarteritis, resolution, is much more likely to
take place, and as Dr. Baqoui himself admits, further on in his paper, "similar conditions are to be found in humans, who make perfectly good re.

It is important, therefore, that these acute changes which are distinct, separate, from the essentially chronic, degenerative atheromatous process, should be distinctly recognised. In the atheromatous process, which is generally regarded as chronically inflammatory in its origin, (but by others as purely degenerative), the initial changes—the cell-prolifer.

ation—take place in the deeper layers of the intima coat.

Before leaving this subject, however, it may be as well to note, that calcification of the coats of the vessels may occur, quite independently of the atheromatous process. Changes of this character may take place in a comparatively rapid manner. In cases in which the salts of lime are removed from one part and deposited in another—the process known as "extensive calcification."

The degenerative changes in the muscular tissue of the heart, which can generally be traced to imperfect blood supply, are very commonly met with in cases of anaemia pectoris. The most frequent are fatty degeneration, fibrosis, muscular substitution, with dilatation. Further, in his statistics, mentions that in 12 out of his 45 cases, there was found unusual softness of the heart; in the 16 cases in the same table, mentioned under coronary disease.
no doubt, in some at least, a close examination would have revealed degenerative changes in the heart substance.

Valvular disease, it is found frequently associated with angina, especially anemic lesions permitting of suppuration, with a rigid, a dilated condition of the aorta. In Sir John Forbes 45 cases, 16 had suffered from valvular disease.

The fact that the various pathological conditions allowed to occur frequently exist without giving rise to any symptoms of angina pectoris, clearly point to some other cause, as the common cause, of the disease—what this common cause is, is a subject which will be considered in its proper place, under the pathology of the disease.

II. Changes in the nervous structures in the thorax, found associated with angina pectoris.

Changes in the cardiac region, generally of a vascular character, have been found in a few cases. That statistics are wanting on this subject, is no doubt in a measure, due to the fact, that the attention of pathologists, has till lately, been mainly directed to the condition of the heart vessels, as the probable source of the disease. In the neglect of possible changes, often of a microscopic character, in the nervous structures. Lancearne 19, has published three cases, in which he found vascular changes to have taken place in the cardiac plexus. In all
of his cases, he found the aorta, coronary arteries, degenerated, and the portion of the pleura most seriously involved, was that, in immediate contact with the aorta, which was found to be vascular and partly hidden by evagination. The microscope showed the contents of the nerve tubes, to be granular, the result of compression, by the development of numerous nervous nuclei between the tubes. These cases are carefully reported by Sandrullat, and genuine cases of am尼亚 pectoris, as far as one can judge from the course, and symptoms — all three patients were above their fortieth year. That these changes in the pleura are secondary to the aortic disease, there can be little doubt, but the study of them, is important, in connection with the fact, that numerous cases of simple atheromatous degeneration of the aorta, complicated with coronary obstruction, do not always rise to symptoms of am尼亚 pectoris.

Dr. Haime \(^{20}\) in 1841, was the first, to publish a case of nerve lesion, in connection with this disease. This case is referred to by Romberg \(^{21}\), at considerable length, and will repay careful consideration. The patient a man, 36 years of age, suffered from paroxysmal intermissions in the cardiac pulsations, with intense pain on both sides of the chest, extending up rapidly to the neck, and head, the attacks gradually increased in frequency, and he died in a "state of torpor" — probably...
comatose condition. Dr. Heine, with Professor Ronicz, 
ansky & Strese, repeatedly examined the patient, 
& diagnosed, "a pseudo-plastic tumour, involving 
the upper cervical plates, or the cardiac nerves, 
with hypertrophy of the cervical spinal cord." 
The autopsy was made by Prof. Kotschansky - Em 
Rombuy's "Nervous Diseases of Man" Vol II p. 342-343. 
for a full report of the lesions. The main 
structural nervous changes noted with, were as 
follows: - the branches of the cardiac plexus, + vagus. 
the phrenic nerve, were compressed by pigmented nodules + lymphatic glands; the thoracic lymphatic glands were enlarged, + pigmented, convoluted with hard masses, containing concretions. The 
phrenic nerve was pressed on, by one of these 
the edge of a trunk, near the root of the lung. The heart was of the normal size 
but tough; the lungs were oedematous.

Dr. Haddon 22. in the Edinburgh Medical 
Journal, also describes a somewhat similar 
case: atheromatous disease of the aorta was 
found - a pigmented gland was seen compressing 
the phrenic nerve.

III. Changes in the abdominal organs.

Degenerative changes in the liver + kidney, have 
been met with in cases of anemia pepticus in 
which no other lesion could be found, + have 
been credited with originating the paroxysms in 
reflex irritation.
Etiology of Angina Pectoris.

Among constitutional disorders, gout + rheumatism are most frequently associated with this disease.

In a paper recently published in the British Medical Journal, Dr. John Duckworth 23 attempts to prove the rheumatic character of gout, and points out its analogy with other nervous: to instances it makes himself very tendency, custom + paroxysmal symptoms - the pyrexia being paroxysmal - the fairly good health enjoyed between the attacks. He states also the fact that specific causes are often present, yielding rapidly to specific treatment. There is no doubt that gouty subjects suffer frequently from severe pains in the cardiac region - what is commonly known, or dreaded, as "gout in the stomach" + "heart." These pains frequently assume the character of paroxysmal anguish, or lead ultimately to sudden death. A retrogression or metastasis of the complaint, according to Ironsman, need not be considered to take place - rheumatism of the cardiac region being developed in the same manner as any of the other neuralgias from which gouty subjects suffer. In such subjects also we find that the heart + vessels are liable to undergo degenerative changes, especially of that character + in those positions of the circulatory system, found most modern so frequently associated with angina pectoris - the first portion of the 2nd & coronary vessels. Ironsman in his lectures brings out the cor...
Between the two diseases very forcibly, and illustrates his arguments by several well-chosen cases by. The case of John Huntin is also one to the point; it is related at some length in Reynold's Synopsis of Medicine, Vol II, p. 322. In three successive

spring after his forty-fifth year, Huntin suffers from attacks of gout. But in the fourth, instead of gout, he had an attack of aminia fœtida which lasted for three quarters of an hour, and which was called "gout of the stomach." These attacks grew

worse, became more frequent towards the close, and he died during a paroxysm excited by a "silent rap." The heart in the autopsy was found normal, and amiasic. The coronary arteries, aorta, mitral and aortic valves are undergoing calcaneous degeneration.

Rheumatism does not appear to have such a close relation as gout to aminia fœtida. With rheumatism, the relation depends alone on the fact that those subject to it, are liable to acute disease of the heart and aorta, with the subsequent chronic changes those lesions undergo. With gout, we have seen something in superadded to these local changes - a marked neurasthnie tendency.

In some cases aminia fœtida has been shown like other nervous disorders to have a de
cidely hereditary tendency. Trousseau relates a case mentioned by Hamilton, that of a soldier suffering from the disease who assured him that his father, two brothers, and a sister, had suffered from similar symptoms.
Enulbing in Zwischen's Cyclopaedia has also traced this tendency.

Age and diet have some prominence as etiological factors. The disease is more frequently met with in advanced life. Forbes 11 states that out of 24 cases he had collected, only 12 were below the fifth year. Heberden in his commentaries claims to have seen 100 cases, all of which, with a single exception, were in patients near or past their fiftieth year. Ironsides 9 states that it is almost exclusively attacks individuals above forty, although it has been met with in younger persons. He mentions that Heberden has found it in youth, that and Hamilton mentions it did not spare childhood. Glaucoma angina, has undoubtedly been met with in young subjects, such cases have been reported from time to time in the journals, but they are always considered exceptional. As more recent instances may be mentioned Dr. George Balfe's 16 case already alluded to in a man, age 64; Dr. Sprungfeld's 17 case in a woman, age 27; a case reported by Dr. Wilson in a farmer, servant, age 23.

As regards sex, males are more frequently affected than females. Forbes 11 gives the proportion as 11 to 1. Heberden only met with three females in his hundred cases. Enulbing considers that this disproportion may arise from the fact that males are more exposed to certain direct causes that may originate these attacks—e.g., exposure to cold, great strain...
of life, excessive smoking etc. Trousseau 6.3. alone among the authorities I have consulted objects to this point - he does not consider it proved, that makes any more subject to angina pectoris than females; the cases he brings forward in lecture 
22. certainly comprise a fair proportion of female patients.

Eulentory is of opinion that in this disease as in other neuralgias, anaemia is a powerful etiological factor. Dr. Eoy. Balfour 23. also favours this view in his book on "Diseases of the Heart and Aorta" he states that in his experience, anaemia of either local or general origin, is a predisposing cause. Among the exciting causes of the paralysis he places the depressing influences of tobacco and noxious poisons. Any cause leading to the alteration of the blood, will act in a similar manner. For this reason no doubt high living with insufficient exercise, is found so frequently associated with this disease, as with gout; the blood being rendered impure.

A high social position, is found frequently associated with angina, probably for a similar reason. In very poor, or those who have to work hard for their daily bread, are almost exempt from this disorder - they are certainly not so frequently attacked.

Epilepsy, according to Trousseau 6.3. must be regarded as a predisposing cause; the evidence of any etiological connection between the two diseases is not considered sufficient by
any other authority to assume notice. We shall return to this subject however in treating of the pathology of asperia pectoris.
Pathology of Angina Pectoris.

In treating of this difficult subject, I shall first consider the various views on the nature of the disease entertained by different authors, and then go into the ultimate pathology of the affection with its relations to the innervation of the heart and vessels system.

The majority of authorities in medicine have either considered angina pectoris as a pure neurosis, or connected it as closely, more or less closely, with certain organic lesions in the heart vessels.

Hitherto in treating of this subject, recognised clearly that it was distinct from any inflammatory process—be gave it a place among the disturbances or spasms, recognising its nervous origin. In the mode of treatment which he favoured, we see this well brought out—he discouraged blood-letting, recommended supporting, and antispasmodic treatment. Since his time, a variety of views on the pathology of this disease have been supported by different writers, some of which it will be merely necessary to mention, others will require a more careful consideration.

Parry as we have seen, considers that the true pathology of the disease, was in the majority of cases to be traced to obstructive disease of the coronary arteries. In his "Essay on the Symptoms and Causes of Syncope Exsanguine," he considers the disease as merely a modified form of Syncope—differing only in this fact, that severe pain is superseded. (Quirner 48) in examining this view of Parry's, points out, that while
There are symptoms in common: the fact that the mind usually remains clear to the end, that the pulse is present throughout the attack, separates it distinctly from a simple attack of syncope.

Bucsi, according to Goumon, held peculiar views on this subject: he considered the true pathology to be a disease of the lower cervical or upper dorsal spinal cord, liable to be aggravated by disease of the heart or vessels. The cardiac pain to which he believed were reflected impressions from the true seat of the disorder.

Latham's von Duesch and Mussau and others again stated that the symptoms of death depend on spasm or cramp of the muscular tissue of the heart, accompanied by severe neuralgic pain, originating in the cardiac plexus. Bucsi, 26 also favours this view, as in support of it it is

Staines: the severe pain that may arise from the same cause, in other organs supplied by the sympathetic, which is a similar condition, e.g. bowels, cystic ducts &c. He also agrees with Lauder Brunton (whom Linné we shall have to consider presently) from the vasomotor symptoms manifested in most cases - the coldness of the general surface, followed by perspiration & secretion of urine - that an essential part of the disease, is a derangement of the vasomotor mechanism, leading to a rise in the blood pressure, & a consequent increase in the work thrown on the heart. Cahn & Traube, constantly before Lauder Brunton took up the matter, had both
expressed opinions to the effect, that angina pectoris should be classed with the vaso-motor nerves; he
referred that the normal vaso-motor centre itself was stimulated.

Mr. Stokes of Dublin referred the phenomena to a sudden accession of vitality in a heart weakened by degenerative changes arising from some sudden call for increased activity.

Mr. Hyden regards the disease as essentially a neurosis, involving primarily the cardiac nerves, and reflected to the spinal nerves through the sympathetic. Mr. A. B. Sansom also holds a similar view, and regards angina pectoris as especially indicative of degeneration of the heart.

Irouseau, in treating of this subject, publishes some interesting opinions peculiar to him. In his lecture on epileptiform neuralgia of the fifth nerve (a disorder which he has never known to be permanently cured), he compares angina pectoris with it, and with epileptic vertigo, and points out the analogy between them. In Chapter XIX, p. 392, he expresses his opinion on the pathology of angina: he considers that although angina pectoris is often symptomatic of organic disease of the heart, it is often, still in some cases of undoubted angina, no lesions are to be met with. Therefore it is not essentially due to organic disease, but is a neurosis, often associated with cardiac and vascular lesions. But these lesions only afford an opportunity for the development of the neurosis.
The disease to be a neuralgia of the branches of the vagus.

On referring to the method of treatment of anopia adopted by Ranconneau at Tours - p. 611. - (on the chemical hypothesis of its origin in calcareous deposits in the aorta, by repeated + long. Continued doses of bicarbonate of soda + belladonna.) Ironsonan reminds his hearers that the Latin word is a favourite with him in epilepsy, + that the good results obtained from its administration, in anopia pectoris, and not be wondered at, for that disease "in in many cases merely an epileptiform neuralgia, or a kind of aura epileptica." In another place, he states that in his experience, in a large number of cases, anopia pectoris is "an expression of the vertiginous form of epilepsy, or in other words, an epileptiform neuralgia." He arrives at this conclusion from comparing the paroxysms of the two diseases, their mode of invasion, course, + termination, together with the nervous symptoms developed during the anopia paroxysms in certain cases. In support also of this connection between the two disorders, he states - points denied by other authorities - that it is not uncommon to find sufferers from anopia pectoris becoming afterwards epileptic, + also that epileptics frequently suffer from symptoms of anopia pectoris. He supports this statement in his lecture by a single instance - p. 602. - in which the characteristic symptoms of anopia are not well developed. In concluding his lecture, Ironsonan draws attention to the fact, that in
Certain cases the paroxysm originates in the arm, a symptom closely resembling the "aura" of epileptic subjects.

In considering these views of Trousseau's, we cannot but admit the resemblance between the character of part of the pain in angina, that experienced in the neuralgic of the fifth nerve, to which he has given the name epileptiform—its sudden and severe onset, and rapid cessation. The relation to epilepsy is not clear, nor in fact most of the authorities on nervous diseases dispose with him entirely on this point. The epileptiform symptom occasionally met with at the close of a paroxysm in angina pectoris, can be explained in a much more simple manner, as we shall see in treating of the ultimate pathology of the disease.

Laennec, Chapman, Depoortis, Romberg and others, regard the disease as essentially a neuralgia of the heart. Laennec calls it "neuralgia Cordis;" he thought that in some cases the vagus and its branches only were affected, in others again the sympathetic cardiac branches. Romberg places this disease among the visceral neuralgias, and treats of it under "hyperaesthesia of the cardiac plexus."

Lauder eminent has demonstrated in one case at least, the fact that during the paroxysm there is increased tension, rise of blood-pressure in the vascular system, followed towards the close of the attack by the opposite conditions.
This case, already alluded to, is fully described in the "Transactions of the Clinical Society of London" Vol. III pp. 191-199. Spirometrical tracings were repeatedly obtained during a paroxysm, & contrasted strongly with those obtained at other times. These experimental results, led Brunton to conclude, that increased vaso-motor action was present & to employ amyl nitrite for the relief of the symptoms. In a paper in the British Medical Journal for March 31, 1877, he states, that the disease consists in a disturbance of the regulating mechanism of the heart & vessels. As a result of these investigations, it is now pretty generally admitted, that increased intravascular tension is present during the paroxysm, in some cases of anginapectoris.

Dr. George Balfour believes, that the blood pressure is above the normal in all cases of angina, but that it rises specially during the paroxysm, he does not consider proved.

In a paper in a recent number of the Edinburgh Medical Journal, Dr. George Balfour expresses his views on the pathology of this disease. He considers the true pathology to lie in some form of interference with the blood-supply of the muscular tissue of the heart, or the intra-cardiac ganglia. In his experience, he has never failed to trace the disease to this source. He states that it is of no consequence what lesion of the heart or vessels is present, so long as the supply...
of blood to the heart. Endurance is now interfered with
no symptoms of angina are developed—more
or less pain arising, according to the extent of
the interference with this supply. While
admitting, that an occasional cause of these
paroxysms has been traced to lesions of the
coronary branches of the vague, + sympathetic.
Such cases as those reported by Hein20 and
Harrison21, he considers, that even in these
cases, there is diminution in the amount of
blood supplied to the heart substance, brought
about by nervous influence, direct, or reflex.
In support of these views, he reports a fatal
case which came under his care, + which
I have already drawn attention to in dealing
with the etiology of this disease. The
patient was a male, 55 years old, the only lesion
found with after death was a “translucent
atheromatous thickening” of the inner coats of
the aorta immediately below the aortic cusps.
Healing is contraction of the cusps + orifices of the coronary arteries. A chronic lithis.
graph of the heart is prefixed to the paper.
Dr. Balfour also draws attention to the
fact, that in those cases of anaemia in which
cardiac pain is complained of, this pain de-
pears on meal nutrition of the heart, or re-
sult of the general anaemia. The pain be-
ing relieved by the removal of this condition
by suitable means. Such cases as I have
included under Dr. Walsh's term "pseudo angina"
at the beginning of this paper, he considers very closely related to true angina pectoris.

These views of Dr. Balton's derive additional support from the experiments of different observers on artificial interference with the circulation through the coronary vessels. That the motor function of the heart depends for their continued power on the free supply of blood through the coronary vessels, is clearly demonstrated by the experiments of Chirac, which were repeated by Brichstetter 33. and von Bezold 34. on the effects of artificial interruption of the coronary circulation on the cardiac contractions.

In a paper in the "Jenaer" on exclusion of the coronary arteries by Delhi 35. of Oldenburg, refers to the more recent experiments communicated to the Académie des Sciences, by M. de la Poche-Fontaine, in Roussy, on the same subject. The effects, according to all these observers, of such interference with the cardiac blood supply, is to diminish gradually, progressively, the force and regularity of the contractions, until ultimately to cause complete arrest of the heart in diastole - the normal regular rhythm gradually returning after the blood is again permitted to enter the vessels.

In concluding this portion of the subject, I after a careful examination of the different views expressed on the pathology of angina pectoris, I consider, that we must regard the disease as essentially a neurosis, more or less closely connected in different cases with...
Causes affecting the nutrition of the heart & the intra-cardiac ganglia. The view of the pathology expressed by Dr. Balfour—that angina pectoris is solely due to coronary obstruction, proportional to the degree of its severity, to the amount of obstruction present—cannot I think be unreservedly maintained. In opposition to this statement, I can refer to cases of marked coronary disease, in which no symptoms were present during life, & to cases of fatty & other degenerative change in the heart. Substance, not with daily in every pathological feature, directly traceable to imperfect blood-supply, in which not a single paroxysm of angina was experienced during life. The case of Dr. Chalmers, reported by Dr. Wardroper in 36, will serve as an illustration of extensive fatty degeneration without a symptom during life. Dr. Hayden, in a table he gives in his work "On Diseases of the Heart & Aorta," found, that out of a total of 95 cases of deaths from fatty degeneration of the heart, in only 8 cases, were symptoms of angina pectoris present. It is only I think by considering the disease a neurosis that we can include all cases of Angina under one general division. Here are cases on record of undoubted angina pectoris, in which after death, skilled observers were unable to detect any lesion of the heart or vessels. The cases also in
which microscopical nerve lesions are met with, and which may yet be multiplied by careful inves-
tigators, also direct special attention to lesions of the nervous structures, as originating causes.
Trousseau 5 has pointed out, that "nerveless may be impaired on organic lesions, and be independent
of them, since these lesions are persistent and cannot therefore be regarded as the essential
condition, and true cause, of nervous disturb-
ances, which are of a transient character."
The number, and variety also of the pathological
conditions associated with the disease tend to
make one doubt their physiological value, and
when we consider still further the number of times, lesions, to all appearances identical
recur, without giving rise to the character-
istic symptoms of epilepsy, one doubts as to their value does not diminish.
The Ultimate Pathology of Angina Pectoris.

In considering this portion of the subject we derive great assistance from the more recent physiological discoveries, particularly in that portion relating to the effects of the paroxysm on the nervous mechanism of the heart.

In employing physiological facts however, in explaining pathological phenomena, great care is required: the complicated nervous mechanism of the heart, cannot after pathological conditions (often of an inscrutable nature) have been developed in it, be expected to yield results, similar to those elicited in an experimental stude, on irritation of portion of its structure. Stimulation, which in a normal condition of the whole, would yield a certain series of results, may in a pathological condition produce no effect whatever; may, may possibly give rise to a totally different set of phenomena.

The leading symptom of the disorder we are considering is pain, originating in the cardiac region. This pain is generally thought to be a neuralgia of the cardiac plexus.

Before however entering into an examination of this point, it is at least interesting, to try for a few moments to understand, what constitutes the subjective sensation known as pain. To say that it consists in a neuralgic condition of the particular set of branches involved, does not assist us; it merely adds another name which means nothing. "Nerve-ache" or "pain in a nerve", or only helps us temporarily to cover our ignorance of the matter. Rounding in dealing with this subject, adds still another name — he calls it a
"hyperesthesia"; + angina pectoris, as we have seen, he treats of, as "hyperesthesia of the cardiac plexus". Pain however, consists in something more than a mere excitation of the normal function of a sensory nerve - there is a clear distinction. If we will consider the point, between supersensitiveness, and pain - the one is an active, the other a more or less passive condition.

In a paper by Dr. Granville, in a recent number of the Lancet, (a paper which he himself admits is prematurely published, in order to secure the results of his private investigations, of the last few years,) this question is putted into, for the purpose of giving relief in neuralgia. Dr. Granville invented an instrument, which he calls a "percuteur," with which, he taps the superficial course of a painful nerve. By means of screws, this instrument can be regulated, so as to give a known number of shocks per second. The effects of tapping a healthy nerve with this instrument, resembles the sensations produced on the application of a weak electric current; if continued, muscular twitchings are excited in the part. In considering the mode of action of this instrument, in the relief of neuralgia, he came to the conclusion, that the relief experienced, was due, not to the shock of percussion, as was generally supposed, but to the establishment of a new set of vibrations, in the nerve itself. In concluding the paper, he states, that in this
opinion, it will be found, that all nerve action - normal or abnormal, will be found to be vibratile. That neuralgia, in a condition in which the normal vibrations have been replaced by abnormal, the benefit resulting from the use of his invention, lies in the fact that a new set of vibrations, are made to take the place of the abnormal series, which are causing the pain, and thus a change in the rhythm results, and the pain is relieved. He considers that these vibrations, "consist in the movements of the parts of the nerve within its sheath." Sharp, acute, pain, he compares to a high note in music, aspiring a series of short rapid vibrations, while dull pain, to low notes, in which the vibrations are longer. He has also found from experiments with his invention, that relief is more quickly brought about in dull pains, by rapid action of his instrument, than vice versa. There is no doubt that the interrupted current acts sometimes magically in cases of lumbago, and other neuralgias, such action might be accounted for on the above hypothesis; nerve action also, has been considered as something analogous to electric stimulation - especially motor action, and electricity itself (with light, heat, sound, etc.) are considered by some as varieties of motion. I have little doubt, that these investigations of Dr. Cuvier's, are a step in the right direction, in the study
of great importance to the medical profession: his paper is well worthy of a careful perusal.

But to return to the subject of the pain of angina pectoris: it is thought by some to originate in the heart; by others, in the cardiac plexus of nerves. The sensory nerves of the heart are derived chiefly from the branches of the vagus; that the sympathetic can convey painful sensations on severe stimulation, there can be no doubt, for it is now generally ascribed to be a mixed nerve deriving its sensory fibres through the communicating branches from the central nervous system. The heart, like other organs supplied by the sympathetic, is not very sensitive to pain, but under special conditions pain of a severe character may arise, particularly if the irritation be severe and continuous. This can be easily illustrated in the case of the abdominal organs: the irritation caused by the presence of food in the alimentary canal, does not give rise to a conscious impression, a reflex act only takes place, & peristalsis is the result. At times however, from some irritating cause, not only does the reflex act take place, but there is super-added to it, a series of subjective sensations, known as colic, (due to over-exertion of the muscular tissue of the bowel,) more or less severe, depending on the exciting cause. Similar sensations result in the gall ducts & ureters from the presence of concretions in those tubes.

As regards the originating cause of the pain in angina, opinions seem to be divided. The
majority however follow Hberden, & consider it due to
spasm, or spasmotic, or irregular, contractions of the
muscular tissue of the heart, due to defective cir-
crely of blood to an already weakened organ, with
over-distension of the cavities. This over-distension
according to some, is due to a rise in the blood-pres-
cure, others again consider that the vaso-motor
nerves of the heart itself are stimulated, & cause
an initial weakness, giving rise to over-distension,
which is followed by the irregular spasmodic con-
tractions, which originate the pain. Ditten 39.

Considers the immediate cause, due to obstruction
to the return of blood by the cardiac veins, from
the over-distended state of the right heart, & the
attempts made by the organ to overcome this
condition. Edindor 10. 11. disappears totally with
this view, & believes that occasionally the pain
is ischaemic, but more frequently due to injec-
tion & pressure on the constituent parts of
the cardiac pleura; he avoids giving an opinion
on the probable originating cause, only stating,
that changes in the rhythm cannot be the sole
cause, though they may produce the feeling of
constriction.

The exact originating cause cannot be decided
on in our present state of knowledge; there is
however much to be said for the view attributing
the initial step in the paroxysm to disturbances
in the regulating nervous mechanism. The
early accession of the pallor of the face, & other
parts, in due according to Dr. Morton 39. & Euden.
...powerful inhibition of the heart, due to vasomotor influences as has been generally supposed, which would require time - measures at least by seconds - to produce similar results; we shall enter into this question when inquiring into the effects of the paroxysm on the heart. I merely mention it now as a possible initial cause of the paroxysm, the pallor of the face is certainly one of the earliest symptoms.

The feeling of dread, as if something terrible was taking place, must be attributed to pain communicated through the sympathetic system of nerves. Romberg has pointed out that such sensations are characteristic of the sympathetic. As illustrative facts he draws attention to the fearful, or "overpowering sense of prostration as if the roots of life were threatened," resulting from a blow over the gastric pleurae or on the testicles. Some years ago I received a blow in the latter region, from a cricket ball, and I can testify is the fearful and insensible impressions produced in my mind, which were quite distinct from, much more difficult to tolerate, than the mere sensation of pain, which was hardly appreciable during the period the more overpowering sensation of dread continued.

The pains in the nerves of the limbs and other parts which usually accompany the paroxysms of angina pectoris, are to be
explained on the ground of the transference of impressions from the original seat, through the various connections that exist. The brachial pains through the communicating branches of the nerves forming the brachial plexus — the 5th to 8th cervical and 1st dorsal nerves — with the mid-Thoracic and inferior cervical ganglia of the sympathetic, which send branches to the cardiac plexus. The cervical pains are to be accounted for through the connections which exist between the anterior branches of the four upper cervical nerves and the superior cervical ganglion on each side. So also with the diaphragmatic pain through the connections of the phrenic nerve with the cervical sympathetic through the fourth and fifth cervical nerves.

The variations in different cases as to the positions affected by these radiating pains are, I think, regulated by the intensity of the paroxysm; if slight, the pain in the cervical region may only be affected; in more severe attacks, these sensations may pass down to the lower extremities.

The question naturally suggests itself in studying this as well as other nervous disorders — whence arises the intermittent character of the suffering? Why should it not be continuous? We have been told, that in the intervals between the paroxysms in the early stages of the disorder, the patient feels generally in his usual health, & is able to
attends to his duties, if not of a kind requiring unceasing muscular exertion; a paroxysm will suddenly seize him and pass off again, leaving him free from pain, & the other symptoms associated with it. This intermission in the phenomena is characteristic of most nervous disorders, & is a strong point in the argument of those who insist on the neurotic origin of angina pectoris. Why these intermissions should occur in nervous disorders, is a point of great difficulty to determine, & one which is impossible in our present state of knowledge to fully account for. Ironsoun in treating of the neuralgias in his clinical lectures, relates two interesting cases of malignant uterine disease, in which not only was there an intermission in the paroxysms of suffering, but a distinct periodicity occurred. In another case, related on the same page (489), a case of uterine fibroids, paroxysms of intense pain occurred daily, between noon & 2 P.M., leaving the patient exhausted, but quite free from pain, till the same hour on the following day. In his chapter on angina pectoris, in discussing this subject, a case of cerebral cancer is referred to, in which severe pains accompanied with epileptic convulsions returned every day at the same hour. In all the above cases serious pathological conditions existed involving the destruction of some or less tissue, in all of them the lesions giving rise to the symptoms of pain were constantly present, but the symptoms of—
Their presence assumed a paroxysmal, *in some a periodic type. iPhone's Chapter on "Epileptic Neuralgia" + "Simple Neuralgia" - Chap. IV p. 103-116. + Chap. XVII p. 479-512 - will well repay a careful study to those interested in this question.

The variations met with during the paroxysms, in the disturbances of the motor functions of the heart require consideration. As we have seen, in certain cases, no effect whatever is apparent in the rate of the cardiac rhythm - in others (* these form the majority), the beat is quickened, + somewhat feeble, + irregular; in a third the pulse is slow, strong + full.

The exceedingly close physiological relations between the various portions of the nervous system which regulate the heart, preclude the idea that that any one is alone affected, to the exclusion of the rest, in a paroxysmal affection like auricular stenosis. We must therefore consider the relations between them in attempting to account for the variations of the conditions present during a paroxysm in different cases.

In connection with this, we must not forget a point already alluded to, that when perhaps microscopic pathological lesions are developed in the nervous tissue, lesions which we have no means of discovering during life, we cannot expect to meet with similar results from causes which in experimental physiology give rise to a certain definite series of phenomena.
In explaining the causes of the variations in the heart-beat during a paroxysm in different cases, we must consider that the entire cardiac mechanism is involved more or less simultaenously; that is, one portion being involved must through the various, intricate, connections between the whole, react to a certain extent on the rest.

The fact, that in the majority of cases, the heart-beats are quickened or feeble, can be explained on the hypothesis of paralysis of the inhibitory fibres of the vagus. From the fact that the pain at the heart, or deadly pallor of the face, are present almost at the same moment, at the onset of the paroxysm, I am inclined to agree with Dr. Morton, that the latter is not due to vaso-motor stimulation (as is generally supposed, which would take at least a few seconds to become an accomplished fact), but to violent, or sudden inhibition of the heart, resulting probably from the intense pain. The sensation expressed by most patients, that at the beginning of the paroxysm they feel as if the heart was standing still, also lends support to this hypothesis, for such it is. No one having recorded the state of the pulse, at the very commencement of the paroxysm. Dr. Morton has shown, that at the beginning of the paroxysm in epilepsy, this inhibition of the heart takes place. By several personally attested instances, although I cannot say I saw the two diseases as related to one another.
yet the sudden pallor or face in both disorders, may I think bear a similar explanation.

From physiological investigations, we know the effect of stimulation of any sensory nerve on the inhibitory fibres of the vagus - they are stimulated reflexly into action, and inhibition of the heart in the result. We also know if the stimulation is severe the heart ceases to beat and arrested in diastole. Even if the stimulation is now continued, the heart after a time recovers, begins to pulse again, no amount of stimulation applied to the inhibitory fibres will bring about inhibition again, till a period of rest is allowed. The inhibitory fibres therefore are in a State of paralysis. The heart being fast, not being controlled by this regulating mechanism.

This is what I believe is brought about during the early portion of the paroxysm - the continued pain, has no effect on the inhibitory mechanism. It being in a State of paralysis. The heart, having recovered from the shock of intense inhibition, beats more quickly.

The question why the heart should beat more rapidly during a paroxysm with an increased blood-pressure present, can, I think be explained in a similar manner. We know from the investigations of many you Bungard, that in a normal state of the nervous mechanism, an increase of the blood-pressure leads to reflex inhibition of the
heart - or as Murray & Haldane have stated it, "the rate of the beat, is in inverse ratio to the arterial pressure." In a healthy condition therefore, increased vascular tension, will lead to stimulation of the inhibitory centre, and diminution in the rate of the cardiac pulsations. In a heart during a paroxysm of angina pectoris, however, we have seen how the inhibitory mechanism is paralysed. This being the case, what is the result of the increased blood pressure? The effect is to stimulate the intra-cardiac motor ganglia to increased action, both by increase of rapidity of the circulation through the coronary arteries, (always present when the pressure is raised) and also by increase of pressure on the aortic arch, leading to reflex irritation of the intra-cardiac motor ganglia. At the same time also, the accelerator nerves of the heart running to the sympathetic are brought into action - some authorities however object to this last factor.

The quickening of the pulse during a paroxysm may be accounted for by supposing that any of the following results are brought about in the nervous mechanism of the heart: inhibitory paralysis, stimulation of the accelerator nerves of the sympathetic, stimulation of the intra-cardiac motor ganglia. We have seen that an increase in the blood-pressure accompanied with paralysis of the inhibitory mechanism will lead to quickening of the rhythmic contractions, a fall in the blood-pressure
also leads to quickening of the heart-beat, by stimulating the accelerator nerves of the sympathetic, etc. (diarrhea)

The slowing of the pulse occasionally noticed during a paroxysm, may be accounted for by the opposite hypothesis to those we have just mentioned: viz. diminution of the inhibitory fibres of the vagus (the initial diminution at the beginning of the paroxysm not proving sufficiently strong to produce paralysis). Partial paralysis of the vitia cardiae motor ganglia, from imperfect blood-supply, an increase of the blood-pressure, the inhibitory mechanism being in action - a right inhibition resulting. To.

In dealing with this subject, Quedenburg (following the example of Landolt) has divided the disease into four types, according to the portion of the nervous mechanism supposed to be involved during the paroxysm; each of these varieties he considers may be affected in two ways - either by irritation or paralysis; they are: 1. Disturbance of the automatic system of nerves; 2. Reflexive system; 3. Sympathetic system; 4. Vaso-motor system. We shall refer to this division again in dealing with the varieties of classification of ampic patients.

As regard the mode of arrest of the heart during a paroxysm, different views have been entertained: Garrard's and others that it is possible that vaso-motor spasm of the coronary vessels may account for it.
In learning to paralyse the nitric-saccharine motor mechanism, death resulting from asphyxia.

Dr. Balfour is inclined to agree with Austin in attributing death to inhibitory paralysis, or gradual exhaustion of the force of the contractions, indicated by the condition of the pulse towards the close. As I have attempted to show, this inhibitory paralysis is a direct result of powerful stimulation of the inhibitory mechanism at an early stage of the paroxysm; so in my opinion the cases of extremely sudden death during the initiatory stage of the paroxysm — a class of cases not infrequently met with — are to be accounted for only by the belief, that intense inhibition of the heart takes place, the result of the pain, the organ being in a weakened condition. (Generally from interference with the blood-supply to its tissues) is unable to recover itself, as a more healthy heart would do, or death is the result.

A case which came under my care while a student has impressed this view strongly on my mind — the patient in recounting his sensations always dreaded the first portion of the attack, repeatedly assured me that if he had not something warm at hand, for local application, he would certainly die at the beginning of the paroxysm — he felt as if his heart had stopped beating; although I have been present during a paroxysm, I was never fortunate enough to see its commencement, or discover whether the subjective sensation
he described so frequently, has any foundation in fact—within the heart and really cease beating for a time or not. The manner of his death, however, was very sudden, and by his wife, was attributed to failure to procure his favourite local application at time—a bag of hot sand kept constantly at hand, which afforded him more relief than the inhalation of the nitrite of amyl, probably from the fact that it has not been freshly prepared.

Admitting that severe inhibition is the cause of the sudden death in these cases, it may be argued that the inhibition is the result of the increased blood-pressure, due to vaso-motor action present during the paroxysms which lead to reflex stimulation of the inhibitory centre. (known.) My objections to this view are: First—That pain is a more powerful stimulant to the inhibitory mechanism; and second, that vaso-motor changes are gradually brought about, as an all muscular movements resulting from sympathetic action. They do not take place in an instant of time, and the cardiac inhibition produced by this cause would be more gradual and only provided for by the other portions of the nervous mechanism, before it had become completely established. In addition to the support of my view, I can bring in instances of sudden death from reflex powerful inhibition from blows on the hypochondriac affecting the solar plexus, & we have seen
that the sensory fibres of the sympathetic are
involved in the pain of the angina pectoris.

The placid and relaxed condition in which
the heart is found after death during a paroxysm
does not help us to any conclusion as to
the manner in which it has ceased to beat.
A similar condition of the organ might be
expected to be found, if death results from
inhibition, or from asystole due to paralysis
of the motor ganglia.

The respiratory symptoms met with during
a paroxysm require a little attention. The
breathing is generally quickened and shallow
and may even be arrested during the scene of
the suffering. These symptoms of dyspnoea
are in most cases due as I have already
pointed out to fear of aggravating the suffering
by the act of moving the respiratory muscles.

The feeling of "Want of breath,"
as experienced by many during the paroxysm,
is in some cases undoubtedly due to changes
taking place in the lobes of the respiratory centres,
characterised by a diminution of the respiratory
activity. Cases in which these changes were watched during a paroxysm
have been reported by Dr. Denio 35 and
Dr. Lockhart Clarke. In these cases in
which a marked symptom is an increase
in the systemic vascular tension, more
or less of the stimulation may result from
the backward pressure.
The nervous symptoms occasionally associated with the paroxysms of an aura or pectoris - the vertigo, spasm, convulsion, coma &c. - as strongly insisted on by Jussieu as indicating its close relation with epilepsy - deserve some notice. They can, I believe, be referred to causes interfering with the cerebral circulation tending to the production of anaemia, or are purely functional disturbances, resulting from that condition of the nervous centres. The experimental researches of Brown-Séquard, Sir Ashton Cooper, Marshall Hall, &c. &c., have shown, that when the brain is rapidly deprived of blood, by compression of the vessels, or haemorrhage, epileptic symptoms with a comatose condition are quickly induced, passing off as quickly when the current is resumed. Professor H. Nottmack, of Jena, in treating of the ultimate pathology of epilepsy in Zinsser's Encyclopædia, draws special attention to this subject & to the experiments of Kussmaul and Remak on the same lines as those alluded to.

In connection with this part of the subject, I cannot refrain from referring to the extraordinarily comprehensive, & interesting lectures of Dr. Walter Scott's, as present being reported in the "British Medical Journal", on "The Influence of the Circulation on the Nervous System." In these lectures he contends, that there is no sense...
disorder as congestion of the brain, and that most
of the symptoms described under this disorder
in the first book, are really the result of an
anemic condition of the organ. For the
various symptoms arising from an anemic
condition of the brain, I should refer to the
number of the journal for April 16, 1841.
for more proper. Before concluding this series
of lectures, let us take up the question we have
just considered.
Classification of Angina Pectoris.

In the classification of this disease, the points generally selected for a basis, have been the variations in the effects produced on the nervous mechanisms of the heart and vessels by a paroxysm.

Sandoz has analyzed these symptoms from a physiological point of view, and divided the disease into four classes:

I. Disturbance of the excito-motor nerves.
II. Disturbance of the cardiac branches of the vasa.
III. Reflex excitement from the abdominal organs.
IV. Disturbance of the vaso-motor system.

Eulenberg, as I have already mentioned, follows Sandoz's classification in part, while admitting that the different types are apt more or less to be combined, or even into one another in the same case. I cannot do better than condense, or tabulate, his classification, from his paper on anginapectoris in Zimmer's Cyclopaedia, Vol. XIX., p. 589.

Each of his classes may assume two forms, that is, they may either be affected by irritation or paralysis, giving rise to opposite results:

I. Excito-motor cardiac, ganglionic angina pectoris — from direct lesion of the automatic excito-motor ganglia.
   1. Irritation — giving rise to increased rapidity of the pulse.
   2. Paralysis — giving rise to retardation of the pulse.
II. Regulator angina pectoris - from lesions of the branches of the vagus.
   1. Direct neurotic of vagus - may assume either of two forms.
      a. Irresistible urge, t' increase of force of heart-beats - feel hard pulse. Sometimes temporary arrest of heart.
   1. Paralysis - acceleration of pulse.
   2. Vagotropic neurosis of vagus - originating in disease of abdominal organs, with symptoms of irritation of vagus.

III. Ectopic motor sympathetic angina pectoris - from lesions of the accelerator nerves of the heart, which run with the sympathetic. Symptoms as in first form.

IV. Vaso-motor angina pectoris - from affection of the vaso-motor nerves which run with the sympathetic. (Shock.) May assume two forms:
   1. Irritation - contraction of vessels, increased pressure, with normal, or but slightly increased pulse, symptoms of arterial anaemia, paleness + coldness of the skin.
   2. Paralysis - with the opposite set of symptoms.

The simplest, and at the same time most comprehensive, method of classifying this disease, is adopted by Professor Espiner-Stewart in his lectures on medicine: He divides the disease...
said these classes as follows:

I. Pure neuralgia of the nerves of the heart, and its neighborhood.

II. Inflammatory changes affecting the nerve ganglia connected with the aorta, and perhaps the heart itself.

III. A condition due to stagnation of the blood arteries, and consequent over-distension of the chambers of the left side of the heart.

Class I. includes the cases I have treated of under Dr. Walsh's name of "pseudo-angina," and also the case of "angina sine dolore," in which, although there is no distinct pain present, yet a dull aching, or numbness, is experienced in the cardiac region.

Class II includes such cases as are described by Dancer and Trenchard.