The nervous affections of the heart : being the Morison Lectures delivered before the Royal College of Physicians of Edinburgh in 1902 and 1903 / by George Alexander Gibson.

THE NERVOUS AFFECTIONS OF THE HEART

With the author's compliments.

THE NERVOUS AFFECTIONS OF THE HEART

BEING THE MORISON LECTURES DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH IN 1902 AND 1903

BY

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EDINBURGH AND LONDON

YOUNG J. PENTLAND

1904

TO

SIR WILLIAM TURNER, K.C.B.

M.B., D.Sc., LL.D., D.C.L., F.R.S.S. Lond. and Edin.,

PRINCIPAL OF THE UNIVERSITY OF EDINBURGH,
PRESIDENT OF THE GENERAL COUNCIL OF MEDICAL EDUCATION AND REGISTRATION,

IN GRATEFUL ACKNOWLEDGMENT OF UNVARIED KINDNESS
DURING MANY YEARS
THESE LECTURES ARE DEDICATED BY

THE AUTHOR

PREFACE

THE following Lectures were delivered before the Royal College of Physicians of Edinburgh during the years 1902 and 1903. They were published in *The Edinburgh Medical Journal* during these years. They are now reprinted with very few alterations, the only changes being some verbal modifications intended to render the perusal of the work easier, and a few additions which should make its meaning clearer.

It is a pleasant duty to return thanks to my friend Dr. Alexander Morison, for suggesting my appointment to the post of Morison Lecturer, and to my colleagues in the Council of the Royal College of Physicians, for confirming this nomination. Fully realising the eminence of those who have preceded me in this office, it was not accepted without misgiving, and my appearance in this position has only been rendered possible by the recent extension of the field covered by the Lectureship.

3 DRUMSHEUGH GARDENS,
EDINBURGH, *9th July* 1904.

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SECTION I.—SENSORY DISTURBANCES.

Lecture I.—Clinical.

Founded in 1864 by Sir Alexander Morison of Bankhead, a former President of the College, this Lectureship was originally intended to meet a want in the medical curriculum by supplying a series of lectures upon insanity. With the development of medical education during the final quarter of last century, the subject of insanity obtained a recognised place in almost every medical school, and the proposal therefore commended itself to the authorities of the College that the scope of the Morison Lectures might be widened with advantage. During the last twenty years, accordingly, many interesting courses of lectures have been delivered upon other branches of medicine than insanity, the only stipulation now being that their subject shall be in some way connected with the Nervous System.

Taking advantage of these recent provisions, it is my intention to devote the present course of lectures to the Nervous Affections of the Circulation. No words of mine are needed to prove the profound importance of the subject thus selected for these lectures. Every part of the circulation is so closely bound up with the nervous system, that reciprocal actions are of constant occurrence. Responding to every stimulus and suffering from every disturbance brought to it by the nervous mechanism, the circulation is subject to a great variety of disorders impressed upon it from without. We may in truth say that the heart and vessels, in circumstances of nervous storm and stress, must be regarded as if surrounded by the fabled robe poisoned by the blood of Nessus.

And, conversely, when the organs of the circulation have undergone morbid changes from causes not primarily arising within the nervous system, they induce disturbances, whether dynamic or organic, in the nervous mechanism. In these different ways, action and reaction are continuously in operation, and no attempt to grasp the affections of the circulation can hope to meet with success in which the influence of the nervous system is not allowed full weight.

The first three lectures will be occupied solely by sensory disorders; motor disturbances will be left for consideration during the three following these, and the lecture of to-day will be entirely given to the clinical aspects of sensory affections.

Before entering upon the discussion of any of these branches of the subject, you will perhaps allow me very briefly to bring before you the history of our acquaintance with the affections to which we are about to devote ourselves. A short account of the various steps by which our present knowledge has been gained will certainly render it easier to grasp the full significance of the facts which will come before us. In this short historical sketch, reference will only be made to work of real utility, and the chief import of each author will, as far as possible, be brought forward.

Amongst the "Hippocratic Treatises"[1] there are apparently references to painful affections of the heart. There is, nevertheless, some doubt, not merely in regard to the exact meaning of the observations, but even in respect of their author, seeing that by Daremberg[2] the Coan treatise is accepted as the work of Hippocrates, while by Adams[3] it is regarded as of a date anterior to his epoch. Since the earlier work of Lancisi,[4] it has been generally believed that the passage referred to really deals with cardiac pain, but this view is not universal. The philosopher Seneca,[5] in his fifty-fifth letter, gives a

[1] "Coacæ Prenotiones," cclxxxvi. Amstelœdami, 1660, p. 226.

[2] "Œuvres choises d'Hippocrate," 2e Édit., Paris, 1855, p. 179.

[3] "The Genuine Works of Hippocrates," *Syd. Soc. Translation*, London, 1849, vol. i. p. 64.

[4] "De subitaneis mortibus," Romæ, 1709, p. 100.

[5] "Opera," Basileæ, 1529, p. 126. The description given by the philosopher is as follows:—"Omnia corporis aut incommoda aut pericula per me transierunt: nullum mihi videtur molestius. Quid ni? aliud enim quidquid est, ægrotare est: Hoc est animam agere.—Ego vero et in ipsa suffocatione non disii cogitationibus lætis ac fortibus acquiescere."

graphic description of some symptoms which appear undoubtedly to be those of angina pectoris, although it must be admitted that subsequent authors have not been at one upon the subject.

In the quaint old history written by Mézeray,[1] there is a description, amongst the remarkable events of 1599, of the sudden death of Gaspard de Schomberg after severe dyspnœa. This fact has often been cited as an instance of angina pectoris, in which there is obviously a misconception. It is of interest to observe that Huchard[2] states that the son of Gaspard de Schomberg died from heart failure preceded by pain in the chest. From the account of the death of Mr Hyde, the father of the first Lord Clarendon, which occurred at Michaelmas 1632,[3] it is obvious that he must have perished of the same affection. The account of Mr Hyde's illness and death is well worth lingering over. He evidently suffered from chronic interstitial nephritis, and laboured under attacks of agonising pain in the left arm, which were accompanied by deathlike pallor. His son goes on to mention his having been in the habit of saying—"that He had passed the Pangs of Death, and He should die in one of those fits." How like the description of Seneca! He adds further, that "He had the Image of Death so constantly before him in those continual Torments, that for many Years before his Death, He always parted with his Son, as to see him no more." In these remarks there may be seen the same gloomy anticipations which were afterwards, in two different epochs, experienced by John Hunter[4] and Charles Sumner.[5] In the work of Lancisi already referred to, the symptoms of angina pectoris are seen to have been well understood by him.[6] His contemporary Dionis[7] is perfectly clear in his account of similar

[1] "Abrégé chronologique ou extrait de l'histoire de France," Paris, 1676, tome vii. p. 358.

[2] "Traité clinique des maladies du cœur et des vaisseaux," Paris, 1893, 2e Édit., p. 590.

[3] "Life of Edward, Earl of Clarendon, written by himself," Oxford, 1759, p. 9.

[4] Everard Home, in "A Treatise on the Blood, by the late John Hunter," London, 1794, p. 45.

[5] Taber Johnston, *Boston Med. and Surg. Journ.*, 1874, vol. xci. p. 365.

[6] *Op. cit.*, p. 89.

[7] "Dissertation sur la mort subite," Paris, 1709. This work has not been accessible to me; it is fully quoted by Gélineau, "Traité de l'Angine de poitrine," Paris, 1887, p. 9.

symptoms as well as in his narrative of two cases, and Hoffmann shortly afterards placed on record an undoubted case of angina pectoris.[1] This brings us down to the well-known instance of the patient described by Morgagni,[2] who died at the age of 42, in the year 1707. The characteristic distribution of cardiac pain, the sudden termination of life during a paroxysm, and the discovery of aortic lesions at the subsequent post-mortem examination, render this case the first serious contribution to the subject. Time will not permit me to dwell upon the claim of modern French authors on behalf of Rougnon[3] as the original observer of angina pectoris. His work has been very fully analysed by me elsewhere.[4] It is perfectly true that he describes pain in the anterior part of the chest followed by sudden death, but it must be remembered that Rougnon attributed the whole condition to ossification of the costal cartilages, and thereby shows such a complete misconception of the entire condition as to render his observations valueless when compared with the previous work of Morgagni. The proposal, therefore, of Huchard,[5] to associate the name of Rougnon with the disease is one calculated to provoke a smile. Far otherwise is it with the great man who inaugurated the study of the affection, and whose name will probably for all time be associated with cardiac pain, notwithstanding that, as we have seen, he was not by any means the first to describe angina pectoris. In his first communication, Heberden[6] published the investigations of more than twenty years, dealing with the cases of something like twenty patients, and containing an excellent description of most of the symptoms now known to us. In his "Commentaries,"[7] published a year after his death, he states that he had seen not less than 100 cases of the affection, of which there occurred three in women and one in a boy twelve years old; all the others were in men of above or nearly fifty. Changes in the coronary arteries associated with præcordial pain, although undoubtedly first published by Fother-

[1] "Opera Omnia," Genevæ, 1753, p. 248.
[2] "De sedibus et causis morborum," Venetiis, 1762, tomus i. p. 283.
[3] "Lettre à M. Lorry," Besançon, 1768, p. 1.
[4] "Diseases of the Heart and Aorta," Edin. and Lond., 1898, p. 758.
[5] *Op. cit.*, p. 593.
[6] *Med. Trans. Roy. Coll. Phys.*, London, 1772, vol. ii., p. 59.
[7] "Commentarii de Morborum Historia et Curatione," Londini, 1802, p. 308.

gill,[1] appear to have been previously noticed by Jenner, who addressed a letter to Heberden in which they were described. Jenner did not publish his observations on account of his solicitous consideration for the feelings of John Hunter, who had about that time begun to manifest anginous symptoms; they were, however, discussed by a private medical society in Gloucestershire, and afterwards embodied in a communication addressed to Parry, which was given by him to the world in his celebrated work.[2] The work of Parry is interesting, not only by reason of its description of Jenner's views, but also on account of his own opinion, that cardiac pain may frequently be the result of stress and strain.

Allan Burns[3] opened an entirely new chapter by the enunciation of the fact, now thoroughly recognised, that interference with the blood supply of the heart, as of any other part, can produce severe pain. He is therefore entitled to our respect as having forestalled the later speculations of Charcot[4] and Potain.[5] Laennec[6] may be referred to in passing as having expressed the opinion that angina pectoris should be regarded as a form of neuralgia. With the interesting contribution of Forbes[7] begins the acknowledgment of two main varieties of cardiac pain, one being organic in origin and serious in import, the other of functional nature and hopeful prognosis. The eloquent lectures of Latham[8] cannot be passed over in silence, for, although they do not add anything conspicuously new to the subject, they give a most luminous account of the state of knowledge obtaining at the date of their delivery, while the narrative of the case of Thomas Arnold is most interesting. With Cahen[9] and Eichwald[10] there began to be recognised the possibility that cardiac pain might be

[1] *Med. Obs. Soc. Phys.*, London, 1776, vol. v. p. 233 and p. 252.

[2] "An Inquiry into the Symptoms and Causes of the Syncope Anginosa," London, 1799, p. 69.

[3] "Observations on some of the most frequent and important Diseases of the Heart," Edinburgh, 1809, p. 138.

[4] *Gaz. méd. de Paris*, 1859, p. 283.

[5] "Dict. ency. d. sc. med.," Paris 1866, Sér. i. tome iv. p. 347.

[6] "Diseases of the Chest," *Forbes' Translation*, London, 1834, p. 648.

[7] "Cyclopædia of Practical Medicine," London, 1833, p. 87.

[8] "Diseases of the Heart," London, 1846, vol. ii. p. 359.

[9] *Arch gén. de méd.*, Paris, 1863, pp. 428, 561, 696.

[10] *Würzb. med. Ztschr.*, 1863, S. 249.

produced by the struggles of an over-taxed organ to surmount the obstacle producing stress. The facts discovered by Lancereaux[1] led to the recognition that the nervous connections of the heart might be seriously affected. Although in all his cases there were aortic lesions, yet it must be acknowledged that the presence of neuritis is an interesting fact. The observations of Landois[2] on the reflex origin of anginous pain, and of Nothnagel[3] on vaso-motor disturbances as its cause, are of deep importance, while Brunton's[4] contribution regarding the increase of blood pressure during the paroxysm, in some cases at least of the affection, must be allowed a position of much prominence in any historical sketch. Gairdner's masterly article[5] upon the subject is not only replete with suggestive facts, but is also characterised by interesting speculation. In any work dealing with this subject, it will for all time deserve and obtain close study. To one matter attention may at present be directed, *i.e.* his suggestion of the term *angina sine dolore* for many cases of cardiac failure ending in sudden death without pain. The remarkable contributions of Huchard, contained in numerous papers, mostly collected and summarised in his great work on "Diseases of the Heart,"[6] are characterised by a skilful analysis of clinical appearances and of their pathological explanation. One of the most valuable additions to our knowledge was made by the observations of Mackenzie[7] on the increased sensitiveness of the skin over the areas of pain, a subject worked at in great detail also by Head.[8] To both of these authors reference will be more fully made in the next lecture, as their explanations of the cause and nature of the subjective and objective sensory disturbances require the most careful study. In both of Balfour's works[9] upon cardiac affections, there are to be found

[1] *Gaz. méd. de Paris*, 1864, p. 432.

[2] *Cor.-Bl. d. deutsch. Gesellsch. f. Psychiat.*, 1866, S. 2.

[3] *Deutsches Arch. f. klin. Med.*, Leipzig, 1867, S. 309.

[4] *Lancet*, London, 1867, vol. ii. p. 97.

[5] Reynold's "System of Medicine," London, 1877, vol. iv. p. 535.

[6] *Op. cit.*, p. 513.

[7] *Med. Chron.*, Manchester, 1892, vol. xvi. p. 293, and *Lancet*, London, 1895, vol. i. p. 16.

[8] *Brain*, London, 1893, vol. xvi. p. 1, and 1894, vol. xvii. p. 339.

[9] "Clinical Lectures on Diseases of the Heart," London, 1876, p. 275, and "Senile Heart," London, 1894, p. 114.

numerous valuable observations on angina pectoris. It is a source of genuine satisfaction to find myself entirely at one with him in declining to accept any separation of the affection into real and false. The arguments which he adduces in favour of his conclusions are unanswerable. To Morison's interesting contribution[1] we owe a great deal. Although it has been believed by many that the blood pressure in angina pectoris is often considerably reduced during the spasm, yet no one, so far as my information goes, had been able absolutely to demonstrate that the suspicion was correct. Morison, however, by a careful investigation, has been enabled to prove the fact. To the lectures of Osler[2] upon this subject it is a pleasure to refer, although this feeling would have been greatly enhanced had he not unfortunately given his adhesion to the separation of angina pectoris into the two varieties of true and false.[3]

In the consideration of the different features of sensory disturbances, it will certainly be the most convenient, if not, indeed, the only possible course to pursue the analytic method. The different varieties of sensory cardiac disorders are not separated from each other by hard and fast lines, but pass one into another by insensible gradations. No two cases, moreover, ever present exactly the same group of symptoms. It therefore seems advisable for me to invite your attention to each different symptom in turn. My intention is not to attempt a full description of the whole complex of symptoms, but to analyse each, and attempt later to explain it.

We shall, in the first place, take up the subjective disturbances, which may be said to form the foreground of the clinical picture. Of such symptoms vague uneasiness is the most common; it is, indeed, quite frequently met with. It is often nothing more than an indefinite sense of discomfort about the chest, with no distinct localisation and no particular characteristics. Also of frequent occurrence are sensations of numbness or deadness about the thorax and upper extremity, extremely vague in distribution, and far from definite in nature. Somewhat more distinct are feelings

[1] *Edin. Hosp. Rep.*, 1895, vol. iii. p. 250.

[2] "Angina Pectoris and Allied Conditions," Edin. and Lond., 1897, p. 1.

[3] It need not be pointed out that a division into *organic* and *functional* is not the same as into *true* and *false*.

of cold or of heat, which patients also occasionally complain of. One of the most remarkable features is a sense of oppression. This particular symptom is commonly noticed, but it was never more vividly described than by Matthew Arnold, who, in one of his letters,[1] refers to "the sense of having a mountain on my chest." Closely allied to this variety of subjective disturbance is a feeling of constriction or tightness of the chest, which has been graphically portrayed by many sufferers. These descriptions are

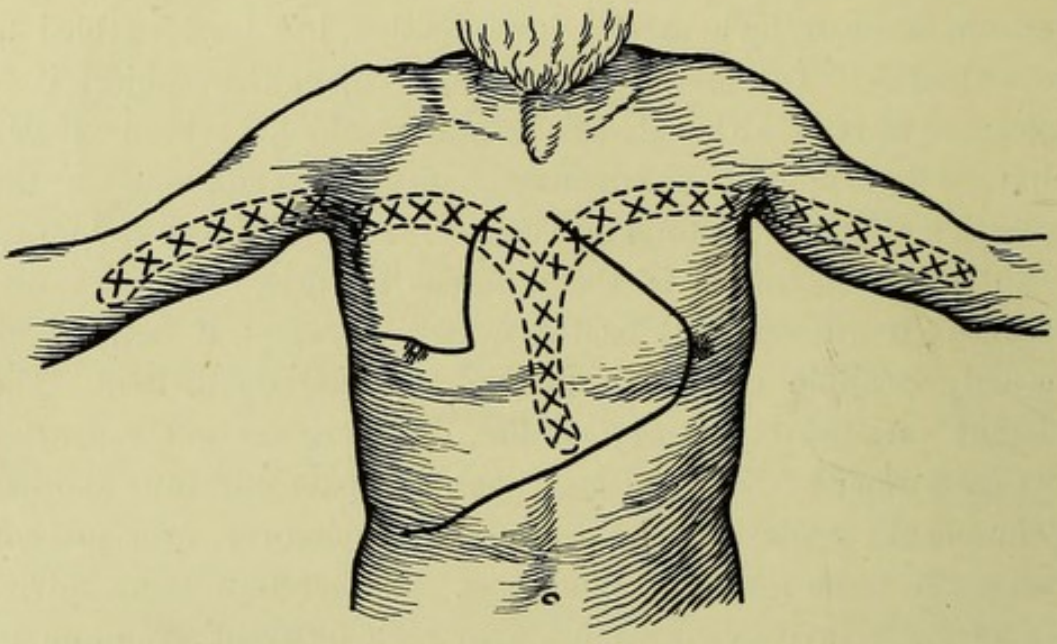


FIG. 1.—Man, æt. 60, suffered from a feeling of constriction, with some paroxysms of pain beginning at the xiphoid cartilage, passing upwards and then outwards as far as the elbows. There was no very definite area of tenderness, but sensibility was exalted over the area marked by the crosses. The patient had arterial sclerosis, but no aortic valve disease, and the cause of the angina was obviously coronary disease.

dramatically summed up by Balfour,[2] as being "a feeling as if a mailed hand grasped the chest in the cardiac area and squirted through its fingers flashes of excruciating agony." This utterance vividly brings forward the close association between the sensation of constriction and the feeling of pain. The pain indeed may surpass any other of which we have knowledge, for those who have experienced the most severe agony from disease elsewhere, and who have also passed through the anguish of angina pectoris, state that there is no comparison between them.

[1] "Letters of Matthew Arnold," London, 1895, vol. ii. p. 278.
[2] "Senile Heart," 1894, p. 124.

The distribution of the pain varies very widely. It is not infrequently confined entirely to the upper part of the chest, within the distribution of the first six thoracic segments of the cord. Its area is often confined even within the præcordia. It is in other instances found at a little higher level, and to right or left of the cardiac region, more commonly the latter. But it manifests a well-known tendency to radiate in certain definite directions. Patients frequently complain of pain at one, or other,

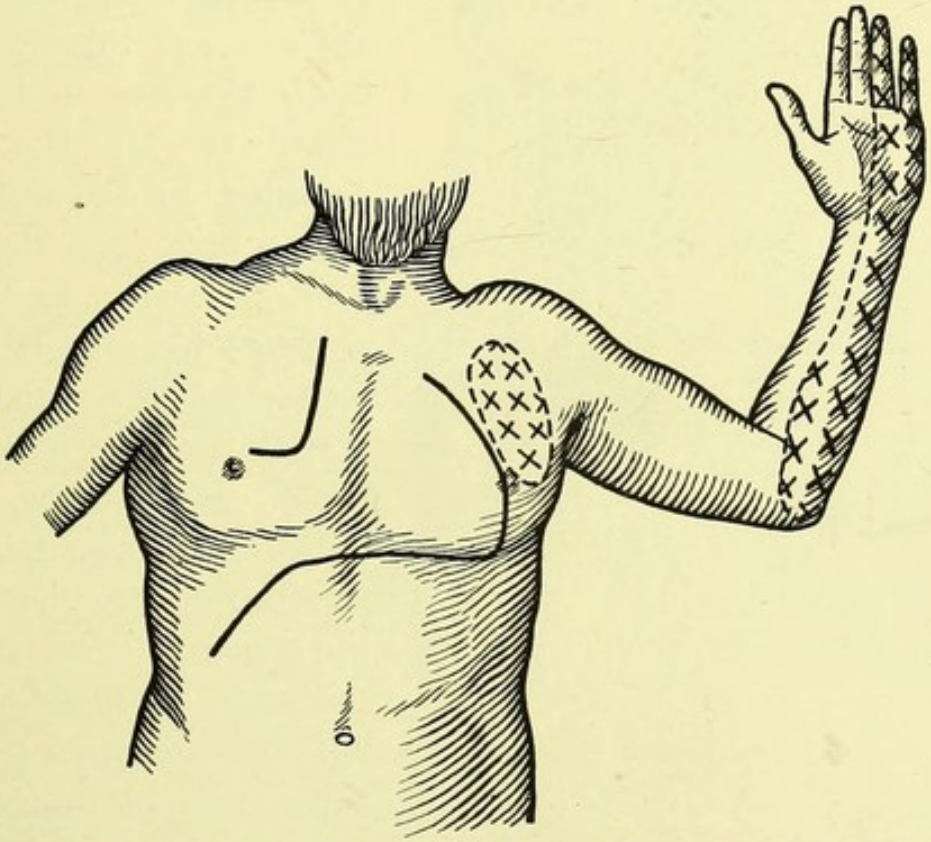


FIG. 2.—Man, æt. 46, with paroxysms of angina, and constant tenderness over the areas marked by crosses. There was arterial degeneration, but no aortic valve lesion, and the diagnosis was coronary disease. Some years later he developed thoracic aneurysm.

or both, of the angles of the scapulæ (see Fig. 6). In other cases it may occupy one small spot at, or close to, the vertebral column (see Fig. 8), but more commonly the pain shoots upwards to one or other of the shoulders, particularly the left. In many cases the painful sensations are experienced in the upper extremities, especially along the ulnar aspect of the arm and forearm, while in a smaller proportion the sensory disturbances may even reach to the extreme limits of the lower extremities. As the pain occa-

sionally shoots towards the inguinal region, and may seem to reach the scrotum, a suspicion of renal calculus has sometimes been entertained. Painful sensations are at times felt in the neck and head, as Head has fully described, showing the implication not only of the upper cervical, but also of the cranial nerves. Occasionally, as was indeed observed by Heberden, the pain is only experienced in the arm, and this was certainly the case in the instance of Mr Hyde, as described in the autobiography of his son, the first Lord Clarendon, to which reference has already been made. Sometimes the pain is felt in the epigastric region, and has at first caused the presumption of an attack of gastralgia. Not infrequently, when this is the case, a sensation of "globus," analogous to that experienced in hysteria, is complained of. A few pictures (see Figs. 1-10), taken from patients suffering from angina pectoris, may be shown at this point in order to illustrate the distribution of painful affections.

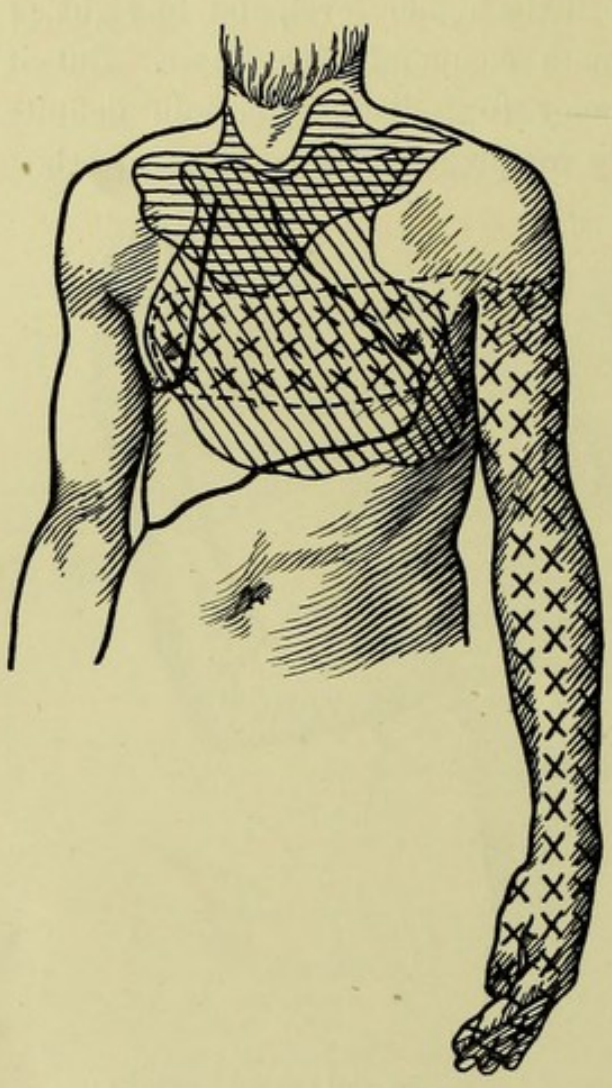


FIG. 3.—Man, æt. 37, suffering from severe paroxysms of angina pectoris and considerable tenderness. There was aortic valve disease. The area of audition of the systolic murmur is shaded horizontally; that of the diastolic murmur is diagonally shaded; the painful and tender areas are marked by crosses.

Over and above these disturbances, which fall under the definition of painful, are a few attendants of a somewhat different type. The sensation of faintness is often present, and may be only a feeling of sinking, or it may actually amount to a consciousness of the nearness of death. The first notice of such a

sensation was published by Heberden,[1] and is contained in a letter addressed to him by a medical man who wrote a description of his own case under the pseudonym "Unknown." In this account he distinguished between the painful sensation and the prescience of death. It is a matter of much interest that, anticipating sudden death, he gave Heberden his sanction to examine his body, which was done by John Hunter within three weeks of the date of the letter.

This sense of impending death dwarfs all other symptoms by its overwhelming character and stupendous importance. It is a conviction that the last enemy is near, and is, as a rule, free from craven fear to meet him. As a matter of fact, the state of mind in many instances might almost be defined as a condition of animated expectancy. It was well phrased by an old military man of my acquaintance; asked by a mutual medical friend, when the end was evidently near, what he was feeling, he tersely replied, "Intense curiosity." There can be little wonder that when fearful anguish has rendered further suffering unendurable, any prospect of release must seem welcome, for, as the pagan poet says:[2]

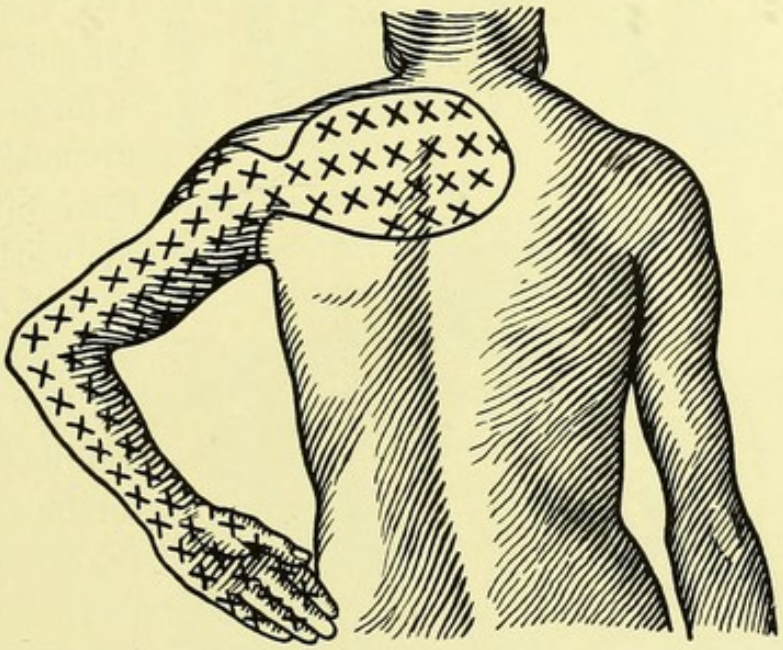
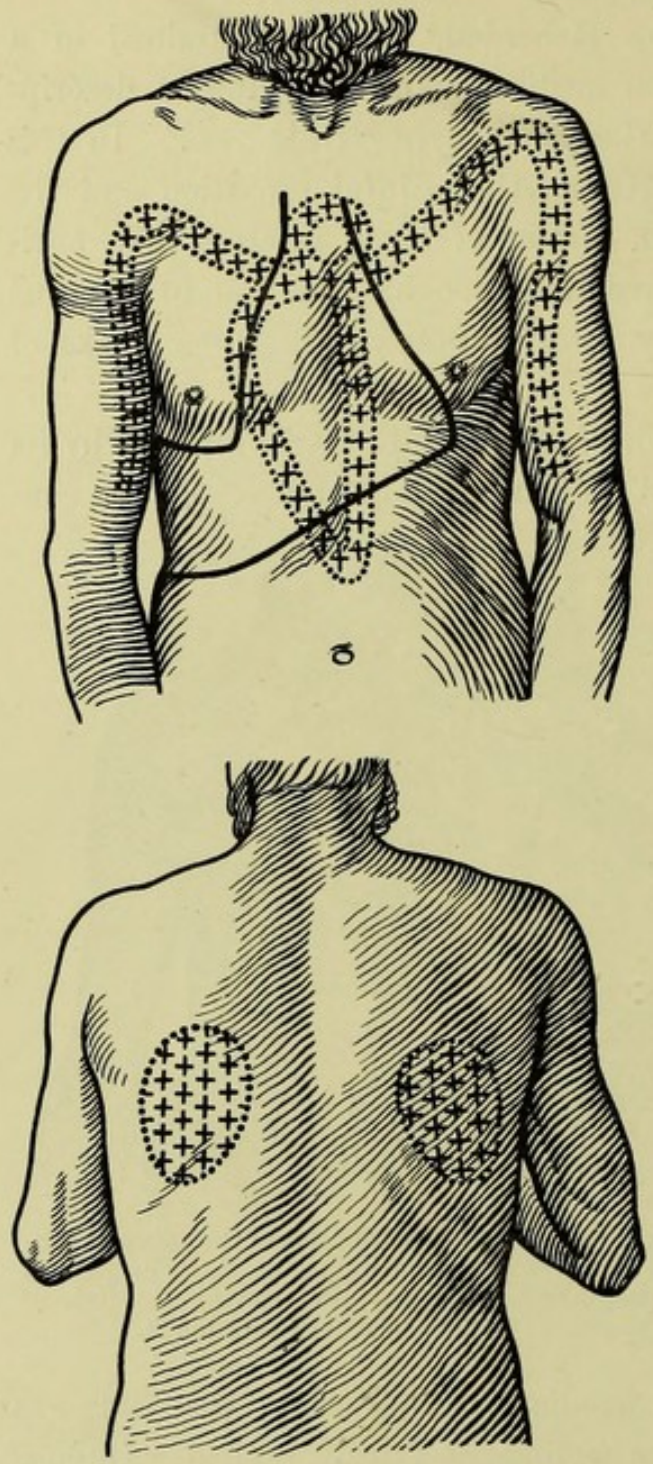


FIG. 4.—Shows the posterior distribution of the pain and tenderness in the case pictured in Fig. 3. The patient died instantaneously the day after leaving hospital, in which he seemed to have improved very much.

"Rebus in angustis facile est contemnere vitam."

Sometimes after a dreadful paroxysm of pain and its concomitants

[1] *Med. Trans. Roy. Coll. Phys.*, London, 1785, vol. iii. p. 1.
[2] Martial, "Epigrammata," xi. 56. 15.



FIGS. 5 and 6.—Man, æt. 54, suffering from aortic obstruction and incompetence. The paroxysms of pain always followed the curious distribution shown in the picture, beginning with the circle, passing down the sternum, upwards again, and radiating outwards to the elbows, and backwards to the shoulder blades. The tenderness was most difficult to delimit. The patient died very suddenly soon after leaving hospital.

has come to an end, the victim, even when the hand of the "Fell Sergeant" is upon him, may be inclined to say with Agag,[1] "Surely the bitterness of death is past." The contemplation of the fate of such sufferers must in truth render it undoubted that to them the last visitor must come in the guise depicted by Dürer in his picture, "Der Tod als Freund." This indeed has been often expressed by the victims of the malady, such as Mr Hyde and Mr Sumner.

But a sudden termination may take place in the total absence of the dreadful agony of severe cardiac pain. Without any suffering—indeed, with no subjective disturbance of any kind — death may occur. This is one form of the *angina sine dolore* of Gairdner. To pass away under such conditions may well be accounted worthy of the ancient adage, "Sic sine morte mori."[2]

It can assuredly be no matter for surprise that

[1] 1 Samuel xv. 32.

[2] The origin of this epigram has never come under my notice. It is quoted by Smollett in his "Don Quixote," Glasgow, 1803, vol. iv. p. 311.

those who are in the toils of an affection liable to assume such tremendous proportions should have many other accompaniments to their sufferings. Several cerebral disturbances have a tendency to make their appearance. Distressing giddiness is by no means rare as a concomitant of anginous seizures, while dimness of sight, dulness of hearing, or even visual and auditory hallucinations may occur. Instead of the mental anguish—*angor animi*—a more merciful visitor may come to the patient in the guise of diminished consciousness, or the mental powers may be disturbed by delusions. It is probable that this group of symptoms has its origin in a failure of nutrition of the brain, and that, although associated with the seizure, it is not closely connected with it.

The objective features of sensory disturbances furnish most interesting opportunities for investigation. It might very naturally be expected that this aspect of the field of inquiry, being free from the uncertainties of subjective idiosyncrasy, should be unattended by the dubieties which beset the observer in trying to assess the value of symptoms which are known to the patient alone. But on account of the variable characters of the clinical picture in different instances of the disease, the analysis of objective symptoms is not entirely devoid of difficulty; it has to be remembered also that out of many attacks during the lifetime of a patient, but few may be observed by the medical attendant; a natural instinct of humanity, moreover, will always lead the latter to expend his energies upon an attempt to relieve, rather than to investigate, the symptoms.

The aspect presented by the patient during a paroxysm varies within wide limits. The attitude may be that which has for long been held up in our schools as the type—the body fixed in one position, whether standing or sitting, and slightly bent forward in an attempt to relieve the feeling of oppression. But to any one who has been called upon to witness many instances of this affection, it is well known that such is far from being the commonest attitude. Much more often there is a greater degree of action in the scene. The sufferer not infrequently assumes the position of a man who, by straining every muscle, attempts to shake off an incubus, and the attitude may even resemble that of the central figure in the celebrated group of Laocoon and his two sons. Instead

of this, the patient may pace the floor in the effort to find relief; he may rock backwards and forwards or from side to side while in a sitting posture; or he may seek to assuage his torments by assuming various forms of recumbent posture. There is, in short, no characteristic attitude in the affection.

It is even so as regards the expression of the face. Although in almost every instance the lineaments reflect a painful condition, yet the varieties of countenance presented by patients are manifold. In one case there may be the expression of fearful anticipation; in

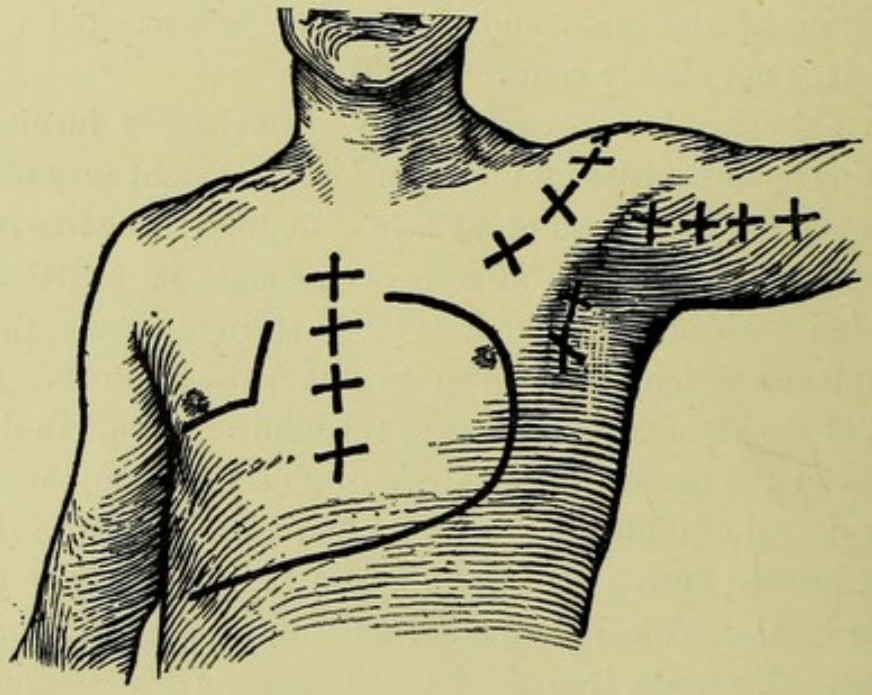


FIG. 7.—Man, æt. 32, suffering from paroxysmal pains and tenderness over the area marked by the crosses, always brought on or increased by exertion. The symptoms were due to aortic disease.

another of resigned acquiescence in whatever the future may have in store.

The tint of the skin is far from uniform. In a considerable proportion of cases of this affection there is a deadly pallor, but, speaking simply from my own experience, it is quite common to find the surface suffused by a bright flush, while in some of the most serious examples there is profound cyanosis. Its moisture is also of variable quantity; at times the skin is absolutely dry, and again, in other instances, it is covered by abundant perspiration. When referring to the objective nervous symptoms, attention will

be directed to certain trophic changes which the skin sometimes undergoes.

Varying digestive troubles may beset the patient. Closely associated as many attacks of angina pectoris are with gastric distension, it is not difficult to understand why hiccough and eructation should take place. A sensation analogous to the globus of hysteria has been mentioned under the purely subjective disorders. Intestinal flatulence rarely assumes noteworthy proportions, yet it at times attains a magnitude worthy of the fine old phrase meteorism.

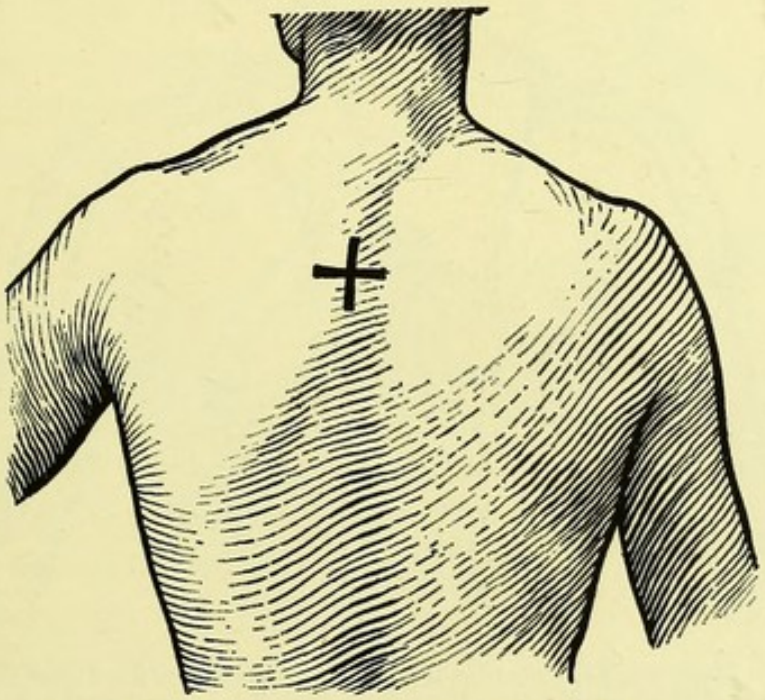


FIG. 8.—Same patient as represented in Fig. 7, to show the small area of pain and tenderness behind.

The condition of the respiration has long been known to be altered during paroxysms of the affection. Sometimes the patient may seem to forget to breathe, and, as in the case of John Hunter,[1] to breathe only by an effort of the will. Again, breathlessness may be prominent, and hurried breathing may be constantly present. Sometimes the patient seems almost as if afraid to breathe in case of increasing the sufferings, but it is a constant observation that, when a long breath is drawn in, it seems to afford a certain amount of relief. The voice is sometimes feeble, or

[1] Everard Home, *loc. cit.*

aphonia may be one of the striking symptoms, and this seems to me to be most frequently the case in those instances presenting such symptoms as hiccough and dysphagia.

The heart-beat and pulse-wave have formed subjects of controversy for a long time, and there can be no doubt that the essential cause of the differences of opinion which have occurred is to be sought in the fact that no two instances of the disease present exactly the same features with regard to the circulation. The pulse sometimes exhibits a very considerable increase of

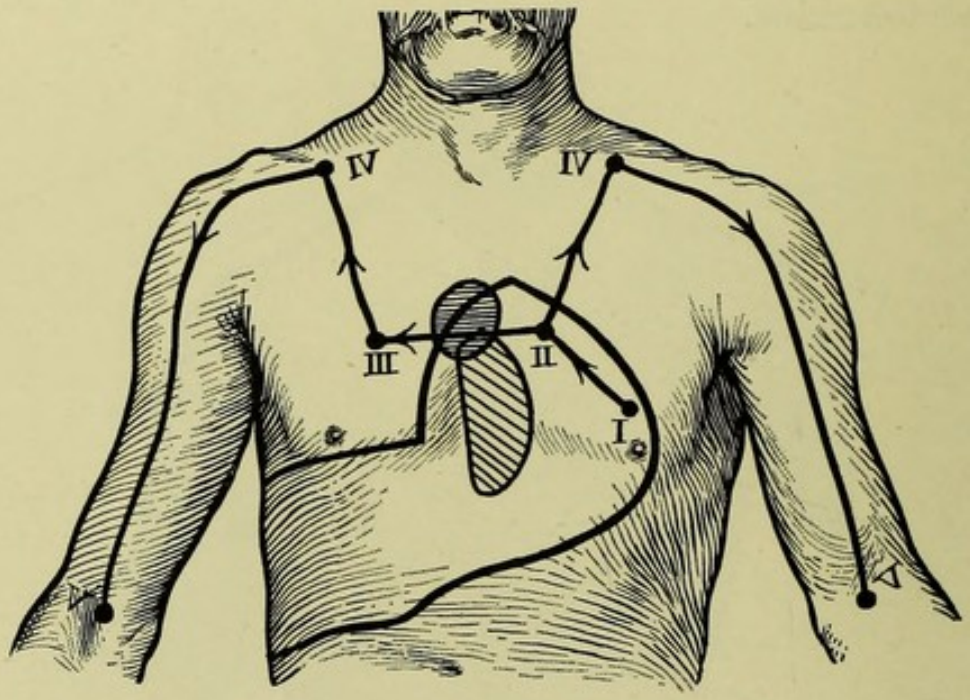


FIG. 9.—Man, æt. 35, suffering from aortic disease. The figure shows the areas of the systolic and diastolic murmurs, as well as the size of the heart and liver. The pain arose at I., darted to II. and III., and then simultaneously ran upwards to IV., and onwards to V. The exact area of the pain could not be definitely made out.

pressure. That fact is freely admitted by all observers. When this is the case, the arterial pulse gives every feature that we are accustomed to find as the result of high pressure. It is regular in rhythm, infrequent in rate, and has sustained resistance. But it is at least as common to find an arterial pulse absolutely the converse of this in every respect. Low pressure may be revealed by its ordinary features, yet it is more usual to find pulsation showing characters intermediate between those of high and low pressure. The pulse is very often indeed found to be one in which high pressure has obviously been present but is failing. It may

be perfectly regular yet unduly frequent; and although the resistance on the first application of the finger seems to be considerable, yet it is found on analysis not to be sustained. Sphygmographic evidence very clearly proves that this is the case. To any one who wishes convincing facts bearing upon this aspect of angina pectoris, the observations of Morison, previously referred to, may be heartily commended. It need hardly be added that the condition of the arterial wall is very diverse; while in most cases of organic angina pectoris the arteries show a certain degree of sclerosis, its extent is subject to great differences. In the various functional varieties of the affection the walls of the vessels may be practically healthy.

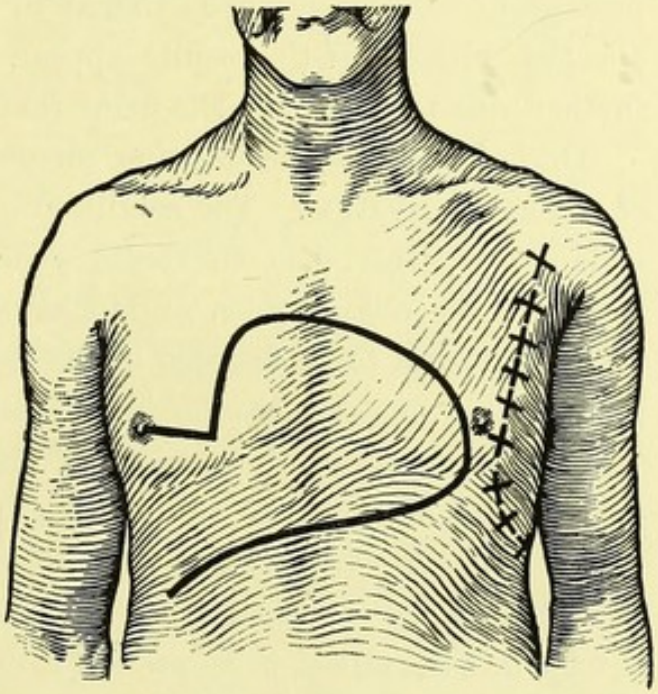


Fig. 10. — Man, æt. 32, suffering from severe pain, accompanied by tenderness, and always much increased by exertion, along the area marked by crosses. The case was one of tobacco angina. Four ounces were smoked daily.

The appearances furnished by the condition of the heart are exactly parallel to those yielded by the arterial pulse. There is almost, if not quite invariably, some cardiac enlargement due to dilatation. The cardiac impulse may be forcible and sustained, but it is, on the whole, more likely to be feeble and brief, as is so common in dilatation. Similarly, the heart sounds undergo considerable variations. While in one case there may be a low-toned and long-continued first sound at the apex, it may be in all respects the opposite of this, and a short, slapping sound may be found there. In the majority of cases of organic angina pectoris, the aortic second sound is accentuated, and it may considerably transcend in loudness the second sound in the pulmonary area. In many cases, however, attended by imminent cardiac failure, in which the right ventricle is seriously implicated, the pul-

monary second sound is much louder that that in the aortic area. It is unnecessary to add that, in the various functional varieties, the cardiac sounds may be approximately normal.

The renal secretion is often modified in a direction analogous to what is so often found in nervous affections. It is, especially after an attack, frequently found to be copious and pale, while disturbances, as regards control, may be observed. Retention of urine, more especially, is occasionally met with. It is not necessary to point out that when some primary disease of the kidneys is present, or when they have suffered in consequence of the cardiac affection, characteristic results appear. In the anginous attacks, further, due to glycæmia, the urine reveals the presence of glucose.

One of the most interesting phenomena in angina pectoris is of recent observation. The condition of the sensibility of the skin, tested objectively, has, in recent years, yielded most interesting results. The observations of Mackenzie and of Head, previously referred to, and to be detailed in the second lecture of the course, have led to a new era in the investigation of the affection. Over considerable areas of the surface of the body, corresponding, with more or less accuracy, to the regions in which pain is subjectively felt, there is an exaggeration of sensibility. This hyperæsthesia is most commonly experienced over the upper intercostal nerves, but it may be ascertained to be present over part of the neck and arm as well. The tenderness may be discovered by the application of varying degrees of pressure with a blunt or with a sharp instrument, or by gently pinching the skin with the finger and thumb. Its exact distribution and explanation will be considered in the next lecture of this course, but it may be well to mention in this place that in some instances, after pain and tenderness have been present for a considerable time, the latter is succeeded by diminution of sensibility.

Occasional trophic effects result in consequence of the nervous disturbances. The skin is sometimes seen to be thin and wasted, and, in certain interesting instances, one of the most striking of which has been narrated by Eichhorst,[1] wasting of the muscles,

[1] "Handbuch der speciellen Pathologie und Therapie," 5^te^ Aufl., Wien und Leipzig, 1895, S. 231. One clear instance of this condition, produced by functional angina pectoris, has recently been under my care.

corresponding to the distribution of the ulnar nerve, was observed.

An essential point, ever to be kept in view in studying these various symptoms, is their paroxysmal character. There is nothing periodic in their appearance, and the paroxysms, whether induced by definite causes, or appearing, so far as can be ascertained, without any exciting cause, are free from anything rhythmic.

Turning, in the last place, to the consideration of the different varieties of angina pectoris, it is necessary once more to emphasise the absurdity of the common phraseology which would divide the affection into true and false. To use such phraseology is to assume that the group of symptoms is a definite disease, whereas the contrary is now universally recognised. It is earnestly to be hoped that the term pseudo-angina will, before long, be banished from modern medicine. It ought to be relegated to the limbo of archaic notions, as an entire anachronism in the twentieth century of our era.

The most satisfactory classification is based upon a subdivision of the different varieties of cardiac pain into two categories—organic and inorganic. It is not pretended that in the latter class there are no structural changes. Certain of the toxic varieties undoubtedly result from something more than mere chemical union of a poison with the tissues; they depend upon definite alterations in the anatomical elements. The fact, no doubt admitted by all, that treatment brings about complete recovery in almost every variety of the inorganic class, must not be allowed too much weight, as, even in the organic class, complete recovery may ensue by means of appropriate management. The natural conclusion must therefore be, that no absolute line can be drawn between the two great classes.

Amongst the organic type must be grouped instances of the affection dependent upon alterations about the origin of the aorta; changes in the walls and lumen of the coronary arteries; modifications in the structure of the myocardium, of which fibroid, fatty, pigmentary, and gummatous lesions are the most common; the presence of pericarditis; the existence of aneurysm; the growth of tumour.

The inorganic varieties fall into toxic and neurotic classes. The former, or toxic class, may be due to chemical or microbic influences. Amongst the former, tobacco certainly holds the foremost place ; but alcohol, in this country, is certainly responsible for a considerable proportion of cases; tea, on the other hand, judging by my own observations, is an extremely rare cause of painful cardiac symptoms; certain of the endogenous chemical poisons are also potent, more particularly those which are connected with the symptoms commonly classed under the terms gout and diabetes. Amongst the microbic factors, influenza is certainly the most powerful, but malaria, diphtheria, and typhoid fever also furnish instances. The latter or neurotic group is a somewhat indefinite class; it practically resolves itself, however, into four varieties. There are cases in which reflex agencies are the cause of the symptoms; in which vasomotor instability is the dominant factor; in which neurasthenia forms the determining condition: and, lastly, in which hysteria is responsible for the onset of the affection. In every real instance of neurotic angina pectoris, it will be found that there is some cardiac weakness, and an attempt will be made in the next lecture to show how the complex of symptoms is brought about by debility of the heart and instability of the nervous system.

Under the classes just mentioned it is possible to place every known form of angina pectoris. It is not a difficult matter to divide and subdivide the varieties so as to obtain an imposing list. Every one who is familiar with the works of Gélineau[1] and Huchard[2] will remember with what indefatigable industry this has been done by these authors It has been no part of the aim of these lectures to deal with points of diagnosis or prognosis. Enough has been said on the present occasion to show what are the principal features of the affection, and to state the problem which will await us on our next meeting.

[1] *Op. cit.*, p. 358. [2] *Op. cit.*, p. 561.

Lecture II.—Pathological.

The problem awaiting our consideration to-day is to explain how pain produced by disturbances of structure or of function in an internal organ has its chief conscious localisation on the surface of the body. The heart is no exception to a general rule. It is only one of many viscera producing analogous sensory effects. Since the observations of Harvey[1] on the exposed heart of the son of Lord Montgomery, it has been well known that the organ is not sensitive to ordinary stimuli in health. We have, therefore, to consider how disturbances of sensibility can be produced by abnormal states of the viscera, and how these disturbances reveal themselves by peripheral manifestations.

In the course of our inquiry we shall have to devote attention to the nature of the nervous connections, to formulate a scheme of their component neurons, to analyse the causes of the afferent impulses, and to attempt an explanation of the sensory effects—such are the most convenient lines upon which our investigation may best proceed.

Before entering upon these different aspects of the subjects, it will be helpful to glance at the development of our knowledge of the nervous relations of the heart, and this not merely for the purpose of assisting the elucidation of those matters with which the first section deals, but with a view further to simplify the succeeding lectures.

The earliest discoveries regarding the innervation of the heart were made by the Webers.[2] They ascertained that the vagus nerve possessed an inhibitory action upon the heart, and concluded

[1] "Exercitationes de Generatione Animalium," Londini, 1651, p. 156.

[2] Wagner's "Handwörterbuch der Physiologie," 1846, Bd. ii. S. 42.

that the sympathetic nerve probably produced opposite effects. This conception remained in great part speculative, since neither of the two brothers was able to show the course of any accelerator fibres. Amongst the contributions of Schiff,[1] which were largely controversial, he showed that the muscular tissue of the heart did not respond easily, or even at all, to stimulation during standstill. He appears to have based his opinion that the vagus was the motor nerve of the heart—readily exhausted, however, by excessive stimuli—upon the fact that weak stimulation resulted in acceleration, while strong stimulation brought about inhibition. This acceleration, resulting from weak stimulation of the vagus, was particularly observed by Wundt and Schelske.[2] From these observations it was regarded as probable that the vagus contained accelerator as well as inhibitory fibres. The result of the investigations of Coats[3] was to show that the action of the heart was weakened as well as retarded on stimulation of the vagus.

The course of the accelerating fibres was ascertained by von Bezold.[4] As the result of his experiments, it was clear that the accelerating fibres must leave the spinal cord for the heart at the level of the first thoracic ganglion. Cyon[5] by destruction of this ganglion put a stop to any accelerating effect of stimulation of the cervical spinal cord. Schmiedeberg and Ludwig[6] finally settled the anatomical relations of the accelerator nerves.

It is impossible to overestimate the importance of Gaskell's long series of papers[7] upon this subject. He showed that the vagus, when stimulated, not only produced acceleration of rate but also augmentation of force of both auricular and ventricular contractions. He further found that stimulation of the sympathetic fibres joining the vagus produced acceleration, while stimulation

[1] *Arch. f. physiol. Heilk.*, Stuttgart, 1849, Bd. viii. S. 166 u. 442.

[2] *Verhandl. d. naturh.-med. Ver. zu Heidelberg*, 1859, Bd. ii. S. 12.

[3] *Arb. a. d. physiol. Anst. zu Leipzig*, 1870, Bd. iv. S. 176.

[4] "Untersuch. über die Innervation des Herzens," 1863, S. 89; and *Untersuch. a. d. physiol. Lab. in Würzburg*, 1867, Bd. i. S. 181.

[5] *Arch. f. Anat. Physiol. u. wissensch. Med.*, Berlin, 1867, S. 389 u. 403.

[6] *Arb. a. d. Physiol. Anst. zu Leipzig*, 1872, Bd. vi. S. 34.

[7] *Trans. Internat. Med. Cong.*, London, 1881, vol. i. p. 254; *Phil. Trans.*, London, 1883, vol. clxxiii. p. 993; *Journ. Physiol.*, Cambridge and London, 1884, vol. v. pp. 46 and 362; *ibid.*, 1886, vol. vii. p. 1.

of the vagus within the cranium gave rise solely to inhibitory effects. The histological differences between the two sets of nerves were also fully investigated by Gaskell; and it may be said briefly that, while the inhibitory fibres are medullated, the accelerating are non-medullated. The vagus fibres, as far as the heart, are medullated, but as no medullated fibres are present in the auricle or ventricle, it is clear that the ganglion cells of the heart have the same relation to the medullated fibres of the vagus, as the ganglion cells of the ganglion stellatum to the medullated fibres in the white connecting branches from the second, third, and fourth dorsal nerves. The conclusion from these facts is obvious, that the intrinsic ganglion cells of the heart are members of the same system as the extrinsic cardiac and vasomotor ganglion cells. The observations of Heidenhain,[1] which immediately followed Gaskell's earliest results, supported the anatomical conclusions to which he had been led. Löwit[2] showed that stimulation of the vagus after the administration of muscarine produced contraction, while Gaskell,[3] MacWilliam,[4] and Mills[5] demonstrated that the nerve produced different effects on different parts of the heart. As the result of a long series of observations, Langley[6] showed that degeneration occurred in the sympathetic nerve, in consequence of section of the anterior roots. It seems probable that the grey ramus is partly post-ganglionic and partly afferent from the sympathetic chain to the spinal ganglia. This, however, is only a belief, and is not absolutely proved by experiment. Ramón y Cajal[7] seems to have shown that fibres pass from the sympathetic into the spinal ganglia and break up there, and Dogiel[8] has apparently traced out the course of fibres arising from afferent cells in the sympathetic ganglia. Berkley[9] has spent much time and labour over the investigation of the nature of the nerve endings in the heart, and Morison,[10]

[1] *Arch. f. d. ges. Physiol.*, Bonn, 1882, Bd. xxvii. S. 383.
[2] *Ibid.*, 1882, Bd. xxviii. S. 312.
[3] *Journ. Physiol.*, Cambridge and London, 1887, vol. viii. p. 404.
[4] *Ibid.*, 1885, vol. vi. p. 192.
[5] *Ibid.*, 1885, vol. vi. p. 246 ; and 1886, vol. vii. p. 81.
[6] *Ibid.*, 1890, vol. xi. pp. 265 and 509 ; and 1892, vol. xiii. p. 786.
[7] *Arch. f. Anat. u. Entwcklngsgesch.*, Leipzig, 1893, S. 319.
[8] *Arch. f. mikr. Anat.*, Bonn, 1898, Bd. lii. S. 44.
[9] *Johns Hopkins Hosp. Rep.*, Baltimore, 1895, vol. iv. p. 80.
[10] "The Nervous System and Visceral Disease," Edin. and London, 1899, p. 10.

from his indefatigable researches, has pointed out the varying characters of the ganglia and their connections in health and disease.

The explanation of the localisation of cardiac pain, by the consideration of the relations of the cardiac nerves to the spinal cord, was undoubtedly suggested by Lussana[1]; this writer traced out the connections of the cardiac nerves, the spinal cord, and the brachial plexus. His views were further elaborated in an extremely suggestive paper by Sturge,[2] and the segmental distribution of the pain was very clearly indicated by Ross.[3] The brilliant and original researches of Mackenzie,[4] upon the presence of exalted sensibility as well as of painful sensations of visceral affections, may be regarded as having inaugurated a new epoch. The subject has been still further elucidated by the beautiful and elaborate observations of Head.[5]

Having thus very shortly shown how our present knowledge has been gained, we must in the next place deal with the course of the nervous connections.

The upper cervical ganglia of the sympathetic chain are connected with the glosso-pharyngeal, pneumogastric, and hypoglossal nerves, as well as with the upper four cervical spinal nerves, and give rise to the superior cardiac nerves. These nerves, which in many instances receive additional branches from the sympathetic cord, take the same course on both sides of the neck, behind the carotid sheath; they have connections with the pneumogastric nerves, and their external and recurrent laryngeal branches, but differ in their distribution after entering the thorax. The right superior cardiac nerve passes along the innominate artery, giving branches to the vessels in its course, towards the back of the aorta, and ends in the deep cardiac plexus. The left superior cardiac nerve accompanies the left common carotid artery to the arch of the aorta, to terminate in the superficial cardiac plexus. Occasionally it ends in the deep cardiac plexus instead of taking its usual course. The middle cervical ganglia, lying near the inferior thyroid arteries, have connections with the fifth and sixth

[1] "Monografia delle Nevralgie Brachiali," Milano, 1859, p. 218.
[2] *Brain*, London, 1883, vol. v. p. 492.
[3] *Ibid.*, 1888, vol. x. p. 333. [4] *Loc. cit.* [5] *Loc. cit.*

cervical spinal nerves, and give rise to the middle cardiac nerves. These nerves pass down behind the carotid sheath, communicating with the recurrent laryngeal and the superior cardiac nerves, and, entering the thorax in close proximity to the subclavian vessel, end in the deep cardiac plexus. The lower cervical ganglia, lying between the last cervical vertebræ and the first ribs, receive communicating branches from the two lowest cervical spinal nerves, and give origin to the inferior cardiac nerves. These inferior cardiac nerves pass inwards, communicating with the recurrent laryngeal and middle cardiac nerves, behind the subclavian arteries, to end in the deep cardiac plexus. Sometimes, on the left side, the inferior blends with the middle cardiac nerve.

The vagus nerves also give rise to cardiac branches. The cervical cardiac branches are usually divided into the upper, of which there are several, small in size, ending in the sympathetic cardiac nerves; and the lower, single on each side, of which the right, joining a sympathetic cardiac nerve, ends in the deep, while the left terminates in the superficial cardiac plexus. The thoracic cardiac branches take their origin on the right side from the pneumogastric trunk itself, and from the recurrent laryngeal nerve; on the left side, they arise from the recurrent laryngeal nerve. These nerves end in the deep cardiac plexus.

The great network known as the cardiac plexus is, for convenience of description, regarded as consisting of two parts—the superficial and the deep—which are intimately connected. The superficial cardiac plexus occupies part of the space between the arch of the aorta and the right pulmonary artery. It receives, as we have seen, the left superior cardiac nerve from the sympathetic, and the left lower cervical cardiac branch of the vagus nerve. It furnishes some small branches to the anterior left pulmonary plexus and terminates in the anterior coronary plexus. The deep cardiac plexus lies between the posterior aspect of the aortic arch and the trachea, and above the bifurcation of the pulmonary artery. With the exception of the two nervous branches ending in the superficial plexus, all the cardiac nerves—sympathetic and pneumogastric—terminate in the deep plexus,

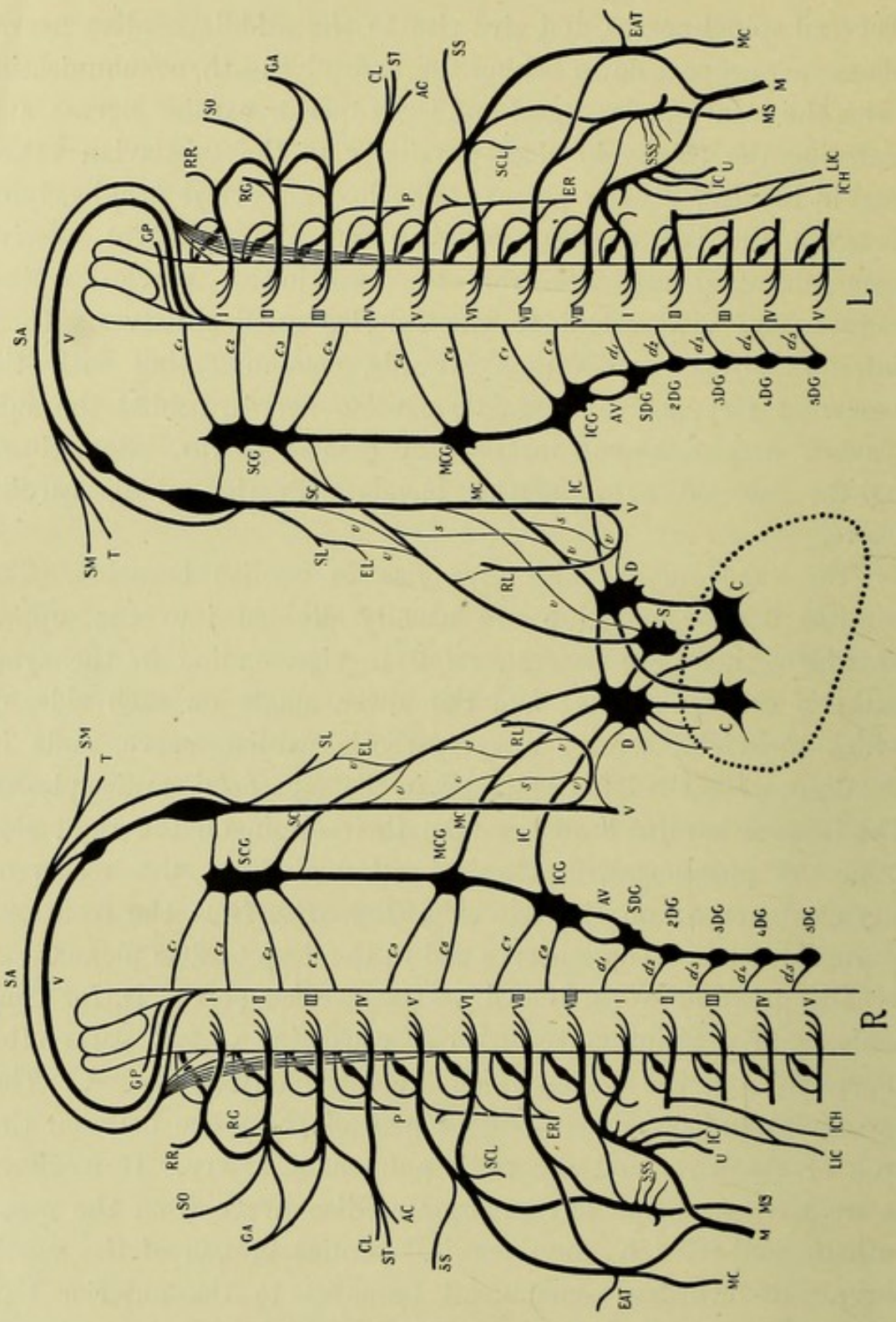


FIG. 11.—Connections of cardiac nerves.—D, deep cardiac plexus; S. superficial cardiac plexus; C, coronary plexus; SGC, superior, MCG, middle, and ICG, inferior cervical sympathetic ganglia; SDG, stellate ganglion; and 2–5 DG, other dorsal sympathetic ganglia; AV, annulus of Vieussens; SA, spinal accessory with branches to sterno-mastoid and trapezius muscles, SM and T; V, vagus; GP, glosso-pharyngeal, SL, superior laryngeal, EL, external laryngeal, and RL, recurrent laryngeal nerves; *v*, *v*, *v*, branches from vagus to cardiac nerves; *s*, *s*, *s*, communicating twigs between sympathetic cardiac nerves; RR, branches to recti; SO, small occipital; GA, great auricular; SC, supra-

which is much larger than the superficial, and is commonly regarded as having a right and left division. It gives off right and left branches. On the right side the plexus furnishes some filaments to the anterior pulmonary plexus and posterior coronary plexus, together with many branches to the right auricle, but most of the fibres unite with those from the superficial plexus which form the anterior coronary plexus. On the left are some filaments going to the anterior pulmonary plexus, and a few

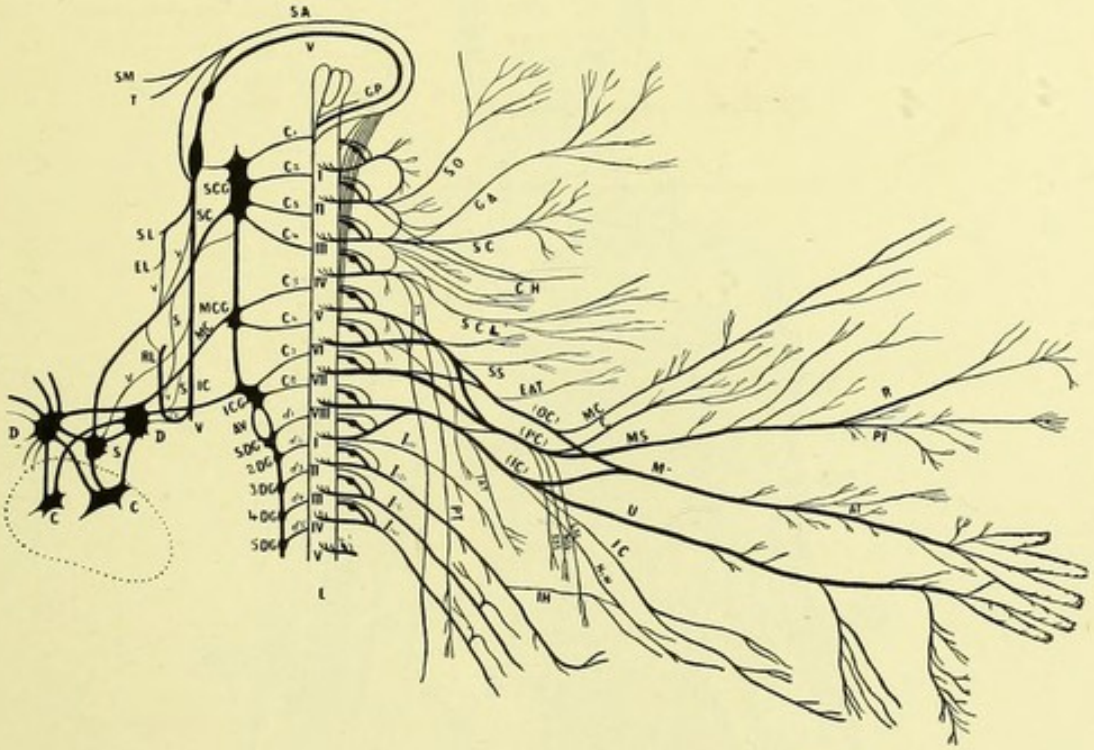


FIG. 12.—Connections of cardiac nerves.—For description, see Fig. 11.

branches to the superficial cardiac plexus, but most of the branches terminate in the posterior coronary plexus.

The anterior coronary plexus, formed by branches from the

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clavicular ; CL, clavicular ; ST, sternal ; AC, acromial ; RC and CH, communicating with hypoglossal ; P and Ph, phrenic ; SS, Suprascapular ; SCL, subclavian ; ER and PT, nerve of Bell ; EAT, external anterior thoracic ; SSS, subscapulars ; LSS, MSS, SSS, course of subscapulars ; MC, musculo-cutaneous ; (OC), (PC), (IC), outer, posterior, and inner cords of brachial plexus ; M, median ; C, circumflex ; MS, musculo-spiral ; U, ulnar ; IC, internal cutaneous ; LIC and NW, lesser internal cutaneous ; ICH and IH, intercosto-humeral ; R, radial ; P, posterior interosseous ; AI, anterior interosseous ; I (1), I (2), I (3), I (4), four upper intercostals. The spinal cord is represented as divided into two lateral halves, right and left ; the Roman numerals denote the segments corresponding to the eight cervical and upper dorsal nerves, whose anterior and posterior roots are shown, the latter characterised by their ganglia ; the connections between the spinal segments and sympathetic ganglia are marked c^1–c^8 and d^1–d^5.

superficial and both divisions of the deep cardiac plexus, accompanies the left or anterior coronary artery; the posterior coronary plexus, mostly derived from the left, but in part also from the right part of the deep cardiac plexus, follows the right or posterior coronary artery in its course.

In Fig. 11, which is taken from my work on "Diseases of the

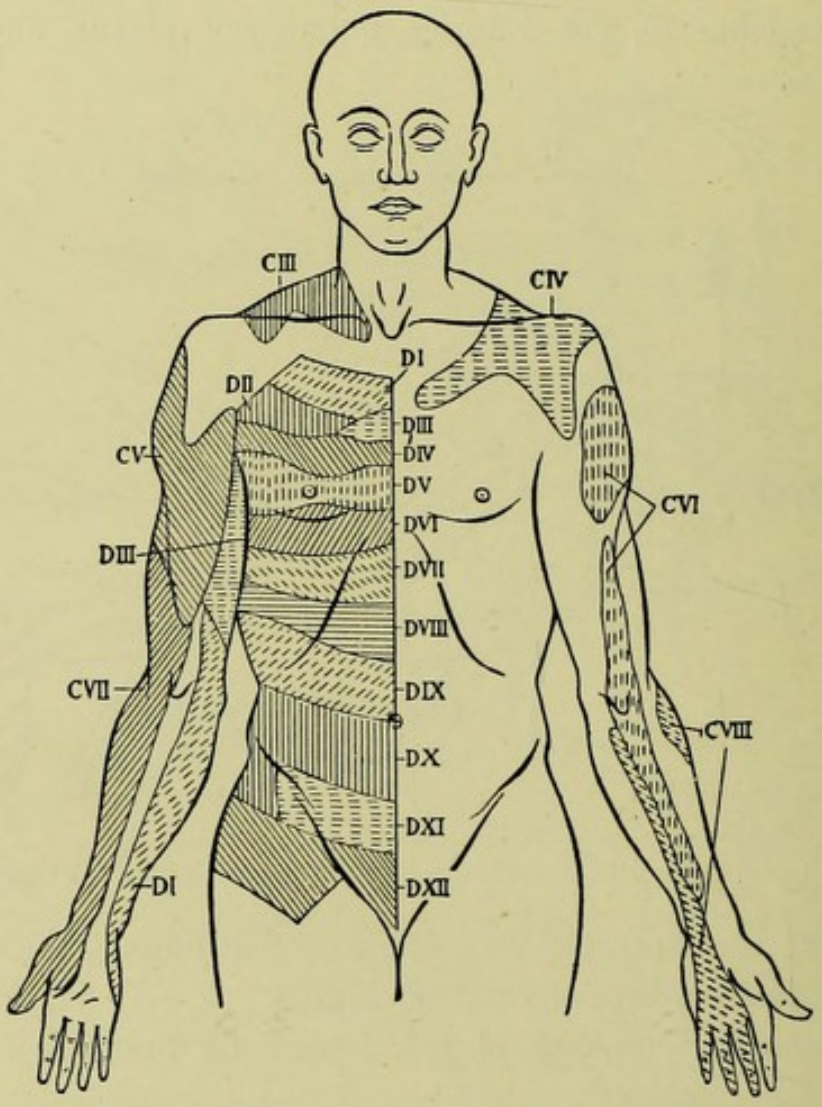

FIG. 13.—Cutaneous representation of spinal nerves.

Heart," all these facts are given diagramatically, and the descriptive references which accompany it explain the whole relations and render the entire scheme easy to grasp. Fig. 12 gives the branches of the spinal nerves arising from the cervical region of the cord, in addition to the left half of Fig. 11. It is unnecessary to mention these branches; they are denoted in the explanation of the figure.

The lettering of both of these illustrations is the same in so far as each is concerned.

To render the nervous supply of the surface clear, it will only be necessary for me to include two diagrams, compiled from the papers of Head, previously referred to, which speak for themselves. It may be mentioned, in passing, that Mackenzie and Head do not

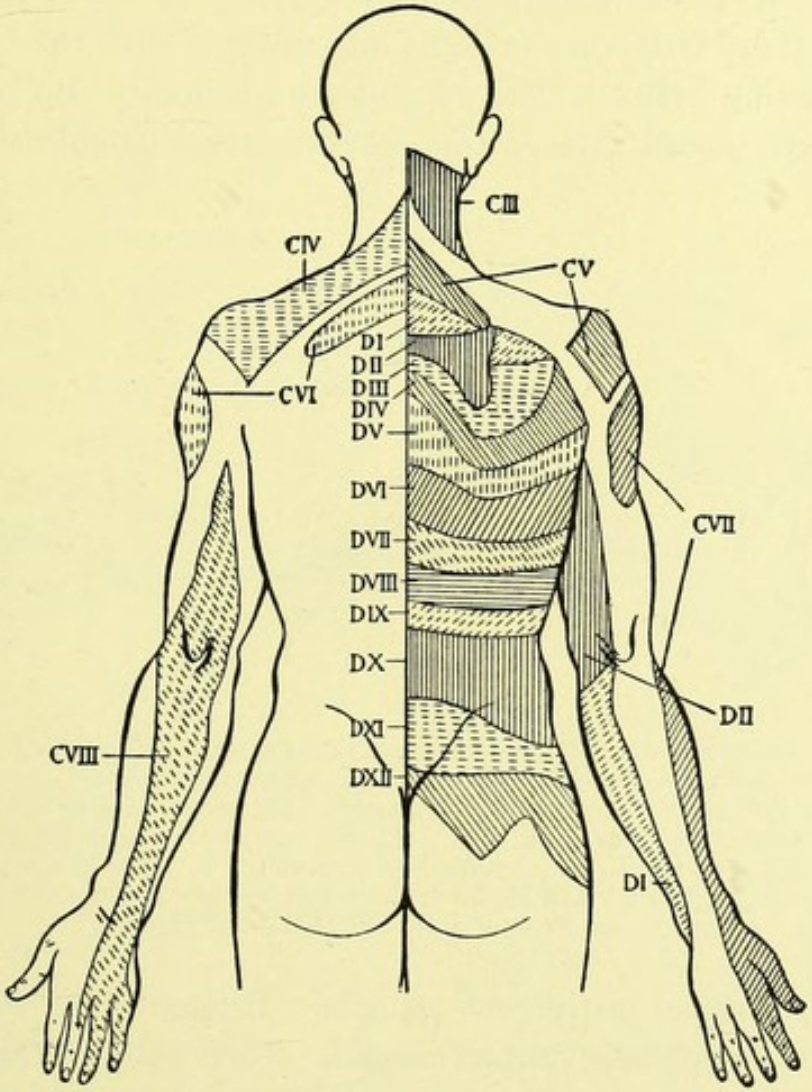

FIG. 14.—Cutaneous representation of spinal nerves.

see eye to eye in regard to the theory of segmental division and distribution; this, like some other very interesting speculations, does not at present demand our consideration in the practical study of cardiac pain. So far as concerns the matter of hyperæsthesia, it may, however, be stated in a word that, although the exalted sensibility has always a fixed central area, its boundaries are often somewhat indefinite.

The exact nature of the anatomical relations between the sympathetic and spinal systems must receive a few words of explanation. It is scarcely necessary to recall to your recollection that the sympathetic system consists of vertebral and pre-vertebral ganglia, the former including the ganglia of the sympathetic chain —possessing the special arrangement of a connection with spinal nerves—and the latter comprising the various visceral or splanchnic ganglia; these latter are brought into relation with the former by nerves passing between the two, and are frequently also connected with others, placed further from the vertebral ganglia, which are

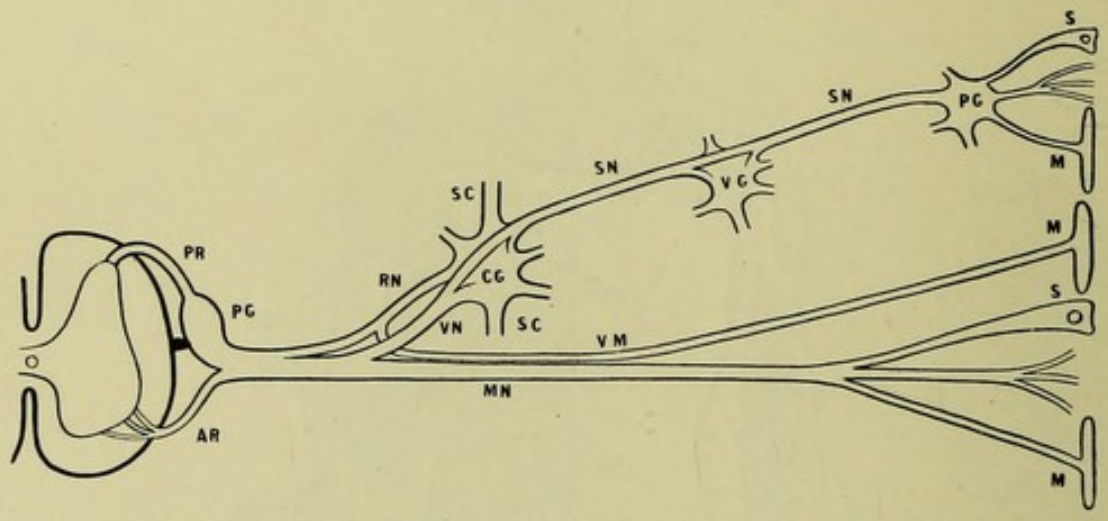

FIG. 15.—Connections of spinal and sympathetic systems.—PR, posterior root; AR, anterior root; PG, ganglion of posterior root; VN, visceral nerve or white ramus; RN, revehent nerve or grey ramus; VM, branch of grey ramus going to vessels; CG, sympathetic ganglion; SC, SC, connections with other ganglia of gangliated cord; SN, SN, sympathetic nerve; VG, visceral ganglion; PG, peripheral ganglion; S and M, its sensory and motor end organs; MN, mixed "spinal" nerve; S and M, its sensory and motor end organs.

commonly termed peripheral ganglia. Belonging to this latter group are the intrinsic cardiac ganglia. Their cells, as Gaskell has shown, belong to the efferent cardiac fibres of the vagus, just as some of the cells in the inferior cervical and first dorsal ganglia belong to the efferent fibres of the augmentor.

As is well known, each vertebral ganglion of the sympathetic cord is connected with a segment of the spinal cord by two communicating links—the "rami communicantes." The white ramus, often termed the visceral nerve, has its origin in the anterior root, which passes directly to the ganglion. The grey ramus, or "revehent nerve," brings the sympathetic ganglion into connection with the

posterior root, and on its path gives off a branch to join the mixed spinal nerve, and to proceed onwards along with it. These relations are exhibited in Fig. 15, which is modified from an illustration given by Foster.[1] It seems now to be quite clear, both from the results of physiological experiment and from the study of the course of degeneration after section, that the white ramus is mainly efferent; while the grey ramus—except in so far as concerns the branch which passes to the corresponding spinal nerve—is afferent.

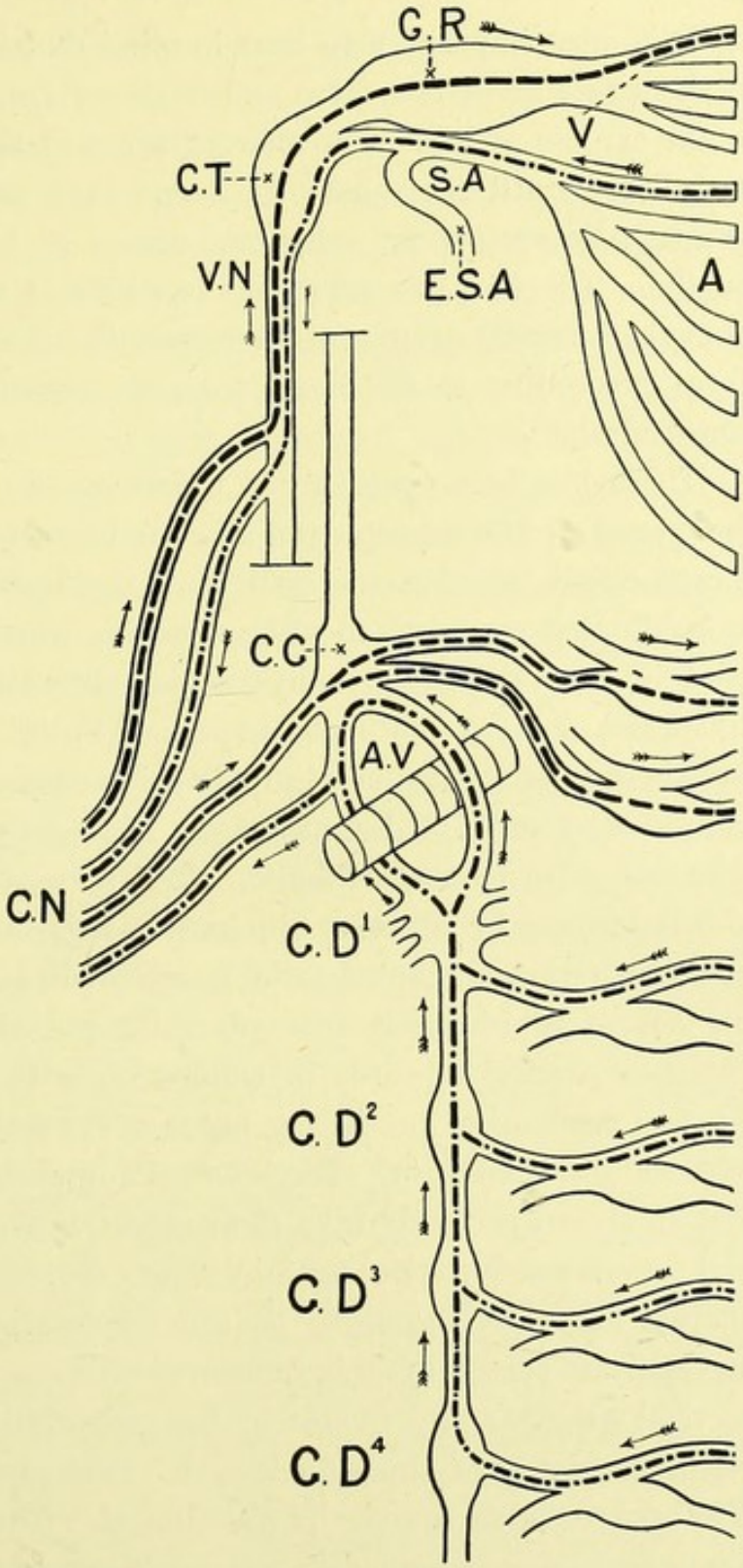

FIG. 16.—Shows the afferent and efferent impulses—the former are represented by the simple interrupted black line; the latter by the dash and dot line. The arrows also assist in showing the course of the impulses. V, vagus roots; GR, ganglion of the root; GT, ganglion of the trunk; VN, vagus nerve; A, accessory roots; SA, accessory nerve; ESA, external accessory nerve; CN, cardiac nerves; AV, annulus of Vieussens; CG, inferior cervical ganglion; GD^1, GD^2, GD^3, GD^4, thoracic ganglia.

[1] "Text-book of Physiology," 5th edition, London, 1888, p. 170.

It is most important to bear in mind that there are differences in the structure of the vagus and of the sympathetic fibres passing to the cardiac plexus. The efferent fibres of the vagus system are medullated until they enter the heart—the efferent fibres of the sympathetic system are only medullated as far as the vertebral ganglia. The fibres found in the heart itself, proceeding onwards from the intrinsic ganglia, are non-medullated, and the sympathetic fibres proceeding to the heart from the cervical ganglia are also non-medullated.

There has been considerable difference of opinion in regard to the course of the afferent and efferent tracts by which connection is established between the heart and the nervous centres. It is probable that some of the discrepancies which may be detected even in the most recent physiological literature are due to the difficulties inseparable from experimentation on sensory tracts. Certain channels which must supply the course of afferent impulses, as proved by undoubted clinical facts, have not yet received support from any experimental evidence. It is no part of my duty to-day to bring forward conflicting opinions in regard to these points still under dispute, and a more useful purpose will be served by showing the course of impulses now generally recognised. The afferent impulses passing upwards in connection with the inhibiting and dilating mechanism run in the vagus nerve and reach its centre in the medulla oblongata. The efferent impulses belonging to this system almost certainly take their origin in the accessory nucleus, and pass onwards to the vagus by which they reach the heart. The afferent impulses belonging to the accelerating and constricting mechanism, travel by the inferior cardiac nerve to the inferior cervical ganglion, and thence by the grey rami to the seventh and eighth segments of the spinal cord. It is possible that they may also take a course round the annulus of Vieussens and reach the upper thoracic segments, but nothing is known of this, and it is not necessary to suggest it. The efferent impulses of this system flow downwards from the chief vasomotor centre in the medulla, and outwards from the spinal cord by the white rami of the first, second, third, and fourth thoracic segments, to the corresponding ganglia of the sympathetic system. Proceeding upwards in the gangliated

cord, they pass round the annulus of Vieussens, and by means of the inferior cardiac nerve reach the heart.[1] These relations are represented in the diagram, Fig. 16.

In the next place, we must inquire whether we have sufficient evidence upon which to attempt the formation of a scheme by which the nervous connections may be expressed in modern terms. Taking up the inhibitory mechanism, let me recall to your recollection that the afferent channel lies in the vagus nerve. It must therefore be obvious that the neuron must have its centre in the ganglion of the trunk or the ganglion of the root. The efferent neuron belongs to the accessory system, and probably has its centre in the accessory nucleus. Its nerve fibre is medullated. As previously shown, the nerve fibres in the heart itself are non-medullated and must belong to another—which may be called the peripheral neuron. It therefore follows that, as represented in Fig. 17, there is one afferent neuron and two efferent, the former of which may appropriately be termed central, and the latter peripheral. Similarly, on turning to the accelerating mechanism we may recognise the afferent fibres in the inferior cardiac nerve, reaching the seventh and eighth cervical segments of the cord.

[1] It may be well to amplify in some degree the remarks made in the lecture upon this particular subject. Of afferent nerves belonging to the heart and proceeding to the spinal cord, we have no experimental proof. If we may judge afferent nerves by those belonging to the visceral system of the abdomen, they are always medullated up to the posterior root ganglion. In the case of many of the abdominal nerves, indeed, these medullated fibres are of large size. Reasoning by analogy, it might be expected that the afferent cardiac fibres must also be medullated. When working at the fibres of the dog's heart, Gaskell was much struck to find that all the nerves from the annulus of Vieussens to the heart were non-medullated, and any medullated fibres could always be traced to the depressor or to the vagus nerve. He therefore concluded that the anatomical evidence confirmed the physiological; that both the pressor and depressor fibres reached the central nervous system by the channel of the vagus nerve. In a private communication, however, Dr Gaskell informs me that the spinal heart nerves are only a special example of the system of vaso-constrictor nerves, which are rigidly confined to the great thoracic outflow of organic nerves, and therefore, if the blood vessels possess afferent nerves, the heart, in virtue of its connection with that system, must possess such nerves. He believes that there is evidence of the possession by the blood vessels of spinal afferent nerves, especially connected with the vasomotor centres, while there is no evidence to show that the blood vessels possess any number of medullated fibres. He therefore regards it as possible, indeed probable, that afferent nerves, at least from the coronary vessels, should accompany the accelerator, and so account for the clinical results.

It is beyond doubt that the centre of this afferent neuron is in the posterior root ganglion. The efferent impulses pass, like those of the inhibiting mechanism, through two neurons. The central of these, which is medullated, certainly has its centre in the lateral grey matter of the spinal cord, and has its synapse in the gangliated cord of the sympathetic. The peripheral neuron—non-medullated—takes its origin at this point and reaches the heart.

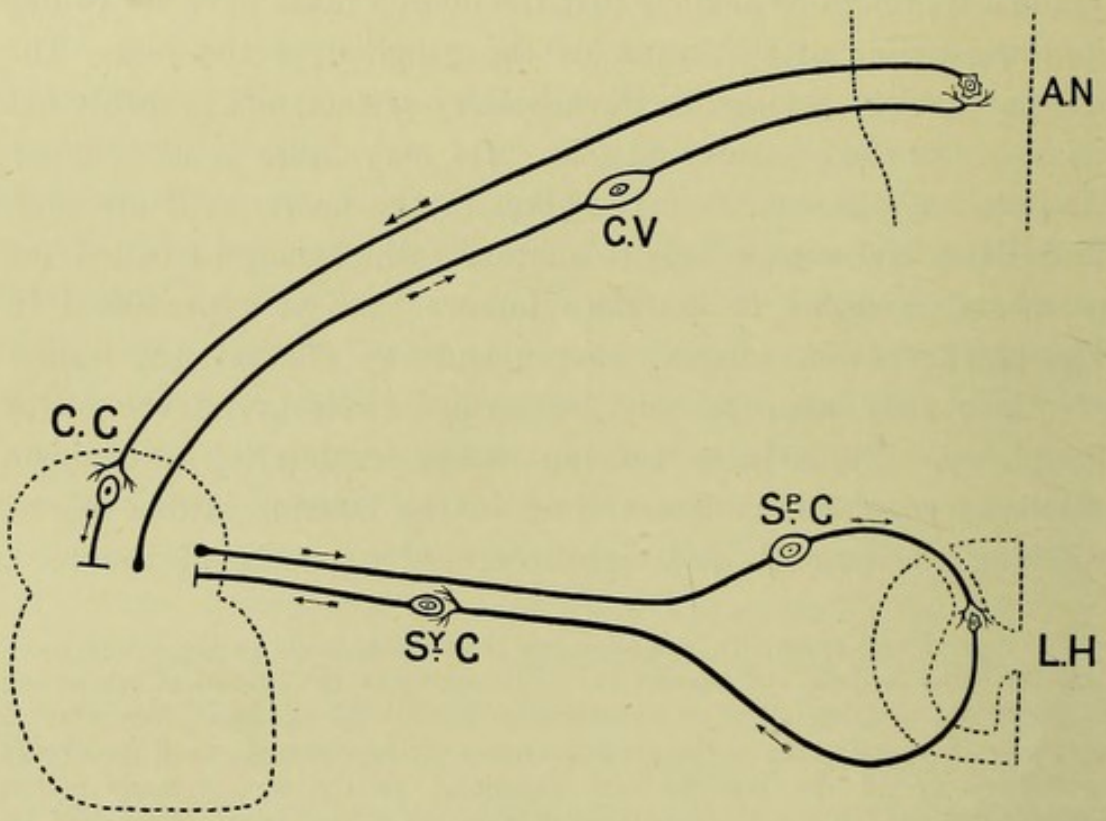

FIG. 17.—Arrangement of neurons in cardiac nerves.—CV, centre of the afferent vagal neuron, in ganglion of the root or of the trunk of the vagus nerve; AN, accessory nucleus, the probable centre of the central efferent neuron; CG, cardiac ganglion or centre of the peripheral efferent neuron; SPG, posterior root ganglion or probable centre of the afferent neuron; LH, lateral horn of grey matter or centre of the central efferent neuron; SYG, sympathetic ganglion or centre of the peripheral efferent neuron.

The causes of the afferent impulses reaching the nerve centres fall into different classes, corresponding more or less closely to the varieties of angina pectoris mentioned in last lecture. Some of these causes are as easy to understand as they are to recognise; others are almost equally difficult of explanation and of detection. This we shall find to be particularly the case as regards toxic influences, reflex stimuli, and neurotic conditions.

But few words are called for in order to explain the organic changes which cause angina pectoris. The aortic, coronary, and myocardial lesions lead to secondary changes in the condition of the heart wall by interfering with its nutrition. This may occur either by lessening the blood supply which reaches the tissues of the heart—including the nervous mechanism—or by producing direct irritation of the sensory nerve endings. In all lesions belonging to this group cardiac pain is of frequent occurrence. Pericardial affections are sometimes absolutely latent, at least in so far as concerns sensory changes. No doubt it is quite common to find some pain and tenderness over the præcordial region in acute pericarditis, but anything approaching the typical symptoms of fully developed angina pectoris is undoubtedly rare. Occasionally, however, a case presents itself with the characteristic distribution and special features of a true anginous seizure. Similarly, aneurysms and tumours within the chest may give rise to a very considerable amount of thoracic pain. It is, nevertheless, uncommon to find a close similarity to the typical symptoms which we have been discussing. In addition to these more definite changes in the circulatory organs, it must be added that, not infrequently, in conditions of failing nutrition of the heart and of acute or chronic dilatation—all of which may present few or no structural alterations—a considerable degree of cardiac pain may be observed. The mechanism is here somewhat difficult to grasp, but probably there is in all such conditions a deficient nourishment of the nerve fibres, and, in the next place, a demand for more energy than can be supplied—consequently, cardiac strain.

Very different in many ways are the toxic influences. Alcohol, tea, and tobacco—the latter more particularly—may undoubtedly give a great deal of uneasiness, and even severe pain, in the præcordia; this may have a characteristic distribution, and may even be attended by considerable hyperæsthesia. Tobacco has certain special effects upon the heart which will meet us in our future study of the motor disturbances of the circulation; these do not call for any remark at the present time. The same is even more true of lithæmia, and it has been described also in glycæmia,

no instance of which, however, has ever come under my notice. It is probable that in all these cases there is a certain amount of intimate chemical union between the toxic agents and the nervous tissues. That this is to some extent true must be admitted, as in certain cases of several of these types the pain, as close observation has convinced me, often sets in without any exertion or gastric distension,—without in fact, any ascertainable exciting cause. In some of them, however, and this is particularly the case in regard to alcohol, it is highly probable that the mechanism is to be sought in a weakened state and dilated condition of the heart muscle itself. The microbic agents seem almost invariably to bring about their effects in a twofold manner—by producing a greater degree of sensitiveness of the nervous mechanism, and by inducing a feeble and dilated state of the heart. Thus the least excessive demand for increased energy results in pain.

Difficulties increase upon us when we attempt an explanation of the different types of neurotic angina. That true angina pectoris results from reflex, vasomotor, neurasthenic, and hysterical causes is now beyond the region of doubt, but cases belonging to all these varieties are extremely rare. It is easy to understand that reflex stimulus may with a feeble heart give rise to cardiac pain, seeing that the heart may be face to face with a demand for an expenditure of energy beyond its capabilities, and we occasionally meet with it in the apparent absence of any structural changes in the heart and vessels. The vasomotor type of angina pectoris is, even in a variable climate like our own, extremely uncommon. Cases at all resembling the original description given by Nothnagel,[1] with chilliness of the surface, and lividity or pallor of the extremities, followed by cardiac pain, are but seldom seen.[2] Nevertheless, in another sense, vasomotor angina is not uncommon. In cases of cardiac failure it is far from rare to find an arterial spasm which, by throwing more work upon the enfeebled heart, leads to the painful symptoms. To sum this

[1] *Loc. cit.*

[2] Since the delivery of this lecture, Mackenzie has published his deeply interesting work—"The Study of the Pulse," Edinburgh and London, 1902—where, page 78, there is stated a view practically identical with the opinion here expressed.

matter up, it need only be added that in the organic varieties of angina, vasomotor changes may be added to the structural causes.

In neurasthenia and hysteria, anything like anginous symptoms is really seldom found. When such symptoms develop, they are commonly induced by some inter-current disturbance, very commonly of an emotional nature. In these two groups it is probable that in addition to a condition of irritable weakness of the nervous system, to use that excellent phrase, there is also some debility which, in the way above described, gives rise to the painful effects.

The last part of the quest is now reached. We have to seek an explanation for the fact that, in consequence of impulses flowing to centres from the heart, which is notoriously insensitive, there is pain with tenderness on the surface of the body. Why is the pain felt on the surface? The sensory stimulus is carried to the cord in the region with which the sensory nerves are connected, and in that segment it comes into relation with the nerves for painful sensation belonging to the segment. "But," as Head[1] has so well put it—"The sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment, the diffusion area is accepted by consciousness, and the pain is referred on to the surface of the body instead of on to the organ actually affected." This means that in the education of the nervous centres they have been taught by experience to refer disturbances to irritation of the sensory areas in connection with them, from which impulses have most commonly started.

The explanation of tenderness in association with pain which is given by Head, is worthy of careful consideration, and merits general acceptance. Impulses passing to the cord from a diseased viscus produce a disturbance in the segment to which they pass, and induce a condition of unstable equilibrium, so that any stimulus applied to the area connected by sensory nerves with the segment will be more powerful in its effects and will give rise to exaggerated sensations.

The greater frequency of pain in the left half of the chest can only be due to the fact that the left ventricle and its attachments

[1] *Loc. cit.*

are connected mainly with the left coronary and left deep cardiac plexuses, which, along with the entire superficial cardiac plexus, are in relation with the left nerve centres. The left ventricle and aorta are, on account of the higher pressure to which they are continuously subjected, much more liable to degenerative changes than the right ventricle and pulmonary artery. The left side of the heart and its adnexa are, moreover, much more liable to sudden stress and strain when any sudden demand is made upon the cardiac energy. That this is correct seems to me demonstrated by the fact that, in an instance of disease of the right side of the heart, Morison[1] found the pain to be confined to the corresponding side of the body, while an instructive case under my own care, in which the cardiac failure began with chronic bronchitic troubles, and dilatation of the right chambers, manifested the earliest anginous symptoms on the right side; these were followed by typical seizures affecting both sides, including the arms.

[1] "Observations on some points in Dextral Valvular Disease of the Heart," Edinburgh, 1880, p. 5

Lecture III.—Therapeutical.

No matter how interesting may have been the observations and speculations on the character and nature of sensory disturbances with which the two former lectures were concerned, they must remain barren of practical utility if they do not lead to some conclusions which will be helpful in the relief of suffering. It is, fortunately, no mere figment of the imagination that modern measures are able to alleviate the symptoms of these affections, and to obviate the tendency to death with which they are so closely united. It is natural that at least as much scepticism should exist in regard to the therapeutics of such disorders as prevails with respect to the treatment of other nervous diseases. It will nevertheless be possible to show good grounds for the belief—nay, for the certainty—that the sensory affections of the circulation are in truth and in deed wonderfully amenable to careful management. We may go farther, and fearlessly assert that in not a few of the worst cases, even when death seems to stare the patient in the face, judicious treatment brings about recovery, which may for several years remain unimpaired. From the aim with which we set out in this course of lectures, the remarks upon treatment must be suggestive rather than exhaustive. A rough sketch is alone possible within the limits assigned to us; a complete picture is out of the question. It would add much to the value of these remarks if it were practicable to give some details of cases in illustration of the principles laid down; nothing more than brief references will, however, be permissible. Following the method adopted in the two previous lectures, it will render the

subject more complete if we spend a few minutes in a retrospect of the development of our knowledge of the measures adopted in these affections.

Heberden,[1] without condescending upon great details, simply suggests the employment of stimulant and sedative remedies. He points out the uselessness of bleeding, purging, and other depressing measures, and recommends the employment of various ammoniacal, alcoholic, and ethereal substances; above all, of opium. Laennec[2] believed in the use of magnetism, and gave particular directions for the application of steel plates, so as to ensure that the "magnetic current" should traverse the affected part. He nevertheless admits that the method is not infallible—an idea shared by his contemporaries and followers—and recommends in addition blistering, bleeding, and antispasmodics. Hope[3] gives full directions as to relieving the stomach of any distension by carminatives, antacids, and even emetics; the use of counter irritants and derivatives; and finally the employment of antispasmodics and sedatives. Latham,[4] in addition to the same general line of treatment, strongly urged the employment of opium,[5] and Stokes introduced the administration of chloroform. A new epoch was inaugurated when Brunton,[6] basing his views upon physiological observation, introduced the employment of amyl nitrite, upon which subject a large amount of work has been done by subsequent authors. In this connection it is but right to refer to the valuable investigations of Hay[7] upon nitrite of sodium, which he was the first observer to study. The employment of the iodides, which, as a remedy for circulatory affections, is chiefly due to Balfour,[8] marks another and most important step in advance. Recent observations upon these drugs, although providing us with conflicting statements, have nevertheless assisted us in attempting to arrive at their method of acting. Amongst those observations the most important are

[1] *Loc. cit.*
[2] *Op. cit.*, p. 652.
[3] "Diseases of the Heart," London, 1832, p. 481.
[4] *Op. cit.*, vol. ii. p. 397.
[5] "The Diseases of the Heart and the Aorta," Dublin, 1854, p. 489.
[6] *Loc. cit.*
[7] *Practitioner*, London, 1883, vol. xxx. p. 179.
[8] "Clinical Lectures on Diseases of the Heart and Aorta," London, 1876, p. 367.

those of Germain Sée[1] on the relative utility of the different iodides, of Huchard[2] on the permanence of the effects of the iodides and their applicability to different varieties of organic angina pectoris, of Fraser[3] upon the real nature of the action of the iodides, and of Stockman[4] upon the mode in which the iodides affect the organism. The observations of these two last-mentioned authors will be more fully dealt with in the sequel.

It may be taken for granted that in a considerable proportion of cases, no matter what may be the type of the affection, the practitioner is called in to minister to the patient during a paroxysm. His duty is therefore at once to relieve the sufferings produced by the attack. It will always occur to the careful practitioner to ascertain whether there be attendant conditions that may perchance underlie or induce the paroxysm. To condescend upon particulars instead of dealing with generalities, let me mention that considerable acidity of the stomach, or moderate distension of the hollow viscera, may by irritation be a reflex cause of an attack of cardiac pain. In regard to the latter factor, it may be added that, when extreme, a distended condition of the stomach and intestines may be a direct mechanical source of disturbance. Interference with the blood current through the lungs is an adequate agent in certain cases, by increasing the stress on the right, just as disorder of the various excretory channels, such as the skin, or the kidney, or the bowel, is an efficient factor, by augmenting the stress on the left side of the heart. Further, the amount of muscular exertion and fatigue, and the extent of mental occupation or exhaustion, must be considered and estimated. In each and all of these directions the proximate agent in the causation of the attack must be if possible ascertained and obviated. Antacids and carminatives for digestive troubles, remedies which will relieve bronchial affections and stimulate excretory channels—such measures will readily occur to the skilful practitioner. The consideration of muscular and mental exertion is of more importance from the prophylactic than remedial point of view, and will be referred to later.

[1] "Thérapeutique physiologique du cœur," Paris, 1893, p. 247.
[2] *Op. cit.*, p. 662.
[3] *Trans. Med. Chir. Soc. Edin.*, 1895, New Series, vol. xiv. p. 152.
[4] *Glasgow Hospital Reports*, 1899, vol. ii. p. 64.

In addition to such measures, certain agents act directly on the circulation, and modify its condition. For this purpose various means are at our service. Amongst the remedies falling under this head the vaso-dilators hold a prominent place. As was, indeed, shown long ago by Fagge,[1] they are not of universal utility, but in those instances attended by, if not indeed consisting in, an increase of blood pressure, they are undoubtedly of much importance. There are two main series, the nitrites and the iodides. Amongst the former are the nitrites of amyl and butyl, or, more correctly, of isoamyl and isobutyl. Amyl nitrite, administered by inhalation, has for years had the pre-eminence amongst the members of this series of drugs. Given in a dose sufficient to produce its physiological effects — dilatation of arterioles, diminution of pressure, and acceleration of heart—it is of the greatest usefulness in terminating many attacks. Isobutyl nitrite has also more recently been frequently employed by inhalation in the painful affections of the heart. Both may be administered in doses of from 3 to 5 minims, and the most convenient method of exhibition is by means of glass capsules. It is difficult to assess the relative value of these drugs, and indeed it seems probable that their effects are largely dependent upon the idiosyncrasies of patients themselves. Each of them has certain disadvantages, such as the sensation of fulness of the head, which often leaves headache and giddiness behind it. These troubles, although, in my own experience, more frequently found to result from butyl, are sometimes even more marked in the case of amyl.

Ethylic iodide, or hydriodic ether, administered by inhalation, is, during a paroxysm, also of great value. The usual dose is 5 minims, and it, like the nitrites, may be employed in glass capsules. Its action is probably due to the liberation of free iodine, which is rapidly absorbed by the blood. It is impossible to say in any given case whether this drug or one of the nitrites will be most serviceable. The remedies can only be judged by their results. Unfortunately, iodide of ethyl, like the nitrites, has the same disadvantages in the occurrence of headache and giddiness. A useful combination is to be found in the capsules of ethyl iodide

[1] *Lancet*, London, 1867, vol. ii. p. 260.

and chloroform, each containing 5 minims of the former and 10 of the latter. The addition of the chloroform has certainly seemed to me to minimise the discomforts often produced by the hydriodic ether.

The administration of powerful stimulants is often necessary. The various forms of ethylic alcohol in common use are of importance. Occasionally brandy and whisky prove troublesome, on account of certain of the particular agents which give bouquet and other characters to the stimulant. In such cases, the *spiritus tenuior* of the Pharmacopœia is of real use. Sulphuric ether, spirit of ether, and Hoffmann's anodyne, as well as the combinations of ammonia with alcohol, are also of great utility; it is sometimes also necessary to go the length of administering ether subcutaneously.

In many a case, such measures require to be superseded by the employment of anodynes, of which general anæsthetics, such as chloroform and ether, take the first place, the subcutaneous administration of morphine being occasionally demanded by the conditions present. Many paroxysms are only amenable to such measures, and there need be no reluctance in their employment, seeing that, in the dosage employed, they can only act as stimulants to the circulation, as well as sedatives to the nervous system.

It has been known, almost from the earliest observations, upon cardiac pain, that respiratory exercise is useful during the paroxysm. Indeed, John Hunter, in his graphic sketch of his own condition,[1] mentions his belief that he had prevented himself from dying by deep breathing. There can be no doubt that deep respiration does to some extent alleviate the suffering in many cases, in those which are characterised by extreme arterial pressure, as well as in those attended by marked cyanosis, but more particularly in the latter group. How are we to explain these undoubted facts? It seems probable that these forced respiratory efforts act by unloading the right cavities of the heart, and also by relieving the pressure upon the aorta, although this latter effect must of necessity be comparatively insignificant. Lastly, before leaving the question of management of the paroxysm, it seems almost needless to add that

[1] *Loc. cit.*

change of position and selection of the posture most comfortable for the patient will require attention, while abundant fresh air must be secured, if the patient has an attack in the house. The application of heat externally to the chest may often be of use.

It may be assumed that in the preventive treatment of every variety—but above all, in the management of such as appear to belong to the organic type—the practitioner will give full directions regarding general measures, many of which consist in the usual treatment of heart failure. Food, sufficient in amount and quality for the wants of the system, and yet at the same time of a nature so as to cause no digestive embarrassment, such as abdominal distension; fluid in ample supply, so as to secure the flushing of the tissues; exercise[1] to such an extent as may stimulate the energy of the muscular, and maintain the efficiency of every other system; rest, in so far as may be necessary for the recuperation of the body; occupation, of a kind that will prevent the mind from dwelling upon the symptoms of disease; sleep, sufficient for the adequate repose of every part of the body—such are the general measures which will naturally occur to every one in attempting to obviate cardiac pain. It may be added that modification of surroundings is often of paramount importance. This appears to be useful, not only by alteration of occupation, but even by the mere change of environment.[2] It may be well, at this point, to express my conviction that several modifications of exercise are of great importance. The use of regular and systematic employment of the muscles of the thorax and abdomen cannot be over-estimated. Forced inspiration and expiration, especially when attended by appropriate movements of the shoulders and arms, will be found of real value in stimulating the circulatory organs; while forced contraction, followed by relaxation of the muscles of the abdomen in a

[1] It may be remarked in passing, that Heberden mentions the case of a gentleman who became relieved of further attacks of angina pectoris, by taking half-an-hour's exercise every day in sawing wood!

[2] Since the delivery of this lecture, Dr Burton-Fanning of Norwich has communicated an interesting fact to me which bears out the statement made above. Six years ago, he kindly asked me to see a medical friend in Norfolk, who suffered from severe cardiac pains, which we concluded to be of gouty origin. Under the treatment upon which we agreed, he improved to a considerable extent, but did not obtain entire relief until he disposed of his practice and settled in a different district.

rhythmic manner, will likewise be found to exercise a most beneficial effect upon the contents of that region. As the abdominal viscera are apt to become engorged with stagnating blood, when there is too little exercise, the rationale of these remarks will be easily apparent.

The employment of massage, in the case of patients who are unable to take much active exercise, is of the highest value, by the promotion of all the tissue changes. In utilising this particular method of treatment in cases of heart disease, the medical attendant must carefully watch the effect of massage, lest the blood should become surcharged with the products of metabolism, and the condition of the patient should, in consequence, be rendered worse. In addition to massage, the resistance exercises, which have been so much in evidence during the last decade, may be employed before the patient is able to take active exercise. These exercises should always be, like massage, carefully watched by the medical attendant. In patients who are not in such a bad case, it is well to enjoin a certain amount of out-door exercise, carefully watching the result produced.

Over and above all these general means, we have to consider the details of special management, and the appropriate drugs for particular conditions. Here the important fact must not be overlooked, that the different varieties of cardiac uneasiness own a complex causation. This primary consideration renders it necessary to take up different groups of the affections, and deal with them separately.

Turning, in the first place, to the organic types of the affection, it may be laid down as the first principle that the causes must, if possible, be removed by the employment of alteratives, aided, according to circumstances, by the administration of cardiac tonics, and supplemented, when necessary, by the use of vaso-dilators. Among the different alteratives, there are none which in any degree approach the iodides in value. A great deal of admirable work has been expended upon the attempt to discover how the iodides produce their beneficial effects. It has been held by many authorities, and the view has received the sanction of Balfour,[1]

[1] *Op. cit.*, second edition, 1882, p. 459; and "The Senile Heart," London, 1894, p. 275.

that they owe their efficacy to the reduction of pressure in the arteries, as well as to the retardation of the pulse rate. Inasmuch as these effects are found, experimentally, to be insignificant, this view cannot be seriously maintained. It is more probable that the belief of Fraser[1] is true, *i.e.*, that these drugs owe their utility to the removal of morbid products. That this action does take place cannot for a moment be seriously doubted. An interesting explanation, suggested recently by Stockman,[2] is worthy of careful consideration. Reasoning from the facts known as to the action of iodine upon the thyroid gland, he argues that the effect of the iodides is produced by a stimulation of thyroid secretion. That there is a considerable element of probability in this suggestion will be allowed by every unbiassed reader, and there can be no doubt that many of the effects produced by thyroid extract in circulatory affections which are attended by high pressure, afford a considerable amount of evidence in favour of Stockman's view. But it is only right to add that it is obviously not the whole truth. Thyroid extract, when employed by me in cases of arterial degeneration and cardiac pain, has proved disappointing when compared with the iodides.

The important question naturally presents itself—In what form is iodine most successfully administered? A considerable amount of labour has been expended upon the attempt to discover whether the different combinations possess effects varying according to their chemical compositions or molecular weight. So far, it seems unlikely that any practical result will accrue from such inquiry, and the selection of that form which is of most use is still based upon unadulterated empiricism. The iodides of potassium, of sodium, of strontium, of calcium, have each of them voluble advocates, with almost as much to say in favour of one as of another. The iodide of potassium still holds its own, and probably will long continue to do so, as that which is on the whole most useful. It may be administered in solution along with various remedies which will disguise its nauseous taste, or it may be given in the form of compressed tabloids, which may conveniently be carried about in the pocket. It sometimes happens that this drug is not well

[1] *Loc. cit.* [2] *Loc. cit.*

tolerated, in which case one of the other iodides may be employed, or the potassium salt may be combined with arsenic—in itself an admirable cardiac tonic—which has a remarkable power of averting iodism. Cases occasionally occur in which none of these compounds of iodine can be borne, and when this is the case it is advisable to fall back upon hydriodic acid, which produces all the beneficial effects of an iodide, and may usually be administered for lengthy periods without producing any untoward effects. The syrup contains 1 per cent. of hydriodic acid by weight, and may be given in 1-drm. doses, gradually increased and well diluted.

It need scarcely be added that in many instances, when there is any failure of the heart, preparations of iodine must be combined with cardiac tonics, and the whole treatment in such cases must be that of heart failure. It is probably also unnecessary to point out that in all cases in which the employment of the iodides is indicated, their effects are aided by carefully watching over the channels of elimination. A mild mercurial aperient for a few days, and a saline draught every morning, are powerful aids in the treatment of the organic types of cardiac pain.

When it is found that there is any tendency to a rise of pressure in the arterial pulse, it is certainly good practice to administer one of the vaso-dilators continuously in small dosage. Here, again, the selection of a particular drug must be based upon the effects which are found to follow its administration. Nitroglycerin, or trinitrin, is one of the most powerful, if it is not indeed the most potent, drug of its class. Administered in a dose of from $\frac{1}{200}$ to $\frac{1}{50}$ gr., it produces most remarkable effects as a vaso-dilator. Its most convenient administration is in the 1 per cent. solution, of which a dose of from $\frac{1}{2}$ to 2 minims may be given at first. In many instances as much as 10 minims may be exhibited every four hours. The tablets, each containing $\frac{1}{100}$ gr., form a very useful mode of exhibition. When desired, the drug may be given hypodermically, the usual solution being made by adding to 5 minims of the trinitrin solution 2 minims of 90 per cent. alcohol, and making up to 12 minims with distilled water. Of this, from 1 to 4 minims may be administered hypodermically. Erythrol tetranitrate, or tetranitrin, is often used in a dosage of

1 to 2 and 3 grs. The best method of administration is by means of the tablets, each of which contains ½ gr. or 1 gr. Mannitol, pentanitrate or pentanitrin, and hexanitrate or hexanitrin, are also sometimes employed in the same doses as the erythrol compound, and, like it, can be best exhibited in tablet form, each tablet containing 1 gr. All these drugs are nitrates, not nitrites, but they undoubtedly act by setting free nitrous compounds when absorbed. It is generally believed that nitroglycerin is most rapid and powerful in its effects, while the mannitol compounds are slower and weaker, the erythrol preparation holding an intermediate position, and that the results are in an inverse ratio as regards duration.

After very considerable experience of all these remedies, the conclusion has been forced upon me that in nitroglycerin we possess the most satisfactory, from every point of view. It may be wondered why sodium nitrite has not been referred to. In my hands it has not proved worthy of comparison with the remedies just discussed.

In those cases of cardiac pain resulting from dilatation of the heart, whether from some previous debilitating disease, or from simple strain produced by excessive stress, the management requires a similar line of treatment to that appropriate for cardiac failure. When such cases occur in those who may be assumed to be free of any arterial and cardiac degeneration, abundant rest, with graduated exercise, and the administration of strophanthus with nux vomica, will induce gradual and continuous recovery.

When we turn to the varieties of cardiac pain dependent upon toxic conditions, we find ourselves face to face with some problems even more difficult of solution than those with which we have been dealing. It is sometimes comparatively simple, nay even perfectly easy, to determine the nature of the poison which has produced the uneasiness. This is the case, for instance, in certain well-marked forms of angina pectoris which follow influenza and other microbic diseases. It is, on the other hand, at times extremely difficult to discover the secret poison in those forms which depend upon the presence of certain chemical poisons, *e.g.*, tobacco

and alcohol. It is, further, a matter of the utmost difficulty to be certain that our inferences are correct as regards the metabolic products which may be supposed, in cases of irregular gout, to be the cause of painful cardiac affections.

In all these toxic varieties of cardiac uneasiness there are three points to bear in mind—the elimination of the poison, the restoration of the cardiac energy, and the restitution of general nervous tone. It need not be insisted that in cases dependent upon the chemical poison which has been used by the patient, such as tobacco or alcohol, the further employment of the poison must be absolutely prohibited. In those varieties which depend upon the presence of a toxin, or other result of some microbic agent, the removal of the poisonous material can best be influenced by stimulating general metabolic activity. It is a common experience that in such cases time is the most important factor in bringing about the beneficial result, and that it is of no use to attempt to hurry the processes of nature unduly. Long-continued rest with massage at first, passive exercises later, and graduated active exertion subsequently, should be enjoined. The use of baths, with this end in view, will be found beneficial; the stimulation of every channel of elimination should further be practised. In addition to employing any sedative remedies which may appear to be called for by the immediate symptoms, it might be expected from reasoning by analogy, that the employment of strychnine would promise the best results. This, indeed, has been taught us by universal experience. The drug may be given in the ordinary way internally, but it acts much more powerfully when administered subcutaneously. It certainly produces effects far more valuable in the majority of cases of toxic angina pectoris than any of the drugs belonging to the digitalis series. There is one class of the toxic variety for which, practically speaking, a complete antagonist can be found, *i.e.*, that in which the cardiac pain is dependent upon the presence of gout. When we are able, in consequence of the presence of some of the protean manifestations of this condition, to assume with reasonable probability that the symptoms depend upon its presence, we shall find that the administration of the specifics, as they are called, is followed by a gratifying

diminution and even disappearance of the cardiac symptoms. In the robust or sthenic type of the gouty diathesis, the administration of colchicum along with alkalies will be our sheet-anchor. Sometimes these may be employed along with digitalis or strophanthus. In the weak or asthenic, guaiac with cinchona, to which, in many instances, a small amount of colchicum may be added, will be found most helpful. Here again the addition of one of the cardiac tonics may seem advisable. Although much inferior to the drugs just mentioned, piperazin, lysidin, uricedin, and other modern representatives of the solvent class, may be taken advantage of.[1]

The reflex variety of angina pectoris, if indeed such a condition really exists as a distinct entity, will entail in the first instance a careful search for the cause of the disturbance. As has already been said, the addition of reflex disturbance to organic and toxic varieties of the affection is very common, and it may be that such a condition as pure reflex angina does not exist. It seems to me, nevertheless, somewhat doubtful. All sources of reflex disturbance will require to be investigated, no matter with what particular system of the body they may be connected. As a simple example let me mention distension of the abdominal viscera. When such conditions are discovered and rectified, recovery, as a rule, ensues; but the removal of the symptoms will be facilitated by the

[1] In my opinion the reason for much scepticism as regards the utility of colchicum in this age is to be sought in the insufficient quantities which are administered. In those of early middle life who are prone to the uric acid diathesis, and who, from the nature of their occupations, obtain insufficient exercise, while their appetites probably would be termed excellent, præcordial uneasiness is extremely common, and, unless checked, bodes more serious trouble in the future. When the group of symptoms under discussion presents itself, it can be checked and removed by careful regulation of diet and arrangement of exercise; the administration of a mild mercurial followed by a gentle saline remedy—both to be repeated every third or fourth day; and by the exhibition of colchicum with an alkali. No doubt, seeing that idiosyncrasies sometimes exist in regard to colchicum, it is always wise to begin with a small dose, such as 10 minims of the wine with 5 gr. of citrate of lithium. But the point which seems to me worthy of urging is, that if these drugs are tolerated—as they are in nine of every ten patients—the quantity of colchicum may be doubled and doubled again. My own general practice is to give for two days 30 minims of colchicum wine and 10 grs. of lithium citrate well diluted, before breakfast and dinner, and thereafter one dose of that size before dinner only. The beneficial effects do not show themselves satisfactorily unless a certain amount of diarrhœa is induced.

employment of such drugs as lessen reflex irritation. Here the bromides play a most important part. Of them the one to be employed must be selected on general principles. In many cases hydrobromic acid is of even greater utility, and its combination with strychnine will at once improve the tone of the nervous system, while diminishing the tendency to reflex disturbance from irritation.

The painful symptoms dependent upon vasomotor angina pectoris, if this again can really be held to constitute a special and separate affection, will receive most benefit from the use of the nitrites. In such cases thyroid extract has undoubted utility, especially when there is much tendency to symptoms resembling Raynaud's disease. Even here, nevertheless, whether thyroid extract be given or not, it is advisable to employ nitroglycerin.

The neurasthenic and hysterical varieties of cardiac pain yield to the general management of the conditions with which they are connected, and it would lead us too far afield if we were to follow out in detail the management of these conditions. Suffice it to say, that the modern and scientific treatment of such affections yields gratifying results in the disappearance of the cardiac as well as of the other symptoms.

SECTION II.—MOTOR DISTURBANCES.

LECTURE IV.—RATE.

THE first three lectures were devoted to the sensory disorders of the heart and the sensory symptoms of cardiac disease; for the remaining lectures we have left for discussion the disturbances of motion. Although the sensory disturbances of the heart are, in themselves, sufficiently complex, the difficulties which surround them fade into insignificance as compared with the troubles that beset the problems connected with the motor disorders of the heart. We shall, indeed, doubtless discover that these lectures will be all too short for the purpose of their consideration, and it will be necessary to restrict our purview to such aspects of the subject as are of real practical importance. Before entering upon the matters which are to occupy our attention, let me, by way of short preamble, mention how deeply engrained upon every literature are references to the movements of the heart. Looking back into the past, frequent references to this subject are to be found in the great epic of Homer.[1] They are to be seen in the philosophic pages of Lucretius[2] and the classic lines of Horace[3]; they are to be read in the prophecies of Isaiah[4] as in the plays of Shakespeare.[5] The movements of the heart thus appreciated by the individual have therefore given a very large amount of imagery to the poetry of all time. It is, nevertheless, quite beyond doubt that the thorough study of this subject is of comparatively recent date. It must, however, be admitted that the pulse underwent a very careful analysis in a

[1] "Iliad," Book I. v. 225.
[2] "De Rerum Natura," Book I. v. 922.
[3] "Odes," Book I. Od. 23, v. 8.
[4] Chap. ix. v. 9.
[5] *King Lear*, Act II. Sc. iv. v. 163.

comparatively early age. Aristotle[1] discovered the pulse to be simultaneous throughout the body; Herophilus[2] found that the pulse and heart beat were synchronous; Rufus of Ephesus[3] gave a very excellent clinical analysis of different kinds of pulsation; yet from the days of Galen,[4] who wrote seven treatises upon the pulse, down to the time of William Harvey,[5] there was no alteration in the teaching as regards it. During even more recent times there must have been no consideration at all of motor disturbances, for, until the publication of the magnificent work of Senac, in the first half of the eighteenth century, little notice was given to the subject. In that work there is a very fair discussion of palpitation and of syncope[6]; that is practically the earliest of the scientific attempts to make out changes in the action of the heart. Morgagni,[7] who followed shortly afterwards with his monumental work, gave a whole epistle—the twenty-third—to palpitation and heart pain, to which there will be occasion to refer later. In that epistle he advanced still further in the analysis of motor disturbances. Even when we take up the work of Laennec,[8] we find how very little attention was devoted to this subject, and it is only in the days of Walshe[9] and Stokes[10] that the consideration of this subject had an effective commencement.

Before we enter upon any of the disturbances, it is advisable very briefly to refer to some preliminary matters. It is unnecessary to make any remarks upon the innervation of the heart. This was thoroughly described last winter from all points of view; there are, however, a few other subjects that must be recalled to your recollection to-day. The first matter arresting our attention is connected with the cause of the cardiac movements. These were originally considered by Haller[11] to

[1] "On the Parts of Animals," trans. by Ogle, 1882, p. 205.
[2] Trans. by Daremberg, *Rev. scient.*, Paris, 1881, tome i. p. 12.
[3] Trans. by Daremberg and Ruelle, 1897, p. 219.
[4] "Opera omnia," 1542, tom. i. p. 823, etc.
[5] "Exercitatio anatomica," 1628, p. 24.
[6] "Traité du cœur," 1749, tome ii. p. 481.
[7] "De Sedibus et Causis Morborum," 1761, tom. i. p. 235.
[8] "Diseases of the Chest and Mediate Auscultation," trans. by Forbes, 1834, p. 655.
[9] "Diseases of the Lungs and Heart," 1851, p. 426.
[10] "Diseases of the Heart," 1854, p. 492.
[11] "Elementa Physiologiæ," 1757, vol. i. p. 430.

be automatic, and due to an inherent power of contraction belonging to the heart itself; this view was also held by Senac,[1] although he modified it in regard to the external sources of interference. When Remak[2] discovered the ganglia at the junction of the sinus and of the auricle, Bidder[3] those between the auricle and the ventricle, and Ludwig[4] those between the two ventricles other views prevailed, and Rosenthal[5] formulated the theory that the heart's beat was due to an automatic action on the part of the intrinsic ganglia of the heart, and not to the muscle itself. With the progress of time these views were modified. The observations of Eckhard,[6] and Foster and Dew Smith,[7] upon the various effects produced by experiments on the ganglion-free apex, and the further investigations of Bowditch[8] and Merunowicz,[9] on the same part with nutrient fluids, altered our conceptions. The result of these observations was the theory that continuous impulses flowed from the ganglia in the sinus and produced the rhythmic action of the heart. Gaskell's experiments showed that each part of the heart has a rhythm of its own,[10] and that the special rhythm of each part is due to a morphological difference in structure. Engelmann[11] found the ganglion-free veins to be the starting-point of the cardiac movements, and we have therefore come back once more to the conception that the movements of the heart are due to its own inherent automatic peculiarities. But we know that these movements of the heart are under the domination of the nervous system from outside. These very ganglia that have been referred to are now known to be simply the outlying or peripheral ganglia of the efferent cardiac fibres of the vagus nerve. They can interfere with the cardiac action in various ways, but never originate it.

In recent times there have been some interesting and im-

[1] *Op. cit.*, tome i. p. 329.
[2] *Arch. f. Anat., Phys. u. wiss. Med.*, 1848, S. 139.
[3] *Ibid.*, 1852, S. 172. [4] *Ibid.*, 1848, S. 139.
[5] "Bemerkungen u. d. Thätigkeit d. automat. Nervencentra," 1875, S. 1.
[6] *Beitr. z. Anat. u. Physiol.*, 1858, S. 145.
[7] *Journ. Anat. and Physiol.*, 1876, p. 735.
[8] *Ber. d. k. Sächs. Gesellsch. d. Wissensch. z. Leipzig*, 1871, S. 682.
[9] *Ibid.*, 1875, S. 254. [10] *Journ. Physiol.*, Cambridge and London, 1883, vol. iv. p. 43.
[11] *Arch. f. d. ges. Physiol.*, 1897, Bd. lxv. S. 109.

portant observations on the ways by which the rate and rhythm of the heart may be modified. Amongst the observations to which reference must be made are those of Wenckebach[1] and Cushny.[2] But it is only right to say that, antecedent to their observations, Mackenzie[3] had come to similar conclusions.

Wenckebach points out that the results of stimuli depend upon the region acted upon as well as the time of stimulation. The accompanying scheme (Fig. 18) shows the rhythm of the great veins entering the sinus, of the auricle, and of the ventricle; in it each beat is normally separated from the succeeding beat by a space equivalent to 20 units of time. If the ventricle is stimu-

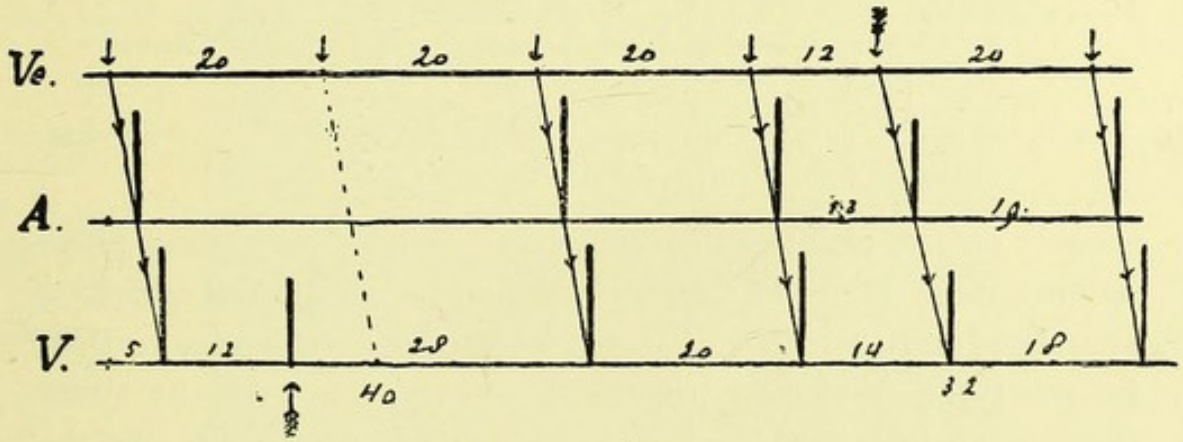

FIG. 18.—Scheme of pulsation of vena cava, auricle and ventricle. For description see text.

lated electrically as at the arrow below, it contracts before its time, but there is not the very least interference with the subsequent rhythm. The sole result is that the ventricle does not again contract during that cycle, but the succeeding pulsation takes place exactly 40 units of time after the original pulsation. On the other hand, if any impulse is applied to the sinus as at the arrow above, at a shorter period of time than the 20 units, contraction takes place, and it is at once carried from the sinus to the auricle and on to the ventricle, so that there is a shortening of this period; the subsequent period, however, returning to the normal.

[1] *Ztschr. f. klin. Med.*, 1899, Bd. xxxvi. S. 181; 1899, Bd. xxxvii. S. 475; 1900, Bd. xxxix. S. 293; and reprint from *Verhandel. d. k. Akad. v. Wetensch.*, Amsterdam, 1903, p. 2.

[2] *Journ. Exper. Med.*, N.Y., 1899, p. 327.

[3] *Journ. Path. and Bacteriol.*, Edin. and London, 1894, vol. ii. p. 314.

The next figure (Fig. 19) is intended to show that so long as the pulsation of the sinus itself is not interfered with, the rhythm persists; when a stimulus is applied to the auricle just a very short time before it would be stimulated by the impulse which comes down from the sinus, it causes the auricle to beat slightly before its right time, 18 units instead of 20, and the ventricle also

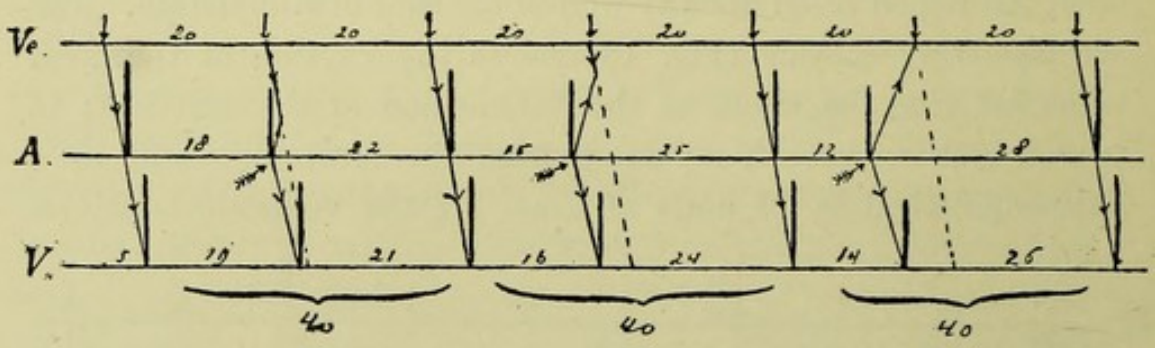

FIG. 19.—Scheme of pulsation of vena cava, auricle, and ventricle. For description see text.

slightly before, 19 instead of 20. But the next succeeding phase occurs exactly after the lapse of 20 units. When the auricle is stimulated a little earlier, if the stimulus does nothing more than intercept the downward passage of the impulse from the sinus, there is no interference with the subsequent movements, and the

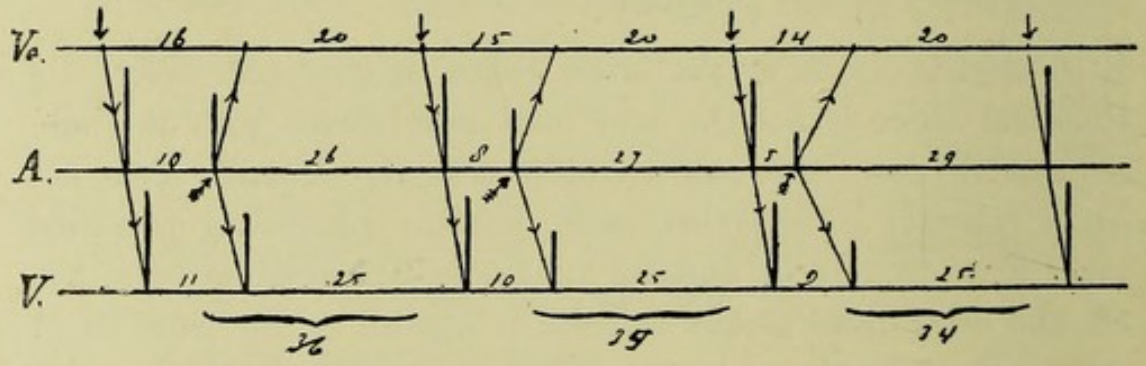

FIG. 20.—Scheme of pulsation of vena cava, auricle, and ventricle. For description see text.

same thing when it is so early as 12 units instead of 20 after the previous beat—so long as the movement does not come before the normal time of contraction of the sinus.

If, however, there should happen to be a stimulus, as in Fig. 20, which takes place at an earlier period, so that it can travel up and not merely intercept but occur before contraction of the sinus, then

the whole phase comes to be considerably shorter. A stimulus after 16 units, followed by the normal 20, gives in all 36 instead of 40 units for the two cycles; and in the same way, if after 15 units, the result of two cycles is 35 units; and, after 14 units, it is 34.

It is clear that if any stimulus occurs, as soon as the refractory period has come to an end, there is a considerable shortening of the phase of contraction.

The observations of Cushny are of much interest in this connection. The accompanying diagram is a tracing taken from his

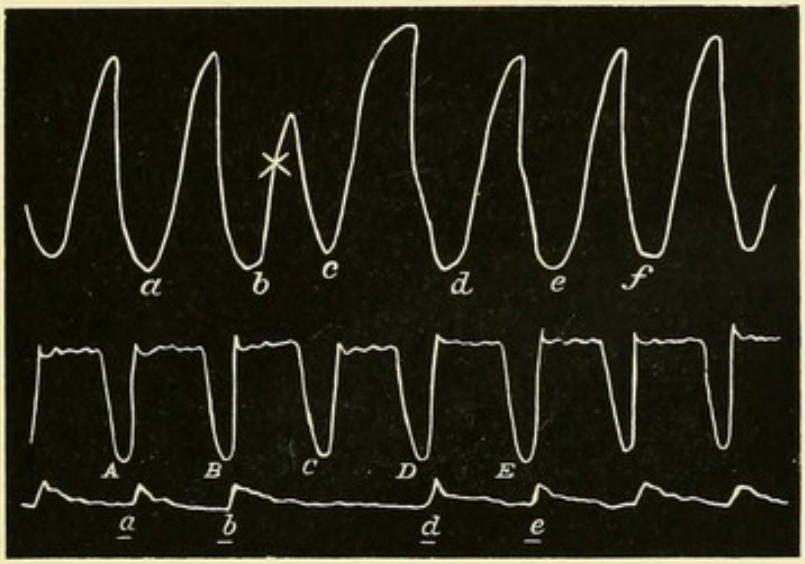

FIG. 21.—The upper tracing was drawn by the ventricle, the next by the auricle, the third by a sphygmograph attached to the carotid artery. During systole the auricular and ventricular levers made a stroke downwards. During diastole they rose again. Stimulation applied to the ventricle at × causes a premature systole of the ventricle, and a missed beat in the carotid, but has no effect upon the auricular rhythm.

work. In it there is a tracing of the radial pulse below, and curves of the pulsation of the auricle and ventricle above. Unfortunately the auricular and ventricular curves are to be read from above downwards—the downstroke is contraction in each case. Now, when a stimulus is applied to the ventricle just before the time when it ought to contract, it does contract, but it contracts feebly, as at *c*, in Fig. 21. It produces no effect upon the auricle whatsoever, but it causes the drop of a beat of the radial pulse. The next figure (Fig. 22) shows a different state of matters, because here the stimulus was applied to the auricle before its

normal time of contraction. There was therefore a short ineffective auricular pulsation followed by a short ventricular pulsation, and the result was the missing of a radial beat. We learn from these observations, that if any stimulus is applied to the heart before the normal time, the premature systole—the early occurring imperfect systole of Mackenzie—takes place, and that it causes a considerable interference with the action of the heart. What that interference is we shall see more clearly in the next lecture, because it is the cause of many forms of irregularity which must then be considered.

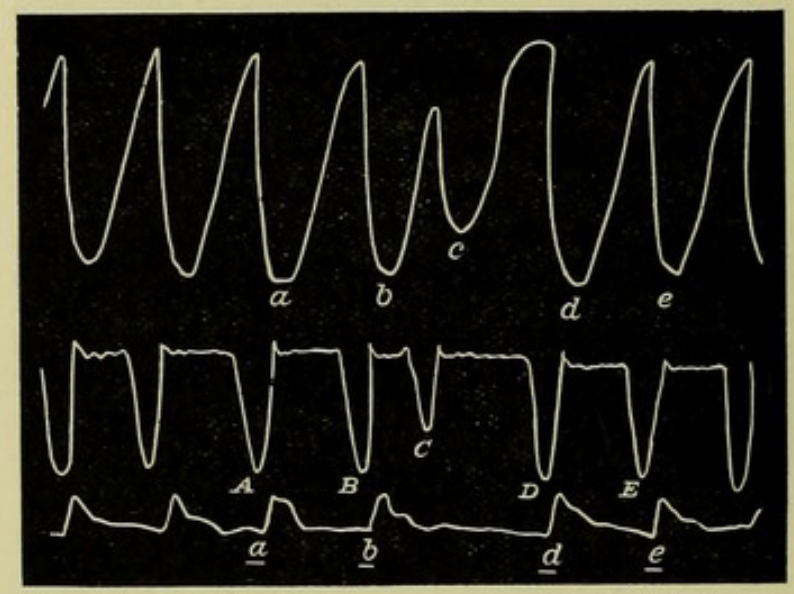

FIG. 22.—Inscribed in the same way as Fig. 4. The electric stimulus was applied to the auricle, and there results a premature systole of the auricle, followed by a premature systole of the ventricle, while the beat is absent from the carotid.

The changes in the rate of the action of the heart constitute the subject which is to concern us for the most part to-day. The changes are twofold: the rate may be accelerated or it may be retarded. As retardation is the easier to grasp, we shall deal with it in the first place. Here perhaps you will allow me to say, parenthetically, that the term slow pulse, often applied to this condition, is objectionable. It is a misuse of the word slow, just as it is a misnomer to speak of a quick when a frequent pulse is meant. It is a total misapprehension of phraseology. If a man crosses the street quickly, it does not mean that he crosses the street often: thus you get the exact meaning of what quick and frequent imply.

Unfortunately, even in the most recent works upon the pulse, this error is perpetuated. It may be pedantic to stand out for purity of phraseology, but, at any rate, my conscience will feel clearer after this expression of my opinion on the subject.

Diminished frequency of the heart's action is commonly called bradycardia. Clifford Allbutt,[1] in his observations upon functional disorders of the heart in his text-book of medicine, falls foul of this word bradycardia, but he allows that tachycardia is a valid term to employ. It is difficult to understand what he means by this. Bradycardia is as much a fact as the opposite condition—this, however, by the way. Bradycardia is sometimes a constitutional peculiarity; we find in almost every work that touches upon this subject, reference to the well known statement that the great Napoleon exhibited the features of bradycardia almost all his life. His pulse had a rate of forty beats per minute, with almost unvarying regularity. It is to be remarked that, as Balfour[2] points out, Napoleon was an epileptic; we know that epilepsy and bradycardia are often associated together. A personal peculiarity, then, is sufficient to give a pulse of very greatly diminished frequency. In addition to idiosyncrasy, infrequency of the pulse may be produced by various stimulations of the inhibitory mechanism; it is very commonly produced in this way by vagal influence. By inhibiting the contraction of the sinus and auricle, such agencies necessarily set the pace for the rest of the heart. These various disturbances may be produced by direct or indirect irritation of the vagus; it is, however, no part of my present intention to-day to analyse with pitiless iteration the various causes which may lead to direct and indirect interference. Suffice it to say that the vagus may be directly interfered with by any cause which presses downwards upon the base of the skull, interfering with the vagal region. This is well illustrated in many a case of increased intracranial pressure. It may further be interfered with by direct pressure on the trunk of the vagus nerve itself, containing as it does accessory fibres, and Czermak is well known[3] to have had the power to press his vagus back upon the cervical vertebræ, thus stopping his

[1] "A System of Medicine," 1898, vol. v. p. 832.
[2] "The Senile Heart," 1894, p. 95. [3] Schmidt's *Jahrb.*, 1868, S. 273.

heart entirely, or regulating its rate, as he liked. Let me say, further, that it seems almost undoubted, that, by the influence of the will, this may sometimes be done. Colonel Townsend, as is described in Cheyne's work,[1] had apparently the power at will of retarding or accelerating his pulse rate; while it is undoubted that Indian fakirs may send themselves to sleep for long periods,[2] and allow themselves to be immured in sepulchres, from which they are afterwards disinterred but little the worse for the experience —they seemingly possess the same faculty. Indirectly, anything which acts upon the vagus mechanism may produce this retardation; and hepatic, gastric, renal, and even more distant causes of irritation, acting reflexly through the vagus system, may interfere with the normal rate of the heart and reduce it.

Toxic agents must be referred to briefly. There are many such toxic agents. We shall see further on that tobacco is one of the most powerful substances that exercise a malign influence over the heart, as is not infrequently shown by a diminution of its rate. Other toxic chemical agents and microbic influences exert similar power, and by acting on the heart itself, or upon the inhibitory mechanism, or upon both, diminish the frequency of the pulsations. Weakness of the cardiac muscle is not at all uncommon, following acute febrile disturbances, as in diphtheria and influenza. Both of these diseases frequently leave as a sequel considerable reduction in the rate of the heart; this is not so much by means of vagus influence as by direct action upon the muscle of the heart itself, because the force of the pulse is more modified than would probably happen if mere vagus influence were solely at work. And there is one other important point. Any increase in the resistance which the blood experiences on leaving the heart tends to reduce its frequency of action; and it is a pleasant duty to pay a tribute to MacWilliam, whose researches are the most convincing, the most thorough, and the most satisfactory we have ever had upon this subject.[3] Those researches prove to us that when there is any increase of the outflow from the heart, the organ is of necessity diminished in its frequency of action. This is

[1] "The English Malady," 1733, p. 307.

[2] Braid, "Observations on Trance," 1851, p. 9.

[3] *Proc. Roy. Soc. London*, 1893, p. 469.

quite in accordance with clinical experience; it is well known that if there is increase of blood pressure, the heart beat is usually diminished in frequency. These are, without dwelling too long upon them, the most important of the causes which lead to diminution of the frequency of the action of the heart.

In an exceedingly interesting case of bradycardia under my observation, the rate of the pulse has usually been from 22 to 28 beats per minute; tracings of the pulse, however, show minute waves intermediate between those appreciable by the finger. It is a matter of importance to note that, between the cardiac sounds which are accompanied by the palpable pulsations, there are faint sounds, occurring once, twice, or even thrice, in the interval. The veins of the neck show pulsations along with, and also between, the radial pulse waves. Simultaneous tracings of the jugular movements and

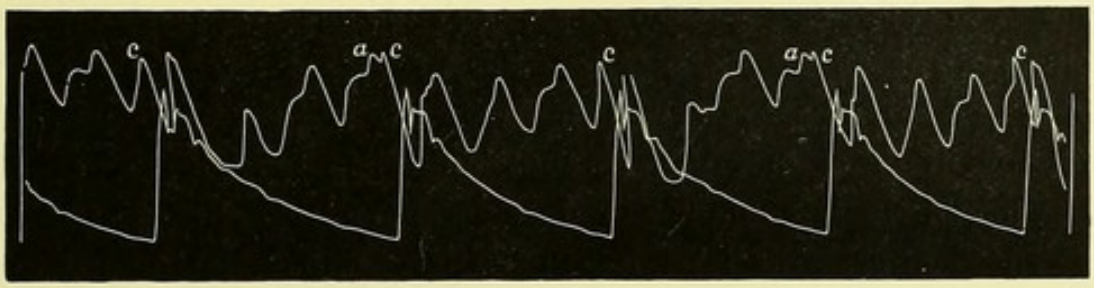

FIG. 23.—Simultaneous tracings from the jugular vein and radial pulse in a case of bradycardia. The upper curve is from the jugular vein, and the lower from the radial artery.

the radial pulse, as shown in Fig. 23, bring out these facts. Now it must be obvious to every one that in such a case as this, the right auricle beats three or four times for each effective pulsation of the left side, or that the auricles pulsate two or three times, without awaking more than a feeble response in the ventricles. This latter supposition is not antagonistic to the assumption of His,[1] which appears also to be adopted by Mackenzie[2] and Wenckebach,[3] that the left auricle can communicate a pulsation to the column of blood in the aorta and arteries, under the high pressure of the arterial system, even with the aortic cusps closed. Far be it from me to minimise the possible effects of auricular contractions. In view of the palpable and audible results of the left

[1] *Deutches Arch. f. klin. Med.*, Leipzig, 1899, Bd. lxiv., S. 316.
[2] *Brit. Med. Journ.*, London, 1902, vol. ii. p. 1411.
[3] "Die Arhythmie," 1903, S. 86.

auricle in mitral obstruction, and of the right auricle in tricuspid obstruction—giving as Mackenzie[1] has shown a distinct hepatic impulse—in view, moreover, of the fact demonstrated quarter of a century ago by Malet and myself,[2] that the auricle produces in health a distinct sound, it must be obvious that auricular pulsations have considerable effects. Yet rational scepticism forbids the unreserved acceptance of the views of His as of universal application.

A reduction of the rate sometimes shows itself by no symptom except the fact that the pulse is much less frequent—that is to say, there is a total latency of the symptoms, while the diminution of the rate often manifests itself in a pulse which is absolutely regular. Occasionally, however, we find a considerable irregularity of pulsation. From the comparison of heart beat and of pulse wave, it is sometimes possible to come to a correct conclusion as to the cause of the infrequency. It may be produced in one or other of two ways. There may simply be a greatly prolonged period of repose, or the effective systole of the heart may be followed by one or more abortive and almost inappreciable pulsations, *i.e.*, some of the beats do not produce an appreciable pulse-wave.

In some cases, at least, it can be proved that the immediate cause of the diminished frequency of the pulse lies in a failure of the wave of contraction to pass from the auricle to the ventricle. To put this fact in other words, a heart block occurs. That the jugular veins sometimes pulsate two or three times for each radial impulse was noticed by Stokes,[3] and this observation has often been verified. In recent times, some important contributions have been made to the subject. The younger His,[4] for example, has published a luminous paper, in which he has proved clearly, that in cases of bradycardia, the auricle has a different rhythm as compared with the ventricle. It is quite true that some authors, such as Jaquet,[5] do not allow this, yet it must be remembered that one positive observation is worth all the negative statements in the world.

In the instance of exceeding importance already mentioned,

[1] "The Study of the Pulse," 1902, p. 195.
[2] *Journ. Anat. and Physiol.*, London, 1879, vol. xiv. p. 4.
[3] *Op. cit.*, p. 302.
[4] *Deutsches Arch. f. klin. Med.*, Leipzig, 1899, Bd. lxiv. S. 316.
[5] *Ibid.*, 1902, Bd. lxxii. S. 77.

this fact of heart-block could not be doubted. The tracing reproduced in Fig. 23 shows this quite distinctly. The upper curve is from the jugular vein, and the lower gives the radial pulse. No one looking at it for a moment could doubt that the right auricle contracts two or three times independently, and then once in harmony with the ventricle. Some of the little waves seen on the radial curve are not quite easy to explain; this is not the place to enter upon a matter of so much difficulty, but in a paper to be published shortly, it may be possible for me to render the subject clear.

When there is great diminution in rate there are usually disturbances elsewhere, and these are generally of nervous nature, often associated with the ordinary symptoms and signs of cardiac weakness. These nervous disturbances may be of the character of faintness; and if the diminution of the frequency should lead to syncope, naturally the faintness and giddiness may become extreme. Along with this, or sometimes instead of it, there are motor disturbances of the whole of the muscular system—in a word, convulsive attacks. These attacks will be referred to in a few minutes more distinctly, and their explanation will then be attempted. Besides such changes as those, it is not uncommon to find that there are mental disturbances. There may be various illusional or delusional changes in cerebral activity. The most important symptoms, nevertheless, are to be found in the consideration of the heart beat and the pulse wave.

When such a condition as bradycardia is found, it is necessary to find out what is its cause; if it is due simply to increased inhibition, it may be removed by appropriate remedies. Such drugs as belladonna or atropine, and the bromides or hydrobromic acid, are the most important for this purpose. If, instead of increased inhibitory influence, there should happen to be toxic effects, these must be as far as possible eliminated. It is sometimes extremely difficult to eliminate a poison. My own personal experience is, that in the case of tobacco, for example, it takes a good six months' abstinence from that weed before its malign influence on the heart begins to disappear, while it often takes six months more before the influence has entirely disappeared. There will be occasion to say a few words in the next lecture as

to different kinds of tobacco and their action on the heart, and to attempt to reach the real reason why tobacco interferes both with the rate and rhythm of the heart. Other toxic agents take a shorter time, but, from the intimate chemical union between tobacco and the nerves of the heart, it is difficult to remove its influence entirely. When we come to such conditions as weakness of the heart after influenza or diphtheria, it is necessary to bear in mind that we have primarily to restore the energy of the heart; and when there is increased resistance to the outflow, it is advisable to lessen it by drugs that will lower the arterial pressure.

There is one special form of bradycardia to which reference must now be made, that is, paroxysmal bradycardia. This has of late years obtained a considerable amount of attention; by the term intermittent or paroxysmal bradycardia is meant a recurrence of greatly diminished frequency of the heart's action. Verily, there is no new thing under the sun. We had, until recently, thought that this condition was first described by Adams[1] of Dublin, and further elucidated by Stokes[2] of the same great school, but, in the pages of Morgagni, two beautiful instances of it attract attention[3]: a grave and honourable clergyman, named Anastasio Poggio, and a certain merchant of Padua, being graphically described as victims of this condition. Burnett[4] and Holberton,[5] moreover, published instances of the affection. Since the observations of Adams and of Stokes, this symptom has been frequently discussed, and Huchard[6] has proposed to term it the Stokes-Adams disease. It is not a disease, but a symptom, and it would be better called the Stokes-Adams syndrome. It consists in sudden accesses of greatly diminished frequency of the pulse, attended by intellectual and muscular troubles. Occasionally it is only syncope that takes place, sometimes it is an epileptic attack. Quite recently some interesting experiences have been allowed me with regard to this affection, since no less than three cases—one in England and two in or near our own city—have been under my observation. It is necessary

[1] *Dub. Hosp. Rep.*, 1827, p. 448. [2] *Op. cit.*, p. 302.
[3] *Op. cit.*, tom. i. p. 70, and tom. ii. p. 420.
[4] *Med.-Chir. Trans.*, 1827, p. 202. [5] *Ibid.*, 1841, p. 76.
[6] "Traité clinique des Maladies du Cœur," 2nd edition, 1893, p. 309.

to explain, if possible, how it is produced. In the very latest work upon these nervous disturbances of the heart, this affection is described by Krehl,[1] who explains it as the result of sclerosis of the coronary arteries. Now the explanation of Krehl is undoubtedly erroneous. There can be no doubt it is due to sclerosis of the cerebral vessels, as is believed by Huchard.[2] The very interesting observations of Webster[3] are in themselves quite enough to disprove the explanation of Krehl. In the tracings which accompany his work, there are no premature or abortive systoles during the period of bradycardia, and if the condition were due to cardiac causes, such would certainly be seen. When we analyse the onset of this symptom, we find that the patient has usually been undergoing some stress leading to strain. Sometimes it may be slight intellectual stress, as writing a business letter; at other times physical exertion, such as flying after a tramway car. It seems to me that the probable explanation is this:—in sclerosis of the cerebral vessels, whenever there is any stress put upon the circulation, the blood supply to the brain is inadequate and ineffective. There is, as a result of that deficient supply, a condition of irritable weakness of the inhibitory mechanism; impulses are sent down to the heart; it is inhibited, and the features of the attack are found.

One interesting point deserving of consideration is the matter urged by Tripier.[4] This author believes that the epileptiform movements precede the infrequency of the pulse, and seems to consider them as the cause of the bradycardia. His views have been stoutly combated by Broadbent,[5] and within the last three years they have received the *coup de grace* at the hands of Webster,[6] whose tracings show clearly that the changes in the action of the heart precede the epileptiform attacks.[7]

As regards the practical subject of treatment, there can be no doubt that rest is the most important therapeutic agent. Due

[1] Nothnagel's "Specielle Pathologie ü. Therapie," 1901, Bd. xv. S. 352.

[2] *Op. cit.*, p. 322. [3] *Glasg. Hosp. Rep.*, 1901, p. 413.

[4] *Rev. de méd.*, Paris, 1883, p. 1001.

[5] "The Pulse," 1890, p. 120. [6] *Loc. cit.*

[7] In the interesting observations of Kussmaul and Tenner ("Epileptiform Convulsions from Hæmorrhage," trans. by Bronner, *New Syd. Soc. Translation*, 1859, p. 7), compression of the carotid arteries is shown to produce convulsions.

attention to the digestive processes is also a matter of real value. Of drugs, so far as my own experience allows me to judge, the most useful are the iodides or hydriodic acid to restore a healthy state to the vessels; nitroglycerin or some other nitrite, if there be any tendency to vascular spasm; and belladonna or the bromides, for the purpose of lessening the action of the vagus.

Turning, in the next place, to increased frequency, or tachycardia, this is sometimes due to personal peculiarities on the part of the patient. There are many individuals in whom the pulse is never below 100. One such patient always occurs to me when thinking of this subject, an Edinburgh shopkeeper known to me for many years, whose pulse has almost never been under 120. He suffers, along with this tachycardia, from agoraphobia. If he finds himself in a large space, like the Grassmarket, he gets into a state of panic, and is never comfortable until he has got into one of the wynds leading into that historic square. In such cases of personal peculiarity there are usually some mental symptoms, analogous to those attendant upon this case.

Diminished inhibitory influence is the commonest cause of tachycardia. This may be induced by pyrexia; by heat, internal as well as external; by poisons of the type of alcohol, belladonna, and tobacco—(though tobacco usually diminishes the heart's frequency, it sometimes increases it); by toxins, such as those of influenza and diphtheria; by organic secretions, as of the thyroid gland, seen in exophthalmic goitre and in artificial thyroidism; mental emotion, and physical exertion—all these causes, by throwing the inhibitory mechanism out of action, cause the heart to act with increased frequency. Increased accelerating influence is very much less certain, but there seems to be no good ground for doubting that, in many cases of pyrexia, this increased acceleration, due to the nervous system, is present; direct cardiac irritation (as in endocarditis, myocarditis, and pericarditis); these are valid causes of increased frequency probably acting in this way. To these must be added cardiac strain, as seen in the irritable heart of soldiers, studied by Clifford Allbutt,[1] Da Costa,[2] and others. Spinal lesions, especially concussion of the lower cervical and

[1] *St George's Hosp. Rep.*, London, 1870, p. 23. [2] *Am. Journ. Med. Sc.*, Phila., 1871, p. 17.

upper dorsal region, will produce similar effects—a patient who was first under the care of Dr. Elder, in Leith Hospital, and afterwards of myself, in the Royal Infirmary, showed this most beautifully. He had, so far as we could make out, a concussion, and there was such a state of instability when he walked a few steps, that he had a tremendous acceleration of the rate of the heart.[1] Reflex influence, lastly, will also set up a great increase in the rate of the pulse, as is often seen in cases of renal mobility.

Tachycardia depends, for the most part, upon a reduction of the diastolic phase. There can be but little doubt that this increased frequency in tachycardia is probably produced, as was suggested by Mackenzie,[2] and shown by Wenckebach[3] and Cushny,[4] by a stimulus which comes to the sinus or auricle before its right time; that is to say, there is either a condition of irritability, or of diminution of control, which allows the normal stimulation of the heart to occur too soon.

Sometimes there is absolute latency of the condition, at other times it is attended by subjective sensations and objective appearances. There may, for example, be palpitation, or even more pronounced sensori-motor disturbance, with physical signs of much cardiac derangement, while mental symptoms are far from uncommon.

In the treatment of tachycardia, removal of the causes weakening the vagus or irritating the heart must be the first indication. Physical and mental repose, with adequate nutrition, must be one of the chief aims. All gastric derangements, and other abdominal disturbances, must be obviated. If there be renal mobility, a bandage must be applied, or surgical measures adopted. Stimulants should be avoided; and the best remedies are to be found in digitalis, strophanthus, and nux vomica, with which may be associated one of the bromides, or hydrobromic acid, when it seems advisable.

It may, however, be absolutely latent. Nothing is experienced excepting by the observer, who makes out that there is increased frequency of heart beat and pulse wave; on the other hand, sometimes there are profound cardiac disturbances, and other

[1] It may be well to add, injuries to the vertebræ in the cervical region usually produce retardation of the pulse. Cf. Hutchinson, *Lond. Hosp. Rep.*, 1866, p. 366.

[2] *Loc. cit.* [3] *Loc. cit.* [4] *Loc cit.*

nervous phenomena. In regard to tachycardia, in general, it is to be treated by getting rid of whatever may be its cause, and restoring the energy of the heart itself.

Lastly, a few words must be said about paroxysmal tachycardia. This complex of symptoms has been much studied since Cotton,[1] Edmunds[2] and Watson,[3] first described it. By the term is meant a recurrent paroxysm of increase in the rate of the pulse. The facts regarding the affection have recently been analysed very fully by Herringham.[4] It is far more common in middle age and after it, but it sometimes occurs in youth; it has even been known in childhood. Its causation is a matter of great difficulty. At present there is no particular view to account for it; but undeniably it must be due, at least in part, to a loss of vagus influence, perhaps also to an increase of the augmentor influence. In another place, a possible analogy to periodic respiration has been suggested by me,[5] —a paroxysmal variation in the functional activity of the cardiac mechanism. Six post-mortem examinations have been made on cases of the affection. Three showed dilatation of the heart, two had interstitial myocarditis, and one revealed fatty degeneration; with these few facts in regard to the morbid anatomy of this affection, we are left to speculate as to its cause.

The essential features of the affection centre in the increased rate of the heart, which is often far above 200 per minute. Tracings of the radial pulse show the condition to depend upon the presence of premature systoles. It may be practically latent, but has sometimes subjective disturbance, while the heart is usually found to be enlarged and its force diminished. Evidence of valvular disease is occasionally present, while secondary symptoms in other organs may be discovered. In attempting to remedy the condition, the same lines must be followed as for tachycardia in general; but the study of posture and the use of a well-fitting bandage may be recommended. In my own experience of this affection, the adoption of respiratory exercises has been productive of much benefit in a few cases.

[1] *Brit. Med. Journ.*, London, 1867, vol. i. p. 629; and 1869, vol. ii. p. 4.
[2] *Ibid.*, 1867, vol. i. p. 721.
[3] *Ibid.*, 1867, vol. i. p. 752.
[4] *Edin. Med. Journ.*, 1897, p. 366.
[5] "Diseases of the Heart and Aorta," 1898, p. 804.

Lecture V.—Rhythm.

In these lectures, it is indeed difficult to avoid, to some extent, a certain amount of overlapping. From the very nature of the subject, it is impossible but that this should, in some degree, take place. The disturbances of the rhythm of the heart, which form the subject of this, have in part been touched upon in the previous lecture, and they will have to be referred to once more in my concluding remarks. It is necessary for me to mention that in regard to some of the disturbances of rhythm it is a misnomer to use the term "nervous affection." As will emerge in the sequel, many forms of altered rhythm are, according to our present lights, produced entirely by changes in the myocardium. As they have, however, to be compared with those modifications of rhythm undoubtedly produced by nervous causes, they must be considered in these lectures.

Starting again on the basis of the automatism of the heart—with the fact that its pulsation is due to its own inherent properties, we have, in the first place, to consider very briefly how the contraction passes from that part of the heart which first beats to the rest of the organ. It used, for a long time, to be a matter of considerable doubt, to be very difficult indeed to understand, how a wave of contraction could pass from the auricular portion of the heart to the ventricular, when, so far as was known, there was no regular muscular continuity between these different parts of the heart. But in quite recent years the researches of His[1] have shown that there is a band of muscular fibres in every mammalian heart, which extends from the back of the right auricle to the region of the attachment of the aortic cusp of the mitral valve. There is,

[1] *Arb. a. d. med. Klin. zu Leipzig*, 1893, S. 23.

accordingly, by this means an absolute continuity of muscular tissue through which a wave of contraction can pass from the auricular to the ventricular part. We have further to remember that the nervous connections, although they have nothing to do with the actual causation of the movements of the heart, may nevertheless exercise considerable influence in conveying the wave of contraction across the organ. We have in these two ways, then, a possibility of the propagation of a wave of contraction from that part of the heart which sets the rate and rhythm at the base to the ventricular part of the organ. Changes in those structures, as will be pointed out later, may quite well interfere with the rhythm of the heart and lead to various disturbances. Not only may the action of the heart be interfered with by those external impulses that have already been referred to in the previous lecture, but in consequence of some alteration in the structure, or even in the metabolism, of the heart itself there may quite readily be disturbances of rhythm. And when we further consider, in addition to such factors as those leading to irregularity, that there may also be differences as regards the stimulus to the heart which is undeniably produced by its fluctuating contents—the varying amount and nature of the blood which its chambers contain—we shall acknowledge with what ease irregularity may be produced.

Irregularity of the heart has undoubtedly been somewhat overestimated in its importance. There can be no question that there are many hearts intrinsically irregular from the cradle to the grave, even in cases when that period covers a very lengthy interval of time. My meaning is that there are many individuals —we meet them indeed daily—who have irregular pulses, and who are not a whit the worse for them. Owing to this common exaggeration of the significance of irregularity, numerous men and women pass the greater part of their lives quite unnecessarily "subject unto bondage." It is absolutely certain that the seriousness of irregularity has in general been greatly exaggerated, and we must recognise the justice of the views on this subject which have been expressed lately by Mackenzie.[1] Let me refer in this connection to the fact that many mammals who are known

[1] "The Study of the Pulse," 1902, p. 106.

to be relatively long-lived, and who are possessed of a large amount of muscular energy, show remarkable irregularity of pulse throughout their whole lives. This is particularly the case with the dog, and more especially with those breeds which we esteem on account of their fleetness and endurance. It is found most remarkably in the staghound, the greyhound, and the collie, all of whose hearts we can make out to be intrinsically irregular.

Now it must be premised very briefly that the normal stimulus of the contraction, which is inherent in the heart itself, is subject, in the first place, to variations in the contents of the auricular reservoirs; and, in the second place, it is subject to interferences caused by the nervous connections. Changes in either of these classes of factors may lead to various types of irregularity. To this subject we must return later. It will be easier to analyse the different causes of disturbed rhythm, after we have considered in what the various types of alteration consist.

We must then, in the first place, briefly consider the varieties of irregularity of rhythm as they may be studied by means of tracings taken from the heart or artery. It is far from my intention to weary you with a long series of cardiac or arterial curves; only a few perfectly typical cardiograms and sphygmograms will be brought before you. There are in the accompanying tracings (Figs. 24 and 25) two common examples of pulse irregularity. In the one case, the irregularity is due to the presence of an early systole—a premature ineffectual systole—most likely caused by structural changes in the heart, or alteration in the amount of blood in it; in the other, it is produced by a lengthening of the period of diastole, which is probably in most cases brought about by inhibitory interference—direct or reflex. In the previous lecture good reasons were adduced for accepting the belief that in the former type of irregularity the heart has been stimulated to contraction too early, as shown by the observations of Mackenzie,[1] Wenckebach,[2] and Cushny[3]; the contraction, however, proving ineffectual, has barely sufficed to open the aortic cusps and allow a wave to be sent into the aorta.

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.*

An observation of Marey[1] illustrates this fact very distinctly, as may be seen in the tracing, Fig. 26. Three cycles of cardiac action are to be seen in the curve, and it will be noticed that they are grouped in pairs, each pair consisting of a larger and a smaller elevation. The dotted line shows the height to which the curve of contraction must rise before the aortic cusps open. It therefore follows that the weaker systole cannot produce any pulsation in the arteries. This is an example of a premature systole without

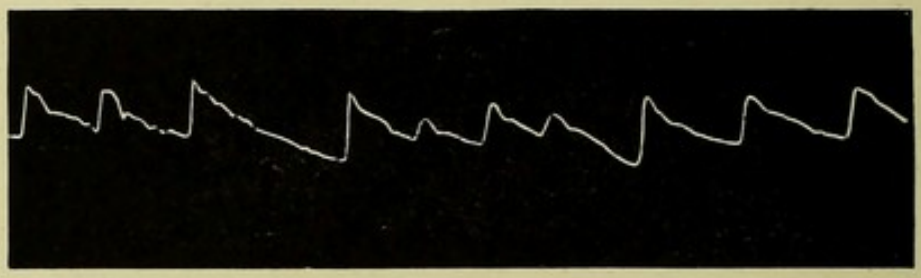

FIG. 24.—Irregularity produced by the occurrence of premature systoles.

arterial pulse. If the early systole were somewhat more powerful, it would give an arterial pulse at once bigeminal and alternate.

There are two main forms of simple irregularity—an irregularity which is arhythmic, and one which is rhythmic. In the tracing, Fig. 27, after a few beats there occurs a premature systole followed by a longish pause. There is, on the other hand, in Fig. 28, a perfectly rhythmic form of irregularity. The heart is beating in pairs of pulsations, but the second pulsation in every group of two

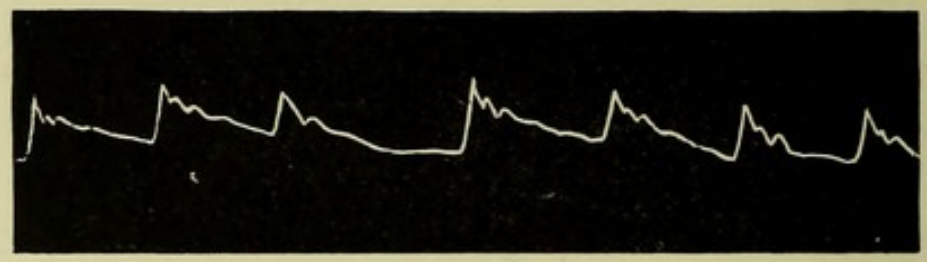

FIG. 25.—Irregularity caused by varying length of diastole.

is a small one, and it is succeeded by a longer pause. In such a form of irregularity the second of the two grouped beats is caused by a premature systole occurring before its proper time, and therefore somewhat ineffective. When we consider such an absolutely

[1] "La circulation du Sang," 1881, p. 122.

arhythmic kind of irregularity, as is shown in Fig. 29, an irregularity which does not only consist in absolute arhythmia of the pulsation, but at the same time manifests a great diversity in the size of each wave—that is to say, a great variation in the amount of force which has produced the pulsation—we recognise that in such a tracing

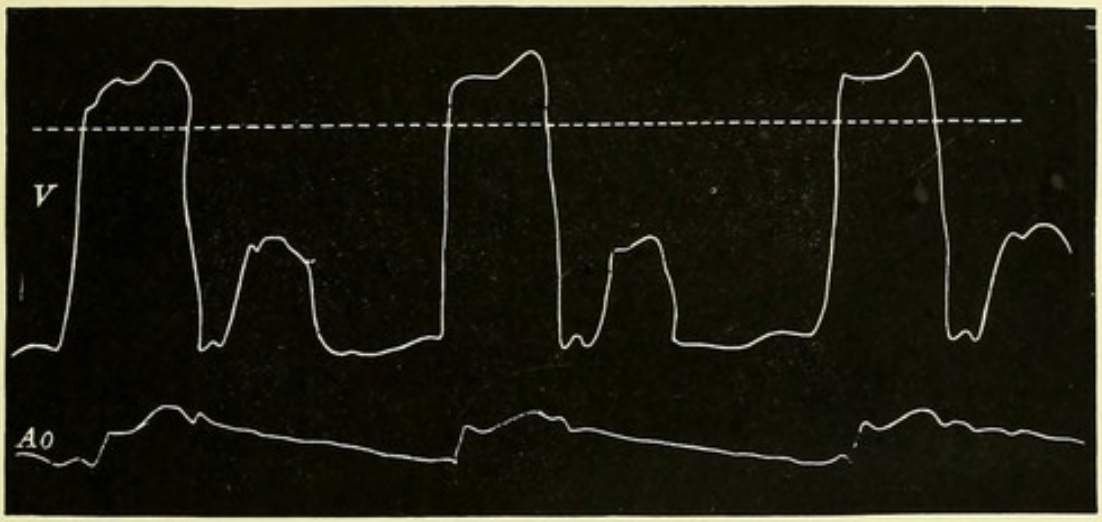

FIG. 26.—Tracing from left ventricle *V*, and aorta *Ao*; the dotted line shows the height of pulsations necessary to open the aortic cusps.

there is a great contrast to what takes place in one of the periodic forms of irregularity; in fact, such a curve gives an approach to delirium cordis.

It is a matter of interest to notice that there are several degrees of the periodic or rhythmic forms of irregularity, according to the

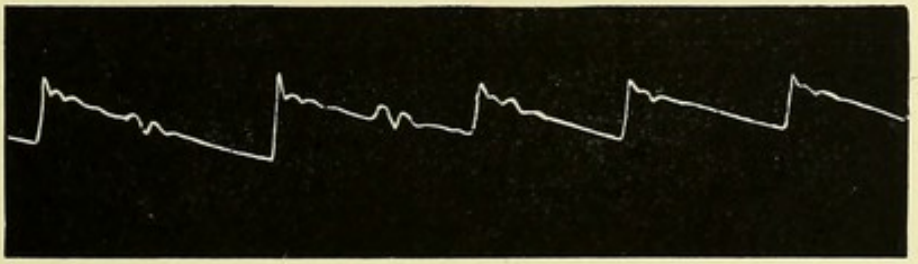

FIG. 27.—Arhythmic irregularity—irregular pulse caused by arhythmic premature systoles; mitral disease.

number of intermissions. There are the pulsus bigeminus, the pulsus trigeminus, the pulsus quadrigeminus, the pulsus quinquegeminus, and even further forms, according to the number of beats in each group. The essential point in these types is that the last pulsation of each group is a premature systole, the occurrence of

which serves to impose on the original rhythm a larger periodicity. In such rhythmic irregularity there is an excellent illustration of the idea so tersely expressed by Herbert Spencer, in his interesting remarks on the rhythm of motion: "Primary rhythms of the organic actions are compounded with secondary ones of longer duration."[1]

In the forms of irregularity of this type, whether rhythmic or arhythmic, the irregularities are usually due to the early and

FIG. 28.—Rhythmic irregularity—bigeminal pulse caused by rhythmic premature systoles; aortic disease.

ineffective contraction of the heart, which has obviously been stimulated in its auricular portion at too early a period. On the other hand, when the irregularity is due to a varying length of the diastole—the usual form of irregularity found in early life—the cause is commonly to be sought in nervous influences. In the pulse disturbances which are found before middle life, this is the most frequent cause which brings about the irregularity. It is not due to any valvular affection, it is not due to any change in the muscular walls of the heart, it is not due to any form of degeneration; it is

Fig. 29.—Arhythmic irregularity—absolutely irregular and unequal pulse; mitral disease.

due to irregular impulses passing down the accessory fibres in the vagus nerve, thus inhibiting the systole, and causing it to be later in its development. In this type of irregularity it is indeed very rare to find any periodicity—there is almost invariably an absolute arhythmia.

[1] "First Principles," 4th edition, p. 261.

The esoteric causes of the various interferences with the ordinary rhythm fall into two well-defined groups—intrinsic, or cardiac; and extrinsic, or nervous. No doubt, as we shall see reason to believe, the two groups sometimes overlap, *i.e.* both are operative in the same individual. A further caution must be expressed. When we trace back the factors to their absolute source, we find that many of them are really the secondary results of more remote causes. For example, as we shall see, some forms of cardiac disturbance are consecutive to arterial changes. Among the intrinsic or cardiac causes are toxic agencies, such as the influence of certain of the toxins which arise in the course of the infections. Of these we have examples more particularly in such fevers as pneumonia, typhus, influenza, and diphtheria. These are far from exhausting the category of such causes, seeing that every acute febrile disturbance may give rise to irregularity of the pulse. One of the most common causes stands next in the list; to it the well-worn term gout may still be applied, although we do not know exactly what we mean when we use it; we may employ it, nevertheless, in the sense of some poisoning of the system produced by faulty chemical processes—an auto-intoxication—of which it is probable that uric acid in excess is only one subsidiary part. Every one knows how common it is to find in the gouty individual a troublesome form of irregularity of the heart. In another category stand three chemical agents—alcohol, tea, and tobacco. These different poisonous substances—to be correct we must allow them to be so—cause varying kinds of irregularity, and produce them in different ways. It is probable that in the case of the toxins, as well as in whatever is the *materies morbi* in gout, there are poisonous results on the nervous system governing the heart, together with toxic effects on the heart muscle itself. In the case of alcohol this is probably also true. There can be no doubt that in an early stage of alcoholic intoxication the vagus is thrown out of action, as the result of which the heart at once races; we must, however, remember that there is also another factor, since alcohol is one of the most potent agents leading to destructive changes in the structure of the heart. Here again, therefore, we have two distinct factors at work. In the case of tea it is a somewhat different causation that gives rise to the ir-

regularity. Although very loth, indeed, to say anything that would curb the use, physiologically at any rate, of that cheering beverage, truth compels me to admit that tea has a great tendency to upset the cardiac rhythm. It does not do so because it produces changes in blood pressure; my convictions are decided on this point, and are based upon a series of unpublished experiments performed by me upon the heart many years ago with theine and caffeine. Tea undeniably gives rise to irregularity through its interference with the nervous mechanism pure and simple. It also produces a tendency to distension of the abdominal viscera; it causes at the same time a weakness and irritability of inhibitory impulses, and these two factors lead to the irregularity to which tea gives rise.[1] As regards tobacco, we know from the researches of Langley,[2] that it has a great tendency to interfere with the synapse between the pre-ganglionic fibre and the nerve cell. It is obvious that in this process of interfering with the connection between the central and peripheral vagus neurons there may be a great amount of disturbance, leading to alteration of the rhythm. Further, as tobacco has the effect of raising the blood pressure, we realise quite clearly how it must necessarily, when abused, lead to disturbance. We shall see in the next lecture that it is one of the most fertile sources of palpitation; meantime you will perhaps allow me to mention that different kinds of tobacco produce very different effects upon the human heart. Certain forms of tobacco are almost innocuous, others are pestilential in the highest degree; and some of the tobaccos that have obtained a very great vogue on account of the literary romance thrown around them, are without doubt the most deleterious of the whole series. There can be no doubt of the fact that of all forms of tobacco the least hurtful is the cigar. The next in point of harmful effect is the pipe, while the cigarette is to be put highest of all in the range of evil possibilities; used as it so often is, by inhalation, it is infinitely worse than any other form of

[1] The observations upon this subject by Percival (*Dub. Hosp. Rep.*, 1817, vol. i. p. 219) are most interesting, and echoed, or rather reinforced, by Stokes (*Op. cit.*, p. 517). Percival states (*Op. cit.*, p. 224) that sometimes tea restores the rhythm of an irregular heart.

[2] *Journ. Physiol.*, Cambridge and London, 1890, p. 277.

smoking. Of other uses of tobacco but little is known to me in any practical manner except as a mere incident.

Enfeebled states of the heart, in the next place, require our notice. Long ago the great Stokes[1] formulated the axiom, "with respect to irregularity, experience shows that this condition is more intimately connected with lesion of the muscles than of the valves of the heart." Such feebleness of the heart may be produced by over-strain and allied conditions. Any long-continued stress leads to strain, strain leads to dilatation, dilatation leads to malnutrition and malnutrition leads directly to irregular action; here it is the heart muscle that is at fault. It seems to me that this is perhaps the best time to try to explain the great irregularity in many cases of mitral disease. In mitral disease, when there is much interference with the left auricle, it is stimulated too soon by the presence of the excess of blood which it contains. This is the first link in the chain. In addition to this, the nutrition of the auricle suffers, leading to structural changes. When the right cavities undergo consecutive dilatation and subsequent organic alterations, the connecting link, the band of muscle fibre which passes from the right auricle to the ventricles, sinks into a state of malnutrition on this account; the combination, therefore, of the weakness of this connecting link, and of the too early stimulus to contraction, leads gradually to the irregularity.

Certain inflammatory affections naturally fall to be considered at this stage. In all forms of pericarditis, but more especially in those which follow acute general diseases, changes of rhythm are common, and are probably due partly to weakening of the muscular walls, and partly to irritation of the nervous connections. It need scarcely be added that when any considerable effusion occurs there is great interference with the action of the auricles—a fact experimentally investigated by Cohnheim[2] and Starling[3]—and much resulting irregularity. Pericardial adhesions, further, act in a quite analogous manner. Endocarditis, for the most part through its attendant muscular weakness, also produces similar effects; and

[1] *Op. cit.*, p. 175.

[2] "Lectures on General Pathology," *New Syd. Soc.*, 1889, sect. i. pp. 21, 31, and 73.

[3] *Lancet*, London, 1897, vol. i. pp. 569, 652, and 723.

myocarditis, whether primary or secondary to pericardial and endocardial changes, is even more potent. Further, of cardiac causes there are the chronic lesions, interstitial myocarditis and fatty degeneration. In such conditions, on account of the interference with the heart, and its consequent weakness, on account, also, probably of the irritation to which the nervous mechanism in the heart is subjected, there comes to be a high degree of irregularity. In many such cases the starting-point is undoubtedly a change in the coronary arteries. Lastly, the heart may fail, as the result of long-continued resistance to the outflow, when there are chronic arterial changes.

The next category does not require any lengthy exposition, reflex and direct nervous influences easily explain themselves through irritation of the vagus nerve. Reflex nervous influences may arise from disturbances of viscera in different parts of the body; direct nervous influences may be purely neurotic or functional in their origin, they may be produced by some irritation of the vago-accessory nerve itself, by interference with the cord or brain, or, lastly, by purely psychical influences as in emotional disturbances. The explanation of the changes of rhythm so produced is to be sought in the fact that there are alterations in the stimuli, modifications in the responses to the inhibitory changes, and possibly accelerator disturbances. As to this latter there is always considerable dubiety. We know little of the accelerator mechanism of the heart when we contrast it with the inhibitory, and in almost every instance where there is alteration, whether of rate or rhythm, it is an inhibitory, not an augmentor disturbance, that causes the change. Here let me mention as an illustration of this statement, an interesting fact brought out by MacWilliam[1] as regards the great difference that exists in two animals, the rabbit and the hare. The former has normally a very frequent, the latter a comparatively infrequent pulse. In the rabbit there is very little disturbance when the vagus nerve is severed, in the hare there is an enormous difference; and it seems probable that the explanation which MacWilliam gives—it is an interesting explanation—is probably quite correct. He shows that the rabbit has no need of staying power or

[1] *Proc. Roy. Soc. London*, 1893, p. 476.

tremendous speed, except over short distances; it does not migrate very far from its home, its burrow is near as a place of refuge; whereas the hare eludes its enemies by speed and endurance, as well as by doubling and twisting. He found from experiments on these two animals, that while there is only a small increase in the rate of pulsation on muscular exertion or on severing the vagus in the neck of the rabbit, there is under the same conditions a very great acceleration in the hare—indeed, it is a common experience to find that the rate goes up to more than 200 beats per minute; irregularity also in the hare is, according to MacWilliam, extremely easy of production.

The clinical features of irregularity chiefly consist in the physical evidence of our senses in examining the heart-beat and the pulse-wave; but it is to be remembered that, along with those various features, there may be a great many attendant phenomena —the patient may suffer from uneasiness or even pain; he may be conscious of weight or heaviness; he may feel tightness or constriction about the chest; instead of these sensations, he may be painfully conscious of the fact that there is a good deal of throbbing and irregularity, with an uncomfortable sensation of pausing or stopping. Some of these phenomena must be taken up in the concluding lecture along with palpitation, and to them it is not now necessary to refer further. Along with such disturbances, there are many kinds of nervous accompaniments which we may also postpone until we deal with the conclusion of our subject.

There are two special forms of irregularity which have to be referred to. The first of these consists in the condition originally observed by Williams,[1] but only generally known since the observations of Griesinger[2] and Kussmaul[3]—the pulsus paradoxus. Under ordinary circumstances it is found that with inspiration the pulse becomes stronger and more frequent. The paradoxical pulse is exactly the counterpart of that. In the pulsus paradoxus with inspiration the pulse loses its force and becomes a little less frequent than during

[1] *London Journ. Med.*, 1850, vol. ii. p. 464.

[2] Unpublished lecture referred to by Widenmann, "Beitrag zur Diagnose der Mediastinitis," Tübingen, 1856, S. 1.

[3] *Berl. klin. Wchnschr.*, 1873, S. 433, 445, u. 461.

expiration. With expiration the pulse again becomes stronger, and somewhat more frequent when it is carefully measured out. The pulsus paradoxus will be referred to in a few minutes at greater length, but let me mention the conditions in which it is most commonly seen—when the pericardial and epicardial membranes are fused together, so that there is obliteration of the sac, along with adhesions round the origin of the great vessels, whereby during inspiration less blood is allowed to pass out into those great vessels, the paradoxical pulse is to be found. It is necessary to grant that this paradoxical pulse is not always confined to that rare condition. In my own experience it has occurred in ordinary adherent pericardium. It also occurs in ordinary pericarditis; in short, we may say that it

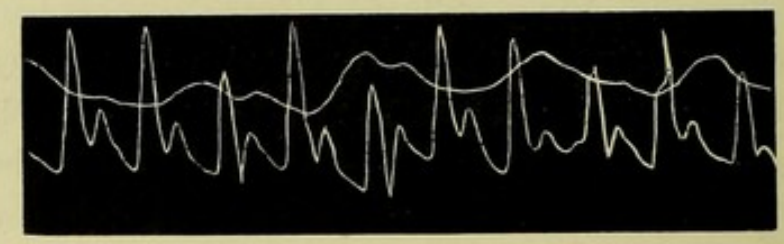

FIG. 30.—Paradoxical pulse from a case of indurative mediastinal pericarditis.

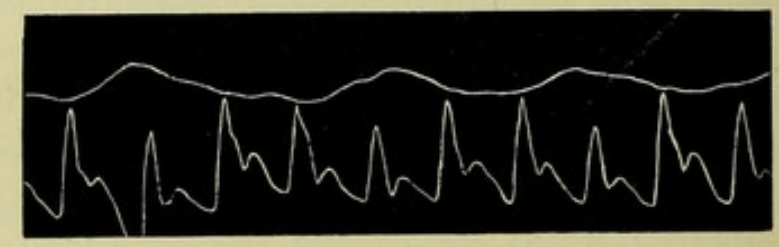

FIG. 31.—Paradoxical pulse from a case of indurative mediastinal pericarditis.

may occur whenever anything lessens the strength of the contraction of the heart, so that during inspiration it is somewhat interfered with in its expulsion of blood into the great vessels.[1] That seems to me to be the explanation of this phenomenon. It is said to occur in perfect health. No example of this has yet come before me. Of this pulsus paradoxus the accompanying tracings (Figs. 30 and 31) furnish excellent illustrations. They are the first two tracings of the condition that have ever been published, giving both

[1] For a summary of views on the pulsus paradoxus, cf. Harris, "Indurative Mediastino-Pericarditis," 1895, p. 61; and *Lancet*, London, 1899, vol. i. p. 1053.

respiratory and arterial curves,[1] and for them my late house physicians, Dr. Bullmore and Dr. Conder, and myself owe our thanks to Dr. Oliphant Nicholson, who is an expert on the subject of graphic records. In the line of the respiration the rise represents the inspiration, and the fall the expiration. The tracings were obtained from a boy in whose case the diagnosis was mediastinal pericarditis. More recently another series of tracings has been obtained by my late house physicians, Dr. Conder and Dr. Henderson Smith, from a male patient, æt. 28, suffering from tuberculous pleurisy and pulmonary consolidation, with undoubted pericardial complications. In the curves so obtained (Figs. 32 and 33) it will be seen that with inspiration the radial pulse almost disappears. In these two last

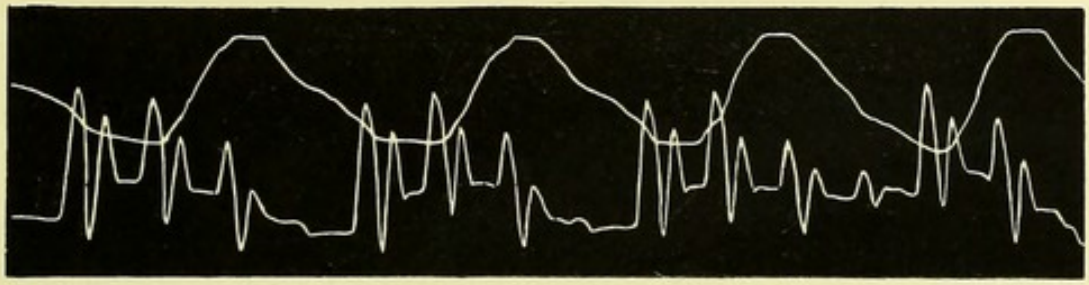

FIG. 32 —Paradoxical pulse from a case of pleurisy and pulmonary consolidation.

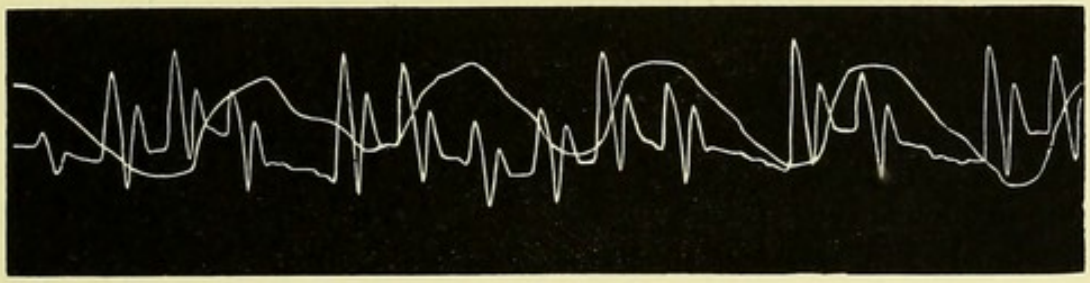

FIG. 33.—Paradoxical pulse from a case of pleurisy and pulmonary consolidation.

tracings it is a matter of interest to note that the pulse is hyperdicrotic.[2]

The second special type of irregularity must now be considered; it is what is called hemisystole. By hemisystole is meant the contraction of one side of the heart, while the other does not pulsate, or contracts somewhat feebly. It is a conception which was sug-

[1] *Practitioner*, London, 1903, p. 212.

[2] Since the delivery of the lecture the patient referred to died, and at the autopsy it was found that the diagnosis was correct—a band of firm fibrous tissue encircled the great vessels. The case has been fully recorded by my House Physician; *v.* Conder, *Edin. Med. Journ.*, 1903, vol. xiv. p. 207.

gested first by Skoda,[1] and afterwards revived by Leyden[2] and Roy.[3] It is an explanation which has been somewhat difficult to accept, on account of what we know as to the structure of the heart. The circular fibres of the heart of both sides are so closely associated, that it is extremely difficult at first sight to grant that the one side of the heart may contract without the other; such was my own belief until a few years ago, when Mackenzie first published his

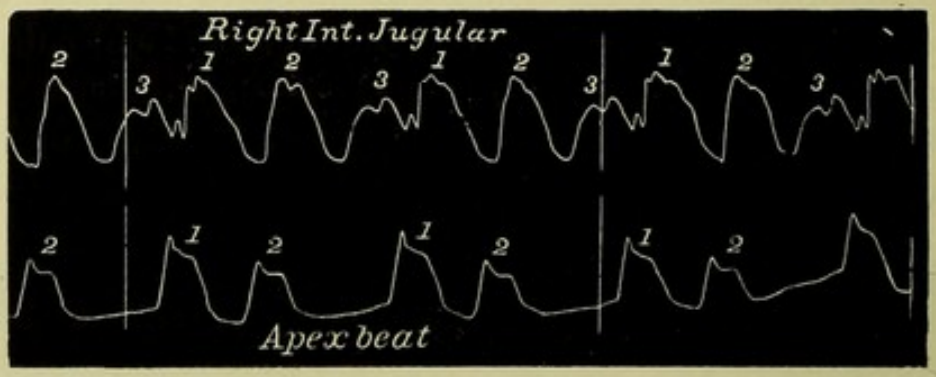

FIG. 34.—Tracings from jugular vein and apex beat in a case of apparent hemisystole.

observations. Mackenzie's work is marked by such absolute accuracy that everything which he publishes must be accepted with a bias in its favour, and these tracings of his go a long way to show that there is a possibility of something like hemisystole. In the tracing shown in Fig. 34, borrowed by permission from the work of Mackenzie,[4] may be seen the movements of the right internal jugular vein and of the apex beat. The interesting point of the tracing lies in the fact that whereas for the first and second contractions of the right side of the heart, as shown by the beat of the right internal jugular vein, there are pulsations also of the apex of the heart—showing that the left and the right sides of the heart were beating simultaneously—during the third pulsation of the right side of the heart there is no corresponding apex beat at all; we must therefore allow that such a tracing as this proves most distinctly that at any rate the left side of the heart may contract so very feebly as to give rise to no tangible evidence of its pulsation.

[1] "Auscultation and Percussion," trans. by Markham, 1853, p. 158.
[2] *Virchow's Archiv*, 1868, Bd. xliv. S. 365, and 1875, Bd. lxv. S. 153.
[3] *Edin. Med. Journ.*, 1878, p. 594.
[4] "Study of the Pulse," 1902, p. 294.

It is perfectly true that such a condition as this can only take place when the heart is in an extremely feeble condition—the state of matters that takes place in experimental physiology when the functions of the heart have been so seriously disturbed that one portion of it alone acts; such a condition as this is only found in the gravest stages of heart failure.

Indications of a converse condition are occasionally seen. In Fig. 35 is a tracing from the right cervical veins, and from the radial pulse, in a case of pericarditis. Now the tracing, Fig. 35, is most interesting, since it shows apparent hemisystole, but not the kind of hemisystole revealed by Mackenzie's tracings,—tracings which, in the main, accord with and support the con-

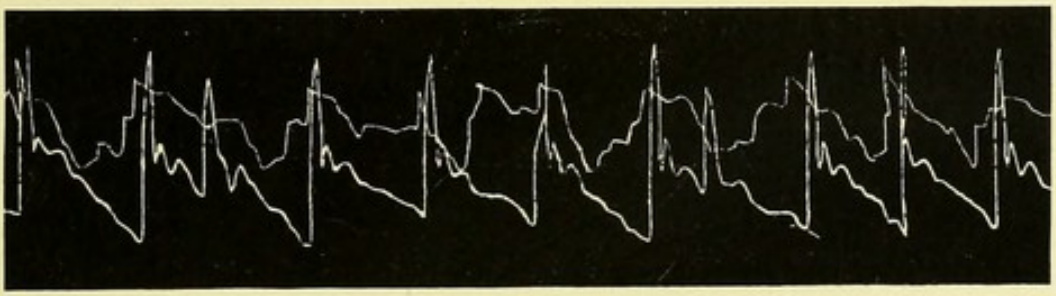

FIG. 35.—Tracing of radial pulse (below) and jugular pulse (above) in adherent pericardium. The second and third, as well as the seventh and eighth radial pulsations, are grouped or bigeminal, but the jugular pulsation is not doubled.

tentions of Leyden and Roy; but it demonstrates that there may be a hemisystole, in which the left side beats without any corresponding contraction of the right. If my interpretation of the curve is correct, it will be seen that twice in that tracing there are actually two pulsations of the radial artery for one of the jugular veins. This can be observed in the two bigeminal or grouped pulsations, second and third, and seventh and eighth, in the tracing. This can only mean that the left ventricle of the heart has, on these two occasions, beat without any corresponding pulsation of the right auricle. The patient from whom the tracing was obtained was nearing his end, and such abnormal pulsations have usually been observed in those who are approaching the termination of life. As the affection from which he suffered was pericarditis, it is possible that there may have been direct myocardial irritation starting an

independent ventricular beat. This is fully dealt with by me in a recent paper.[1]

All that now remains to be said is concerned with the methods of treating pulse irregularities. Once more let me again, even at the risk of being considered wearisome, repeat that the prognostic significance of irregularity is commonly regarded from a point of view which is much too pessimistic. Here, as in all other affections, we must trace the symptoms to their source and treat them according to their causes. When the morbid symptoms are the result of toxic agents, the first indication is to get rid of them. In the case of the microbic toxins time alone will serve to bring about their disappearance, but tonic remedies assist in the process of recovery. The products of faulty metabolism, in gout and its congeners, must be removed by the usual methods. As regards the chemical agents, the first point is to cease using them, and to employ the nervous tonics. When the symptoms are due to weakness of the heart itself, it is very interesting indeed to observe that by strengthening the heart by drugs of the purely cardiac type, such as digitalis and strophanthus, irregularity can be curbed and abolished. This is especially true as regards the irregularity of middle life. With regard to youthful irregularity, its causation is extremely variable. If it is due to some simple emotional or psychical disturbance, the great point is to get rid of that disturbance. If it should be due to a weakness of the vagus system plus some irritation in any other viscus, what has to be done is to allay the irritability of the vagus nerve either by the bromides pure and simple, or by a combination of strychnine and hydrobromic acid, which is found to be extremely valuable, while the irritation elsewhere is allayed. If the proximate cause should lie solely in disturbance of such a neighbouring viscus as the stomach, a very simple line of medication to that disturbed organ, in addition to the hydrobromic acid, will bring the irregularity to an end. There are some other additional points in regard to irregularity, but they fit in more thoroughly with palpitation, and will be discussed in my concluding remarks.

[2] *Edin. Med Journ.*, 1903, N.S., vol. xiv. p. 219.

Lecture VI.—Force.

The concluding part of our subject is now before us; we have to consider variations in the force of the heart. The study of these variations must, in the nature of things, necessarily trench to some extent upon the subjects which we have passed in review during the previous two lectures, inasmuch as variations in force almost always include modifications of rate and alterations of rhythm. As already pointed out, it is therefore scarcely possible to avoid a certain amount of overlapping in discussing these conditions. Variations in force are in physiological circumstances conditioned by a great many different factors. They are under the control, under these conditions, of the nervous system. Whenever the heart is subjected to any excessive strain, an impulse is sent up by the afferent vagus system—the analogue of the depressor nerve in the rabbit—as the result of which an impulse, descending by the accessory fibres, lessens the force of the heart. This mechanism constitutes a natural safety-valve which prevents the heart from manifesting an excessive, and possibly destructive, degree of action in the attempt to overcome the strain to which it is subjected. Such is the anabolic action of the vago-accessory mechanism. There is a possibility in the augmentor system of increasing the force of the heart—a katabolic action; that side of the physiology of the circulation, however, is by no means so well known. From the results obtained by modern physiologists, we know that, as regards its force, the heart is almost entirely governed by the vago-accessory system of nerves. Experiments which have been carried out during recent times upon this subject, show us that, whether in respect of stimuli applied to, or demands made upon, the heart, section of the accelerator and augmentor nerves has comparatively

little effect upon the force of the systole. Any operation, on the other hand, by which the functions of the inhibitory nerves are abrogated, invariably produces very remarkable effects; there cannot, in short, be a greater contrast in the whole realm of visceral innervation than is shown by the behaviour of the inhibitory and the augmentor nerves. Such are the most important of the nervous influences which rule the force of the heart. It is, moreover, to be remembered that the force of the heart is subject to considerable variations, according to a number of different influences which are at work upon it, some apparently almost irrespective of the nervous system. Whenever the pressure that the heart has to overcome is reduced, the force of the heart becomes correspondingly diminished. When the resistance is increased, the force of the cardiac activity is augmented; but if the resistance to be overcome by the heart reaches an excessive level, the force suddenly comes to an abrupt end. It seems, therefore, that just as a certain mean of blood pressure is required to call forth the properties of the arterial walls, so a certain degree of resistance is necessary to bring out the full activities of the heart. It therefore follows that both extrinsically and intrinsically the force of the heart, like its rate and rhythm, is subject to great variations.

The main object of this lecture is to discuss the increase, decrease, and inequality of the force of the heart. It is not necessary to dwell at length upon the various alterations which lead to such changes. They have really been so fully dealt with in the two previous lectures that they may only be adverted to briefly. The same structural changes in the heart and blood vessels that were mentioned are operative here. Analogous, if not identical, toxic effects upon the heart and blood vessels, conditions of stress leading to strain, and lastly, the influence of the nervous system, especially the inhibitory mechanism—all these need only be referred to very shortly. The various abnormal conditions as to the force that will occupy us during this lecture are chiefly summed up in the terms palpitation, tremor, syncope, and asystole.

By palpitation is meant a change in the action of the heart resulting not only in a great increase in its rate and force, but in a subjective sensory disturbance, so that the victim of palpitation be-

comes conscious of a certain amount of discomfort, the most prominent feature of which is throbbing in the chest. It is thus sensori-motor. In my remarks on angina pectoris, the subject of palpitation was briefly mentioned as being in some degree analogous. Palpitation, consisting in acceleration of rate, as well as increase of force in the action of the heart—presenting, moreover, a sensory as well as a motor aspect—necessarily touches upon a good many subjects in cardiac physiology. The chief cause which leads to palpitation is diminished vagus control, and this is brought about in a variety of different ways. In the first place, it may be by the direct influence upon the lower centres of some emotional disturbance higher up. It is well known that this symptom of palpitation is far more common in the first than in the second half of life. It is very much more common in the fair sex than in that which is called the sterner sex, and it is found that year by year, as we advance in life, the tendency to palpitation becomes less, if there be no visceral disturbance. Of emotional disturbance in early life it is necessary to say but little. We all know that during early life the emotions pass through a somewhat prolonged period of stress and strain—the *Sturm und Drang* of Goethe is particularly applicable to the promising bud of adolescence and the opening flower of adult existence. But, in addition to the gentler emotional disturbances which are particularly confined to that period of life, we have also others of a harsher type, such as anger—assuredly one of the most powerful factors in inducing an attack of this kind; many other mental disorders are likewise powerful in the causation of palpitation.

One particular nervous condition is very commonly attended by a feeling of palpitation, *i.e.* hysteria; it is, however, necessary to point out there is such a symptom as hysterical palpitation, so called, which is not to be discovered objectively by physical examination. Clifford Allbutt, in his most interesting and picturesque Goulstonian Lectures, did well to draw the attention of the medical profession to the fact of "palpitation which is never perceived by the stethoscope."[1] In cases of hysteria it has been a common custom to speak of pseudo-angina pectoris, or false angina pectoris; good reasons were assigned in my first series of lectures for the belief

[1] "On Visceral Neuroses," 1884, p. 21.

that any one who persists in perpetrating such a phrase is guilty of an absurdity, and is wanting in common sense. We might speak with less inaccuracy of pseudo-palpitation than of pseudo-angina pectoris. Far be it from me to employ the one term or the other; both are ridiculous and absurd, but it is a greater error to use the phrase pseudo-angina pectoris, than it would be to coin that of pseudo-palpitation. The hysterical condition, then, whatever that may be, is a cause of a sensation of palpitation. To any one desirous of reading some interesting observations on hysterical vagus neurosis, the work of von Noorden[1] may be confidently recommended.[2] The converse of the picture, or a hysterical manifestation of cardiac origin, is very rare indeed.

Of a direct influence upon the vagus mechanism exerted by some gross cerebral lesion, we do not, although cases of palpitation thus caused occasionally occur, know so much. Such lesions are more likely to produce simple bradycardia or simple tachycardia. Consideration of this fact is enough to cause a suspicion, if not more, that in palpitation there must be an exaltation of the augmentor, with a depression of the inhibitory, nerves. Reflex disturbances of every kind may produce palpitation. Such disturbances may arise from viscera not only directly connected with the vagus nerve, but from organs only indirectly connected with it, and situated in more distant parts of the body—*e.g.*, kidney, ovary, or uterus. It is common, however, to find that palpitation is more dependent upon such states as disordered gastric digestion or biliary irritation, changes which obviously are directly connected with the vagus system. Conditions of weakness of the myocardium, in the next place, may lead to palpitation, not only because they beget an irritable state of the heart muscle itself, but also because they produce a state of instability in the nervous mechanism of the heart. In this category must be placed all acute or chronic affections which impair the integrity of the myocardium. Here again it is once more incumbent upon me

[1] "*Charité-Ann.*, Berlin, 1893, S. 249.

[2] Since the delivery of these lectures, Huchard (*Journal des Practiciens*, 1903, p. 276) has wisely pointed out the possibility of error in considering cases of epilepsy and hysteria to be of cardiac origin, when the fact may be that the circulatory and nervous symptoms are only fortuitously connected.

again to mention the different toxic influences—microbic and chemical. It is very common to find that some of the poisons of the acute infective diseases leave behind a great tendency to palpitation, partly from weakness, but partly because they have really poisoned the nervous control over the heart. Once more, it need hardly be said, we meet our three old friends, alcohol, tea, and tobacco. It is unnecessary to say much about these three poisons, seeing we considered their action when we last met; their influence in producing palpitation is absolutely analogous to their action producing a simple acceleration of rate.

Then, lastly, amongst the causes of palpitation are all kinds of physical stress. Amongst soldiers, especially young soldiers, strain has been recognised since the well-known observations of Clifford Allbutt,[1] Myers,[2] Da Costa,[3] Seitz,[4] and Fräntzel.[5] Another cause was written about not very long ago, by Cocking of Sheffield, in a paper which was contributed by Snell,[6]—a paper which referred to the effects of occupation on miners. Cocking's contribution was in regard to some of the disturbances of the heart's action found in miners. We frequently, in the wards of the Royal Infirmary, where we have coal miners from Fife, the Lothians, and Lanarkshire, find in those patients that palpitation is a prominent symptom. Even within the last few weeks an interesting case of this kind was in my ward. In such cases as these we are left to consider that very likely the particular stress is brought about by the patient lying for hours together upon one side, very commonly the left side, to leave the right arm free, and using a pick all these hours in order to get the coal out of the seam. In this connection it may not be out of place to recall the fact that excessive physical stress is frequently associated with other causes of disturbance,—in short, the patient is often found to have been a worshipper at the shrines so quaintly mentioned by Lancisi.[7]

[1] *St George's Hosp. Rep.*, London, 1870, p. 23.

[2] "Diseases of the Heart among Soldiers," 1870, p. 22.

[3] *Am. Journ. Med. Sc.*, Phila., 1871, p. 17.

[4] *Deutsches Arch. f. klin. Med.*, Leipzig, 1873, S. 485, and 1874, S. 143, 279, 433, 583.

[5] "Die idiopathische Herzvergrösserung," 1889, S. 3.

[6] *Trans. Sanitary Institute*, 1895, p. 105.

[7] "De Motu Cordis et Aneurysmatibus," Lugd. Batav., 1740, p. 256. It may be well to give the picturesque imagery: "Venditor Piscarius annorum 45, habitus

Palpitation is, then, a greatly increased force of the action of the heart of which the sufferer is conscious. The question next arises, How does it come on, and under what circumstances is it found? Here many points are inexplicable. It occasionally occurs in the middle of some violent exertion. More commonly it arises during some profound emotional disturbance. There are probably few of us of the sterner sex here to-day who have not known the discomfort of having to deliver an after-dinner oration —who have not risen to our feet with the sensation as if the heart were throbbing in the neighbourhood of the throat, rendering any oratorical effort a practical impossibility. Such emotional disturbance often brings it on. But there is another side to this picture: it is not uncommon to find a patient, who has practically been living a quiet life in all respects, waking up in the middle of the night with a painful attack of palpitation. Sometimes we find that the dietetic errors, so graphically portrayed by Sydney Smith,[1] have been the root of this mischief, but sometimes there are absolutely no such eccentricities—no defiance of digestive functions—and the unfortunate sufferer awakes with the sensation as of an engine in his interior, with its governors removed, racing to destruction. Such conditions as these are probably to be explained in this way, that during sleep, when we know the wheels of being move slowly, the nervous system is reduced below its ordinary standard, and the vagus control, intrinsically weak in such cases, falls below the level that is necessary to keep the heart within due bounds even during the period of sleep. This is simply thrown out as a suggestion: there is nothing, so far as is known to me, that will prove or disprove it.

Palpitation, as regards its symptoms, is a conscious sense of discomfort connected with the chest; the prime symptom is really the painful throbbing. It is often attended by a feeling of fulness, as if the chest were possessed of contents far beyond its possibilities, and there is very frequently with that a sen-

carnosi vitæ omnino libertinæ Baccho nimirum, Dianæ, Neptuno, ac Veneri frequenter indulgens, ex qua postrema pluries contraxit luis stigmata, quæ fere incurata suis confisus viribus imprudentissime contempsit."

[1] "Memoir of the Rev. Sydney Smith," by Lady Holland, London, 1855, vol. i. p. 125.

sation of tightness, of constriction, and even occasionally of soreness radiating upwards, particularly to the left shoulder. Along with this, some of the symptoms which are common in hysteria are not infrequently found—the sensation of globus, a ball rolling up towards the throat and threatening to choke the individual. In addition to such features, there is very often a feeling of anxiety, together with singing in the ears, flashes of light before the eyes, deep sighing respiration, and occasionally giddiness, which renders it difficult for the patient to stand upright. Many of these sensations are graphically depicted by the master hand of Rousseau.[1] On objective examination, there is very greatly increased force, as well as rate, of the heart's action. Sometimes in cases of palpitation that we are called in to see, we can make out the heart's sounds at some distance from the chest without a stethoscope.

With such varying symptoms and different causation, the treatment of palpitation must be conducted upon somewhat diverse lines,—the cause must be ascertained, and if possible it must be removed. When it consists in simple gastric disturbance, nothing is easier than to get rid of the palpitation by correcting the irritation, whether it be a simple gastric catarrh or one of the secretory affections of the stomach that modern medicine has analysed with patience and success. These must be, in the first place, corrected, but at the same time it is necessary to lessen the afferent impulses passing up by the vagus channel, and to brace up the whole nervous system in every way. The one combination of drugs which is always found to be most useful for such a condition as this, in addition to our rhubarb, soda, and bismuth, consists in strychnine with hydrobromic acid. The acid is much more useful than any other form of bromine, such as we so commonly use in the bromides. But this is only to touch upon the fringe of the subject. If there be another reflex cause, such as undue mobility of the kidneys—a common cause of palpitation —then the viscus which is at fault must be put back in its right place by one measure or another. If it is from some more distant organ, the disturbance must likewise be found out and corrected.

[1] "Confessions," book vi. pp. 82 and 113, London, 1897.

If there should happen to be a weakness of the heart as well as of the nervous system, the heart must be toned up, and I need hardly say that strophanthus, digitalis, nux vomica, and iron are the most important remedies for the purpose. When we come face to face with alcohol, tea, and tobacco, we must proceed exactly as we saw it is necessary to do in regard to irregularity; we must cut them off altogether if possible, or as nearly so as may be practicable. For the palpitation arising from alcohol, digitalis and strophanthus are sovereign remedies, with the usual regulations as to rest and diet. For that induced by excess in tea, these drugs are also useful, but not so effectual as in the case of alcohol; they are only of doubtful advantage. And for these last two strychnine stands far ahead in value of either digitalis or strophanthus. Lastly, when physical stress leading to strain has set up palpitation, necessarily a long period of rest is required.

Turning, in the next place, to tremor, it must be stated to be quite a different condition from palpitation. This is not usually an affection of young persons. It is much more common in the middle of life, and sometimes lasts for a very considerable time afterwards. To this sensation of trembling in the chest various literary men have given a good deal of prominence. It is spoken of, for example, by Sir Walter Scott[1]; and many other men of letters have adverted to the extreme discomfort of this trembling action of the heart. There can be no doubt that Balfour[2] is in accord with usual experience when he says that he has never once, in the course of his existence, come across a tremor of the heart produced by emotional disturbance. Since he published his work on "The Senile Heart," about eight years ago, this point has been watched by me, and my own experience leads me to coincide entirely with him. When thinking over the matter recently, and looking over notes upon the subject, no case presented itself in which tremor cordis had come on as the result of emotional disorder. It is, nevertheless, essentially due to a loss of vagus control. It is produced by nervous disturbance without doubt, but not by psychical excitement. It is at times induced reflexly,

[1] "Journal," Edinburgh, 1890, vol. i. p. 153.
[2] "The Senile Heart," London, 1894, p. 68.

it is sometimes caused directly through nervous affections, but it is far more commonly the result of those debilitated and degenerative conditions of the heart muscle to which middle life is prone. Like palpitation to some extent, it nevertheless produces symptoms which are quite different. In palpitation the heart throbs violently, shaking the patient, sometimes shaking the bed; here, however, a quite different state of matters prevails. The individual affected feels as if he had a timid bird fluttering within the bosom. That is the kind of sensation to which many of us in middle age are not altogether strangers. This weak fluttering or feeble trembling of the heart is often attended by another most disagreeable sensation—a sensation as if the heart stopped and might not recommence its action again; it is therefore by no means uncommon for those who suffer from it to pass into a condition of considerable mental distress. There is in most instances a good deal of anxiety attendant upon it. Sometimes there is a considerable tendency to faintness, giddiness, and some of those other disturbances so common in palpitation. With these nervous accompaniments, moreover, there is a great tendency to deep breathing; profound suspirious respiration is one of the external evidences of the condition. On physical examination in cases manifesting the symptoms of tremor cordis, there is an irregular feeble fluttering action of the heart, which may be made out by the hand as well as the ear; an irregular weak quivering action, rising and falling in its intensity, sometimes more and sometimes less obvious to the observer, while the patient often shows some mental apprehension in his face. We are not to regard the condition as by any means a symptom of great danger. Many men begin to suffer from it in the fourth or fifth decade of life, and the symptom pursues them continuously or intermittently until the termination of a good long life many years afterwards. It certainly was so in the case of Sir Walter Scott; it has been so in the case of many lesser men since his day. The condition is to be relieved by bracing up the muscular substance of the heart, by getting rid of any depressing cause that may be present, and in this twofold manner the trembling may be led to disappear altogether.

Syncope, or fainting, has been known since the days of Seneca, who, in one of his letters to Lucilius,[1] speaks of the disagreeable feeling it produces. It also has received adequate treatment at the hands of many brilliant litterateurs, and it is well to mention, as one giving a very excellent description of it, that old cynic Montaigne; in one of the essays in the second book he gives a dramatic narration of his own sensations as he passed into a condition of syncope and emerged out of it again—the attack being induced by an accident. Syncope is most common amongst women, amongst patients of a nervous temperament, amongst those who are anæmic or who are feeble from any debilitating affection; and its proximate cause, its determining factor, is not infrequently some emotional disturbance. Hearing some gruesome tale, seeing some ghastly sight, or even the remembrance of something of the kind, may precipitate an attack. The senses of smell and taste may likewise induce attacks. These are direct factors, and we shall see that this category of causation comes up again before us in a few minutes in considering asystole. Reflex influences also lead to fainting; all kinds of disturbances of the internal organs, such as the passage of a biliary or of a renal calculus; in short, any painful impression conveyed upwards, may be the reflex cause of faintness. It may be produced also by a direct loss of blood, which lessens the power of the heart muscle, and also reduces the stimulus to contraction by diminishing the contents so that it fails to respond. It need hardly be added that a bloodless condition of the brain also brings the depressor mechanism into play. The symptoms produced are a feeling of faintness in the first instance, a sensation of uncertainty often with giddiness, an impression of objects being indistinct and sounds distant, with a state of mental confusion which, if we are to trust Montaigne,[2] is very far from being disagree-

[1] "Opera," Basileæ, 1529, p. 126.

[2] "The words of Montaigne may be given in full: "Il me sembloit que ma vie ne me tenoit plus qu'au bout des levres; ie fermoy les yeulx pour ayder, ce me sembloit, à la poulser hors, et prenoy plaisir à m'alanguir et à me laisser aller. C'estoit une imagination qui ne faisoit que nager superficiellement en mon ame, aussi tendre et aussi foible que tout le reste; mais, à la verité, non seulement exempte de desplaisir, aius meslee à cette doulceur que sentent ceulx qui se laissent glisser au sommeil."—"Essais," Livre II. chap. vi. p. 391. Edition Firmin-Didot, 1882.

able. The words of the essayist would lead one who has never himself yet had the experience of fainting, to believe that the process on the whole is not an unpleasant one. Walshe,[1] who expands this idea in one of his most eloquent chapters, speaks of it as being much more likely to be attended by pleasurable than by painful sensations.

The objective appearances are those of pallor, with a sharp or pinched condition of the features, and dilatation of the pupils, coldness of the surface, and diminution of respiration. It need hardly be added that on closer scrutiny the heart beat is feeble and flickering, and the pulsations in the arteries are often practically non-existent. It is probable that the heart, during syncope, is not at rest, but has the fibrillary movements to be described in asystole. Such is the condition of syncope. It is obviously brought about by exactly the opposite set of conditions to those which produce palpitation; the impulse comes down the inhibitory nerve, whether as the result of depressing influences sent up, or of direct influences upon the centre, as the result of which impulses passing downwards cause inhibition.

It is to be treated by putting the patient in such a condition as will allow gravitation to aid the return of blood to the heart, and increase the difficulty of sending the blood from the heart, so as to augment the normal stimulus to the auricles, which will lead them to a resumption of their ordinary rhythmic contraction. It need hardly be added, that if there should happen to be any prolonged syncope, such as might be dangerous to life, it is necessary to rouse the patient by means of appropriate stimuli to the surface, and to the special senses, by the use of injections of such substances as ether, and by the passage of a galvanic—not a faradic—current, to penetrate through the tissues if possible, and act directly upon the cardiac mechanism. These are the appropriate means by which syncope is to be relieved. As to minimising any tendency to it, this can best be done by stimulating and increasing the tone of the whole nervous system, and further by increasing the nutrition of the cardiac muscle.

We now come, in the last place, to the final subject with which

[1] "Diseases of the Heart and Great Vessels," fourth ed., 1873, p. 189.

we have to deal, that is, to the subject of asystole. For a good many years most of the works which bear upon sudden death—from the earlier work of Lancisi,[1] down to the monumental work of Brouardel[2] in the last decade of last century—have been familiar to me. In all these works we fail to get an adequate presentment of the reasons why a patient dies suddenly of heart disease. MacWilliam, to whose excellent work it has been my duty to pay a very well-merited tribute, has put matters very clearly as regards the mode of death in asystole.[3] Let me premise that there can be no doubt that Stokes[4] was perfectly right in his protest against the popular conception that sudden death is very common in heart disease. This remark of that great man has been abundantly justified, and we now recognise that sudden death is not of extreme frequency in such affections. One or two special diseases are, as we know, prone to sudden dissolution, but these form a great exception to the vast mass of cases of heart disease. And, as we grow older, our prognosis in cardiac affections—although it will probably remain for a long time one of the opprobria of practical medicine—this prognosis, as we grow older, becomes without doubt rather more hopeful than when we start out upon our life work as physicians. Well, according to MacWilliam, when asystole occurs, the heart does not suddenly stop beating, but is thrown into a series of fibrillary contractions—weak, purposeless vermicular movements, instead of co-ordinate movements of the mass of auricle or ventricle such as occur under healthy circumstances. The work referred to sets beyond all possibility of doubt that this is the normal way in which the heart ceases its activity. MacWilliam shows us that different animals have various tendencies in this respect. Lower animals amongst the mammalia, such as the rat or the hedgehog, may show this condition of fibrillation of the muscle for a few hours, after which a perfectly healthy heart beat is resumed. But in the higher animals, such as the cat and the dog, this is not possible; a comparatively short interval of time after such a fibrillation has been started in the heart, it is no longer possible to bring it back to its normal

[1] "De Subitaneis mortibus libri duo," Romæ, 1709.
[2] "La mort et la mort subite," Paris, 1895.
[3] "*Brit. Med. Journ.*, London, 1889, vol. i. p. 6.
[4] *Op. cit.*, p. 133.

healthy activity. MacWilliam believes this to be probably the state of matters which occurs when the human heart also fails, even when the end comes suddenly. It is known from the observations of prison physicians that when a man has been hanged his heart does not stop instantly with the crushing of his medulla oblongata; it goes on beating sometimes for twenty minutes in a feeble and irregular fashion, and finally flickers out. We may take it that this, occurring as it does for the most part in a strong, healthy man, is an expression of that fibrillary or vermicular action. Now, in the human being, such a condition as this may be brought about by a variety of different causes. It may be sudden or ingravescent. It may be produced by profound mental disturbance. All of us are conversant with the story of the French king who, for some error on the part of his jester, held a mock trial, when he was condemned to death. A masked headsman and a block in solemn black were introduced. The jester was made to kneel at the block, but instead of beheading him the executioner simply sprinkled a few drops of water on the back of his neck, and the whole Court, thinking this an excellent jest, broke into a fit of laughter. To the consternation of all, however, the jester never moved a muscle, he was as dead as Julius Cæsar. This is a well-known historical instance of profound emotional disturbance terminating the existence of an individual who apparently was in perfect health.[1] It is a very curious circumstance that extremely joyful emotions are almost as serious, or, as is sometimes stated, even graver, in their effects in this way than those which are profoundly depressing.

Other causes than emotional disturbances are reflex influences acting upon an enfeebled circulation. The profound impression produced by the passage of gall stones, in a patient with a feeble

[1] On the conclusion of this lecture, the chairman kindly recalled to my recollection that a similar episode had occurred in Aberdeen. One of the servitors of the University had made himself obnoxious to the undergraduates, and was consequently arraigned before a mock tribunal and condemned to death. With every circumstance of the solemnity befitting the occasion, the servitor was brought to the block, and, almost as in the case of the French jester, was gently flicked on the back of the neck with a wet towel. A similar tragic termination occurred in this instance. The secret was well kept, but some inkling of the facts leaked out, and for long afterwards in the streets of Aberdeen, any youth bearing the semblance of a student used to be greeted by the mob with the grim pleasantry: "Who killed Downie?"

heart, has been reported from time to time as the cause of death, and other similar visceral disturbances may bring about reflex results equally disastrous. Then a direct result upon the vagus nerve may at once stop all further pulsations of the heart. The Valsalva experiment, of trying to expire deeply with the nose tightly held and the mouth closely shut, has been known to lead directly to death by pressing upon the heart, and it may be added that one of the not infrequent modes of death that physicians meet with is in emphysematous patients with weak hearts and dilated stomachs, in which the heart is subjected to pressure both from below and from above, and is quickly brought into a condition of standstill. There are, besides, a large number of causes in the heart itself. There are such modes of death as sudden occlusion of the coronary vessels, rupture of the wall of the heart, and the sudden effect which may be produced upon it by direct violence. These are all factors which may lead to this condition of asystole. It seems to me that it cannot be any longer doubted that the explanation given by MacWilliam is the correct one as to the mode of operation of these causes.

When we see any one who obviously is standing within the shadow of death from threatened asystole, what are we to do to avert the impending disaster? We must remove as far as possible all the causes which may be depressing to the action of the heart; we must relieve the stress and consequent strain to which the heart is subjected directly or indirectly; every means by which the action of the vagus nerve will be abrogated should be employed, so that there shall be no interference of the regulating mechanism, and we must also use such drugs as we know to be powerful agents in bringing about a return of the contractile force. In cases of heart disease, we often encounter a combination of circumstances leading to asystole, and we frequently have the intense satisfaction of warding off an alarming attack of the kind and seeing the patient restored to comparative health even for years. This is especially the case in varieties of valvular affections in which the mitral cusps are affected. One very obvious cause, increased pressure within the arterial system, we can always remove by the use of the iodides, or by the use of the nitrites; and

by removing this resistance to the circulation, we can thereby aid the heart in keeping up the circulation and in losing the tendency to this fibrillation. At the same time, by the use of the cardiac tonics, along with the alteratives and vascular dilators, we achieve a double purpose.

Finally, let me add that in all enfeebled conditions of the heart, such as are so commonly found to underlie the symptoms we have been engaged in considering, careful hygienic and dietetic regulations must be enforced, while periods of rest and exercise should be definitely arranged. One very important matter is the employment of regulated movements of all the skeletal muscles, and in particular of those of the trunk, so as to ensure the thorough action of the respiratory apparatus and the adequate support of the abdominal organs.

In these lectures it has been my aim to be suggestive rather than exhaustive, because in the short space of six lectures upon the nervous affections of the heart it has been impossible to do otherwise. The various points which have been brought before you in these lectures will, it is to be hoped, not be altogether destitute of practical importance, while the scientific interest found in such researches has at least in some measure been laid before you.