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SENSORY & MOTOR DISORDERS OF THE HEART

ALEXANDER MORISON


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THE SENSORY AND MOTOR DISORDERS OF THE HEART

THEIR NATURE AND TREATMENT

BY

ALEXANDER MORISON, M.D., F.R.C.P.

SENIOR PHYSICIAN TO THE GREAT NORTHERN CENTRAL HOSPITAL; PHYSICIAN IN CHARGE OF HEART CASES TO THE MOUNT VERNON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST AT HAMPSTEAD AND NORTHWOOD; LATE PHYSICIAN TO THE CHILDREN'S HOSPITAL, PADDINGTON GREEN, AND TO THE ST. MARYLEBONE GENERAL DISPENSARY

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PREFACE

THE title of this book sufficiently indicates its contents. It purports to set forth conceptions of the nature and treatment of sensory and motor disorders of the heart. Both these subjects have, during the last three decades, received much attention, and the observations, suggestions, and conclusions given in the following pages are based upon an experience extending over more than thirty years.

Views of the nature of cardiac sensibility and motion have, during some portion of this period, owing to anatomical and physiological research and the introduction of additional methods of examination both in the laboratory and at the bedside, undergone considerable change. The writer, therefore, does not consider the space he has devoted to this aspect of the questions involved excessive; nor does he expect the views expressed by himself, which are rather conservative, to be endorsed by all. They will probably be considered too mechanical by some, and the share he ascribes to the nervous system, in its relation both to cardiac sensibility and motion, greater than the data at present acknowledged by physiologists warrant. He is content to leave the determination of this point to the future, without abating his conviction that the rôle of the nervous system in these respects has been unduly minimized by those who differ from him.

The clinical methods which he has chiefly employed are those of inspection, palpation, percussion, auscultation, and pathological investigation, to mention which is to recall names which may in time be obscured by those of others pursuing different methods, but which he is inclined to believe will survive for some time to come. The graphic method has, however, also been used to illustrate some points, but the value of diagnosis and prognosis by photography has not been emphasized. The data given by the latter processes are, with few and unimportant exceptions, more conveniently

obtainable otherwise, and the instruments involved are unlikely to come into general use.

The minute attention given in recent years to the study of cardiac irregularities, with which the names of some British physicians, and notably that of Dr. James Mackenzie, are prominently associated, has served to distinguish more precisely from one another varieties of arhythmia or, as the writer has preferred to term the condition, extrasystolia, without regarding the maintenance or otherwise of the so-called sinus rhythm. This nomenclature he does not expect will meet with the approval of all.

On the whole, in the prognosis and certainly in the treatment of the disorders with which this book deals, he is of opinion that the sagacity and skill of some masters of cardiology who now rest in the Walhalla of Medicine, have not been surpassed by any recent work. The greater safety, however, which has been secured for the more serious operations of surgery by the life-work of a great Englishman affords the hope that some mechanical impediments to the action of the heart which are beyond the power of the physician to deal with may, by the combined efforts of the physician and surgeon, be more successfully treated now and in the future than has hitherto been possible.

In conclusion, the writer feels that some apology is due for a perhaps too dominant personal note in these pages. The substance of his observations and remarks has, however, already been given in lectures and in articles in the medical press, forms of communication in which it is difficult for the individual altogether to obliterate himself. While acknowledging his indebtedness to all the journals which have allowed him to utilize material which has appeared in their pages, and which is acknowledged in the text, he has especially to express his obligation to the proprietors of the *Lancet* for allowing him to rewrite in a measure and use the chapters dealing with the Sensory Disorders of the Heart, which appeared in that journal as 'Lectures on Cardiac Pain.'

14, Upper Berkeley Street,
London, W.
November 20, 1913.

CONTENTS

PART I

THE NATURE OF CARDIAC ACTION

PART II

THE SENSORY DISORDERS OF THE HEART

PART III

THE DISORDERS OF CARDIAC MOTION

ERRATA

Page 85, line 7, *for* 'Allen-Burns' *read* 'Allan Burns.'
,, 55, line 4 and elsewhere, *for* 'Vacquez' *read* 'Vaquez.'

THE SENSORY AND MOTOR DISORDERS OF THE HEART

PART I

THE NATURE OF CARDIAC ACTION

CHAPTER I

THE NODAL SYSTEM: ITS ANATOMY

CONCEPTIONS of cardiac action have within a comparatively short period undergone so much modification that it is necessary, before discussing disorders of cardiac sensibility and motion, to consider as shortly as possible, but with sufficient fulness, the scientific bases on which current views of cardiac action rest.

The time-old controversy between myogenists and neurogenists, in which Gaskell intervened with so much deserved weight, casting his influence into the scale against the neurogenists, gained an added interest by the discovery of a new fact. Gaskell's conclusions had been drawn chiefly from the study of cardiac action in the reptilia, in which the continuity of muscular fibres between the auricles and ventricles was apparent. But there was a difficulty in applying his views to those vertebrates in which there was, in adult life, an apparently complete severance of muscular continuity between the upper and lower chambers.

This hiatus was filled by the discovery by Kent and His of a junction between auricle and ventricle through the

medium of a muscular structure of peculiar character, which came to be known as the 'auriculo-ventricular bundle.'

It is not necessary, at the moment, more than to mention that Kent described yet other junctions between these chambers by ordinary muscular fibre. To this I shall return.

The peculiarity in the character of the auriculo-ventricular bundle consisted in its being, as it has been termed, 'embryonic,' and it was and is regarded by many as the remains of the aboriginal cardiac tube of the early embryo.

While the discovery of the bundle satisfactorily disposed in the minds of the myogenists of the difficulty as regards the muscular conduction of motion from auricle to ventricle, it still remained to account on myogenic lines for the incentive to *auricular* action.

Keith and Flack disposed of this difficulty by the discovery of a constant nodule consisting of tissue resembling in many respects that of the auriculo-ventricular bundle, and situated in the groove between the superior vena cava and the appendix of the right auricle.

Finally, Wenckebach, Thorel, and some others, have described still other junctions between the sinu-auricular and auriculo-ventricular nodal structures.

It does not appear to have concerned the myogenists greatly what was the intrinsic nature of the pre-auricular contraction of the great veins and coronary sinus entering the heart. That this point, however, cannot be neglected I shall endeavour to show later.

It has been the fate of muscle, regarded as automatic in various situations in the body, to have been also thought at one period of the knowledge of their existence to be independent of neural control, and, indeed, sometimes to be free from the intrusion of nerves into their structure.

To this rule the auriculo-ventricular bundle has been no exception. As regards the sinu-auricular node, on the other hand, situated as it was found to be among ganglia and nerve fibres, this view was never very comfortably entertained. Both structures being regarded as portions of the original cardiac tube, which pulsated prior to the invasion of the heart by the nervous system, it is not unnatural that such

views of the nerveless automatism of these structures should have been regarded as possible. This innervation, to which I shall have to refer in greater detail, has now been satisfactorily established, and some other facts which I shall mention render it probable that the conception, in any case of the auriculo-ventricular bundle, as a part of the original cardiac tube, may also have to be abandoned.

The sinu-auricular node, situated, as its name implies, between the sinus as represented by the superior and inferior venæ cavæ and the auricular appendix, is neither so elaborate nor extensive a structure as the auriculo-ventricular node and tract. It consists of a muscular tissue in which the nuclei are large, and the cells so approximated as to make them appear more numerous than in an equal area of ordinary muscular tissue. The striæ of the fibres are indistinctly marked, it is definitely subepicardial in position, and processes from it pass into the auricle in the interstitial spaces between the ordinary muscular fibres; while some, likewise, pass upwards into the musculature of the termination of the superior vena cava.

If the fibres which Wenckebach describes[1] as uniting this node to the ordinary auricular muscle be those I have mentioned as issuing from the node into the intermuscular spaces of the adjacent auricle, I can confirm his observation; but, as I have stated, they likewise penetrate the caval musculature.

Some have ascribed a much more extensive range to the fibres of the sinu-auricular node. Thorel[2] establishes a connection between the sinu-auricular and auriculo-ventricular nodes by way of the coronary veins and coronary sinus. It is difficult in the normal adult mammalian heart to escape the impression that the node of Tawara and the bundle of His are related to the structures at the mouth of the coronary sinus; but in the persistent embryonism of the case of foramen primum which I have described in the *Journal of Anatomy and Physiology* for July, 1913, I have been able to show that the node of Tawara has no connection whatever

[1] *Archiv f. Physiol.*, 1907.

[2] *München. Med. Wochensch.*, No. 4, 1910.

with the coronary sinus, and in the latter itself I have found no nodal texture.

In addition to these principal connections claimed by Thorel, he also describes another passing down the inter-auricular septum, together with some fibres from the neighbourhood of the foramen ovale, which, he asserts, join the node of Tawara. These also would find it difficult to

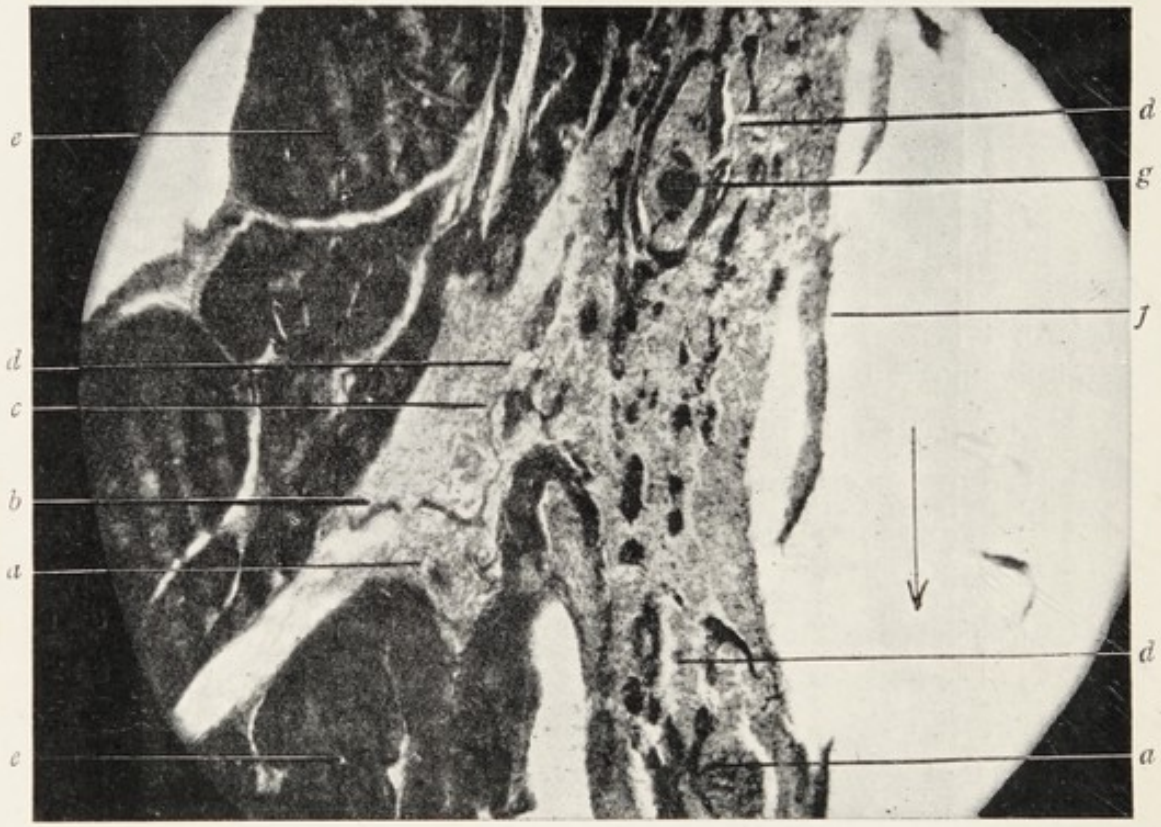

FIG. 1.—THE SINU-AURICULAR NODE IN THE PIG.

a, *a*, Large nerves ; *b*, smaller nerve ; *c*, fibril of the same ; *d*, *d*, muscle of the node ; *e*, *e*, auricle ; *f*, epicardium ; *g*, artery.
Arrow points to auricle. (Obj. $\frac{1}{3}$ in.)

reach that node in the case I have mentioned, inasmuch as the interauricular septum below the level of the foramen ovale was absent.

The sinu-auricular node is supplied with blood by branches of the right coronary artery, around some large trunks of which it may in places be observed to cluster.

Although it had long been observed to be situated in a neighbourhood in which the ganglia and nerves of the cardiac plexus are frequently encountered, the first published account of its intimate innervation appears to have been by

myself.[1] B. S. and Adèle Oppenheimer, however, investigated its innervation rather prior to myself, although their results were not published until later.[2] It is innervated in the manner of ordinary cardiac muscular fibre—a fibre passing into varicose fibrils and meandering around and among the muscle cells. This innervation is most easily detected in the ungulates (Figs. 1 and 2). The Oppenheimers only found vaso-motor nerves in the node of the human subject; but doubtless, as Engel, to whose researches I shall refer, found, in the case of a comparatively fresh human heart, as regards the auriculo-ventricular bundle, further research will also show in this case a more pronounced innervation.

FIG. 2.—INNERVATION OF THE SINU-AURICULAR NODE IN THE PIG. (HALF DIAGRAMMATIC.)

a, Nerve ending in fibril *b*; *b'*, fibril of another fibre; *c*, *c'*, *c''*, moniliform nerve fibrils; *d*, *d*, muscle cells of node.

The *auriclo-ventricular* node, situated in the normally developed heart under the septal cusp of the tricuspid valve, is usually stated to arise by a short root near the opening of the coronary sinus into the right auricle, and to divide into two branches near the pars membranacea septi, the right branch passing by way of the moderator band for further division and distribution in the right ventricle, and the left piercing the membranous septum below the non-coronary cusp of the aortic valve for distribution to the left ventricle. Discovered, as I have said, by Kent in 1893, and independently by W. His, junior, who described it more fully, it was examined and described in still greater detail by Tawara, whose monograph was published in 1906. To no one in this country are we so much indebted for our knowlege of the anatomy of these structures as to Professor Arthur Keith.

[1] *Journ. of Anat. and Physiol.*, July, 1912.
[2] *Journ. of Exp. Med.*, vol. xvi., No. 5, 1912.

Consisting of a texture resembling that of the sinu-auricular node, which is now appropriately termed 'nodal tissue,' it is said to form a junction on the one hand with auricular muscle, either by growing into it or by auricular tissue coalescing with its nodal structure; and on the other with the ventricle by passing downwards, and ultimately, according to Tawara, by being transformed into ordinary cardiac muscle. I have, however, also seen branches of this nodal tissue near its origin seek the interstices between muscle bundles as trunks of nerves do, and then proceed for further distribution.

Vaquez[1] very interestingly points out the conclusions to be drawn from the shorter and longer course of strands of the bundle of His before they reach their ultimate distribution to the ventricular myocardium, and refers to the work of Refisch, Saltzmann, and Hering. These concluded from their observations that the apical portion of the heart contracted in systole before the base, and that the papillary muscles supplied by the shorter distance strands of the bundle contracted before any other portion. Vaquez regards this as proof of the earlier effect of the auricular stimulus through the shorter strands.

But there are even shorter strands which penetrate the top of the septum ventriculorum. Saltzmann, indeed, states that the septal base precedes the papillary muscle in contraction, while the mural or external base succeeds it![2] He, however, wisely remarks that conclusions drawn from his experiments on the eviscerated heart are not to be regarded as final, and it must be borne in mind that all the nodal textures are controlled by the nervous system.

Like the sinu-auricular, this node also is supplied with blood by the right coronary artery, and may be observed at times to be placed like a collar round large branches of that vessel (Fig. 3).

Tawara, who first fully described the structure as a whole, was also the first to describe in a measure its *innervation*. He did not, however, use any special stain for nerve tissue,

[1] 'Les Arythmies,' Paris, 1911, pp. 47-51.

[2] *Skand. Archiv. f. Physiol.*, vol. xx., 1908.

and did not determine the nature of the ultimate distribution of the nerves seen.[1]

The first important contribution to the innervation of the bundle was Wilson's.[2] Using the vital methylene blue method, he described ganglia and plexuses in the bundle, chiefly of the ungulates; but he also gave no delineation of the ultimate termination of the nerves on the muscle cells of the structure. His method was, however, very fruitfully

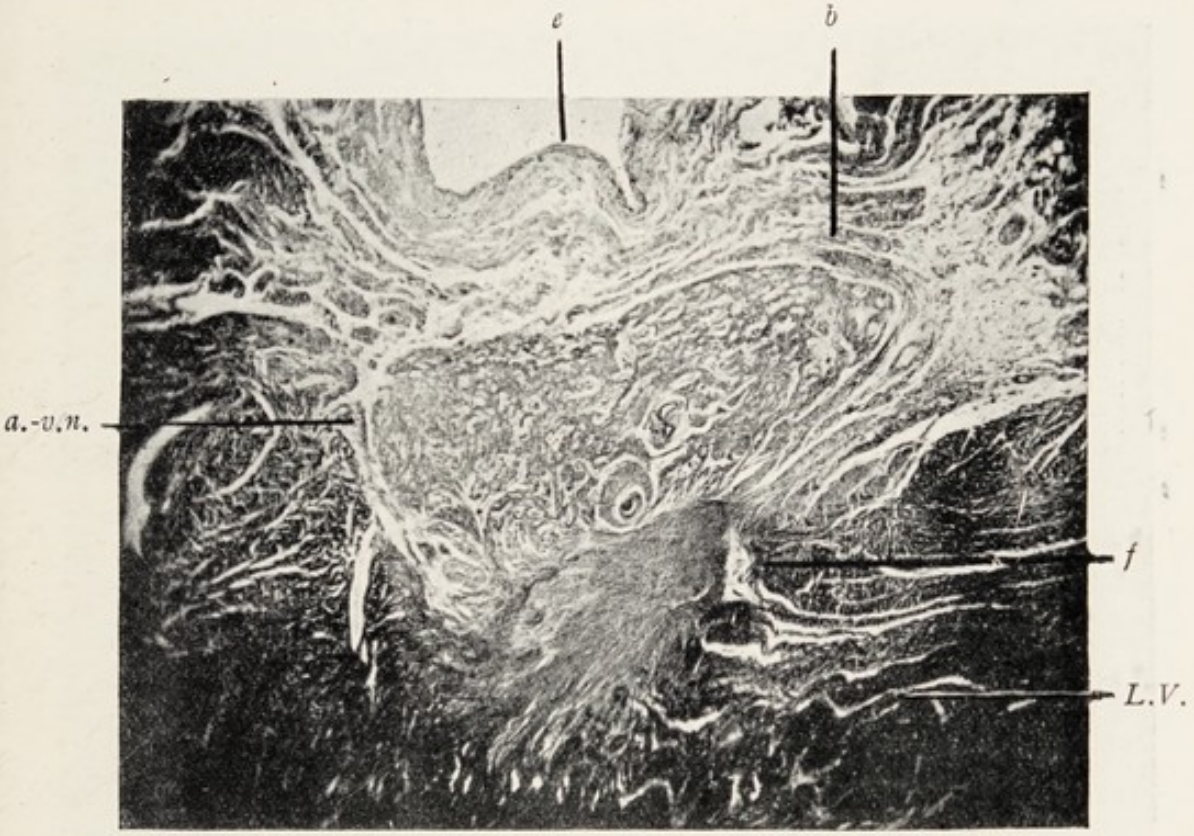

FIG. 3.

a.-v.n., Auriculo-ventricular node, with central artery (right coronary) and many arterial points; *e.*, epicardium; *b.*, boundary of auriculo-ventricular node, with longitudinal nodal fibres; *f.*, fibrous body; *L.V.*, left ventricle. (Obj. 1 in.)

practised by Irmgard Engel in 1910.[3] She worked under Aschoff in Freiburg, and examined ungulates, carnivora, and man.

The ungulates, as is well known, have the bundle largely developed in all its elements. Engel describes and delineates ganglion cells in which the axons pass into varicose fibrils,

[1] 'Das Reitzleitungssystem,' etc.

[2] Proc. Roy. Soc. B., 1909, lxxxi. 151.

[3] *Beiträg. z. Path. Anat. u. z. Allg. Path.*, 1910, xlviii., 499.

and are directed both towards the apex and base of the heart, but chiefly towards the base. By another method she determined the important point that a very considerable number of medullated nerve fibres in the calf follow the bundle without branching or alteration until the papillary muscles are reached, when they are distributed in the ventricular wall. She also shows the ultimate termination of fibrils to the cells of the Purkinjean fibres. Finally,

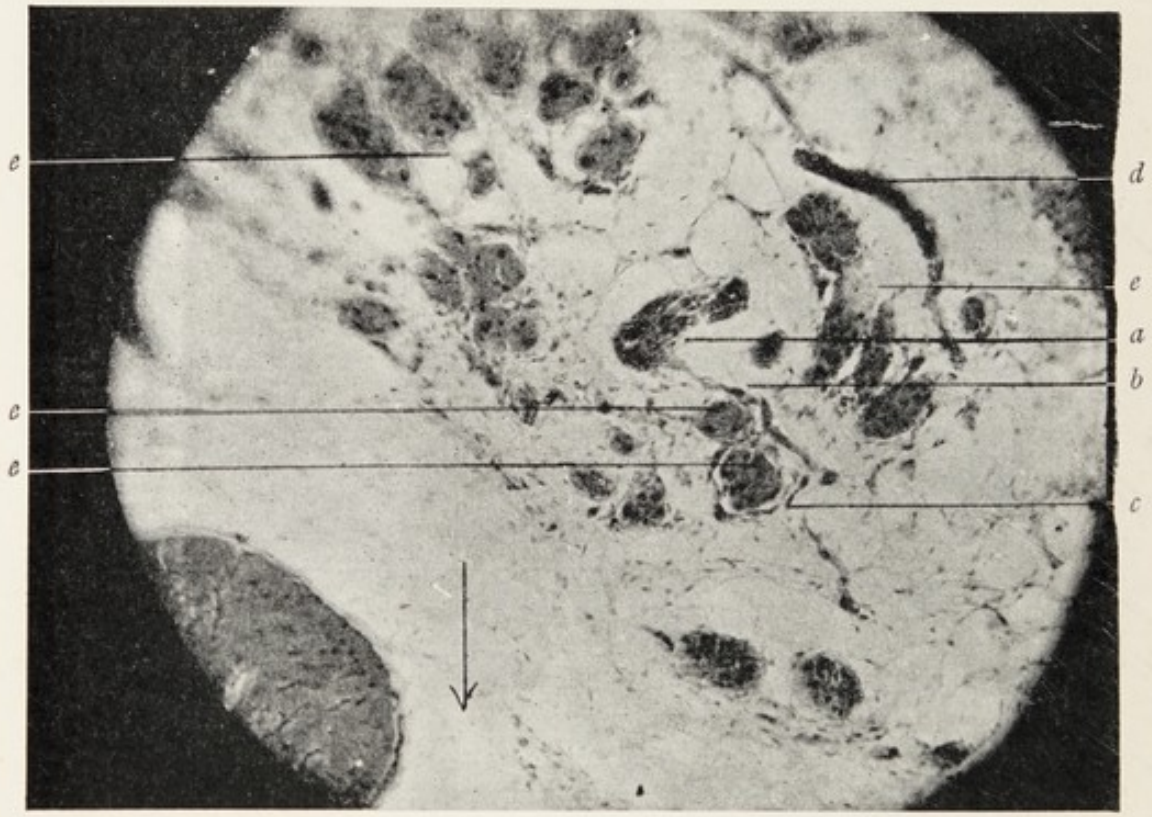

FIG. 4.—AURICULO-VENTRICULAR BUNDLE IN THE SHEEP.

a, Transverse section of large nerve; *b*, fibre arising directly from the same and becoming fibrillar; *c*, primary plexus around *e*; *d*, large nerve; *e*, *e*, groups of Purkinjean cells.

Arrow points to ventricle. (Obj. $\frac{1}{6}$ in.)

she was fortunate enough to find in a human heart, examined one hour after death, a rich innervation of the auriculo-ventricular bundle.

Unaware at that time of Engel's work, and not finding that Wilson had delineated the final termination of the nerves on the cells of the structure, I examined the matter for myself, and published my results in July, 1912 (*loc. cit.*). I used a modification of Sihler's hæmatoxylin method which

I had found successful in staining the nerves of the intra-cranial artery, a that-time automatic structure, and in the phase of being regarded as without nerve control.[1]

I examined the sheep, pig, and man, and found that in the sheep the nerve terminations on the muscle cells were of two kinds—namely, by varicose fibrils winding around and on them, and by triradiate nodal points closely applied to them and resembling sensory terminations (Figs. 4, 5, 6 and 7). In

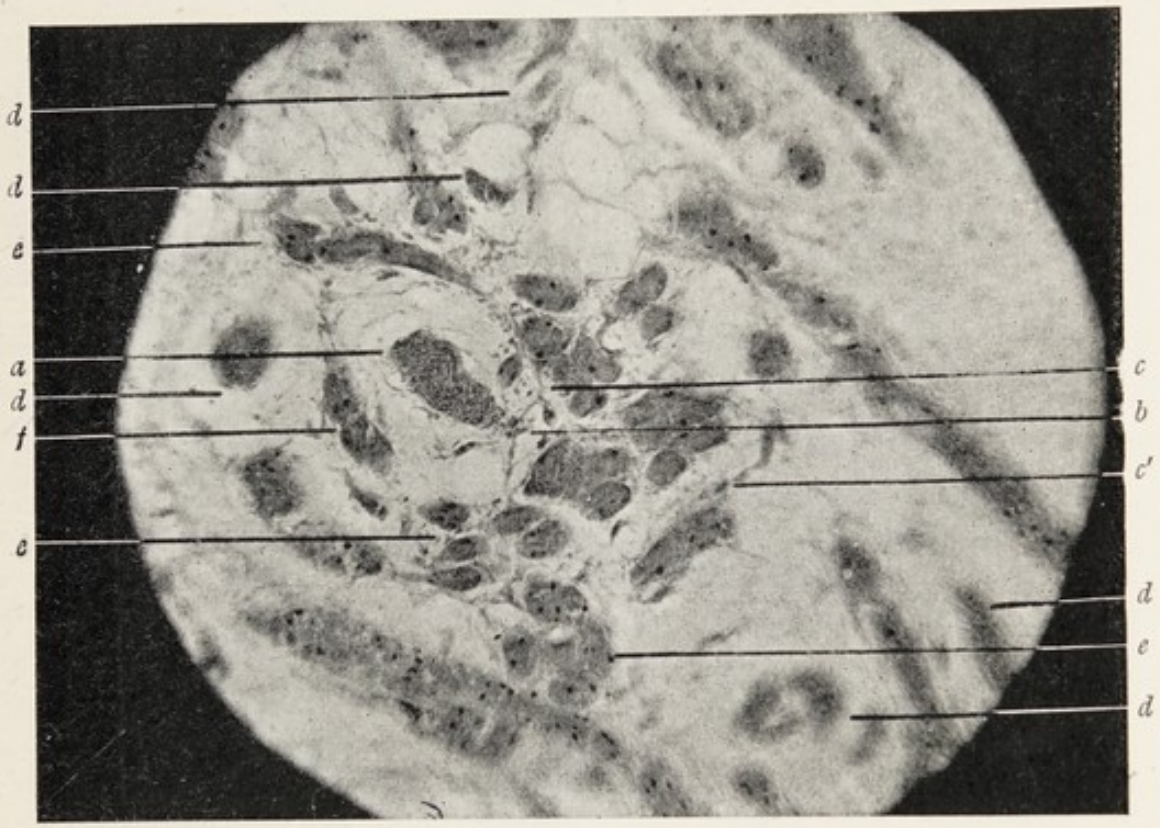

FIG. 5.—AURICULO-VENTRICULAR BUNDLE IN THE SHEEP.

a, Transverse section of large nerve; *b*, nodal point in the same, whence the primary plexus *c* directly springs; *d*, *d*, large nerves; *e*, *e*, groups of Purkinjean cells; *c'*, another primary plexus. (Obj. $\frac{1}{8}$ in.)

both the sheep and pig it was manifest that the bundle and the course it pursued was a highway for large nerves passing for distribution to the ventricular walls. In man, probably because of the necessary precedence of death for a longer or shorter period before the examination of the structure is possible, the nerves are difficult to stain. It may be affirmed with confidence, however, that no large nerves enter the bundle, although they may course near it. In one case, that of a

[1] *Edin. Med. Journ.*, 1898.

young man whose heart was examined ten hours after death, I succeeded in staining ultimate fibrils. But the question of a sufficient innervation of the human bundle may be regarded as having been settled by Engel (*loc. cit.*).

The facts mentioned, taken together, may be considered as having definitely decided the point that these nodes, whether regarded as initiative and automatic in provoking cardiac action, or as conductive of such, are completely under the control of the nervous system.

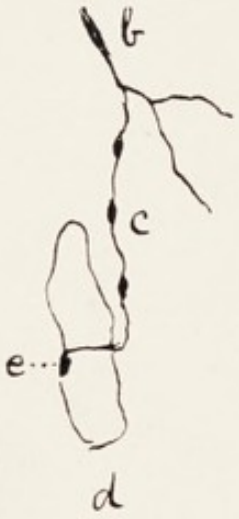

FIG. 6. — PURKINJEAN CELL FROM FIG. 5 (*f*). (HALF DIAGRAMMATIC.)

b, Termination of fibril; *c*, moniliform fibre applied at *e* to muscle cell *d*.

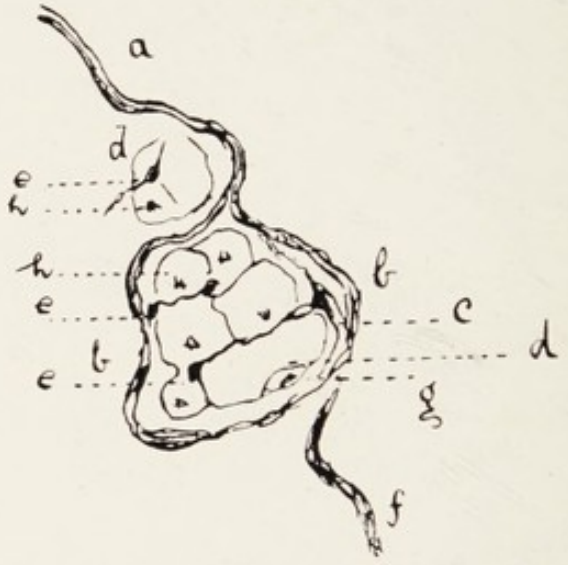

FIG. 7. — REPRESENTATION OF *b* TO *e* IN FIG. 4. (HALF DIAGRAMMATIC.)

a, Nerve fibre; *b*, *b*, primary plexus round *d*, a group of Purkinjean cells; *c*, secondary node growing off from fibril passing between muscle cells and bearing contact nodules, *e*; *f*, fibril continued; *g*, nucleus and nucleolus of a large Purkinjean cell; *h*, *h*, nuclei of other cells.

Are they structures subserving the purposes of the nervous system, or can they act independently of the latter?

Regarded as remnants of the aboriginal cardiac tube, of which they alone have escaped progressive development into ordinary cardiac muscle fibre, one would have expected them to be without the necessity for the elaborate innervation of a later date in embryonic development, with which we have seen them to be endowed. Moreover, we should expect that they would be devoid of the power of expansion

by active growth in later stages of the complete evolution of the heart.

The embryological study of this point is still in its infancy, and further data are necessary to our forming a conclusion based on solid fact. Retzer, from embryological observation in the pig,[1] states that when the

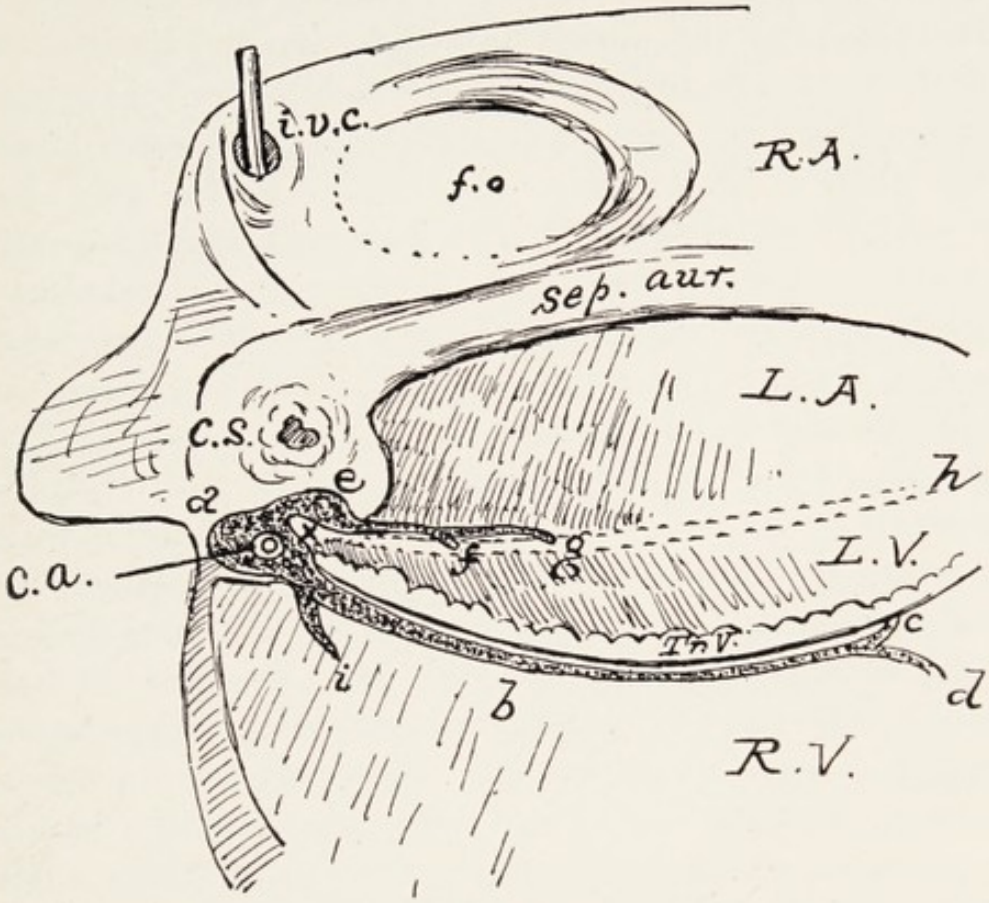

FIG. 8.

R.A., right auricle; *i.v.c.*, inferior vena cava; *f.o.*, foramen ovale; *sep. aur.*, lower edge of auricular septum; *c.s.*, hypertrophied entrance to coronary sinus; *e*, portion of auriculo-ventricular node throwing off diverted branch, terminating in *f* and *g*, in the fibrous boundary *h*, between *L.A.* (left auricle) and *L.V.* (left ventricle); *Tr.V.*, tricuspid valve; *a*, largest part of an auriculo-ventricular node; *c.a.*, coronary artery; *x*, on fibrous structure dividing the node; *i*, *c*, *d*, branches of *b*, the main trunk of the auriculo-ventricular bundle; *R.V.*, right ventricle. (Diagrammatic.)

septation of the heart is about to become complete, and the auricles have been cut off from the ventricles by the growth of the texture of the auriculo-ventricular boundary, the continuity of the upper with the lower heart is preserved by a growth downwards of muscular tissue from the 'septum superius,' through the 'septum intermedium,' on to both

[1] *Anat. Record*, vol. ii., 1898.

sides of the interventricular septum. This downgrowth from the septum superius is, according to him, the auriculo-ventricular bundle. While I cannot agree with Retzer that the downgrowth need be a prolongation of the 'septum superius,' I believe with him that the auriculo-ventricular node and bundle have the power of active growth, and that the bundle invades the ventricles as nerves and vessels do.

On examining the persistently fœtal conditions of a case to which I have already referred, in which the septum primum was absent, and anatomical details of which I have published in the *Journal of Anatomy and Physiology* for July, 1913 (vol. xlvii.), I found that the node occupied a position in the right auricle close to the epicardium (Fig. 8). Divided in its anterior portion by dense fibrous tissue, the structure was cut into two parts. The larger coursed along the top of the completed interventricular septum below the line of auriculo-ventricular valves, and branched to supply both ventricles. The smaller, but yet considerable portion, diverged to the left before the interventricular septum was reached, and, travelling round the posterior part of the left auricle in its lower portion, sought to enter the left ventricle, but failed to do so, ending blindly in the fibrous textures of the auriculo-ventricular boundary. We still require to learn when the first vestiges of the nodal structure appear in the embryo, but the facts mentioned leave no doubt that the auriculo-ventricular node pushes by its processes into the ventricular heart; that is, that it *grows* during a comparatively late phase in the development of that organ, and cannot therefore be regarded as a portion of the primitive cardiac tube, but is an active and developing differentiation of mesoblast. What, then, are the ultimate functions of the nodal textures of the heart?

CHAPTER II

THE NODAL SYSTEM: ITS PHYSIOLOGY

'When the chest of a mammal is opened,' writes Foster,[1] 'a complete beat of the whole heart or cardiac cycle may be observed to take place as follows: The great veins, inferior and superior venæ cavæ and pulmonary veins, are seen, while full of blood, to contract in the neighbourhood of the heart; the contraction runs in a peristaltic wave towards the auricles, increasing in intensity as it goes. Arrived at the auricles, which are then full of blood, the wave suddenly spreads, at a rate too rapid to be fairly judged by the eye, over the whole of those organs, which accordingly contract with a sudden sharp systole. In the systole, the walls of the auricles press towards the auriculo-ventricular orifices, and the auricular appendages are drawn inwards, becoming smaller and paler. During the auricular systole, the ventricles may be seen to become turgid. Then follows, as it were immediately, the ventricular systole, during which the ventricles become conical. Held between the fingers, they are felt to become tense and hard.'

In the description of this succession of phenomena published in the year in which Kent and His discovered the auriculo-ventricular bundle, mention is not made, for obvious reasons, of the action of the coronary sinus. It is not so obtrusively visible with the opened chest, as the structures named, but its design and pre-auricular action are of even greater interest than the pre-auricular contraction of the veins referred to.

The coronary sinus is, embryologically, the representative

[1] 'Textbook of Physiology,' part i., p. 232, ed. vi., 1893.

of the left horn of the sinus venosus, the first pulsatile structure in the embryonic heart. Into it the coronary veins empty themselves—veins which possess valves to guard against reflux. At its auricular orifice the coronary sinus is guarded by the Thebesian valve, a structure which, as Argaud has shown,[1] while acting efficiently as a valve to prevent reflux into the sinus during auricular systole, has not the composition of the membranous structures guarding the arterial and atrial orifices, but, in addition to a considerable musculature, is also well innervated.

The musculature of the coronary sinus is demonstrably continuous with that of the right auricle, and hypertrophies in common with it. Its innervation by ganglia and nerve fibres is abundant. It certainly need have no direct relation to the sinu-auricular node, and as certainly may be quite unconnected with the auriculo-ventricular node and bundle, as my case of foramen primum proves, and it shows no nodal tissue in itself. Its necessarily pre-auricular contraction, as a chamber which may be powerfully developed, is not induced by any preceding muscular stimulus; but, on myogenic lines, it might be regarded as acting in concert with the other representatives of the sinus venosus (superior and inferior venæ cavæ), as stimulating to contraction the auricle with which it is in muscular continuity. As I shall argue later, its action appears to be more reasonably explicable on neural than on muscular lines, and it may prove to be the key to a more satisfactory explanation of the mechanism of the heart-beat. The coronary sinus may, in short, be regarded as the least changed portion of the sinal structures seen in highest development in some pre-mammalian animals. Even the highly organized Thebesian valve may be regarded as a surviving venous valve.

His, junior, appears himself to have been the first to have endeavoured to determine the function of the auriculo-ventricular bundle by dividing it,[2] but details of his method and the results obtained are not so clear as that which was practised and those which were obtained by Hering, whose

[1] *Archiv. d. Mal. du Cœur*, iv., 1911, p. 642.

[2] *Wiener Med. Blät.*, 1894, No. 44.

results are given in *Pflüger's Archives* for 1905, vol. cviii. He found that division of the heart-muscle in the situation of the bundle before it divides into its main right and left ventricular branches was followed by an alteration in the rhythm of auricular and ventricular systole. The ventricle thereafter beats more slowly than it previously did, while the rate of auricular systole remains unchanged.

To avoid objections raised to the method of dividing the structures concerned by the knife, Erlanger produced the same effects by a specially-devised clamp.[1] Paukul,[2] however, considered that he had shown, by a method in which he tied the bundle and its neighbourhood, that, when the bundle was alone enclosed in the ligature, dissociated action of the auricles and ventricles did not occur, and also that the passage of a ligature through these parts without its being tied induced dissociated action of the auricles and ventricles. Paukul's experiments were made upon the heart *in situ*.

Cohn and Trendelenburg, however, aware of Paukul's results, studied the question in the eviscerated heart, artificially fed by saline solution and oxygen. They found division of the bundle when complete to be followed by dissociated action, and when incomplete not to have this consequence.[3] Their method and its results are of high interest and value. Their experiments were performed on ungulates, rodents, carnivora, and apes. While they at times found the production of dissociated auriculo-ventricular action to be difficult and transient in the rodents and carnivora used, and moderate in the apes, the effects of section of the bundle in the ungulates (goat) were very remarkable. In one case a 2 : 1 rhythm resulted; but in two other instances, the cut not being in quite the same situation, although the bundle is said to have been severed, the dissociated auriculo-ventricular rate was in one case 16-20 : 1, and in the other 8-10 : 1. The authors are inclined to attribute this remarkable difference from the other animals operated upon to the tender age of the subjects, which were kids, and express no opinion as to the muscular

[1] *Zentralb. f. Physiol.*, xix., 1905, p. 9.
[2] *Zeitschr. f. Biolog.*, 1908, p. 177.
[3] *Archiv f. d. Ges. Physiol.*, 1910, p. 1.

or neural conduction of auricular impulse. But, knowing as we do the very remarkable difference between the innervation of the bundle in the ungulates as compared with the other animals employed, it is difficult to dismiss without further consideration the possible and even probable influence of a severance of the nerves in and around the bundle in these cases. Engel, as we have seen, has shown that large numbers of medullated nerves course with the bundle in the calf to its termination, while ganglia in it cast their axons both towards the base and apex of the heart. These anatomical conditions were, of course, unknown to Gaskell when he wrote his memorable article on the nature of cardiac action.[1] If, as seems probable, the very remarkable retardation of conduction in the ungulates be in some measure due to the division of the innervation referred to, we cannot suppose that 'conductivity' is alone, in such measure, dependent upon the nervous system, but must regard other qualities attributed to muscle in terms of anatomical metaphysics, to be also dependent upon neural influence, a point which I shall again consider when summarizing the conclusions arrived at from a study of the facts mentioned.

The last point which may be mentioned in connection with the experimental physiology of these structures is that Erlanger (*loc. cit.*) believes he has shown that after complete obstruction of the bundle, stimulation of the pneumogastric nerve fails to inhibit the ventricles, while stimulation of the accelerant nerves still quickens the heart, a conclusion which is endorsed by Hering in great measure[2] and has been found to be the case by other experimenters, and most recently by Cullis and Tribe,[3] the inference being that the ventricles are only inhibited secondarily to the auricles. Cohn, however,[4] has observed stimulation of the left pneumogastric to cause the inhibition of the ventricles without a preliminary inhibition of the auricles, and he, as well as Robinson and Draper,[5] appear to have satisfied themselves, by stimulation of the

[1] Schäfer, 'Physiology,' vol. i.
[2] *Archiv f. d. Ges. Physiol.*, vol. cvii., 1905 *et loc. cit.*
[3] *Journal of Physiology*, April, 1913, p. 148.
[4] *Journal of Experimental Medicine*, xv., 49, 1912.
[5] *Ibid.*, p. 31.

right and left vagi separately, that the former is chiefly distributed to the sinu-auricular node, after removal of which (Flack) the ventricle cannot be inhibited, while the latter chiefly influences the auriculo-ventricular node and tract.

Finally L. Frédérique[1] believes that he has shown by a gradual compression of the bundle of His, somewhat on the lines of Erlanger's compression experiment, that the conductive functions of the muscular element may be placed in abeyance without affecting the inhibitory power of branches of the vagus coursing in the nodal track. Still greater compression, he believes he has shown, abolishes both muscular conduction and nervous inhibition. Does it, however, follow, from the results he has obtained, that neural influence is absent when dissociation is complete without inhibitory action being abolished? May not numbing of the bundle without destroying it interrupt the ventricular reflex resulting in normal ventricular contraction? Perhaps; but the existence of a ventricular reflex mechanism has first to be established. Is there any evidence for such? In the eviscerated heart any mechanical means may excite contraction. The ventricle which has ceased to beat may also by massage be made to contract, as in Arbuthnot Lane's case.[2] But such effects are now attributed to the inherent excitability of muscle, not to nervous action. On what grounds? Because physiologists have not convinced themselves of the existence of a true visceral reflex, and for no other reason. When Erlanger originally experimented, the very *existence* of nodal innervation was considered doubtful. When Frédérique experimented this was no longer a tenable supposition. A little longer, and it is quite probable the bundle may be shown, not only to be the channel for inhibition, but also to form part of the mechanism of a ventricular reflex which becomes automatic on the lines I shall presently suggest.

Physiology of the Sinu-Auricular Node.—In the case of this node, discovered later than the lower structure, and sought for on Wenckebach's suggestion after the discovery of the latter, and a belief on his part in the automatic origin of

[1] *Archiv. Intern. de Physiol.*, 1912, xi. 405.
[2] *Lancet*, November, 1902.

cardiac action, the results of physiological experiment do not impress one with so great a sense of the importance of the structure as they do in the case of the auriculo-ventricular nodal system. It has been shown experimentally that the action of the heart may be slowed or quickened by the application, to the region in which the node is situated, of cold and heat respectively. It has also, however, been demonstrated by Flack[1] that it may be so treated as to be assumed to have been destroyed, without the cardiac rhythm being permanently affected. Flack, however, found (*loc. cit.*) that mild electrical stimulation of the node induced inhibition of the heart, stronger stimulation a mixed effect of acceleration and retardation, and still stronger stimulation no effect whatever. If it be true, as Flack maintains, that stimulation of the vagus in the neck fails to induce cardiac inhibition after destruction of the sinu-auricular node, the fact is interesting, and, if shown to occur without a simultaneous destruction of the chief strands of the pneumogastric nerve, important. But do not the nerve channels suffer in the course of the destruction of the node, whether this be effected by searing, compression, or excision?

One must likewise remember that, preceding the contraction of the auricles, there is a contraction of the large veins entering the heart, including the coronary sinus. Of these the superior vena cava, like the coronary sinus, is endowed with a powerful musculature, which at points is continuous with that of the auricle. The auricle therefore, on myogenic lines, receives an impulse to contraction from both the caval muscle and the other structures entering it, and pulsating prior to it, at the same time as from the sinu-auricular node, if ordinary cardiac muscular fibres be conductive.

I have already stated that some fibres from the sinu-auricular node may be observed in the pig to invade the caval muscle as well as the auricle. Did we ascribe to the nodal texture of the sinu-auricular node, under these circumstances, the power of provoking at once the contraction of the caval muscle and the subsequent contraction of the

[1] *Journal of Physiology*, October, 1910.

auricle, it would imply a double function regulated by the nervous system, with which we now know it to be endowed. But, inasmuch as the caval muscle is itself independently innervated, and the nodal action from this point would not explain the pre-auricular contraction of all the other structures mentioned, this view can scarcely be entertained. That the sinu-auricular node, nevertheless, has a function must be regarded as probable. As a nerveless residuum of the aboriginal embryonic heart, it might have been dismissed as effete; although the constancy of its position, even under these circumstances, would have been remarkable. But as a fully innervated and vascularized organ one must regard it as having a living purpose in the mechanism of the heart-beat. What that purpose is is not clear. Its development in the embryo requires further investigation before its 'pace-making' or any other function can be regarded as settled.

Thomas Lewis distrusts conclusions drawn from destructive methods of experiment and from observations on the dying heart, but believes that electrical stimulation, guided by electro-cardiographic tracings, yields evidence which shows that normally the heart-beat commences at the sinu-auricular node. Stimulated elsewhere than in this neighbourhood, the preventricular wave shows departures from the normal shape. For Lewis this is sufficient evidence that the heart-beat commences at the sinu-auricular node. The observations of earlier workers were according to him coloured by contemporary conceptions of morphology, and placed the initiation of the cycle, as we have seen, higher. 'In the mammalian heart,' he states, 'no additional chamber, in the form of a sinus, exists.'[1] The coronary sinus, however, we have seen (p. 13), is a scarcely altered portion of the cardiac sinus as seen in the cold-blooded vertebrates, while the modification of other parts of the sinus in mammals still leaves them recognizable, and, whatever the shape of the wave of auricular contraction as reproduced either by electrical or mechanical methods, it is preceded by a visible if not registrable contraction of the sinal structures entering the auricle.

[1] Report, Section I., International Medical Congress, 1913.

Pathology of the Nodal Structures. — The evidence as to function, to be derived from a consideration of the consequences of pathological change in the *sinu-auricular node*, is as little positive as is that derived from experimental physiology. Not that certain abnormalities of cardiac rhythm have not been ascribed to pathological conditions observed to affect it. Such alteration has usually been of a chronic inflammatory type. On the other hand, I have myself known it to be completely involved in sarcomatous growth without any disturbance of left ventricular rhythm resulting.[1] The right auricle and right ventricle in this case were very largely immobile from sarcomatous transformation and fixation.

As regards the auriculo-ventricular node and tract, the matter is different. Just as the results of experimental physiology are more definite in this case, so also phenomena associated with pathological changes in these structures are frequently more notable. I say frequently, for there have been cases carefully examined and reported in which no sufficient change has been found to account for the dissociated auriculo-ventricular action which unquestionably existed. Other cases have been reported in which, apparently, destruction of the structure has taken place and no retardation of ventricular rate has been noted.[2]

The conditions affecting the node and bundle have been inflammation (acute and chronic), calcification, involvement in new growth, and obstruction by aneurismal pressure. As one would expect from the experience of experimental physiologists, the pathological changes which are associated with complete and persistent dissociation of auricular and ventricular action are as a rule those which completely obstruct or destroy the continuity of the bundle in a part common to the two ventricles. Here again I say 'as a rule,' for persistent dissociation with bradycardia has been noted in cases in which an exhaustive examination of the structures has shown but partial disease. Josué[3] suggests that these

[1] *Lancet*, January 9, 1909.
[2] Martin and Klotz, *American Journal of Med. Sciences*, N.S. 140, p. 223.
[3] Report, Section I., International Medical Congress, 1913.

varying results of auriculo-ventricular nodal invasion may be explicable in some measure by the results of Frédérique's experiments, to which I have already referred (p. 17).

It is natural that the majority of cases examined satisfactorily should have been those in which clinical evidence of dissociated action (heart-block) has been obtained during life. It is desirable, however, that cases should be investigated which have not presented the usual signs of this condition. In this category, from the absence of ventricular retardation during life, the graphic evidence of delayed ventricular response has frequently not been noted, and changes found in the bundle after death have therefore not had the value for determining the function of the parts which they would otherwise have had. It may, however, be accepted that most cases which have shown a normal rate and rhythm of ventricular action during life have not been the subjects of heart-block. As exceptions to this we may regard cases in which the auricular rate has exceeded the normal, and the ventricular rate, though normal, has been relatively slow in comparison with the auricles. In such cases, however, as Hertz and Goodhart showed, the conditions differ from those obtaining in heart-block with organic disease of the bundle, inasmuch as the ventricular rate is accelerated by atropine,[1] and probably also by other normal causes of ventricular acceleration, such as exertion and emotion. These considerations notwithstanding, there is enough pathological evidence confirmatory of the results of physiological experiment to show that the structures composing the auriculo-ventricular node and bundle have a special relation to the influences provoking a normal rate and rhythm of ventricular action. What are those influences?

[1] *Quarterly Journal of Medicine*, January, 1909.

CHAPTER III

THE ACTION OF CARDIAC MUSCLE

THERE are in the heart three main types of visceral muscle vascular muscle, nodal muscle, and what is usually termed 'ordinary muscle,' but which I prefer to call 'tonic muscle.' Of the last, there are various types distinguished chiefly by calibre.

As these three kinds differ in anatomical structure, so also do they differ in function. Like the nodal muscle and some other structures at one time regarded as automatic, such as the intracranial vessels, the coronary bloodvessels were supposed to be without nervous endowment, and have only comparatively recently been brought into line with the other bloodvessels of the body by being shown to be vaso-constrictor and vaso-dilator at the instance of the nervous system.[1] The innervation of the larger branches of the coronary arteries by nerve fibres coursing in the external coat of the vessels has been easily demonstrable for long. On examining microscopically the coronary arteries in a case of angina pectoris in 1902, I found also an innervation by well-developed ganglion cells situated between the internal and middle or muscular coats of the vessel.[2]

That, like the arteries in the somatic portion of the body, the coronary vessels contain nerve fibres which may evince, under certain circumstances, great sensibility will be maintained when dealing with the sensory disorders of the heart.

The nodal muscle, which in its main masses or centres—the sinu-auricular and auriculo-ventricular nodes—has a

[1] Brodie and Cullis, *Journal of Physiology*, Vol. 43, p. 313.

[2] *Lancet*, vol. ii., 1902.

close relation to the vascular system, we have seen to be very fully innervated, an innervation which, like the nodal system itself, is more developed in some animals than in others, and notably so in the ungulates.

The ordinary, or tonic, muscle, of which the chief mass is in the ventricles, the aspirative and propulsive powers of which are the main factors in maintaining the circulation, is fully innervated.

Although no nodal structure has so far been discovered in the left auricle to complete the bilaterality of the nodal centres of the heart—for the division of the auriculo-ventricular node into right and left ventricular branches secures this bilaterality for the ventricles—the bilaterality of the cardiac nervous system is demonstrable.

Macroscopical anatomy gives us the right and left pneumogastric nerves, the right and left cardiac visceral outflow by way of the stellate ganglia, the rings of Vieussens, and the inferior cervical ganglia; while microscopic anatomy shows collateral or autonomic ganglia at the entrances of the pulmonary veins, as well as at the venous inlets of the right auricle. Large trunks, the greater part, if not all, of which belong to the accelerator and augmentor groups, may also be followed into both ventricles; while clinical phenomena indicate the presence of fibres afferent to the central nervous system, and, as shown by the effect of the emotions, fibres also efferent from the higher nervous system into the heart. The peculiar depressor nerve likewise, afferent to the vaso-motor centre in the medulla, appears to have a special relation to the vascular system.

We have, therefore, in the heart, in association with the three types of musculature named, three modes of innervation, which may be termed respectively the 'vaso-motor nervous system,' the 'nodal' or 'cardio-motor nervous system,' and the 'cardio-tonic nervous system.' In other words, whatever view be taken of the nature of cardiac motion, its manifestations in rhythm, propagation, and force, or tone, are under the control of the nervous system. So much is conceded even by most of those who have advocated the intrinsic possession by the cardiac muscle of the Gaskel-

lian qualities of excitability, contractility, rhythmicity, conductivity, and tonicity—the pentagon of properties by which they still explain both normal and abnormal cardiac action. The heart for them is still automatic, but it is a controlled automaton—as Wenckebach has said, a bridled horse.

Scientific inquiry into the nature of things necessarily commences *ab infero et imo*, and an inquiry into the nature of cardiac action must commence with a study of the evolution of the organ in the embryo.

According to Nicolai,[1] Wagner in 1850 was the first to note that the heart of the chick could be observed to pulsate thirty-six hours after germination, while nerve cells could not be detected in the structures of the primitive heart until the sixth day. That the cardiac tube pulsates before nerve elements can be detected has also been observed by others, including W. His, junior. This fact has been regarded as evidence for the muscular automatism of the developed heart. As Nicolai points out, however (*loc. cit.*), the material which pulsates at the early period mentioned in the chick is not muscle, but an undifferentiated material—the 'contractile protoplasm' of Kölliker. Like the cardiac muscular fibre, and unlike the central nervous system which is derived from the epiblast, the lateral or sympathetic ganglia and their derivatives—the collateral and peripheral ganglia in the viscera—according to Paterson, spring from the mesoblast. This view is not shared by some, but Paterson's facts seem to me difficult to interpret in any other way.[2]

Except in the case, so far as we know at present, of one animal, the king-crab (*Limulus*), the cardiac nervous system is so incorporated with the cardiac muscle as to render its separation from the latter for experimental purposes impossible. In that animal, however, which at times attains a considerable size, the chain of cardiac ganglia lies quite outside the cardiac muscle, and the latter, after careful examination, has been shown to contain no ganglion cells. Carlson found on dividing the connection between these

[1] *Archiv f. Physiol.*, parts i. and ii., 1910.
[2] Phil. Trans. B., London, 1890.

ganglia and the pulsating heart that all movement in it at once and permanently ceased.[1] There is, however, said to be a distinction between the behaviour of the cardiac muscle of *Limulus* and that of other animals, inasmuch as it can be tetanized by faradic stimulation like voluntary muscle.

Without attaching too much importance for our present argument to the interesting anatomical and physiological peculiarities exhibited by the heart of *Limulus*, it is nevertheless a noteworthy fact that cardiac motion in it has been shown to be indubitably of neural origin. It must be borne in mind also, that the observation of the various qualities mentioned as appertaining to cardiac muscle has been made on hearts sufficiently developed to be fully endowed with nerves, and it is an easily observed fact in intra-uterine life that the movements of the fœtal heart are influenced by uterine contraction, just as in extra-uterine life they are modified by respiration. Such modification, both within and without the uterus, can only be brought about through the agency of the nervous system.

I have already mentioned that both the auriculo-ventricular bundle and its intrinsic and surrounding innervation are very notably developed in ungulates, and that division of the bundle has a more striking effect in producing dissociated auriculo-ventricular action in them than in other animals. It is difficult to believe that the severance of this pronounced innervation has not a share in the production of the remarkable crippling of 'conductivity' or ventricular response, observed under these circumstances, or to escape the conclusion that the other properties of cardiac muscle are not likewise dependent upon originative impulses in the nervous system.

In every developed organ except the nervous system three factors may be said to constitute the functional unit—its cell, the innervation of such cell, and its blood-supply. Even in the nervous system, with its double current of afferent stimuli and efferent impulses, the former might be regarded as a mode of innervation of the executive nerve cell. Yet one cannot strictly speak of the innervation of a

[1] *American Journal of Physiology*, vol. xii., 1905.

nerve cell. Here the functional unit is, strictly, double, not triple—namely, the cell and its blood-supply. The nervous system, like the blood, is, then, common to every variety of organic cell. The blood nourishes the whole cell, and is necessary to the exercise of its every property; the nervous system controls, regulates, provokes its every property; for how can it control and regulate the properties of muscle without having the power to restrain or unloose energy which has certain visible manifestations, such as contraction, rhythmical or arhythmical?

The nodal structures in the heart have hitherto been regarded as remnants of the primitive cardiac tube. Their embryological evolution and history, up to their ultimate and persistent form, require further study. The sinu-auricular node is regarded as originative of cardiac motion, the auriculo-ventricular node and tract as chiefly conductive of the auricular stimulus to contraction of the ventricle. Whatever the date at which the auriculo-ventricular node originates, and on this point more information is required, the facts I have mentioned leave no doubt that it grows ventriclewards long after the primitive tube has attained a complex development. That is, it cannot be regarded as a remnant of the primitive tube which has not participated in the progressive development of the rest of that structure, but as a special differentiation of mesoblast which develops, grows, and spreads on its own lines, to exercise a special function.

Like muscle and nerve, it is probably excitable; unlike muscle, its contractility is altogether doubtful. Like nerve, it is probably conductive. Its rhythmicity is, naturally, as doubtful as its contractility, and its tonicity more doubtful than both these properties. Properties possessed by the node must likewise be possessed by the bundle, and, if the rhythmicity and contractility of the general mass of the node be conceivable conditions, it is difficult to imagine what object could be attained by similar properties in the bundle. Tawara[1] concludes, from its anatomical structure

[1] *Loc. cit.*, p. 185.

and relations, that the *conduction* of a rousing stimulus (*Erregungsreizes*) is its only purpose.

Excitability and conductivity, then, are the two qualities with which it may be regarded as endowed, with the former like nerve and muscle, with the latter like nerve. For has muscle an intrinsic conductivity, or is the apparent spread of muscular motion effected by nervous channels? This as regards the auriculo-ventricular node and tract.

The conditions affecting the sinu-auricular node are somewhat different. There is no question of its receiving muscular impulse. It is credited, as I have said, with regulating and spreading it.

Cardiac motion, we have seen, commences visibly in the sinus—that is, in the large veins entering the heart, including the pulmonary veins and in the coronary sinus. The well-developed musculature at these entrances is quite continuous with that of the auricle, and hypertrophies with it when that chamber is hypertrophied. If muscular conduction by ordinary fibre be anywhere possible in the heart, it is here. How much stimulus the auricles require to induce contraction on myogenic lines may be argued; but if the stimulus from the sinal structures be not sufficient for this purpose, they must require a large amount of it, and if sufficient, the action of the sinu-auricular node as a pace-maker would seem a work of superfluity, and is, moreover, in no way applicable to the pre-auricular contraction of the pulmonary veins. The latter, powerfully muscular and continuous with the left auricle, are also richly innervated, and, as I have said, so far no nodal tissue has been discovered in connection with them.

Again, when Stanley Kent first described the fibres which unite the right auricle and ventricle at the point now recognized as the auriculo-ventricular junction, he also described a union between auricle and ventricle at their outer boundary.[1] Kent investigated the point by serial coronal sections in the hearts of small animals such as mice, and speaks of auriculo-ventricular junctions both at the septum and at the outer boundary of both the right and left auricles

[1] *Journal of Physiology*, vol. xiv.

as being easily shown. Keith has stated that he has never been able to discover this outer conjunction, and its existence is not generally admitted. It is, however, clearly demonstrable in several sections which I have made in the heart of the mouse, and, though slender, is quite a possible channel for the conduction of auricular motion to the ventricle, granted the conductivity of ordinary cardiac muscle. I have, however, only observed this junction in the right side of the heart.

By serial sections of the heart, both coronal and sagittal or longitudinal, of the same animal, in which there is no pars membranacea septi, a very close approximation of ordinary auricular and ventricular muscle may be noted, but only a few sections in the series I made show so intimate a coalescence as to justify the belief in the muscular continuity of the chambers. As we cut in sagittal section, we find the continuously muscular septal limb of the auricle fitting like a cap on to the broad end of the septum ventriculorum, in close and firm union with it, but not muscularly continuous. Cutting still more deeply, we pass this point and verge upon the left auricle, with the open ventriculo-aortic orifice. Here we note the muscular fibres of the auricle run like a tongue into the septal cusp of the aortic valve in such a manner as to show that, during auricular systole, when the aortic column of blood falls and rests upon the aortic cusps, the cusp in question is tightened by the action of the auricle in systole. It is, doubtless, thus strengthened to receive the impact of, and to support, the column of aortic blood.

Unlike Kent, I have not, as I have said, found the same close approximation and partial fusion of auriculo-ventricular fibres at the left auriculo-ventricular junction that I have at the right. It is quite possible, even in carefully executed serial sections, to miss such a point ; but my belief, from my own experience is that the conditions in the right and left heart in this respect are not the same.

The reason for the all but complete separation at the right auriculo-ventricular junction, except by way of the nodal structures, and for the absolute separation at the left auriculo-ventricular junction, is, doubtless, the necessity for the

separate action of these chambers to aspirate and propel the blood. In the adult or aerial heart, the relation of the organ to the lungs differs on the right and left. Pulmonary aspiration aids the propulsion of the blood in the right heart, while it retards it in the left, on which side also the enormously greater work of propelling the systemic arterial circulation falls. As the labour involved is, under the circumstances, greater on the left than on the right side, the separation of those cavities is more complete, thus affording both left auricle and ventricle the firm fibrous *point d'appui* which enables them consecutively to propel the arterialized blood with most advantage. Muscular continuity would under these circumstances, apart from any question of the conductivity of muscle, be a mechanical disadvantage in the mammal, although sufficient for the requirements of the reptile, fish, and early mammalian fœtus—animals in which the musculature shows departures from the adult mammalian type sufficiently great to reduce the value of argument from the one to the other.

CHAPTER IV

THE NEURAL AND MECHANICAL FACTORS IN CARDIAC ACTION

SINCE purely myogenic conceptions of cardiac rhythm and action have been entertained, there has been a tendency to reduce the rôle of the cardiac nervous system and to minimize the importance of the purely mechanical factor in the circulation of the blood. The rate, rhythm, and force of the cardiac contractions have been explained by the varying rhythmicity, conductivity, and tonicity of the successive structures of the heart from base to apex of the cardiac tube.

Important as were the discoveries made in our knowledge of the innervation of the heart during the period when the pendulum of opinion over-swung in the neurogenic direction, and important as have been the results of research into the musculature of the organ since it has over-swung in the myogenic direction, we have in the course of the preceding remarks observed that some new facts, both in the innervation and musculature of the organ, are calculated to modify some of the views of myogenists which, before the discoveries of these facts, appeared to rest upon a strong foundation.

Among these new facts may be mentioned the evidence for a different distribution of the two pneumogastric nerves, the complete and complex innervation of the so-called 'automatic structures' in the heart, and the evidence I have myself adduced by anatomical examination of the comparatively late growth and extension of the auriculo-ventricular node and bundle in the embryo.

A few words on some purely mechanical conditions underlying cardiac action seem to me, therefore, required in this

place. The points in the mammalian heart at which there is an unquestionable muscular junction between the component structures are those at which the large veins and the coronary sinus enter the auricle, and where the union between the auricles and that between the ventricles takes place. While in the case of the auricles and ventricles respectively simultaneity of action normally occurs with unhesitating smoothness, with a similar continuity of structure the action of the great veins and of the coronary sinus definitely precedes that of the auricles.

Of these structures the coronary sinus is the most complex, and must be regarded as a *chamber* rather than the mouth of a vein, having, as I have already stated, all the conditions necessary to an active chamber—namely, valves behind it to prevent reflux from it, and a valve in front of it to prevent reflux into it. Here simultaneity of action with the auricle does not occur, for the sinal systole precedes that of the auricle ; but the conditions involved do not require the same forcible propulsion of the contained blood as in the case of the mammalian auricle and ventricle. It resembles more the sinuous systole of the reptilian heart.

Serial sections of the heart of a small animal such as the mouse, which give a more extensive view of the parts concerned, show well the purpose of the intercavital interruption to muscular continuity. The firmly implanted foot, on the one hand, of the septal limb of the right auricle on the interventricular septum, in close connection to but not continuous with it, and, on the other, another portion of the same limb implanted on and muscularly continuous with the left auricle, show the mechanical value of the fixed point, in the one case, for powerful systolic propulsion, and in the other the equally important yielding nature of a junction by muscular continuity, which secures simultaneity of the systole of the auricles.

Similarly, the central fibrous body (in some mammals such as the sheep and horse, further strengthened by transformation into bone), with its continuation into the fibrous boundary separating the auricles from the ventricles, affords the latter the *point d'appui* necessary for their vigorous and

rapid contraction. Muscular continuity in these situations would have produced the sinuous and sluggish contraction of the reptilian chambers, which also, it must be remembered, notwithstanding their continuity, are consecutive in action.

The ossification of the central fibrous body in the ungulates is interesting, as also is the precise position of this cardiac bone. In the sheep it lies usually above the right and posterior cusps of the aortic valve and close on to the musculature of the ventricles. Between it and the atrio-ventricular bundle there is a considerable mass of ventricular muscle, that structure being deeply buried in muscle in this animal. In another ungulate, the pig, having a richly innervated bundle, there is no ossification of the fibrous body, which is itself pierced by the bundle *above* the ventricular muscle, much as the pars membranacea septis is in the case of man. The rat, again, has a deposit of cartilage in the fibrous structure supporting the aortic cusps, but at a considerable distance from the auriculo-ventricular bundle. It cannot, therefore, be supposed that this osseous induration of the central fibrous body has any protective influence for the richly innervated bundle of the ungulate; and it must in all probability merely be an exaggeration of that stiff intercavital separation which is essential to the powerful intermittent propulsion from auricle to ventricle and from ventricle into the arterial system.

At no point of the cardiac mechanism is the importance of the mechanical factor more pronounced than in the ventriculo-pulmonary section of the circulation. Here, also, as in the case of the sinal structures entering the auricle, the ventricular heart is continuous with the bulbar heart or conus arteriosus; but in the mammal there is no such perceptible intermission in action between ventricle and conus as there is between sinus and auricle. The post-natal changes on the establishment of the aerial circulation, which the massive fœtal right chambers demand in size and power, in consequence of the diminution of the labour thrown upon them, are very eloquent of the importance of integrity of the mechanical factor in the normal circulation, and as eloquent of the importance of defect in the mechanical factor in heart

disease which has compensatory and, later, degenerative changes in its train.

The demonstrable muscular continuity between sinus and auricle and auricle and ventricle in the reptile, and the experimental reversibility of muscular conductivity in strips of muscle from the same animal, supplied facts for the theory of the muscular nature of the phenomena of cardiac action in that animal, although, as we know, the innervation of reptilian cardiac muscle is rich. But the auriculo-ventricular separation of the principal chambers in the mammal, for some time after Gaskell's revival of the myogenic theory, left the union of auricle and ventricle in action to the nervous system.

Although Gaskell had argued the essential identity of conditions in cold and warm-blooded animals (*loc. cit.*), it was not until the discovery, as I have stated, in mammals, of the auriculo-ventricular node and bundle, and of the sinu-auricular node, that the missing links were found which advanced the claim of the cardiac muscle to be regarded as independent of the nervous system in the manifestation of the phenomena of the heart-beat. These structures then became the automatic centres for the action of the ventricles and auricles respectively.

In time the separateness of these centres, regarded as residua of early embryonal automatic activity, became less recognized, and the initiation of cardiac motion was left to the so-called ' pace-maker,' the sinu-auricular node; while the auriculo-ventricular structure became a receptor and transmitter, rather than an initiator of ventricular activity; and the whole auricle, or some part of it, came to be regarded as capable of generating the stimulus which might be conducted to the ventricle, and explode energy there, to be expressed as ventricular motion.

The auriculo-ventricular pause was thus to be explained, as in the reptilia, by the interposition of an embryonic strand between auricle and ventricle, which, in accordance with Gaskell's law of conductivity being inversely as rhythmicity, retarded the transmission of the auricular impulse to the ventricles. Tawara, however, on the anatomical ground of

the *length* of the so-called 'primitive texture' of the auriculo-ventricular bundle, before it reaches its final transformation into or contact with ordinary muscle, considered that transmissions of auricular impulse must be quickened rather than retarded, which, if true, would negative the correctness of Gaskell's law, provided always the bundle be considered to have a superior rhythmicity. It is now also very generally acknowledged, as Ivy Mackenzie recently pointed out,[1] that there is only a very superficial resemblance between embryonic and nodal tissue, which, as I have shown and suggested, is more probably a later differentiation of mesoblast subserving the functions of the nervous system. That, apart from any question as to muscular or neural conduction, however, the almost complete separation of the auricles and ventricles from one another by fibrous texture, is the chief cause of the pause between auricular and ventricular systole, can scarcely be doubted, however the ultimate response of the ventricle be explained. The auriculo-ventricular boundary is the line of auricular insertion.

The embryology of the nodal structures, as I have said, still requires much investigation before dogmatism on their essential nature will be permissible; but facts which I have related show that the auriculo-ventricular node, at any rate, is a peculiar innervated structure, which continues to grow into ventricle at a comparatively late period in the development of the heart, and penetrates those chambers on the same lines as do the nerves and vessels of the organ—that there is, in short, an auriculo-ventricular nodal system, innervated itself and accompanied by nerves, whose destiny is to be distributed to the ventricular muscle.

Whether the sinu-auricular node, in like manner, continues to grow in the embryo while retaining its original embryonic character, or whether it be a residuum of embryonic tissue at all, and not a special neuro-muscular organ, cannot finally be determined at present for lack of necessary facts. That its fine fibres penetrate into the interstices between masses of ordinary muscle, in a manner similar to vessels and nerves, is easily demonstrable under

[1] Report, Section I., International Congress of Medicine, 1913.

the microscope; and that the cardiac nodal system, like the cardiac vascular system and the whole mass of ordinary cardiac muscle, is under nerve control, is not now disputed or disputable. The pendulum of opinion, over-swung in the myogenic direction, is at present stationary in its over-swung position, or tending slightly nervewards. The relation to and influence upon the nodal system of the nervous system has now investigators. What, then, is nerve control, and to what extent does the nervous system control the action of cardiac muscle?

CHAPTER V

THE NERVE CONTROL OF CARDIAC ACTION

THERE are in the heart, as I have said, three main types of muscular texture—vascular muscle, nodal muscle, and ordinary or tonic cardiac muscle, whose function is as distinct as their structure; and as all are under the control of the nervous system, the latter must also be modified in its relation to them. The relative anatomical position of these three types is suggestive, and might be diagrammatically represented as a series of concentric circles, of which the innermost is the vascular; that immediately surrounding it the nodal; and the ordinary, or, as I prefer to name it, the tonic musculature, the largest and embracing both the others. The nervous system controlling these three types would be termed respectively the 'vaso-motor nervous system,' of which we know a good deal; the 'nodal' or 'cardio-motor' of which we are beginning to learn something; and the 'cardio-tonic' of which we also know a good deal, though not under that name, but as the 'accelerant' or 'augmentor and vagal nervous systems.' Of all three we require to know a good deal more than we do, even anatomically.

Since the origin of the myogenic and neurogenic controversy, which is not of yesterday, is certainly of to-day, and probably also of to-morrow, it has been the fate of all three to be denied existence in the first place, to be allowed to exist in the next, and to be permitted to have functional activity and influence at last. To what extent, then, does their influence exist?

Beyond the power of the will, all are peculiarly sensitive to the emotions, a fact which, Reid says, the estimable

Haller regretted for the sake of myogenicism![1] A scientific appreciation of the physiology of the emotions scarcely exists except in general terms, although the topic is important, especially, as Huchard[2] and others have pointed out, in its bearing upon cardio-vascular pathology. Varying with the degree and kind of emotion—trivial, pleasant, or painful—the heart's action is immediately and constantly and directly influenced by emotion. Indirectly, it may be said to be influenced also by the intellect and will. For, difficult as the control of the emotions by the will may be, it is not impossible, as constant experience teaches us. The sources where these important influences in the higher centres arise, as also the channels by which they travel to the viscera, including the heart, are imperfectly known. But we know, in the case of the heart and vessels, that they may be accelerant, augmentor, depressant and depressor, pleasurable, or painful in effect, and must find expression in great measure by the same channels as the variation in cardiac action produced by the unconscious influences of a peripheral and physical nature, which provoke adaptation to them, in the cardio-vascular system.

The argument for nerveless automatism based upon the asserted *absence* of the innervation of any structure regarded as automatic, may, we have seen, be dismissed. Such 'absent' nerves are always discovered sooner or later. The chief arguments for the nerveless automatism of cardiac action are based upon observation of the early embryo and upon the study of the eviscerated heart. At the very early period at which the heart of the germinating ovum has been observed to pulsate, the pulsating structure, as Nicolai remarks, is not muscle, but primitive protoplasm. The study of the *properties* of cardiac muscle, even *in utero*, is only possible at a period when muscular structures are fully innervated. Under these circumstances variations in rate and force of action may be observed to be due to such physical influences as uterine contraction, in which the psychical factors referred to above are absent. During

[1] 'Physiological, Pathological, and Anatomical Researches,' p. 2, 1848.
[2] 'Maladies du Cœur Arterio-Scleroses,' 1910.

severe uterine contraction at term, the fœtal heart may constantly be observed to be retarded, quickening on the relief of intra-uterine pressure. Such cardiac variation of action is only explicable, like respiratory variation in aerial life, on neural lines.

The elimination of the psychical sphere secured by fœtal conditions is extended to that of the entire central nervous system by removal of the heart from the thorax. As is constantly demonstrated in the laboratory, the heart under these circumstances may continue to beat for a length of time with little encouragement in reptiles and amphibians; and with care and artificial feeding by saline solution and oxygen also in mammals. Even the human heart removed after death, and kept on ice, has been made to resume pulsation after a considerable period when artificially fed and warmed.[1] There is, therefore, no doubt that the heart contains within itself the mechanism and incentives to sinu-auriculo-ventricular motion. The most obtrusive of the structures thus surviving or revived are, of course, the muscular textures, the movement of which is perceptible to the naked eye and easily registrable.

That the nervous structures also, under these circumstances, retain excitability and conductivity, is shown by the effects of electrical stimulation in the eviscerated and perfused heart. As I have already mentioned, Cohn and Trendelenburg found a notably greater dissociation of co-ordinate auriculo-ventricular action in the ungulates (goat), with their large and plentiful innervation of the 'connecting' structures than in other animals in which this innervation is less pronounced, although they attributed this fact to the youth of the subjects—kids a week old—rather than to the condition of their innervation. Friedenthal[2] divided all the nerves to the heart at intervals without killing the animal, which survived for many months, without unduly disordering the heart's action. Although he mentions Smirnow's section of the depressors, and the consequent degeneration of the sensory nerve endings in the endo- and

[1] Kuliabko, *Pflüger's Archiv*, vol. xcvii.
[2] *Archiv f. Physiol.*, 1902.

peri-cardium, he does not appear to have made any microscopical examination of the structures of the heart, to determine the course of degeneration in this case. Had he done so, he would probably have found the degenerative process cease at the next cell station in the cardiac plexus, although collateral and vagrant cell stations are not recognized as independent at present by physiologists in the case of the heart.

In the case of the stomach and intestines, the solar plexus and the superior mesenteric ganglion are recognized as cell stations, medullated fibres intended for the stomach passing through the lateral ganglia for demedullation in the solar plexus, and fibres intended for the intestines passing through the solar plexus for demedullation in the superior mesenteric ganglion. But the comparison cannot be pushed too far, as there are certain differences in the effects of stimulation of the nerves in the case of the heart and the digestive organs. For whereas the heart is inhibited by stimulation of the vagus, the stomach and intestines are provoked thereby to peristalsis. Stimulation of the sympathetic outflow, on the other hand, accelerates the movements of the heart, but inhibits those of the gastro-intestinal tract.[1] Later researches mentioned by Starling[2] show, however, that the vagus also contains fibres which inhibit the stomach, and the splanchnics some, the stimulation of which augments its movements. Finally, Nicolai quotes Morgan and Row as having found that removal of the submucous plexus in the stomach caused rhythmical action generally in that organ to cease.[3] In connection with the question, however, of the essential nature of cardiac motion and of viscero-muscular motion generally, apart from grosser regulatory effects on movement, Pawlow's ingenious experiments on gastric secretion have revealed the suggestive fact that the psychical stimuli which provoke such secretion run in the vagus, after section of which *such* stimuli cease to induce secretion, even electrical stimulation of the peripheral cut-ends of the nerves having only a slow response from the glands.

[1] Haliburton, Kirk's 'Physiology,' p. 681.

[2] Schäfer's 'Physiology,' vol. i., p. 324.

[3] *Loc. cit.*, p. 39.

As we have seen, in the case of the heart, section of the auriculo-ventricular nodal system induces, according to some, the absence of, and according to all, diminished response from the ventricle to vagal stimulation, which is in this case normally the inhibition or prevention of movement; and, as I have also stated, recent experiments[1] appear to show a closer relation of the upper node to the right and of the lower to the left pneumogastric nerve. While it is necessary at the present stage of this investigation to avoid dogmatizing on the special channels of nervous influence which govern that internal secretion of the cardiac muscle cells which provokes motion, and which is vaguely termed 'stimulus production,' this, which corresponds to the secretion of glandular organs, is no less under the control of the nervous system than is any other secretion.

To give a name to an effect or property does not explain its origin or nature, or even the proximate cause of the phenomenon described. Excitability may be regarded as a property of matter distinguishing the so-called organic from the inorganic, although the distinction is arbitrary and only convenient for discussion, rather than actual, for chemical reaction may be argued to be a mode of activity akin to life. The rhythmicity, contractility, and tonicity of muscle are merely descriptive terms, and its independent conductivity an assumption. For even the reversal of the phenomenon termed 'conduction,' under the circumstances in which it is observed and induced, does not exclude the excitation of other elements than muscle contained in the stimulated structure.

The myogenic theory of cardiac muscular action rests chiefly upon the absence of evidence considered satisfactory of the existence of a true reflex action in the visceral nervous system. Such *apparent* reflexes as Gaskell states (*loc. cit.*) are explained at present, like the hypogastric reflex, by the contiguous stimulation of post-ganglionic efferent fibres. This, however, is not certain, and there are points both in the anatomy and physiology of the visceral innervation which still require further investigation. Even positive

[1] Cohn, Robinson, Draper.

assertions on these points in the past have been shown later to be erroneous. In the only known instance in which the nerve supply of the heart is completely detachable from the musculature—namely, in *Limulus*, the dependence of all the properties of the cardiac muscle on its innervation has been demonstrated by Carlson (*loc. cit.*).

It seems, therefore, reasonable to believe that stimulus production in cardiac muscle may be provoked or checked by the nervous system, that the conduction of muscular movement from one part to another may also be influenced by it, and that the tone of muscle may certainly be augmented or depressed by the action of the nerves supplying the muscle manifesting these variations, modes of action, or properties.

The occasion for such variation, again, is due to external incidents. The muscle cell has to adapt itself to some situation impressed upon it. It may have to quicken, to slow, or to energize in various degree to meet such situation, and the manner of its response is controlled or permitted by the nerve factor in the functional unit, while the perception by the cell of the necessity for variation can only come to it through the nervous system. The muscle factor, therefore, the cell, in the triplex functional unit of blood, cell and nerve, is nourished in proportion to its activity by the first, regulated by the vaso-motor nervous system, and caused to respond in any particular manner by the last, a situation which leaves the nervous factor paramount for executive purposes, and the muscular factor the instrument of its will, so to speak, whether such will be to increase, diminish, or arrest the production of that stimulus, without the generation of which the cellular motion could not occur. In the production of this stimulus material, as in the production of visceral secretion generally, the vagus appears to be especially, although not solely, concerned. The triplicity of the functional unit mentioned belongs no less to the nodal than it does to the ordinary muscle of the heart. It has been suggested by Retzer,[1] and more recently by Graham Brown,[2] that the nodal structures may be akin in function to the muscle spindle in voluntary muscle. 'The general con-

[1] *Anat. Rec.*, vol. ii., 1898.

[2] *Edin. Med. Journ.*, June, 1912.

clusion,' remarks Ivy Mackenzie,[1] 'which a survey of the anatomical data would suggest, is that the two systems—sinu-auricular and auriculo-ventricular—are large neuro-muscular spindles, the one controlling auricular contraction, and the other controlling ventricular contraction. Further, the two nodes are probably independent cardio-regulatory centres whose co-ordination is determined not by the passage of a wave of contraction from the sinu-auricular node through the auricular muscle to the auriculo-ventricular node, but by the controlling influence of the nervous system.' With so much of Mackenzie's conclusion, the argument in the preceding pages is in agreement.

Graham Brown suggests that 'the atrio-ventricular bundle is in reality a mass of highly differentiated receptors timed to perceive the commencement of contraction in the wall of each cavity of the heart, which stimulus, transmitted to local centres, would seem to regulate rhythmically the contraction of each chamber.' This ingenious theory, when made by Retzer, had objections offered to it on anatomical grounds by Gordon Wilson (*loc. cit.*), and when Brown speaks of the relation of the atrio-ventricular bundle to each chamber, the facts already mentioned show the executive effects of its activity to be limited to the ventricles, unless the sinu-auricular and auriculo-ventricular nodes be regarded as connected. The question is, whether it is merely a receptor and transmitter of auricular impulse, or whether it generates during auricular contraction the incentive to ventricular systole.

This point awaits elucidation by further embryological investigation, but a minute study of my case of foramen primum leaves no doubt in my own mind that, in the exercise of its function, the auriculo-ventricular node has no direct connection with the sinu-auricular node, or any of its appreciable outflow. The well-developed node is, in this case, anatomically detached from any possible connection with the higher structure.

As a nodalation of fully developed cardiac auricular muscle is a less probable process than a progressive development of

[1] Report, Section I., Internat. Cong. of Med., 1913.

nodal texture into ordinary muscle, the junction of this node, if its embedment in auricle be regarded as such, is more probably made by the nodal structure with the auricle than by the auricle with it. The node, in the case I have mentioned, and which represents a persistent fœtalism, appears to be essentially a subepicardial structure, like the sinu-auricular node, intruded between auricular and ventricular muscle. It remains to be shown by further embryological work whether, in the normal development of the heart, its original position, below and external to the coronary sinus, is carried forward towards the position it usually occupies in the fully developed heart, as the interauricular septum becomes complete.

A noteworthy feature of the auriculo-ventricular node is its rich arterial vascularity. Surrounding a large trunk of the right coronary artery, by branches from which it is directly supplied, this arterial supply is in some parts so rich as to suggest a very considerable functional activity of the structure. Its size, moreover, which is considerably greater than that of the sinu-auricular node, appears to bear some proportion to the ventricles, more massive than the auricles, which it has to supply by its prolongations—the right and left ventricular branches or bundles.

Referring again to the phenomena of the heart-beat, as described by Foster (*loc. cit.*), it will be remembered that during auricular systole 'the ventricles may be seen to become turgid. Then follows, as it were immediately, the ventricular systole. . . .' This turgidity is due not only to the filling of the ventricles with blood, but to the coronary vessels also showing a turgid fulness, which is at its height at the moment of and during systole, and which shows a repletion of the coronary vascular system, with raised tension in those vessels. It is this gradually rising intraventricular and intracoronary turgidity which is probably the provocative to the ensuing ventricular systole, as it is certainly the immediate precursor of it. Like the entrance of food into the stomach, when gastric secretion is actively provoked, so in all probability the gradually rising tension of the ventricles provokes the active production of the stimulus material in

the cardio-motor or nodal system of the ventricles, which explodes the contractile energy of the chambers. As, when the vagus is severed, the gastric secretion becomes slow and a bradypepsia is brought into being, so when the bundle is completely severed, the inhibitory power, involving control of stimulus production by the vagus (which shows its secretional influence), is lost or greatly diminished, and a ventricular bradycardia ensues.

It may obviously be objected to this, that auriculo-ventricular activity may for a time take place with regularity when no blood is circulating either in the cardiac chambers or coronary vessels, as in the eviscerated heart. The only difficulty presented by this consideration is the absence of accepted evidence of a cardiac visceral reflex; for, the habitual repetition of an act, even in the voluntary muscular system induces a mode of automatism, as is shown by the swimming and other purposive acts of the beheaded frog. The situation is saved for the nervous system in this case by the recognition of the 'spinal soul'? Is it certain that there is not a *visceral* 'soul'? This is the line which divides the myogenist from the neurogenist, and influences his views not only of visceral automatism, but also his interpretation of the clinical signs of disordered visceral action. Visceral automatism is —these conditions being granted—in other words, *a functional habit* acquired by repetition and inheritance under appropriate stimulation of the intrinsic and essential nervous factor.

In discussing the sensory disorders of the heart, I shall again have to refer to the apparent insensibility of the viscera in many respects, to stimulations such as cutting, tearing, and pinching, but shall point out that such phenomena do not exclude the sensitiveness of the apparently insensible structures to an adequate stimulus, nor the localization in the viscus of referred pain by the best judge—the sufferer.

Notwithstanding the large amount of impressive work which has been done by electrical and toxicological methods (Langley's) to elucidate the relation of the viscera to the nervous system, experience has taught us to be cautious in

accepting finality in physiological conclusions, and the adequate vital stimulus which has been necessary to the establishment of this neural automatism of the rhythmical viscera, may yet find nearer artificial imitation than has hitherto been employed in investigation.

As an example of what I mean, I may refer to the fact mentioned by Sherrington[1] that, although cutting and other gross methods of stimulation of the gall-bladder and duct show no sign of sensibility in these parts, the injection into them of a saline solution immediately causes a remarkable vascular reflex. The presence of a calculus in the duct, in process of extrusion by the muscular and fluid-content pressure of the parts involved, moreover, induces a pain which may be referred by an observer to the abdominal wall, but which is, in the humble opinion of the sufferer, deeper. To these observations reference will again be made in their bearing on certain clinical facts.

The abrupt auriculo-ventricular pause in the cardiac action of mammals on this showing is due to that mechanical separation in the chambers which has been discussed at some length, and the duration of this pause, under normal circumstances, to the time occupied by the rising intra-ventricular pressure and the turgidity of the ventricular section of the coronary vascular system, before ventricular response, actions which become automatic. The auriculo-ventricular fibrous zone, which is the point of insertion for the auricular muscle, becomes also, during auricular diastole, the insertional *point d'appui* of the ventricular muscle during its systole. The difference in *mass*, too, of the three sections of the heart—the sinus, the auricle, and the ventricle—has an influence on the duration of that contraction; but the less definite the intercavital separation, the more sluggish and sinuous is sinu-auriculo-ventricular contraction. This is best exemplified by the reptilia in which, according to Gaskell (*loc. cit.*) and others, stimulation of the vagus fails to inhibit, and the absence in them of a depressor nerve shows that the cardiac sluggard is indifferent to vaso-motor relief.

Current conceptions of the muscular conductivity of the

[1] Schäfer's 'Physiology,' vol. i., p. 854.

auriculo-ventricular junction make the node of Tawara a jumping-off place for the auricular impulse, not a highly developed structure taking the initiative, under nervous excitation and control, in elaborating the stimulus to ventricular action. The endeavour to find an anatomical continuity between the upper and lower nodal structures is a consequence of the conception of muscular continuous action from base to apex of the cardiac tube, from sinal rhythm to ventricular rhythm, which is the key the myogenist uses in deciphering normal and abnormal manifestations of cardiac motion. When we come to consider the latter, I shall endeavour to show that phenomena presented by such actions, normal and abnormal, are quite as validly explicable on that conception of the nature of cardiac action which has been set forth in the preceding pages.

This also I believe to be a surer guide in the prognosis, and a safer and more rational aid in the treatment, of heart disease than a view which, satisfied with the time relations of certain events in the cardiac cycle, and content with the manifestations of muscular force, undervalues the influence of more mechanical factors in the heart's action,[1] and depreciates the importance of a neural factor which may vary rhythmically and arhythmically the action of the heart, as it also may disastrously arrest, as by a bolt from the blue, the whole cardiac function.

[1] Mackenzie, 'Oliver-Sharpey Lectures,' *Brit. Med. Journ.*, 1911, vol. ii., p. 796.

CHAPTER VI

THE CORONARY CIRCULATION AND THE CARDIAC BLOOD

In the preceding pages the muscular and neural factors in cardiac action have been sufficiently dealt with for the purposes of this book. Before closing this scientific preface, some consideration of the third factor in the triplex functional unit underlying organic action—namely, the blood and its circulation—will not be out of place.

The cardiac muscle is nourished in mammalians by the coronary arterial system which usually springs from two vessels arising from the aorta behind the two anterior pouches of Valsalva at the root of that vessel. These arteries sometimes arise by a common trunk and then divide into two main branches. At times, also, the heart is only nourished by one coronary artery and at others there are, as we shall have occasion to recall, three or even four coronary arteries. The place of origin of one or other artery may likewise vary, and Luschka cites a case in which the right coronary arose from the right subclavian artery.[1] The left coronary artery is almost invariably considerably larger than the right, where it issues from the aorta, and the pressure in it is consequently greater. Hyrtl affirmed that anastomosis did not occur between the two arteries, and others have restated and stereotyped his opinion. In 1883, however, Dr. Samuel West[2] determined, by injecting hearts with carmine-gelatin, that there was free anastomosis between the vessels, and in the same year Dr. J. Wickham Legg independently came to the same conclusion, and published his observations in the

[1] *Anatomie des Menschen*, 1869, p. 402.

[2] *Lancet*, June 2, 1883, p. 945.

Bradshaw Lecture of the Royal College of Physicians.[1] Dr. Legg considered that the anastomosis was by way of the apex, not, as maintained by some, in the sulcus at the base of the heart. I was aware of Dr. West's general conclusions, but had forgotten the details of his experiment, when I investigated the matter for myself in 1902, and was culpably ignorant of Dr. Legg's work until after my experiments were finished. Consequently, some points in my procedure differed from that of both these physicians, although the conclusions I arrived at were practically the same. The hearts of children I found most suitable for the investigation in consequence of the greater transparency of the epicardium and the absence of accumulated fat. Like Dr. West, I found hearts which had been kept in fluid for a time, so as to allow rigor mortis to pass off, were more easily injected than fresher organs. My experiments were made under hydro-pneumatic pressure in the ordinary manner and under warm water at 50° C. Gelatin variously coloured was the material used. The arteries were injected either simultaneously or alternately. The heart of a boy six years of age, who was accidentally killed, was simultaneously injected by way of both arteries—the left with carmine-gelatin and the right with ultramarine-gelatin. This done, the red tube —that tied into the left coronary artery—was allowed to lie loose in the water, while the blue tube—that inserted into the right coronary—was injected under pressure. Presently, fluid was observed to issue from the loose left tube, at first purple and then blue in colour; the ultramarine-gelatin had reached the left coronary artery. The process was then reversed and the right tube was allowed to lie loose while the left coronary was injected under pressure with carmine-gelatin. Presently, the fluid issuing from the right tube was observed to become purple and then red; the injection had reached the right coronary artery.

To determine the *mode* of anastomosis, the heart of an infant sixteen months old was taken. The right coronary was ligatured near its origin and the nozzle of the injection apparatus was tied into the left coronary artery, which was

[1] *Medical Times and Gazette*, 1883.

injected with ultramarine gelatin. The whole heart, with the exception of a small portion near the commencement of the right coronary, where the ligature was placed, was injected, and, unlike Dr. Legg, I found the anastomosis between the two vessels took place, not only by recurrent vessels ascending from the apex, but also by vessels which descended from the transverse sulcus and crossed in the sulcus on the posterior aspect of the heart from the left to the right main trunk. On the whole, I found injection of the coronary circulation by way of the artery on the left more easy than by way of that on the right. Nor was the permeation of the injected material limited to the heart only, but travelled upwards in the intervascular cellular tissue, and helped to nourish the coats of the large vessels, especially those of the pulmonary artery in my preparations. This ascending distribution of the coronary vessels probably establishes communication with other branches of the aorta, especially the bronchial arteries, a circumstance which may in some instances be an important factor in maintaining the nutrition of the heart.

Thus Dr. West's and Dr. Legg's experiments, of which my own may be regarded as a repetition and confirmation, show beyond cavil that the arterial system of the heart anastomoses freely. This freedom of communication on the arterial side is matched by the collateral pliancy of the coronary venous system, as shown by a remarkable case published by the late Dr. Moxon of Guy's Hospital in the Transactions of the Pathological Society of London.[1] The case was that of a young man, nineteen years of age, in whom the main coronary sinus was quite obliterated by the pressure of a hydatid cyst, at a point near the normal entrance of the sinus into the right auricle. Notwithstanding this obstruction at so essential a point, the cardiac muscle showed no undue venosity, and a committee appointed by the society determined that the blood had returned freely to its appointed destination by way of the foramina of Thebesius in the right auricle.

Free as is the provision for circulation through the walls

[1] Vol. xxi., pp. 99-100.

of the heart, Dr. Sibson observed[1] that during ventricular systole, arteries and veins alike became turgid and subsided during diastole. This is evidence of momentary pressure on the smaller arteries and of the forward bound of the blood-stream during diastole, at which moment, also, the recoil of blood on to the aortic cusps plays the part of valves at the arterial orifices of the coronaries, and urges forward the momentarily impeded column. In his Bradshaw Lecture already mentioned, Dr. Legg recorded the result of experiments in which one or other, or both, coronary arteries had been ligatured during life. The general experience of those who performed these experiments appears to have been that, while ligature of both vessels was followed by cessation of cardiac movement within a slightly shorter period than when only one was tied, the effect of the single ligature was quite as deadly. An older writer (Chirac) seems to have been the only dissentient from the general opinion, while Cohnheim, likewise quoted by Dr. Legg, found that ligature of one vessel caused sudden cessation of the heart's action in 100 seconds. In view of the very free circulation in this system which has been shown to exist, this result is difficult to explain on a purely hæmic hypothesis. That death should be the result from this cause when both vessels are ligatured we can readily admit, for the hæmic factor is then quite, or almost quite, cut off, but that one vessel should kill on this hypothesis is not easy of explanation. The result is probably due to the suddenness with which an important section of the cardiac circulation is interrupted.

We have touched upon the nature and conditions of the blood-vascular system of the heart. The importance of the quality of the blood itself must be emphasized and the beautiful and necessary provision mentioned, whereby the incessantly active organ, by the short-circuiting of the coronary system within the larger cycle of the systemic vessels, is constantly supplied with the very first of the newly oxygenated vitalizing fluid. Indeed, while it is customary to speak of the pulmonic as a modified system in the circulation from its intrinsic peculiarities, and of the portal system as

[1] 'Med. Anatomy,' col. 73.

distinct for the same reason from the general systemic arterio-venous circulation, but of the coronary vessels as merely a portion of the latter, I think it might be argued that the intrinsic peculiarities of the circulation through the heart, while they cannot place it in a category apart, justify our using the term 'coronary system.' Our knowledge of the share taken by the lymphatic system in this connection is too imperfect for fruitful discussion in this place. All the anatomical and physiological facts, however, which have been mentioned have an important bearing on our appreciating the nature of the sensory and motor disorders of the heart and in guiding us in their treatment.

PART II

SENSORY DISORDERS OF THE HEART

CHAPTER I

INTRODUCTORY

It is a well-recognized fact that circumstances which readily provoke pain in the somatic area of nerve distribution fail to do so in that of the visceral nervous system. Organs may be touched, handled, and cut without the subject evincing sensibility or suffering. To this rule the heart is no exception.

Physiologists teach us, indeed, that even normal subcutaneous textures such as tendons and muscles have little sensibility. This general insensibility of the viscera is, however, conditional. Under certain circumstances, we know that visceral pain of the most acute character may be experienced. The liver, which may be studded with nodules of malignant disease and cause the patient little discomfort, may also evince all the agony of hepatic colic. The intestines, which may be exposed and handled without causing pain, may be the seat of painful spasms from the stimulation of retained material. The heart which may painlessly harbour a hydatid cyst, may evince on occasion such an agony of pain as to cause the sufferer to seek relief in suicide.

To some extent these differences in behaviour are probably due to a difference in the *degree* of stimulation necessary to provoke pain in a viscus, but it is also probable that some structures entering into its formation are more endowed with afferent nerves capable of *painful* sensibility

than others. The comparatively insensitive or differently sensitive elements would in these circumstances require a greater degree or peculiar kind of stimulation to induce pain, if it could be induced at all, than those endowed with nerves of another type, which would transmit painful stimuli more readily and more markedly.

It has appeared to me, from a clinical consideration of the subject, that those parts of the viscera which have the least amount of yielding elasticity, such as the gland ducts, the bloodvessels, and the orifices of hollow organs, are more frequently associated with pain when disordered than other parts of the viscera, but they are probably not the only sites at which the stimulus to pain may arise.

Observation and thought have produced a variety of views on the nature of visceral pain. Some believe that the viscera, as well as those parts of the body endowed with direct cerebro-spinal nerves, can evince considerable pain. Others do not deny a degree of visceral sensibility, but regard the manifestation of visceral pain as chiefly parietal. Yet others consider referred visceral pain to be altogether parietal.

Langley remarks:[1] 'The splanchnic nerves, superior and inferior, and the pelvic nerve, when pinched or stimulated electrically, are apparently as sensitive as any nerve in the body, and we know that certain diseases of the viscera give rise to exquisite pain.'

As Herz has pointed out[2] in his lectures on the sensibility of the alimentary canal, due regard must be had in judging the matter as to whether the stimulus to pain is of an abnormal type, such as cutting, tearing, pinching, or searing, or whether it is 'the adequate stimulus'—the stimulus, that is, appropriate to the nerves in question, as light is to the eye, sound to the ear, and so forth.

This appropriateness of stimulus is an interesting subject; but there is probably a more simple explanation of the apparent capriciousness of the incidence of pain and its expression when arising in the viscera, and especially in the

[1] Schäfer's 'Physiology,' vol. ii., p. 688.

[2] Goulstonian Lectures, p. 46.

heart, which holds a special place among viscera. Herz believes that tension is the only cause of true visceral pain, but I confess that of only causes and only explanations, as of most generalizations, one has learned to be cautious.

In the case of the heart it is not possible by physiological experiment to ascertain the incentives to *pain* and its sites of production. That they vary both in kind and in situation might, *a priori*, have been regarded as probable; and experiments performed by Nature and observed at the bedside, which are quite as important as the results of research in the physiological laboratory, give us some assurance that this is the case.

The manifestation of cardiac pain may be peripheral and parietal, or organic and central. The low sensibility of the heart in health to ordinary stimuli—*i.e.*, to such as affect the skin—has led some to conclude that, while the *stimulus* to pain may be deep, its *expression* is superficial, and that all referred visceral pain is parietal.

As Sir William Osler has pointed out, however, the mind may, and in fact does, as in the case of renal colic, refer the pain to a deep source. In his Lumleian Lectures (1910) he also drew special attention to the pain provoked by the impaction of emboli in bloodvessels. But notwithstanding these remarks, indicating a perception of local visceral sensibility, he appears to agree that disorder of the 'Gaskellian function' of contractility lies at the bottom of angina pectoris. It is, however, with such an explanation, as with most others of a *single* kind advanced, that it is only a satisfactory explanation of *some* cases. 'Contractility' is no doubt that property of muscle which most distinguishes it. Without contractility there could be no movement, and no property of muscle can be more frequently affected than its contractility. And yet cardiac pain on the whole is not common, and angina pectoris as a clinical entity rare. Indeed, in some instances an increased failure of contractility appears to be associated with some relief to præcordial pain quite reasonably regarded as anginal. It has frequently been remarked that, with the establishment of mitral insufficiency in non-valvular cases evincing angina, the frequency

of such attacks has diminished, and we know that a dilated heart rarely presents the signs of angina. There is, however, reason in the view urged by some—for example, Vacquez—that in such cases the pain is usually experienced at the commencement of dilatation, and subsides, like that of the bladder when dilating from retention, when the muscular resistance of the organ has been overcome and a paretic condition established.

Huchard regards the most common pathological lesion associated with angina pectoris to be a thickening of the inner coat of the coronary arteries (atheroma), and from the time of Edward Jenner till now disease of the coronaries has been associated with cardiac pain. Yet we know that here, as in the case of contractility, lesion of the coronaries is much more frequently found *without* than with painful symptoms.

The very *variety* of the manifestations of pain argues a difference in its seat and intrinsic nature. For example, cases occur in which the radiation of pain may be to right or left. It may also be more general and to both right and left up to the head and over a considerable surface of the body. In dealing with the clinical history of cardiac pain, I shall give some particulars of a case which exhibited dextral radiation in association with dextral valvular disease, and G. A. Gibson and others have also observed the same radiation of pain to the right.[1] Great pain may, moreover, be rapidly or much more slowly fatal. I shall relate instances of this difference which were alike in many ways, but differed in one important and distinctive particular. Both these cases showed fatty degeneration of the heart, and the patients were of much the same age. But while the apparent cause of the angina in the protracted case was ante mortem thrombosis of the right coronary artery, no discoverable condition could locate the precise site of the stimulus to pain in the other, and it was not unreasonable to assume that it was in the musculature of the left ventricle.

Spasm is no doubt a phase of contractility as well as relaxation; but the possibility of a localized spasm of cardiac muscle is, on hypothetical and experimental grounds, disputed.

[1] 'Lectures on the Nervous Affections of the Heart,' p. 38.

The rhythmical action even of one ventricle apart from the other, except when cut away from its fellow, is denied by many. That, however, this is possible is argued by cases of malignant disease of the organ, which may fix one ventricle while the other continues to act undisturbed. Such a case I have myself published.[1]

Indeed, there are those who regard the affection as spasmodic in nature—the *distentio* of Heberden—and consider that the seat of the spasm in angina pectoris is not in the heart at all, but in the œsophagus or stomach. Verdon has recently ably argued this view.[2] That a severe œsophagismus or gastrismus may give rise to severe pectoral pain may be admitted, but against the view that the acquisition of an anginal habit in the foci of sensitiveness in the spinal cord is the explanation of *all* cases of Heberden's angina pectoris, various considerations may be urged. It is sufficient, however, to recall the fact that many cases show cardiac lesions calculated to distress the organ, and that many attacks are rapidly relieved by agents which act chiefly on the cardio-vascular system.

There is an observation of Kronecker's which frequently elicits the impatience, if not the scorn, of enthusiastic myogenists, but which may have a bearing on this question. Kronecker found that puncture of the heart at a certain point near the base of the septum ventriculorum was followed by the more or less rapid arrest of cardiac motion. This puncture is apparently accidental, as many punctures may be made before the spot is touched. It has been suggested that what Kronecker probably pierced when he stopped the heart was the bundle of His.

As I showed in the first part, in the human heart there is no large innervation of the bundle itself, but a *fine* innervation, derived from the *neighbourhood* of the bundle. In animals in which this structure is well-developed, I have also shown that the anatomical character of the innervation justifies the belief that there is a sensory element. It is possible that in some cases of severe and rapidly fatal angina, the stimulus to pain arising in this source or centre for the

[1] *Lancet*, January 9, 1906. [2] *Lancet*, July 12, 1913.

innervation of the auriculo-ventricular node and bundle may be causal, both of the suffering and the fatal result, and accord with Kronecker's experimental experience. The investigation, therefore, of these structures is desirable. In such a case, however, which I shall relate, I found the bundle certainly sclerosed, but not so peculiarly so, as to distinguish it from others similarly affected, but not manifesting cardiac pain.

It will be maintained in what follows that the sensory disorders of the heart are dependent upon more than one condition affecting the cardiac nervous system, the only feature common to all cases being *pain*. The consequences of such pain in their immediate or postponed influence upon life it will be suggested are dependent upon the *site* and *nature* of the condition provoking it.

Pain is necessarily provoked by some direct incidence on nerve structure, and when we remember the relatively small number, according to Langley, of afferent fibres reaching the posterior root ganglia from the viscera—that is, the afferent visceral nervous system in all probability capable of receiving and transmitting *painful* impressions by way of the white *rami communicantes*—we can conceive that the *accidental* situation of some change or condition producing such incidence upon nerve structure may have an influence in producing the anginous state. This would also account for the comparative rarity of the condition, and for its seemingly capricious absence or presence under apparently the same underlying states. To discover such foci of stimulation, a minute anatomical examination of the heart in fatal cases is necessary, and such a sufficient investigation has not frequently been undertaken.

This larger view of the causes or sources of cardiac pain is supported by Professor Hering of Prague in the able report on the pathology of cardiac failure which he recently laid before the International Medical Congress in London, in which, while granting the possibility of a muscular source of cardiac pain, he also argued from the results of pathological research that it might be of coronary vascular origin. In what follows some evidence of this will be adduced.

It will therefore be maintained that important as is the study of the normal and abnormal physiology of the heart during life by experimental and clinical methods, the minute anatomical investigation of the organ after death has at least equal importance in enabling us to arrive at correct views on the causes of normal and aberrant cardiac sensibility and action.

CHAPTER II

THE PATHOLOGY OF CARDIAC PAIN

As a general term 'cardiac pain' should include all pain arising in or near the heart, the frequently very slight subjective discomfort of a weak or uncompensated heart, as well as pain provoked by inflammation of one or other texture of that organ, or caused, as happens in some instances apparently, by external pressure exercised upon it or neighbouring structures by growths or aneurismal tumours. We have seen, however, that the heart itself may be subjected to much disturbance of this character without exciting pain as a predominant symptom. With the exception of aortic valvular disease, to which special reference will be made in its own place, endocardial lesions are not often the cause of that agony which has gained the designation of 'angina pectoris.' In using the term 'cardiac pain,' therefore, I shall chiefly refer to the latter. The terms are almost equally indefinite. It is natural that effects should obtrude themselves upon our notice and enter into our nomenclature of disease when causes are unknown or disputable. The ancients were temporarily content with such a term as 'dropsy'; we have to employ such expressions as 'arthritis deformans,' 'exophthalmic goitre,' or 'angina pectoris.' Minute research, which is the chief engine of scientific progress, will doubtless in time relegate all such expressions to the museum of words which mark the advance of medicine to more definite knowledge, but that time, in the matter of cardiac pain, is not yet. It seems presumptuous, indeed, to say so, but it can with all modesty be truly asserted that the heart has rarely, if ever, been thoroughly examined post

mortem. If its general proportions and gross lesions have been noted, in many instances its microscopic structure and vessels have been left uninvestigated. If the latter have been more or less examined, the condition of its nervous regulative apparatus has been overlooked. If, again, it has been attempted to throw light upon the latter, the matter has not been exhaustively dealt with—cannot, indeed, at present be satisfactorily dealt with because it is still very obscure. Indeed, the time necessary for such investigations is not at the disposal of the general pathologist, and specialism in research, as in practice, will most probably find a place in the near future. In these circumstances the sketch I propose giving of the pathological conditions associated with cardiac pain must of necessity be imperfect, but there are one or two points in connection with this matter upon which I hope to throw some light which, to me at least, is new, and appears to be not without interest.

The heart, as a whole, of those who die during an attack of cardiac pain is perfectly flaccid when the body is opened, and exhibits *prima facie* strong justification for Parry's use of the term 'syncope anginosa' as applied to these cases. The cardiac muscle in cases which die thus is at times found to be well preserved, even in circumstances which at first sight seem well calculated to induce degenerative changes. Thus the striæ may be well marked, and there may be little evidence of muscular degeneration in cases in which there is much obstruction to the coronary circulation. But, on the other hand, in cases well supplied with pervious and comparatively elastic arteries, the muscle cell may manifest well-marked evidences of fatty degeneration in association with, or independently of, fatty infiltration of the textures—that is, the dissection and rupture of muscular fibres by the penetrating growth of a surplus of fat cells.

The coronary arterial system plays an important rôle in the pathology of angina pectoris, and yet a very puzzling one, like every other structure in connection with that very inadequately investigated subject; for the grossest atheromatous and calcareous change may be met with in these vessels without their having elicited during life any feature

in the syndrome of angina pectoris. Why this is so only future and more careful investigation will fully reveal, but I shall presently mention some facts which I regard as important in this connection.

The atheromatous change in the coronaries is a very striking and important phenomenon. If transverse sections be made of a healthy coronary artery in a young adult, a certain standard proportion may be observed in the relative thickness of its layers or coats. The membrane of Henle, separating the subepithelial from the muscular coat, is the important guide to the histologist and the measure whereby we may determine the degree of the thickening which the vessel has undergone—in other words, the amount of elasticity of which the nutrient vessels of the heart have been robbed. Comparing such a normal specimen which I obtained from the right coronary artery of a well-developed male, nineteen years of age, who died from appendicitis, with that of a man who died at the age of sixty-three years from angina pectoris, two general facts are observed—namely, that the subepithelial layer in the healthy young man bears a very small proportion to the thickness of the muscular layer, while in the older man, who died from angina, the subepithelial layer is much thicker than the muscular, the whole artery being less elastic and the muscular layer hypertrophied when compared with that of the standard mentioned —in consequence of the greater difficulty thrown upon the vascular muscle to contract upon its less yielding substructure. If, again, the latter specimen be compared with the atheromatous coronary taken from a man aged fifty-four years who died from angina pectoris and in whom the atheromatous change was associated with a rigid calcification of the walls of the artery, which in great measure must have prevented the main trunks of the vessel from contracting at all, it will be observed that, while the subepithelial greatly increased layer exhibits well-known atheromatous changes, the muscular coat, unlike that of the uncalcareous but hypertrophied artery, is not thicker, but thinner, than that in the healthy young man taken as a standard. At places, also, this seems directly due to the pressure of atheromatous

nodes. In order that I might prepare specimens of this calcified artery I found it necessary to immerse the portion selected, which, like the others, was taken from the vessel on the right, in dilute hydrochloric acid for nearly a fortnight, in order to decalcify it. In the case from which this vessel

FIG. 9.—ANEURISM OF THE RIGHT CORONARY ARTERY, THE WALL OF THE VESSEL DECALCIFIED, ITS LUMEN CONTAINING BLOOD.

The greatly thickened intima and atrophic media are shown with the head of the aneurism resting on the latter. Occ. 4, obj. 1 in. (Swift's).

was removed there were constant attacks of angina for a length of time before the fatal seizure and in all circumstances as to rest or activity, and yet there was nothing in the calcareous vessel externally to distinguish it from many other calcareous arteries unassociated with cardiac pain. But on

microscopic examination I discovered one fact which I consider of great interest, although I do not positively state that it was necessarily the essential or only cause of the angina. It would, however, be quite as difficult to prove at present that it was not. I was fortunate enough to obtain sections illustrating this point, and I think most will agree with me that these specimens show in a very instructive manner the mode of formation of an aneurism before it has eroded the muscular coat of the vessel and become evident externally. (*Vide* Figs. 9 and 10.) It will be observed that

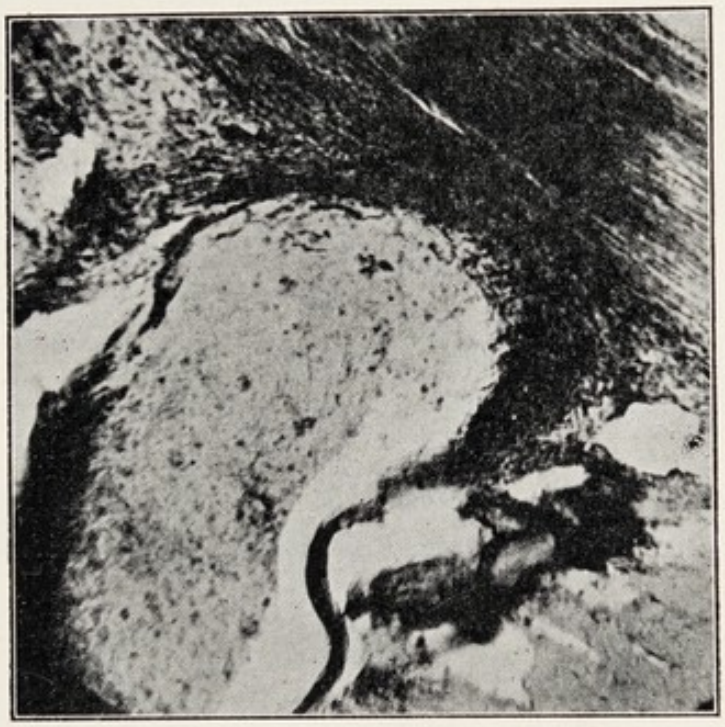

FIG. 10.—THE HEAD OF THE SAME ANEURISM MORE MAGNIFIED. Occ. 4, obj. $\frac{1}{6}$ in. (Swift's).

the internal layer of the vessel has been pushed before the eddy in the blood-stream at its commencement, and that this diverticulum in the stream has been dammed in by protective and proliferative processes in its passage through the connective-tissue layer, until its outer encapsuled end lies in immediate contact with the muscular layer. How many more of these saclets there might have been in the vessels I cannot, of course, say—perhaps no more—but the discovery of one argues the possibility of others. In the immediate neighbourhood of the sac there are encapsuled spaces filled with blood which resemble sections of veins,

but which appear to be portions of the same aneurismal process—portions, that is, of a dissecting aneurism.

I have looked through the indices of the Transactions of the Pathological Society of London from the first to the last volume, and find no mention of such a condition. Nor have I found any record of it elsewhere. It is of interest that this instance occurred in a prolonged and severe case of angina pectoris. It may have a bearing on the explanation of pain in some cases; it may not. The future must decide this point, but in the meantime I regard it as very probable that intravascular aneurism is one of the causes of angina. As we shall learn, when we consider the clinical history of angina pectoris, there are cases in which the neural element of pain—for pain can only be a property of the nervous system—is the predominant feature, and the cardiac failure in all probability precipitated by it; and another class of case in which the apprehension of cardiac failure, the sense of impending death, is associated with little or no local cardiac or referred pain. It is probable that these cases, differing from one another in so important a particular, differ likewise in the details of their local pathology.

The hæmic factor, without local laceration or disturbance of texture, as in the instance I have related, may, it is surmised, play a predominant rôle by temporary—that is, claudicatory—occlusion of a vessel or vessels on the hypothesis first suggested by Allan Burns of Glasgow, or by thrombosis. Certain it is that the coronary vessels may become thrombosed, but claudication is at present merely a hypothesis based upon argument from analogy, the analogy being drawn from vessels in other parts of the body. I shall again refer to coronary thrombotic angina, one of the most painful and fatal varieties of the affection.

I shall later also have a few words to say on the subject of so-called ' angina sine dolore,' and may, with all due respect to those who think otherwise, call in question the right of some of these cases to be classed with Heberden's disease. But to determine this point also, much more minute investigation must be made than has hitherto been the case. That cases of coronary atheroma are numerous,

and of angina pectoris rare, has been remarked by nearly all writers on the subject, but it is equally well known that cases of aortic atheroma are much more common than cases of aortic aneurism. Moreover, even all conditions of aortic aneurism are not equally associated with pain, but there are few cases of aortic aneurism in which there has been no pain at some time or other, however that circumstance be explained. Sir T. Clifford Allbutt has likewise emphasized the relation of aortitis to angina pectoris, and even suggests that evidence supports the view that 'the seat of anginal pain is not in the heart, as every writer seems to suppose it to be, but in the aorta.'[1] The late Sir William Gairdner and other physicians of note, as we shall learn again, have also called attention to pain as a symptom in connection with aortic disease. From Sir T. Clifford Allbutt's opinion that all classical cases of angina pectoris owe their origin to extra-cardiac causes I shall find it incumbent upon me, in the course of my remarks, to dissent; but the views of the authorities I have mentioned necessitate a short examination of the degenerative changes in the aorta, while discussing the pathology of angina pectoris.

It will be remembered that while inquiring into the conditions of the coronary circulation, it was mentioned that the circulation through the nutrient arteries of the heart was systolic in time, and aided by the diastolic tension of the aorta when the propulsive phase of ventricular action was at an end (p. 50). There is thus the brunt of a double impact expended upon the commencement of the thoracic aorta and especially that portion of it whence the coronary arteries spring. It is therefore in no way remarkable that the aortic intima should in these situations show in a very large number of cases which have approached or passed middle age the evidence of wear and tear—that is, of atheroma and its associated processes. The conditions of atheroma, while essentially the same here as those already described in considering the coronary branch, are also modified to some extent, in obedience to the histological differences

[1] Selections from the Lane Lectures. Reprinted from the *Philadelphia Medical Journal*, January 27, 1900, p. 124.

involved and the greater blood-pressure to which the larger vessel is subjected. The inner coat in this vessel, as is remarked by Coats and Auld in their memoir on the subject,[1] is relatively thicker, and the delimitation of the inner from the middle (muscular) coat less strict than in smaller arteries. Hence there is a somewhat greater difficulty in separating the consecutive stages of the conditions, essential and accidental, from one another. These authors agree with Virchow in regarding the process as a whole as inflammatory, but do not exclude a certain conservative function in the proliferation of connective tissue which it entails. 'The lesion in the intima is,' according to Coats and Auld, 'a reduplication of its own tissues.' Atheroma is, in its essence, an overgrowth of connective tissue in the inner arterial coat, usually arising in an insular or disseminated manner in the form of nodes or patches, and tending to degenerate in these foci and to produce retraction of tissue in inflamed areas; hence, among other effects, the narrowing of the coronary orifices behind the pouches of Valsalva. Coats and Auld are equally instructive on the production of aneurism in the aorta, and show from its small beginnings to its ultimate penetration of all the coats of the normal vessel how the process commenced in the intima as a rule, and, followed by its depression or cupping, presses on to and perforates the muscular coat, until the latter forms but the collar to the neck of the sac. I have said as a rule, for Coats and Auld discuss the question of a primary rupture of the muscular coat—a condition which is actually observed under the microscope in association with proliferation, which is in a measure a protective overgrowth of connective tissue. It is feasible to suppose, as they suggest, that in a weakened vessel subjected to so great an impulsive pressure of blood, as is the case in the aorta, such rupture may occur, and be the initial step in the series of changes which lead to the ultimate extra-vascular bulging, when the sac itself is chiefly formed of the thickened and inflamed adventitious coat, the very arteries of which may reveal an obliterative end-

1 'Selected Researches in Pathology,' by A. G. Auld, p. 109. J. and A. Churchill, 1901.

arteritis (*op. cit.*). But a process such as this is not probable in the coronary arteries themselves. Here, as the specimen I have referred to (p. 62) very well reveals, the aneurism is evolved more on the lines of the so-called 'atheromatous ulcer,' a depression in the endothelial layer pressing towards a softened area in the intima, bursting into it, and encasing itself as it pushes its way towards the media in a capsule begotten of proliferative arteritis, there to pulsate against and ultimately to destroy the muscular coat, and in all probability in the process to cause the organism pain. Such, then, is the history of the slow attack, invasion, and overthrow of the ramparts against destruction in an artery by the eroding forces which are inimical to its life, but which are of necessity factors for a time in the life of the organism. To exist is to wear, and to wear is to tear. But in association with cardiac pain attention has been called, especially by Sir T. Clifford Allbutt, to an acute aortitis—an acute inflammation especially of the lower segment of the ascending portion of the thoracic aorta—and I shall postpone a more detailed consideration of this important subject until I come to discuss the clinical history of what I have termed 'aortitic angina,' and the essential cause of which in the majority of cases is specific infection.

In connection both with the acute and chronic changes in the aorta and coronary system we may have inflammatory and proliferative changes in the aortic cusps, due to rheumatic endocarditis in its simple or so-called malignant or ulcerative forms, or, as is argued by other evidences of the disease elsewhere, to syphilis. I shall also have occasion, when discussing the clinical history of the disease, to instance a remarkable case in which a gigantic ulcerative endocarditis affected the pulmonary arterial valves and portions of the pulmonary artery. So much, then, for the vascular changes in connection with cardiac pain. The blood itself is in many cases normal for the age of the patient, but may be spanæmic from one or other dyscrasia, such as paludism, gout, or lead-poisoning.

Trustworthy observations on changes in the cardio-vascular nervous system in association with angina pectoris

are not numerous. Huchard[1] mentions the names of Gintrac, Corrigan, Lancereaux, and Loupias, as having treated of neuritis of the cardiac plexus; of Peter as having met with this condition in association, not only with neuritis of the plexus, but also of the phrenic nerve; and of 'Putjatin, Uskow, Hoffmann, etc.,' as imputing angina pectoris to inflammation of the cardiac ganglia. The detection of the ultimate distribution of the cardiac nerves to the muscular fibres of the heart is a difficult matter even in the fresh tissues of recently killed animals, while their state in the human cadaver is, so far as I am aware, unknown. In Lancereaux's case the cardiac plexus participated in an extremely rich abnormal vascularization at the root of the aorta, and showed microscopically a round-celled infiltration between the nerve fibres of the ganglion which compressed these. These conditions were associated with angina pectoris. Previously to his observations the neuritic and neuralgic theories of cardiac pain appear to have been rather the coinage of their authors' brains than the result of anatomical investigation. As a matter of fact, the nerve trunks of the heart and the ganglia of the cardiac plexuses may be observed in those dying from angina pectoris to be, as a rule, normal for the age of those affected. Thus, any interstitial thickening of the trunks which may be thought to exist is seldom capable of being regarded as inflammatory, and the only change observed in the cells of the ganglia is that of pigmentary degeneration, and a greater difficulty than in young and fresh material of staining the component parts of the cell. Such has been my own experience, and it appears to be the rule; but there are important exceptions in Nature to all that we, in our impatience, regard as 'the rule.' Indeed, in the fatal case of angina from which I obtained the internal aneurism to which I have already referred I met with a cluster of shrunken ganglion cells in an atheromatous patch flanked by calcareous deposit. From a comparison of sections it appears to lie in the same portion of the circumference of the artery as that in which I found the aneurism, but not at quite the same level.

[1] 'Maladies du Cœur et des Vaisseaux,' 1893, p. 598.

These two pathological conditions could not have been far from one another. There is also in the immediate neighbourhood of the head of the aneurism a transversely divided and thickened nerve, which may possibly have some connection with the ganglion I have mentioned.

The following is a description of the specimen (Fig. 11): in a transverse section of the right coronary artery of the case mentioned there lies an encapsuled cluster of cells, each of which has a clear round nucleus with a single

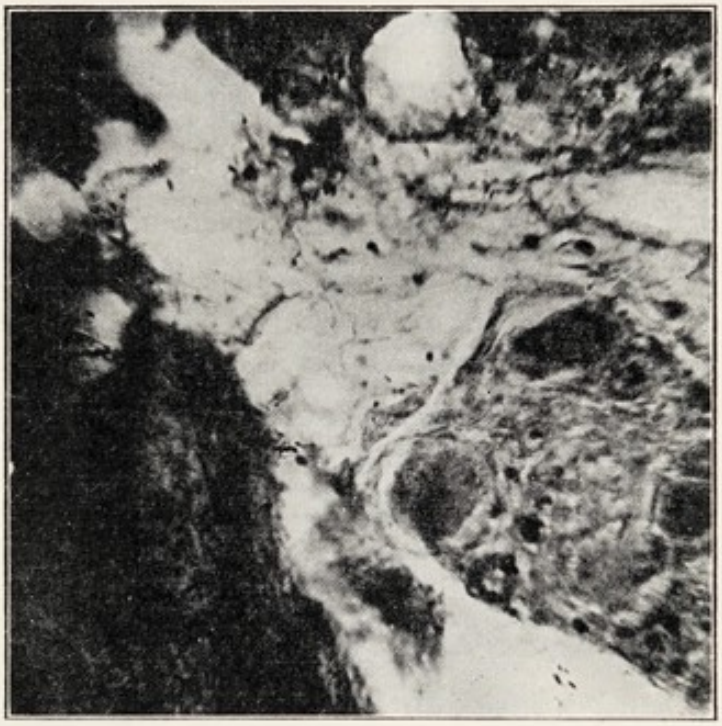

FIG. 11.—NERVE GANGLION, WITH THICKENED INTERSTITIAL TISSUE FROM THE RIGHT CORONARY, SHOWING NUCLEUS AND NUCLEOLUS IN THE CELL HIGHEST TO THE RIGHT.

Occ. 4, obj. $\frac{1}{12}$ in. (oil immersion).

nucleolus, as may be determined by careful focusing in a clear light. The cell body is of a brownish-red colour and has shrunk away to some extent from the walls of its capsule, if it originally quite reached these. The cells are separated from one another by a considerable thickness of intercellular connective tissue. A cluster of seven cells is sharply marked off from the surrounding textures by an encircling capsule. Near the lower end of the cluster and to the left there is a detached cell of the same character as the others, but within a more thickened capsule. The cells, their capsules, and the

intercellular tissue have a different colour from the surrounding textures. The latter on all sides of the cell cluster except to the right is altered intima stained blue by hæmatoxylin. The spaces in its fenestræ are empty, and in one place the space is open at both ends. To the right of the cell cluster there is a space free from texture except at two points where there are portions of an amorphous material stained blue. This vacant space probably marks the former site of a calcareous deposit which has been dissolved by the acid in which the preparation was placed prior to section, and the blue patches necrotic atheroma. The general position of the cell group is in the greatly overgrown and degenerated inner coat and close to the muscular or middle coat. Internal to the well-marked line—the membrane of Henle which separates the media from the intima—and immediately next it there is a layer of material, granular in aspect, in which the circular course of fibres is not evident, and at places in which there are some transversely divided fibres and also small bloodvessels. Interspersed between the outer end of the cell group and this layer, there is a narrow tongue of blue-stained atheromatous necrosis. The colour of the cell group, of its capsule, and of the intercellular tissues approximate most closely to that of the muscular layer, and to that of the vessels and nerves lying external to the outer or fibrous coat of the artery. I have gone thus minutely into the particulars of this specimen in order to demonstrate its neural nature, of which I have no doubt this fact is necessarily an important one in its bearing upon a possible cause of angina; for although the peripheral ganglion itself was probably on the efferent or motor stream of sympathetic innervation, it nevertheless probably contained sensory elements.

With regard to the condition of the nervous system in angina pectoris outside the heart, and the vessels in the immediate neighbourhood of the heart, facts are still much lacking. Huchard refers to a form of angina attributed by Cuffer to the bulb becoming involved by the spread of an ascending neuritis of the pneumogastric nerve, and the lightning pains of locomotor ataxy, probably due to inflam-

matory changes in the ganglion on the posterior root of the spinal nerves, or of sensory centres in the posterior columns of the associated segments of the cord, have at times been regarded as causal of the cardiac crises which occur in some of these cases. But as aortic lesions are likewise met with frequently in the latter, such attacks may really be referable to a nearer cause than to any change in the posterior root ganglia of the spinal cord. Of tissue changes in the higher nervous centres which might be construed as causal of angina pectoris, we know nothing.

The most humble texture in the structures we are considering in relation to cardiac pain is the connective tissue. It is the mortar of the building and, though humble, as important as mortar is to the security of the whole. In it, moreover, channels for nutrition, innervation, and lymph pass. However strong and important the other elements may be, the strength and health of the whole is dependent upon the good quality of that vital cement of the organs. Its thickening under strain and as a consequence of the general stress of life is but the prelude to decay, and the introduction with such decay of the destructive processes of impaired nutrition, the shrinking of its texture and the strangling by it in its vicious decrepitude of the important structures which it holds and keeps together. As life advances, therefore, or in consequence of the abnormal senility precipitated by diseased action, a gradual increase of the connective tissue of the heart and vessels may be observed which soon begins to hamper the action of the organ.

As regards the muscle of the heart and vessels, this responds to the call made upon it by increased growth, by hypertrophy, which we have seen to be a result of atheromatous thickening in the coronary artery, and arises under the stimulus of an impediment to circulation in the heart, and which later, as rigidity is increased and nutrition is interfered with, yields to degenerative changes in the essential muscle cells. This, especially if coupled with an invasion by fat cells in excess, rapidly degrades, as I have already noted at the commencement of these remarks, the

normal character of the muscle. A moderate cardio-sclerosis then passes into a chronic myocarditis, with interstitial cellulitis, obliterative endarteritis, atheromatous change, destruction of vessels, exposure of sensitive structures, painfully exercised function, and the death of the organism.

I shall again refer to the influence of an excess of cardiac hypertrophy, under certain circumstances, in causing an erethism associated with much cardiac pain.

CHAPTER III

THE CLINICAL HISTORY OF CARDIAC PAIN

THE classification of the varieties of an affection which is denoted by a term that has but the significance of a symptom must of necessity be a classification of symptoms. The terms 'true' and 'false' convey little meaning, and the degrees of severity or fatality denoted by such words as 'gravior' and 'mitior' are not much more enlightening, while the use of such general terms as 'vaso-motor,' 'arterial,' or even 'coronary,' is but the indication of dissatisfaction with the indefiniteness of the terminology of the disease usually employed. In view of these facts, and after an inquiry into the physical basis of the affection, it seems desirable that we should attempt a classification, however provisional and transient it may prove, which endeavours, in the light of facts still too few in number, to give a local habitation and a name to phenomena which we are not yet in a position to explain with scientific precision. With these reservations it appears to me that we are in the meantime justified in discriminating pathologically between the modes of cardiac anguish on the lines indicated in the following table:

- Cardiac anguish (angina pectoris).
  - I. With pain (Heberden's disease).
    - 1. Musculo-spastic.
    - 2. Coronary.
      - Aneurismal.
      - Occlusive.
      - Thrombotic.
    - 3. Aortic.
      - Aortitic.
      - Aneurismal.
    - 4. Neuritic.
      - Intravascular.
      - Extravascular.
    - 5. Neuralgic.
      - Intrinsic.
      - Extrinsic.
    - 6. Endocardial (valvular).
      - Aorto-coronary.
      - Dilatational.
      - Hypertrophic.
    - 7. Vaso-motor (? peripheral).
    - 8. Compound.
  - II. Without pain (angina sine dolore).
    - 1. Fear with syncopal signs.
    - 2. Fear without syncopal signs.
    - 3. Syncopal bradycardia.

Musculo-spastic Angina.—'Angina pectoris, as far as I have comprehended its nature,' writes Heberden,[1] 'seems to be related to distension (*ad distentionem*), but not to inflammation.' He then goes into several reasons for coming to this conclusion, and the last of these is that 'it affects some after the first sleep, as is frequently the case in diseases from distension.' The word I have literally translated 'distension' is evidently meant to convey the idea of spasm—spasm like cramp in the leg after sleep. M. Huchard, who in his excellent work bends all his ingenuity towards proving that the sole cause of angina is really a more or less sudden anæmia of the cardiac muscle due to atheroma of, or imperfect circulation in, the coronary arteries, places Heberden's notion among the many exploded theories which he enumerates.[2] Sir T. Clifford Allbutt appears to agree with him on this point.[3] Sir William Osler, however, does not so definitely dismiss Heberden's view as untenable. In his 'Lectures on Angina Pectoris and Allied States,' he writes: 'Pain, the special feature of the angina attack, is explained by the cramp theory. The most intense suffering which can be experienced is associated with muscular contractions of the tubular structures, as in intestinal, biliary, and renal colic, and in the contractions of the uterus in parturition. And, observe, that this agonizing pain is in parts not endowed, so far as we know, with very acute sensibility. Theoretically, there is much in favour of the idea that in the most powerful muscular organ of the body, irregular cramp-like contractions, even if localized, might be accompanied by painful sensations which could attain the maximum intensity present in an angina attack' (p. 120). He, however, considers that this theory cannot account for 'frequently recurring attacks.'

My argument commits me to the support of no single theory of angina pectoris. I have already suggested that the frequently occurring attacks associated with calcareous arteries may be due to a neuro-vascular condition, the chief

[1] 'Commentarii,' p. 312.
[2] 'Maladies du Cœur,' etc.
[3] Lane Lectures, reprint, p. 115.

element in which may be an aneurism which has not yet eroded through the muscular coat of the vessel and gained a position of greater freedom and one less influenced by passing variations of intravascular blood-pressure. I have shown direct evidence of this, as I take it, in the intravascular aneurism already referred to. But circumstances alter cases, and such alteration demands and denotes a difference in the mechanism of the production of pain. That a whole heart could be in a condition of tonic spasm, and yet the pulse proceed more or less altered, is not to be thought of. On the other hand, a rhythmically contracting organ—and we have seen that this rhythmicality is an inherent neuro-muscular property of the heart—may, as it seems to me, be affected by a cramp in limited areas which crushes in its tonic grip the sensory nerve endings of the organ, and, if unrelieved, is calculated to induce by way of the pneumogastric nerve and the structures it innervates a profound and, it may be, fatal inhibition, the visible sign of which is the flaccid diastolic heart found post mortem in those who die in the agony. This tetanic behaviour on the part of the cardiac muscle, the possibility of which is denied by experimental physiologists, while probably favoured by an insufficient coronary circulation in some cases, might, as it seems to me, in view of the inherent neuro-muscular rhythmicality of cardiac muscle, arise from physico-chemical changes in the muscle itself, apart from the *quantity* of its blood-supply. For, such a fatal attack appears to be possible from over-exertion in cases in which the blood-supply to the organ is little interfered with, in which atheroma, either of the aorta or of the coronaries, is not in an advanced degree, and in which muscle fibres are in many portions fairly free from degenerative changes. As an example of cases capable of bearing such an interpretation I may relate the following:

On November 10, 1901, I was hastily summoned to a gentleman whom I had met in society, but whom I had never previously seen professionally. He was sixty-three years of age and had some months before returned from the Far East, where he had spent many years of his life, and where he had comparatively recently married a native, by

whom he had two children. He had suffered, I was told, a good deal from his liver while abroad, and about two years before his return to England had experienced what would seem to have been a severe syncopal seizure. He was, however, pronounced sound after this illness, and in September of 1900 consulted a well-known physician in London who likewise pronounced him organically sound, as I learned from the patient's diary, but stated that he was suffering from an excess of 'uric acid,' and prescribed exercise and an anti-lithatic dietary. He accordingly used chamber gymnastics, and cycled. I learned from his partner in business that he was an energetic and excitable man. He was short, rather stout and bald, and what remained of his hair was grey. He was alert and active-looking for his age, a good billiard player and most careful liver, dieting precisely as he had been instructed, and taking probably more exercise than had been enjoined. He regarded himself as perfectly sound, and from his business arrangements would appear to have entertained the expectation of a prolonged existence. The day before I saw him was Lord Mayor's Day, and he went with his young son to the show, stood in one place for two hours, afterwards visited St. Paul's Cathedral, and carried his child up and down the stairs of the Underground Railway. He retired to rest apparently quite well and slept well during the night, being in this respect one of those cases to which Heberden refers as awaking to pain. At five o'clock in the morning he had a severe attack of pain in the chest, which passed off and which after a time recurred. About eight o'clock he sent for me. I saw him at nine o'clock. He was quite comfortable when I reached him, but told me that the pain he had experienced was very severe, yet he did not seem unduly alarmed about it. Soon after I arrived he was seized with pain in the centre of his chest, with radiation down his left arm to his hand. The attack was severe. He rolled round on his right side, supporting himself on his elbow, and closed his eyes with an expression of agony. His pulse was not quickened, but during the pain it occasionally intermitted. Its palpability varied; it was at times larger and more easily felt, and again smaller and less palpable.

After the attack passed off the pulse was smaller and more rapid than during the persistence of pain. I gave him at once an ounce of brandy without water, as it was the only stimulant available. The attack lasted about three minutes and then subsided, and the patient declared himself to be free from pain. This complete subsidence of pain is a remarkable phenomenon in these cases, and is probably due to the peculiar sensibility of visceral sensory nerves. The respiration was restrained during the agony, but his breathing was not otherwise disturbed or difficult. After a time he had slight recurrences of pain, and I gave him more brandy. This, too, passed off, and he declared himself to feel better. Having prescribed trinitrin and enjoined his remaining strictly in bed, I left him for a short time apparently quite comfortable, to breakfast with a friend a few doors from the house of the patient. While at breakfast I was urgently summoned to him again, and arrived without loss of time. I was informed that he had been feeling fairly comfortable and had asked for some tea and toast, but again experienced a recurrence of pain, and immediately thereafter began to breathe stertorously. When I reached him he was unconscious, but occasionally groaned. His pulse was imperceptible and the heart-sounds could not be heard, while his face was cold and clammy, and tears flowed down his cheeks. I injected strychnine and morphia hypodermically, which had no effect, and used artificial respiration. His breathing, however, became irregular and slower, and he died within fifteen minutes of his seizure. I learned afterwards that two days prior to his death he had had some discomfort in his chest which he regarded as rheumatism, and used some liniment for it, but attached no importance to it. In view of subsequent events, however, it is possible, as the patient had been leading a very active life, that this may have been a prodromic symptom of the angina which slew him in what was practically his first attack. For the series of attacks on the day of his death constituted a more or less continuous status anginosus. I obtained permission to examine the heart after death. The case manifested slight atheroma of the aorta, with a comparatively early stage of that change in

the larger coronary arteries, somewhat more in the smaller fatty infiltration of the cardiac muscle, and fine fatty degeneration of the muscle fibres. I retained the specimen, and in 1912 examined the auriculo-ventricular node and bundle, which I found to be sclerosed, but not more so than it frequently is without any anginous accompaniment.

To consider the painful angina in such a case as due to defective circulation in coronary vessels which were widely patent at their orifices, only moderately thickened in their inner coats in a large part of their course, and pervious everywhere, and to ignore the invasion by fat and degeneration of the muscle itself, as a thorough-going adherent of the coronary vascular theory like M. Huchard would do, appears to me to be unreasonable. On the other hand, there was certainly nothing in the condition of the aorta to support Sir T. Clifford Allbutt's views as to the aortic origin of all angina, and the nerves in this case and the ganglia which I examined might be regarded as normal for the age of the patient. In these circumstances we are justified, I believe, in maintaining that in one variety of painful angina the muscular factor in spasm plays the leading part, and that the simple and obvious interpretation given by Heberden of some of these cases is correct. That such, like most other cases, are compound will be argued later, and then a subsidiary rôle may rationally be assigned to other factors in cardiac action.

Aneurismal Coronary Angina.—Since the day on which Edward Jenner, with that simple yet rare acumen which characterized the first vaccinator, found something gritty strike his knife at a necropsy on a case of sudden death, and thought that a portion of the ceiling had fallen on the organ, but examining more closely discovered the first calcareous coronary artery in connection with such cases, until now, that state of these vessels has been associated in the minds of many physicians with the cause of angina pectoris. Jenner's friend, John Hunter, died from angina, and his heart, which was small and probably atrophied, contained the calcareous coronaries which Jenner was the first to detect in an indubitable case of angina. But many instances

have occurred in which these chalky channels have existed, and yet the organ which has lodged them has at no time evinced the symptoms of angina. One cannot, therefore, use the presence of this condition alone as a factor in the more precise classification of angina. But some state in association with such vessels may well, in some cases, be distinctive of a type of that malady.

It would be hasty and unscientific to assume, because in a well-marked case of the classical variety of angina pectoris a well-developed internal or masked aneurism with surrounding inflammatory processes has been found, and, moreover, in close proximity to autonomic ganglia and nerves, that such a condition must necessarily account for angina in all such cases. I make no such claim. In complex cases of the disorder, and perhaps most cases are complex, other conditions may be more directly causal; but I think we are justified in assuming that the case in which this condition has been found, and which I shall relate presently as typical of the clinical history of this variety of the disorder, gave evidence of no other condition which could be regarded as more directly causal of the very frequent attacks of cardiac pain, than this aneurism and the neighbouring innervation of the vessel. I have, therefore, in the meantime taken this striking feature in the case as the mark of at least one variety of the disease, and have used the term 'aneurismal coronary angina.' Should a closer examination of decalcified coronaries reveal the fact that such aneurisms are common in them without having provoked pain, the term will have to be abandoned. If, however, this does not prove to be the case, it appears to me that we have in such a circumstance a simple and sufficient reason why an aneurism such as this one, fully developed, sub-muscular, and freely communicating with the lumen of the vessel, pulsating with every throb of the heart, and recording on the muscular coat of the artery variations in arterial pressure, should explain, at least in some cases, why one person may have calcareous coronaries without angina, and why another, with apparently the same condition of these vessels, should know all the agony of breast-pang.

What other circumstances in the case I am about to relate could have more directly caused angina? The vessels were much occluded, but the cardiac muscle was well preserved, as I have already demonstrated. We have also noted the excessive freedom of coronary anastomosis, and in this case the right coronary artery had two small orifices, one of which may have been in connection with the third, or supplementary, vessel, which sometimes exists, and is known as the 'artery of Vieussens.' The tension of the patient's pulse was somewhat raised, as will be stated; but the response of his heart's action to the pain was regular, quick, and as normal as that of any healthy heart would be under the emotion inseparable from such distress. Presumably, therefore, the auriculo-ventricular neuro-muscle was unaffected. The organ, as a whole, was neither hypertrophied nor atrophied. The atheroma of the aorta, apart from that of the coronary vessels, was moderate in degree. The valvular apparatus of the heart was competent and unobstructed. It is true that a structure, probably ganglionic and inflamed, was found in the right coronary artery, but it could only have been recurrent fibres passing through the ganglion, not the ganglion itself, which caused the pain if the nerves were involved, for we at present believe that such peripheral ganglia have motor, not sensory, functions. The aneurism, then, would seem to have been the efficient cause of pain in this case, the clinical history of which was as follows:[1]

The patient, a man aged fifty-three years, a gilder, came under my care at the Great Northern Central Hospital on July 25, 1894. He complained of breathlessness, with attacks of pain in the chest, which passed down the left arm to the fingers and up the left side of the head. He had suffered thus for more than seven years. He had never had rheumatic fever, but had, during the ten years previous to my seeing him, suffered from gout in the great toe on four occasions. When he first came under my notice, nought amiss was detected in his heart beyond increased impulse. His pulse-rate was

[1] Particulars of this case were first published in *Treatment* on October 28, 1897.

60, and its tension was raised. Under the use of trinitrin and a mercurial pill the attacks became less frequent, and the patient ceased to attend the hospital in August, 1894. On November 21 of the same year he again presented himself, stating that he had been ill for ten weeks with what he called 'rheumatic gout,' an attack which would, no doubt, have been more correctly described without employing the qualifying adjective. He was rather under medium height, well-built, thin, with a dark complexion, grey beard, and bearing a feeble and suffering expression. On examination his tongue was furred, his appetite was bad, and his bowels were costive. His pulse-rate was from 66 to 72, and its tension was slightly increased. The heart-sounds were clear, the apex-beat was diffused, and there was systolic and diastolic pulsation in the epigastrium. The lung-sounds were normal. His urine was sufficient in quantity and contained neither albumin nor sugar. The arcus senilis was present in both eyes. He still complained of breast-pang, and the following particulars as to its character, situation, and the circumstances in which it occurred, were elicited on this occasion. The pain commenced about the level of the fourth rib on the left side, passed up to the sternal notch, and gave rise to a suffocative feeling; it was then felt in the left shoulder and down the left arm, being felt in the four fingers of that hand, never in the thumb, and most acutely in the ring and little fingers. The pain was also stated to shoot up the nape of the neck and into the head. It was severe, lasting sometimes ten minutes, sometimes for half an hour. The patient could not move when the attack came on, as every step appeared to make it worse. He was compelled to stand up, and could not lie or even sit down. It caused him to breathe deeply, but not quickly. It induced involuntary micturition. When gas escaped by the œsophagus, he experienced relief. This occurred in a minute or two after the commencement of the attacks. He stated that the heart beat 'very fast indeed' during the attack, and as pain passed off it became quieter. The attacks usually occurred after some exertion, but on two occasions had wakened him out of sleep. On awaking thus in pain he observed that his

heart was beating quickly. He felt at times during the attacks as though he would not recover. They caused him to feel cold. He did not, however, perspire after or during the attacks. He had found, during his first attendance at the hospital and while taking trinitrin, that the attacks were of short duration and that he had been much relieved thereby. Five-minim doses of liquor trinitrinæ were prescribed, the mercurial pill was repeated, and he was asked to take a mixture containing nux vomica, chloride of ammonium, and bicarbonate of sodium. Notwithstanding the temporarily

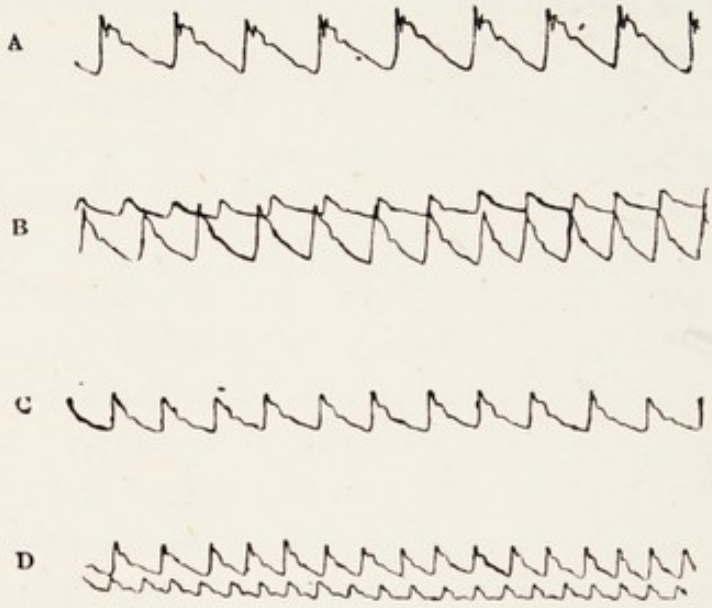

FIG. 12.

A, Tracing of right radial pulse, free from pain and without trinitrin, November 28, 1894. B, The same, free from pain and under trinitrin; upper, full effect of drug; lower, effect subsiding; December 5, 1894. C, The same; pain just passed off; no drug used; January 16, 1895. D, The same; lower, during attack of pain; upper, pain subsiding under trinitrin.

beneficial action of the trinitrin, the attacks of breast-pang continued and became more frequent. To ascertain the sphygmographic character of the patient's pulse, as also the effect upon it of trinitrin, the tracings A, B, and C (Fig. 12) were taken on November 28 and December 5, 1894. The trinitrin was administered in the absence of angina. On December 12 a soft systolic mitral bruit was noted for the first time, and in view of the increased dilatation of the left ventricle which this sign indicated, a mixture containing tincture of digitalis and liquor strychninæ was prescribed,

and on January 2, 1895, the patient felt better, though the attacks were still frequent and the apex bruit was not audible. On the 16th it was noted that the pain tended to spread into the *right* arm. While seated in the waiting-room of the hospital the patient had occasional attacks of angina, and on the 16th, while under examination by me, he had a moderately severe attack. The pulse-rate rose to 114, and fell to 96 when pain passed off. The sphygmogram C (Fig. 12) was taken immediately after relief, no drug having been given. The lower tracing in D (Fig. 12) shows the pulse during angina, and the upper its condition as the attack subsided under trinitrin. After this date the mitral systolic bruit was usually present, and the attacks of angina were frequent and of variable duration—so frequent and so variable in severity that some who saw the case believed that the patient was dyspeptic rather than suffering from true angina. Latterly even the trinitrin, which he regarded as a friend in need, ceased to relieve him to the same extent as previously. Finally, on June 29, 1895, at five o'clock in the afternoon, he was seized with pain in the chest in his own house. He said that he felt as though his heart would burst, remarked that he felt faint, and fell down on his knees. Just previously to this he had taken the trinitrin, said that the drops would be of no use to him this time, and expressed the hope that 'the Almighty would take him.' He spoke no more, and died without a struggle at 5.30—that is to say, in about twenty-five minutes after he was seized with breast-pang. These particulars I ascertained from those who were present at his death.

External and evident aneurism of the coronary arteries is not unknown, but has been rarely reported. In the Transactions of the Pathological Society of London,[1] Dr. T. B. Peacock relates a case the previous history of which was unknown, but in which the patient had an attack of angina on the day preceding death. It was not, however, the actual cause of death, which seems to have been due to a rheumatic attack with sero-purulent pericarditis. In this case there was an aneurism of the left coronary artery as

[1] Vol. i., p. 227.

large as a pigeon's egg, which was buried in the left ventricle at the base of the heart. Peste[1] reported a case which is quoted both by Peacock (*loc. cit.*) and by Huchard[2] in which the patient, a man aged seventy-seven years, died from the rupture of an aneurism of the size of a hazel-nut in an ossified coronary artery. Huchard likewise mentions a case published in an inaugural thesis by Hoffmann in 1886, in which a woman, aged thirty-three years, who suffered from asthma and angina pectoris, exhibited after death, with other changes, aortic valvular disease and an aneurism of the right coronary artery. The conditions indicate this case as having been syphilitic. In the Transactions of the Pathological Society,[3] Mr. J. Jackson Clarke relates the case of a child who exhibited numerous aneurisms on the coronary arteries, which it is suggested were probably either specific or micrococcic. The child had acute endocarditis.

Coronary Occlusive Angina.—Some who are averse to the acknowledgment of a musculo-spastic angina of the cardiac muscle itself do not hesitate to admit the feasibility of a theory which rests upon an intermittent spasmodic closure of the coronary vessels (Huchard), having in its train an ischæmia of the cardiac muscle which eventuates in the angina. With this claudicatory theory I am not at present concerned. By coronary occlusive angina I mean an angina the result of atheromatous occlusion of the coronary arteries. We have seen how as life advances there is a thickening of the internal coat, which, if in a measure protective at its outset, passes in many cases into a destructive phase, leaving in its train impediment to the circulation, with or without additional lesions, involving a solution in continuity of the coats of the vessels. With one phase of this vascular destruction—namely, aneurism of the coronary artery—we have already dealt at some length. On occlusive coronaritis or atheroma I wish in this place to say a few words. Given satisfactory proof of the apparent soundness of the cardiac muscle, of the nervous apparatus concerned with the sensi-

[1] *Archives de Médecine*, 1843. [2] *Op. cit.*, p. 840.
[3] Vol. xlvii., p. 24.

bility and motion of the heart and of the character of the blood itself, and given then a considerable impediment to the flow of blood into both coronary arteries, I am prepared to admit that the chief factor in the production of angina in some cases may be a condition of difficult circulation in the nutrient arteries of the heart on the lines suggested by Allen-Burns in 1809, and elaborated by Potain under the term 'intermittent claudication.'[1] But this is a very different position from that taken by those who, on the detection of a certain amount of atheroma in the coronaries of those who die from angina pectoris, assume without more ado that this state of the coronaries is chiefly responsible for the syndrome of Heberden's disease. It is notorious that the exclusion of the other possible factors in the disorder is in many cases not even attempted, and that even when it is attempted it is done without the exhaustive investigation which alone carries weight or brings conviction to the mind. While, therefore, coronary occlusive angina—a term, perhaps, better and more simply expressed as coronary insufficiency —may be accepted as a variety of that affection, we cannot too soon emancipate ourselves from the thraldom of too narrow a view of the causation of angina, however influential the support given to such a theory.

Coronary Thrombotic Angina.—A man fifty-nine years of age was admitted in a collapsed condition to the Great Northern Central Hospital. He complained of severe and continuous pain at the lower end of the sternum, radiating down his left arm. He was regarded as the subject of a ruptured gastric ulcer, and was placed in a surgical ward. There appears to have been either some doubt as to the diagnosis, or the patient was regarded as too much exhausted to undergo operation, for none was performed—fortunately, as it transpired. Pain was said to have been continuous from the time of admission at 8 p.m. on January 22 until 7 p.m. on January 23, when the patient died.

On examination after death, the heart was found to have undergone fatty degeneration, the coronary arteries were atheromatous, and the right coronary artery contained a

[1] *Clinique Med. d. l. Charité*, 1894.

partially decolourized clot adherent to the vessel, which showed it had been *in situ* some time prior to death. No other conditions accountable for the symptoms manifested by the patient or for his death were discoverable. It was learned later that the patient had occasionally had attacks of angina of a less severe nature prior to his last and fatal attack.

Sir William Church has also recorded[1] a fatal case of ante-mortem coronary thrombosis which, too, was accompanied by severe angina.

In view of the acknowledged fact that severe pain is felt with arterial embolism, and remembering the abundant innervation of the coronary bloodvessels without and, as I have shown, within, it appears legitimate to regard such a condition as that described to be causal of the severe angina manifested. Such cases might as truly be described as cases of coronary colic, as are the severe attacks associated with pain, and it may be collapse, which are due to the impaction of a calculus in the bile-duct or ureter, and termed 'biliary' or 'renal colic,' as the case may be. The heart, like the liver or kidney, may be insensitive to cutting or tearing, but the coronary artery, like the bile-duct and ureter, may evince a severity of pain which leaves little doubt in the mind of the sufferer as to its situation relatively to the surface. Pain is referred to the site of embolic, thrombotic, or calculous stimulation.

As bearing upon this point,[2] Sherrington, as already stated, calls attention to the fact that the insensitiveness of the liver under incision or puncture by the surgeon stands in contradiction to the pain caused by biliary calculi, and refers to the 'ease and certainty with which mechanical stimulation of the duct by a little fluid excites a strong vascular reflex, mentioning the interesting anatomical fact that, while the vagus is the afferent and efferent nerve to the pancreas,' the common bile-duct, though opening together with the pancreatic, possesses afferent fibres from the spinal ganglia. A similar spinal innervation of the coronary vessels un-

[1] St. Bartholomew's Hospital Reports, vol. xxxii., p. 7.

[2] Schäfer's 'Physiology,' vol. ii., p. 854.

doubtedly exists, and it is probable, in view of the radiation of coronary angina, that it is along these nerve channels that the afferent stimulus causing pain travels.

Aortitic Angina.—Syphilitic disease of the heart and aorta, apart from aneurism, has, since Ricord's day, been studied by many authors. In the *Lancet* of 1897[1] there is an article on syphilis of the myocardium by Dr. Sidney Phillips, who refers to previous work on the subject. Sir William Osler and Dr. Mitchell Bruce dealt with the matter in their Lumleian Lectures,[2] and Sir T. Clifford Allbutt has, by publications extending over a lengthened period, made the 'suprasigmoid portion' of the aorta his own.[3] Foreign medical literature also deals with the condition fully.

These facts notwithstanding, syphilitic aortitis with valvular incompetency, though a distinct clinical entity, is a type of valvular disease which is not always recognized as specific, although, with reasonably careful investigation and reflection, it should not fail to be detected.

The patient affected with specific aortic valvular disease is usually a man, and generally comparatively young, but not a youth. His age varies from thirty or thirty-five to about fifty. Even on careful investigation, and as a rule, no history of rheumatic or scarlet fever is to be elicited. Nor is there a history of physical disability in earlier life. The patient has usually followed some active calling, and often that of a soldier or sailor. Ultimately he becomes dyspnœic on exertion, frequently complains of attacks of pain in his chest or epigastrium, and on examination reveals aortic valvular disease with consecutive enlargement of the organ.

If he fail to respond to treatment (as he frequently does at the stage at which he presents himself) and die, the necropsy reveals an atheromatous aorta and puckering of the valves, which, considering the comparative youth of the patient in most cases, and an absence of the history of the usual acute infections which induce heart disease, leads, even when syphilitic infection is denied, to a diagnosis of

[1] *Lancet*, January 23, 1897, p. 223.
[2] *Ibid.*, March 26, 1910, p. 839; and July 8, 1911, p. 69.
[3] 'System of Medicine,' vol. vi.

the consequences of that condition (Fig. 13). More minute investigation of the textures confirms the correctness of this view. Specific aortitis may be associated with a severe and fatal type of angina pectoris.

One of the best descriptions of the disease in association with angina with which I am acquainted is from the pen of M. Huchard, with whose argument founded upon it I am unable to agree.

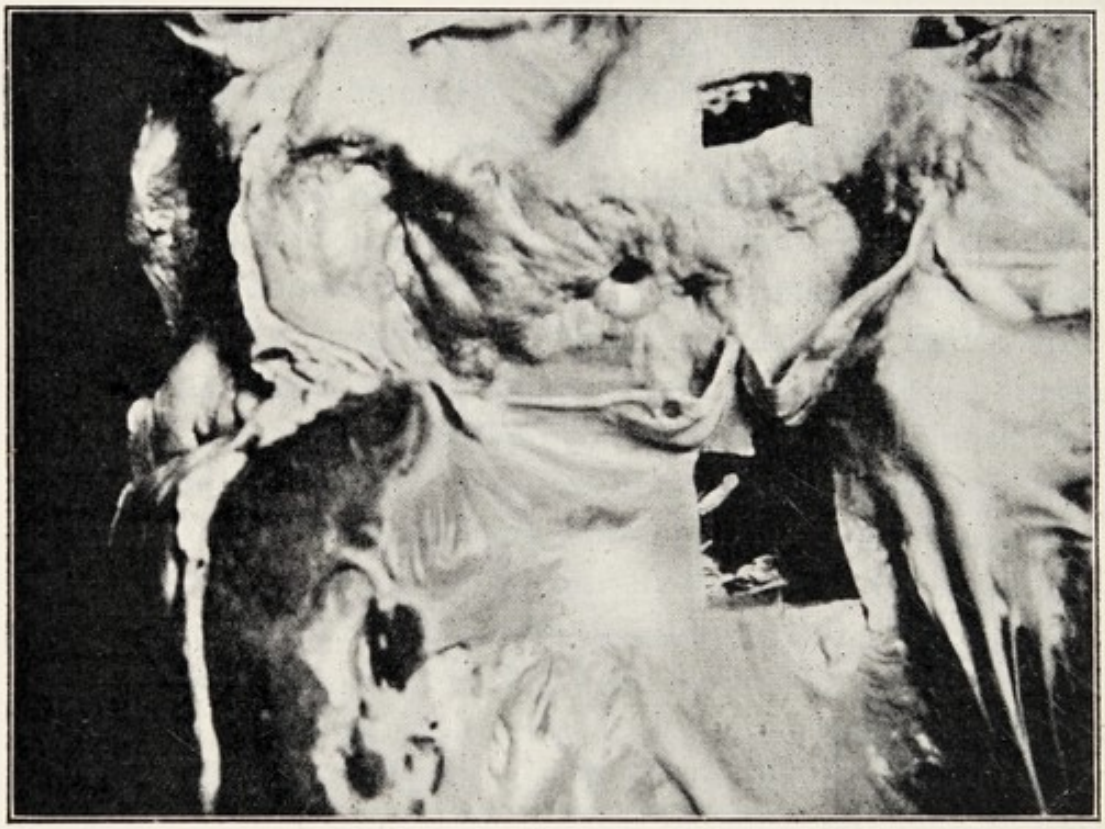

FIG. 13.—SYPHILITIC AORTITIS OF LONG STANDING IN A MAN THIRTY-NINE YEARS OF AGE, SHOWING THE PUCKERING OF THE INTIMA, WITH ENCROACHMENT ON THE CORONARY ARTERY AND SHRINKAGE OF AORTIC CUSPS.

Portions of the vessel and ventricle have been removed, as is seen, for microscopic examination.

Adrien G., thirty-two years of age, a stoker, was admitted into the Hôpital Tenon under the care of Huchard on February 6, 1883. A drunkard, he had contracted syphilis in 1872. For a month he had complained of vague pains in the chest coming on during work, accompanied by radiations into the left arm, the elbow, and the two last fingers of the left hand. The pains come on suddenly, and while they last he has to leave off work. The lungs and the heart are perfectly healthy; neither at the apex of the heart nor at the base in the aortic region can abnormal signs

be discovered. The urine is clear, sufficient, and without albumin. On February 11, at the morning visit, he complained of uneasiness in the præcordial region, with slight pain in the left arm. The patient was able, however, to leave his bed, and the day passed comfortably enough. At nine o'clock in the evening he awoke suddenly, and complained of a violent agony—an atrocious pain with a sense of suffocation in the præcordial region, which caused him to writhe on his bed. The face became cyanosed, and the patient constantly placed his hands over the lower part of his chest, as if to remove an insupportable weight. This painful crisis, accompanied by anguish, lasted with various recurrences till half-past eleven o'clock; at no time during this final attack did the patient complain of pain in the arms. He died in a minute.

Necropsy.—The lungs are much congested, allowing very dark blood to escape on section. Over the course of the aorta from its origin to the pillars of the diaphragm an enormous injection reveals itself as an extremely well-marked vascular network. In the sheath of the artery at its posterior part, at the level of the periaortic pericardium, there are four ecchymoses, situated one at the origin of the brachio-cephalic trunk and the left carotid, and three others about two centimetres from one another. They are each of about the size of a lentil. On dissecting the aorta there is well-marked congestion of the surrounding structures, but the congestion does not reach the pneumogastric nerves. At the final portion of the arch of the aorta where it becomes vertical there is in its interior a large patch of aortitis $3\frac{1}{2}$ centimetres long, following the course of the vessel, and almost 2 centimetres broad. This patch is situated to the left and rather posteriorly in the aorta. It makes a marked elevation on the interior of the vessel, and is not at all calcareous. It has a gelatino-fibrous consistency, pale grey colour, and irregular outline and surface. A fresh section examined microscopically showed a very abundant proliferation of connective tissue in the internal coat, and a well-marked thickening of the middle coat; the outer coat is slightly thickened, and the connective tissue there is more abundant than normal. In the patch of aortitis there is neither fatty nor calcareous degeneration. On continuing to open the vessel in the direction of the left ventricle we find another patch of inflammation just where the left carotid arises. The calibre of this artery is also much diminished, and is scarcely a fourth of its normal size. This patch goes quite round the carotid, but does not project more than half a centimetre into its interior. It also

is fibrous, not calcareous. Still lower we observe at the root of the aorta, over the whole expanse of the vessel, at a height of 3 centimetres and 1½ centimetres above the free edge of the sigmoid valves, a swollen surface, pale grey in colour, with a sinuous outline, rather hard to touch, but not calcareous. It would seem, then, to have been a matter of comparatively recent aortitis. The cardiac muscle was pale, and had a little surplus fat. There were no inflammatory lesions on the mitral and tricuspid valves. The aortic valves were competent and without a trace of inflammation. The cardiac muscle examined microscopically was found healthy. The other organs are much congested; the kidneys, the liver, the spleen, the pia mater, the brain, the bulb—they showed no other change. The cardiac plexus was not examined microscopically, but by the naked eye it was impossible to detect the least trace of periaortic hyperæmia. The ecchymoses which were found in the front of the aorta were nothing more than the results of the suffocative conditions which terminated the attack; they could in no way be regarded as a cause of the angina. The inflammation of the aorta has produced a considerable diminution in the size of the coronary arteries. These arteries themselves have an important peculiarity; thus, we find only one coronary arising at the level of the free edge of the sigmoid valves; its orifice is so diminished under the influence of the endarteritic inflammation that one can scarcely pass the point of a very fine probe through it. Immediately thereafter the calibre enlarges and resumes its normal diameter.

The report of this case, contributed by M. Huchard and M. Pennell, first appeared in the *Revue de Médecine* in 1883, and is among those so industriously collected and published by M. Huchard in his well-known work, to which I have already had occasion to refer. I offer no apology for having translated this fragment literally and transcribed it bodily. It is the work of a master alike of language and of clinical observation. It is also the work of one convinced of the general applicability of a particular theory of angina pectoris. The inflammation did not touch the pneumogastric, the cardiac plexus was to the naked eye free from inflammation, but the blood-supply to the organ was encroached upon in a very remarkable manner. Yet the cardiac muscle, though pale, was normal, and the valvular apparatus of the heart was sound. The coronary arterial

theory of angina M. Huchard considers applicable in this case also.

Now, so long as blood gets into the coronary system at all, the absolute freedom of anastomosis in that system seems to render it a matter of little moment whether a man have one or three coronaries, and whether his arterial orifices gape or are contracted. There may be very considerable obstruction to the coronary arteries at their commencement in the aorta in such cases, with little angina pectoris. Localized portions of the myocardium may become fibrotic or degenerate from being robbed of blood, but this occurs only when the larger vessels are absolutely closed or smaller branches are occluded by peripheral endarteritis. In M. Huchard's case the cardiac muscle was healthy, and we may therefore take it that its blood-supply was not materially interfered with. But no one has more graphically or better described the potential sensibility resident in arteries than M. Huchard, and in the widespread and superbly delineated acute aortitis in this case it seems to me that we have quite a sufficient explanation of the breast-pang of this patient without the importation of any more obscure theory. This case, then, I would submit, was no evidence of the relation of the coronary circulation to angina pectoris, but of the anginal pain associated with an acute aortitis, a variety of the disorder which may be suitably described as aortitic angina.

Such a case offers every support to the views of Sir T. Clifford Allbutt, who has eloquently enunciated the aortitic theory of angina. 'Many years ago,' he writes, 'in the days of my studentship at St. George's Hospital, a case came under my notice which I see as vividly as if the patient were still before me. A man of some thirty or thirty-four years, of vigorous frame and apparently of vigorous constitution, lay propped up in bed in extreme agony. He complained, when he could whisper to us, of intense retro-sternal pain, never absent, indeed, but returning upon him in paroxysms. The pain radiated about the shoulder or shoulders; whether it extended down the arm I cannot remember. The respiration was restrained in dread.

There were no physical signs to betray the presence of the disease within. What I vividly recollect, as if burnt into my mind, is the aspect of the man, bound on the rack in the presence of death, and yet, for the agony at the centre of his being, unable to cry out. Consultations were held, but to little purpose, save to certify that the case, if one of angina pectoris, was a strange one, because of its continuous if still paroxysmal character, and because of the fever with it. Bence Jones, whom no man exceeded in brilliancy and rapidity of diagnosis, declared for acute aortitis; the patient died suddenly soon afterwards, and the necropsy justified Bence Jones's opinion. On the inner surface of the ascending aorta were groups of grey, semitranslucent patches disfiguring the walls of the slack and dilated vessel; and —let this be carefully noted— no other cause of death was discovered. The heart and coronary arteries were healthy.'[1] After discussing acute aortitis he proceeds to describe a case of 'stenocardia' induced by such exercise as walking up a hill, and comes to the conclusion that the pain in such '*and in all such cases is not cardiac but aortic.*'[2]

That the clinical picture which Allbutt has so eloquently word-painted is associated with paroxysms of pain which may justly be included in the category of anginæ pectoris, cannot for a moment be doubted, and that he is fully justified in ascribing the distress to the local inflammation, which was doubtless due to syphilis, appears to be incontestable. But does such a fact, in view of the pathological bases of angina into which we have examined, justify so sweeping a generalization as that which assumes that the pain characteristic of Heberden's disease cannot be originated elsewhere, in muscle, or vessel, or nerve? I would respectfully assert that it does not, but that muscular spasm, aneurismal pressure, and local neuritis, as well as other states to which we have still to refer, may cause pain which is certainly pectoral and retro-sternal, but neither aortic nor aortitic.

[1] Lane Lectures, Reprint, p. 119.

[2] *Ibid.*, p. 124.

Aortic Aneurismal Angina.—It is related of a Dutch boy that, perceiving a leak in some portion of the dyke which has turned a waste of waters into a rich and thickly populated land, the Hollow-land, or Holland, he placed his hand over it, gave the alarm, and held the ocean at bay until the defect had been remedied and the population saved. The narrative is probably true, for it is scientifically explicable. The pressure of fluid is proportionate to the surface it presses upon, no matter what its actual volume. We are not, therefore, surprised when we find the heart and coronary system little affected by the blood-pressure even of large aneurisms in the aorta. What *does* frequently occasion us surprise is that patients should carry with them, sometimes for a lengthened period, considerable aneurisms of the aorta, with all their secondary vascular manifestations in impeded venous circulation, without evincing great discomfort and sometimes having no pain. But this is not by any means always so, and there are probably few cases of aneurism of the aorta which have not, at one time or another during their growth and existence, caused some pain. At times this pain is great and not to be distinguished, apart from local considerations, from angina pectoris due to one or other of the causes we have already considered. 'Many authors from Morgagni downwards,' writes Sir William Gairdner in his memorable essay,[1] 'have recorded cases of thoracic aneurism, having in a more or less perfectly developed form the characteristic symptoms of angina pectoris, and we have already alluded to M. Trousseau as confirming by his large and carefully watched experience the view that such cases very closely resemble, and may, in fact, for a lengthened period and after careful examination, be indistinguishable from what he regards as the truly idiopathic forms of angina. The author of this article,' he further states, 'is able from personal experience to say that no organic disease has appeared to him more frequently to assume the symptomatic characters of angina than aneurism, and he is also prepared to state as the general result of inquiries pursued over many years, and particularly directed to this subject, that even small

[1] Reynolds's 'System of Medicine,' vol. iv., p. 544.

aneurisms arising very near the heart, and especially such as project into the pericardium or compress in any degree the base of the heart itself, are much more apt to give rise to angina-like symptoms than much larger tumours in more remote positions.' This important statement, embodying the experience of one who had every qualification for sound observation, justifies the classification of an important variety of angina as arising in connection with aneurism of the aorta. It is an important fact, also, that those aneurisms which evinced this symptom on the whole most markedly were *small* aneurisms, aneurisms which could not, to the same extent as larger tumours, have penetrated the muscular coat of the vessel and paralyzed its grip on the throat of the sac. Knowing as we do the comparative insensibility of the viscera including the heart, and bearing in mind the fact that considerable pressure may be exerted on that organ without causing pain, it appears more probable that the anginiform symptoms in connection with such aneurisms are due less to their pressure upon the heart than to the neuritic and lacerative processes taking place in the aneurisms themselves.

Intravascular Neuritic Angina. — M. Huchard,[1] after demonstrating how even peripheral nerves will degenerate if robbed of their blood-supply, and coming to the conclusion that the nervous accidents possible in the course of angina are but accidents, neuralgic or neuritic, presses the lesson home and writes: 'There are not several anginæ pectoris, but only one—coronary angina' (p. 652). I admire his eloquence and ingenuity, but am not convinced by his argument. I have already shown in dealing with the pathology of the subject, buried in the wall of the right coronary artery of a patient who died in angina and after long suffering, not only an aneurism, but also an inflamed and thickened nerve ganglion. I do not say that this was the sole cause of the angina and of the vascular disturbances which follow pain in organs under the sway of emotion, but I do not think anyone can say that it was not at least as likely to induce these phenomena at times as any condition of the

[1] *Op. cit.*, p. 645 *et seq.*

cardiac muscle, which was healthy to all appearance, notwithstanding a greatly hampered coronary circulation. That circulatory impediment, or coronary inadequacy, may in a certain proportion of cases play the major rôle in angina pectoris I have already admitted. That it does so in all I deny. It is the more remarkable that M. Huchard should so inexorably limit himself to one explanation of angina because he has evidently carefully examined the condition of the coronary circulation, and admits in one place a free anastomosis of the vessels, but in another appears to agree with Hyrtl, who denied this. He explains the absence of angina in some cases in which the arteries are calcareous and yet the muscle healthy, by maintaining that atheroma of this type may serve to maintain the patency of the vessels, and that a supplementary artery may come to the aid of the threatened muscle. But in the case I have related the muscle was healthy, the vessels were much obstructed by a narrowing of their calibres, and there certainly was a supplementary artery of Vieussens, or fat artery, arising close to the right coronary. The case, nevertheless, had 'true angina' and died in an attack, but the wall of the coronary artery contained both an aneurism and an inflamed nerve ganglion. Therefore I think we are justified in maintaining, contrary to the conclusion at which M. Huchard has arrived, that there is not only one angina pectoris, but that there are several, and that intravascular neuritic angina is one of these.

Extravascular Neuritic Angina.—I have already stated that the extravascular nervous structures when they have been carefully examined in fatal cases of angina have frequently been found healthy, and this is in all probability true in some cases of the intravascular nerves also, but in a certain number of cases, as I have mentioned, the extravascular nerves are admittedly involved. In these we have seen that the affection of the cardiac plexus appears to have been secondary—a consequence of the extension of vascular inflammatory processes to the nervous elements in their vicinity. This was so in Lancereaux's case. In such cases the inflamed plexus may indeed be regarded as contributing

to the angina manifested, but it is only an extravascular neuritic element in the case. In being so, however, this condition is not peculiar, for we shall learn that in the majority of cases the underlying physical basis of angina is complex. Most anginæ are probably compound, with in some cases a preponderant leaning towards one or other of the varieties which have been described. Extravascular neuritic attacks of pain may also be due to the erosion of neighbouring structures by the aneurism and such may even be agonizing and persistent and require the continuous use of morphia. Pectoral pain may likewise result from this involvement of the phrenics by the extension of mediastinal and pericardial inflammation.

We are, however, but on the threshold of our knowledge of the rôle of the nervous system in angina, and many more systematic investigations into the extravascular nervous system in such cases must be undertaken before we can speak with any authority upon this particular point. In 'tabetics,' using the word not as Heberden did when he classed tabetics with peri-pneumonics,[1] but in the modern sense as applied to those having inflammatory changes usually of specific origin in the ganglion on the posterior root of the spinal nerves, and in the columns of Goll and Burdach, we find that the lightning pains experienced in the lower portions of the body have at times their homologues in the region of the thorax. While we have to be on our guard against confusing intercostal manifestations of this kind with pain in the nerve tracts passing from the spinal cord to the heart, there seems no sufficient reason to question the occasional occurrence of such. It is true that in some cases in which this condition (angina) has been reported the patient has also had aortic or aortitic valvular disease, and that both the nervous and vascular lesions have been attributed to syphilis. But the angina has as a rule been explained solely by reference to the aortic lesion, and when the latter exists, that conclusion is probably the most rational. Oberndorfer has recently referred the stenocardia of some

[1] *Op. cit.*, p. 308.

cases of specific aortitis to the disease affecting the *intercostal* arteries at their origin.

But all tabetics have not aortic valvular disease, and when angina of lightning-pain character arises in these, it is quite possible that the source of the affection may be extra-vascular and neuritic—the projected and visceral expression of a central nervous lesion. Indeed, the radiation of cardiac pain outwards, and its spread from the associated segment of the cord peripherally, is in itself an argument in favour of the possibility of a projection of the irritation from the sensory roots of the spinal nerves and the affected segments of the cord inwards. Such a visceral projection of central stimulation might also follow the course of the pneumo-gastric tract, but, as we have less clinical evidence of this than we have of projection from the centres of the sympathetic stream, we cannot with any benefit enter into its further consideration here. In the higher centres of the brain we have no pathological facts to justify our attributing angina pectoris to a central lesion, but in the introduction to these pages the influence of the emotions on the action of all the viscera, and especially upon that of the heart, was pointed out, and Heberden recognized that excitement favoured the onset of attacks of angina (*perturbatione animi augitur*). The central influence, however, in such cases but stirs into activity the local factors in the disease elsewhere.

Neuralgic Angina.—The use of this term raises the question of the propriety or otherwise of employing such an expression as 'pseudo-angina.' What is true angina? Contrary to the aim of some who have spoken and written upon the subject, I have deliberately avoided referring the well-known clinical features of the affection to only one category of physical conditions. Pain and its consequences, when connected with a particular abnormal organic state, may bear a close resemblance to pain and its results when these are associated with another abnormal state—that is, the consequences have a unity which the causes have not. If angina pectoris be one, it is also divisible; but if there be real pain, and the consequences of such pain are manifested in a typical manner by

a series of recognizable phenomena, why regard it as spurious because when associated with a certain condition it is less fatal than when associated with another? As I maintained in a paper on the blood-pressure in angina pectoris, published in the Edinburgh Hospital Reports,[1] 'A case of "true angina" is one in which there is no doubt about the angina, and there is no mistake about the reality of breast-pang in many so-called functional cases. . . . The idea of spuriousness is only permissible in so far as angina is not associated with demonstrable lesion, and because the tendency to it in many cases yields to judicious treatment or wears itself out.' Sir William Osler quotes these passages with approval in his instructive monograph on angina pectoris, and adds (p. 87): 'Herein lies the essence of the whole matter—the symptoms on the one hand indicate the existence of a grave organic, usually incurable malady, and on the other a condition very distressing, it is true, but rarely serious and usually curable. The advantages of thus recognizing a functional group far outweigh any theoretical objections, and in a series of cases the forms are, with few exceptions, fairly well defined.' Now, actual *neuritis* is generally associated in angina with grave organic disease of the structures innervated, and it therefore appears to be preferable to separate these from cases in which no organic disorder is discoverable, and pain and its immediate effects are the chief phenomena. Long usage permits our calling such cases '*neuralgic* angina,' which is perhaps more definite than, and preferable to, such terms as 'false' or 'pseudo-angina.' Far as the nervous endowment of the heart and its vicinity still is from complete elucidation, much progress has been made in unravelling this obscure subject. We know now the mode of ultimate distribution to the cardiac muscle, we know somewhat the innervation of the bloodvessels of the heart, Thoma believes he has demonstrated Pacinian bodies in the vascular wall, the main nerve trunks have been known of old, and, as has been shown, the sources of the visceral nerve-supply have in great measure been successfully traced, and, lastly,

[1] Vol. iv., pp. 246-265.

the comparatively recently discovered nodal structures, once regarded as automatic and nerveless, have been shown, as all automata in time come to be shown, to be completely innervated and under nerve control. Why, then, hesitate to recognize a possible neuralgia of the intrinsic and extrinsic nervous mechanism of the heart ?

CHAPTER IV

THE CLINICAL HISTORY OF CARDIAC PAIN

(Continued)

Endocardial Angina

THE justification for the use of the term 'endocardial angina' to indicate a condition still quite comprehensible by that name, although not expressed in terms of the myogenic cardiology, is the fact that, whatever causes or consequences there may be of valvular disease of the heart, such as atheroma of the aorta or of the coronary arteries, it is indubitable that cases are met with in which the frequency and severity of attacks of angina are coincident with non-compensation. Thus, there may be a storm of angina in aortic valvular disease of the heart during such a period of non-compensation, which practically subsides when the heart has recovered power. In these, as in many other cases, the physical basis of the disorder may be complex, but, the element of non-compensation being the key to the situation, it is a legitimate inference that intracardiac pressure may be one of the factors in precipitating the attack of angina. Whatever consequence of the non-compensation may be selected as most suitable for rearing upon it a theory of the anginous attacks, the central fact remains —non-compensation, or failure of contractility and tonicity.

That a disturbance of the coronary circulation is not absolutely essential to the production of the cardiac pain and its brachial radiation I think I shall be able to prove by relating some particulars of a case of dextral valvular disease which came under my care many years ago, and an account of which I published at the time.

Sir (then Dr.) T. Lauder Brunton in the *Practitioner* for 1891 developed the theory of cardiac distension as a cause of angina pectoris, and that this may be a factor in the production of some degree of cardiac pain in cases such as we are at present considering appears very probable. Professor (now Sir William) Osler,[1] however, pertinently inquires, 'Why, if extreme dilatation is a cause, does it not occur more often? There must surely be some additional factor, or attacks would be of everyday occurrence.' It is not, however, extreme, but sudden, and it may be only moderate dilatation, which is more usually associated with cardiac pain than greater and more persistent dilatation.

The majority, although not by any means all, of the cases of cardiac pain producing the syndrome of angina pectoris in valvular disease of the heart are cases of aortic valvular disease, and of the latter most are mainly regurgitant in character. Angina with endocarditic mitral disease is comparatively rare. In aortic valvular disease, and especially in its regurgitant variety, we usually have a powerful ventricle throwing blood suddenly into the aorta, and as suddenly, of course, into the coronary vessels. The effect of the force of this impulse upon the interior of the coronaries may be broken if there be atheromatous narrowing of their orifices; but if the blood find quick and distensive entrance into these vessels, it is not difficult to conceive, in view of the facts I have already related in connection with the innervation of the coronaries, that the incentive to pain may be here in such cases. This will most frequently be found to be the case when there is considerable ventricular hypertrophy.

But I have met with anginal attacks, as I have stated, in a case of well-marked disease of the valves at the orifice of the pulmonary artery.[2] The cusps in this case were much distorted and covered with cauliflower excrescences, and from their size and impact against the wall of the artery had provoked inflammation there. There were also scattered vegetations on the tricuspid valve and in the conus arteriosus.

[1] *Op. cit.*, p. 122.

[2] Transactions of the Pathological Society of London, vol. xxvii.; also 'Dextral Valvular Disease of the Heart,' Graduation Thesis, 1878, p. 5.

It was manifestly a case of what we should now call infective endocarditis engrafted on an older and rare dextro-cardial lesion (Fig. 14).

The case was one of long standing, and occurred in a young man twenty-one years of age. His cardiac pain radiated, not to the left, but to the right, was characteristic of distension, and not observed by me to be associated

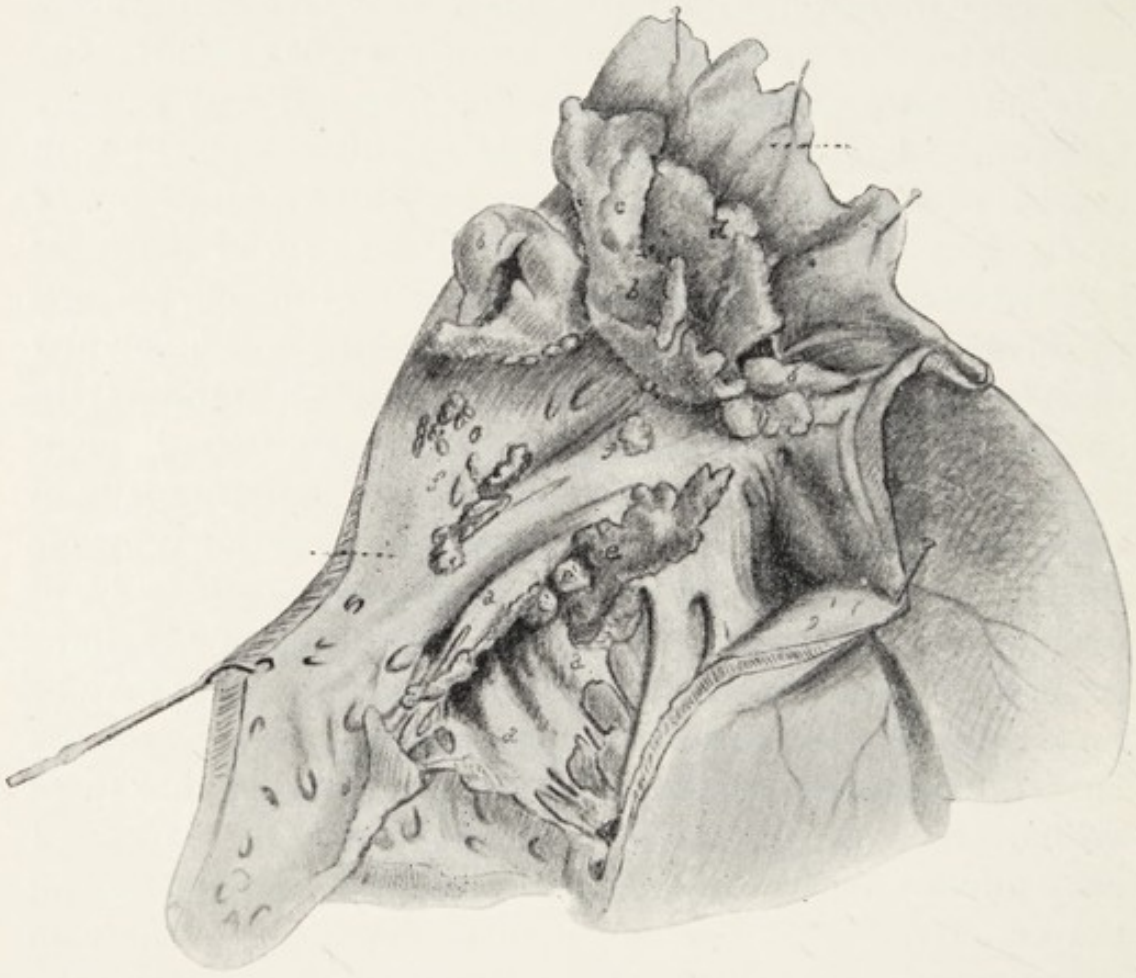

FIG. 14.—DISEASE OF THE PULMONARY AND TRICUSPID VALVES WITH ENDO-CARDIAL ANGINA RADIATING TO THE RIGHT ARM.

with the extreme agony and vaso-motor phenomena which we shall have to consider presently, although the patient stated that the pain was at times severe. There was, of course, in this case no coronary arterial factor to complicate the etiology of the pain. We may conclude, therefore, that a purely local cause, such as distension of a chamber, may have a certain, but not always a very powerful, influence in calling into play the more immediate factors in the production of cardiac pain—namely, painful stimulation of the

cardiac nerves, be it through stretching or through muscular cramp. The severer cases of endocardial angina are, as I have said, usually associated with aortic valvular disease, but severe endocardial angina from sudden distension of the ventricle may also be observed in mitral valvular disease. The sudden ventricular repletion caused by rupture of an arterial cusp is attended with pain in the chest, which is most probably due to endocardial tension.

What are usually the central and consecutive phenomena in endocardial cases? The following case, some particulars of which I shall relate, was published by me in the Edinburgh Hospital Reports[1] in my article on blood-pressure in angina pectoris. It was that of a man, thirty-two years of age, who had never to his knowledge had rheumatic fever, but who exhibited the full clinical picture of aortic regurgitation, and in whom probably rheumatism, as so often happens in early life, had been overlooked. He came under my care in May, 1894, at the Great Northern Central Hospital. In December, 1893, he began to suffer from attacks of pain in the chest, severe in degree, and in the first instance passing up to his left shoulder and down his left arm. As the attacks became more severe, pain radiated even down his right arm, and affected his legs and body generally. He also noticed that when pain passed off a tenderness remained in the præcordia to the left of the sternum, the parietal tenderness to which Mackenzie, Head, Gibson, and others, have called attention. Brandy at one time relieved these attacks, but later it became useless. The pain was relieved by standing up, and became worse when lying down. This fact, in view of the nature of his valvular lesion, is interesting. On the advice of a medical man, he lay in bed on one occasion for three weeks continuously, and rose from the recumbent position for no purpose. He steadily grew worse, and his pain became more severe. He took nitrite of amyl as an inhalation with benefit. He had the usual physical signs of aortic regurgitant disease. The bruit was loud, diastolic, and of a highly musical quality. On November 7, about seven months after I had first seen

[1] Vol. iv., p. 252.

him, he had an attack of angina while I was examining his heart. The organ quickened in action, and the pulmonary second sound became markedly accentuated, while the aortic diastolic bruit became less audible. He then inhaled a capsule of nitrite of amyl. After a short time the accentuated pulmonary second sound became less audible, and the heart's action became slower and slightly irregular, the musical diastolic bruit being occasionally loud and prolonged, and again soft and short, and then pain passed off. The patient persistently refused to become an in-patient of the hospital, as he dreaded recumbency, and, wishing to use the sphygmograph and to examine the patient with more leisure, I asked him to call at my house. The following particulars I transcribe from the article to which I have referred: 'When I had taken a tracing, an attack of severe angina supervened. The patient at once rose and grasped his left wrist tightly with his right hand. I persuaded him to sit down, and took a tracing as well as possible under the circumstances. He crushed and inhaled a capsule of nitrite of amyl while I was doing so. I was thus fortunate enough to get a tracing of his pulse when the drug had begun to act, and another when the pain had just passed off. These tracings, taken in connection with the observations on the patient's heart during the attack which I have already mentioned, appear to me of theoretical and practical interest. The patient did not again present himself at the hospital until January 16, 1895, when he seemed in all respects better. He had gained flesh, been more free from pain, and slept better in the interval between attacks. He consulted me, as he had not recently been quite so free from pain. On examination I found the apex-beat in the fifth space slightly inside the nipple-line, the transverse measurement of the heart 4½ to 5 inches, and the bruit in the same situation as formerly, but, though still distinctly musical, rather less so. A sphygmogram of the right radial artery was taken while the patient was seated, and may be regarded as normal for him (Fig. 15). These sphygmograms are sufficiently instructive on examination. No. 1 represents a characteristic tracing of apparently pure (and uncompensated) aortic re-

gurgitation under low pressure. The pressure was, however, sufficient to produce an accurate reading. Nos. 2, 3, and 4, were taken with the hand in one position and at one application of the instrument. It will be observed that the predicrotic wave, which is well marked near the summit of the normal tracing—that is, of the tracing before angina—has fallen considerably during the attack (No. 2), and tends

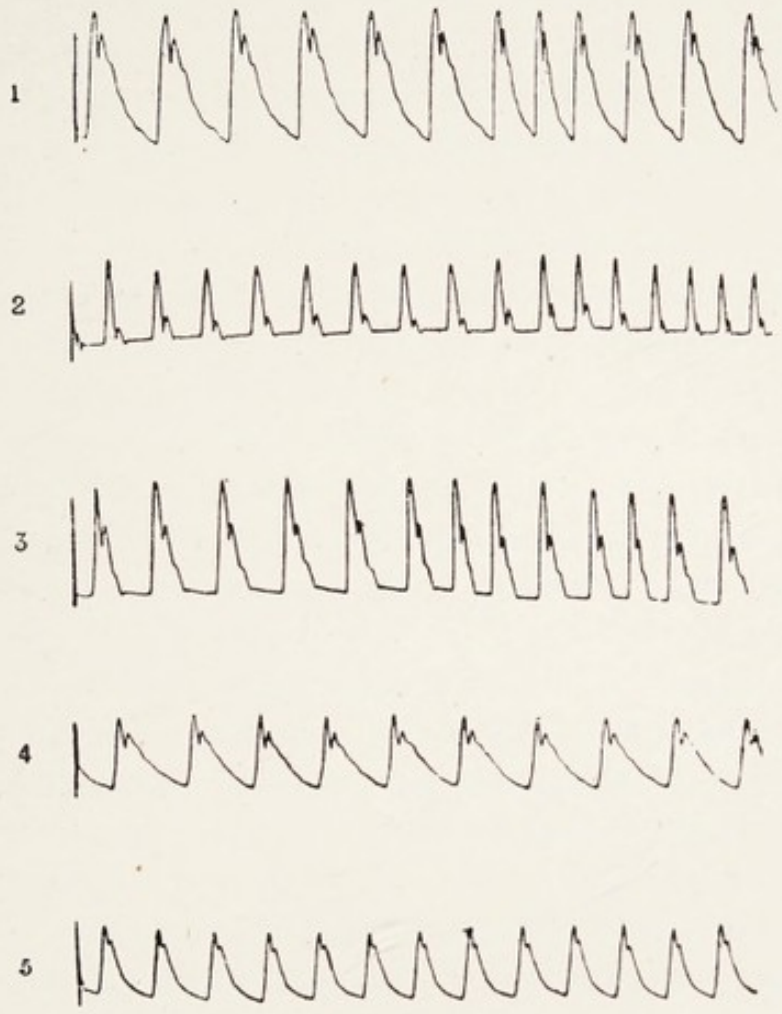

FIG. 15.

1. Aortic reflux; uncompensated; right radial; patient seated. 2. The same; left radial; seated; attack of pain. 3. The same; pain subsiding under nitrite of amyl. 4. The same; pain quite subsided under nitrate of amyl. 5. The same; compensation temporarily established.

gradually to rise after the inhalation of the nitrite (No. 3); while it has all but attained its original level when the patient declared himself free from pain (No. 4). During the paroxysm (No. 2) it will also be noted that the wrist pulse has shrunk away from the instrument during cardiac diastole, but not sufficiently to obliterate its diagnostic undulations;

while in No. 3, when the nitrite had begun to act, it raised the lever in a manner more resembling the normal tracing (No. 1) than that of complete post-nitrite relief (No. 4), when the dilated and refilled vessel pressed with greater force on the lever, as is evidenced by the moderately high upstroke, the return of the predicrotic wave to its normal level, and the full and quiet swing of the whole sphygmogram. A notable characteristic of the tracing of the attack at its height (No. 2), and of that showing commencing relief from the inhalation of the nitrite, is the flat-drag of the dicrotic or prepercussional portion of the sphygmogram. This was not due to accidental impediment, or other disturbance in the action of the instrument, but, in my opinion, to the fact that during that time the collapsed vessel was not in effective contact with the sphygmograph, which recorded an almost straight drag in consequence. This is less apparent in No. 3 than in No. 2, but is nevertheless well marked in it. In No. 4, on the other hand, the vessel has filled so as to be in good and constant contact with the lever, and registers the tracing of normal repletion and pressure.'

If this interpretation of these tracings be correct, and the practical abolition of the bruit, together with the increased accentuation of the pulmonary second sound, be borne in mind, we have to deal in such a case with a heart for the moment dilated and feebly contracting, and a collapsed, not actively contracted or bespasmed, peripheral arterial system. These *consequences*, however, do not explain the more immediate *cause* of the pain. While in such cases the suddenly distended or dilated ventricle, on Sir Lauder Brunton's hypothesis, probably plays a part in the etiology, this factor alone does not, it appears to me, explain the widespread pain, involving in this case both right and left brachio-thoracic nerve paths and extending even to the body generally. In view of all the facts we have gleaned, an important cause of this widespread distress in such an aortic lesion as that just described is probably the distensive throb of the blood cast by a dilated yet hypertrophied ventricle into the aorta and coronary system. To this supposition the same

objection may be urged as that which occurred to Sir William Osler in criticizing Sir Lauder Brunton's theory—namely, that if the above view be correct, why do not these consequences follow more frequently?

Now, in connection with the lesion mentioned, such attacks may occur with considerable frequency, and in the particular case I have quoted they occurred with great frequency, especially when lying down. Thus I have the following note concerning the experience of the patient: 'Went to bed at 10 p.m., slept at once for three-quarters of an hour, when he was awakened by pain in the præcordia, passing down to the left wrist on the ulnar side. Duration of attack five to ten minutes, when he slept again. Was wakened every hour by the same pain.' The general nervous organization of the patient is not to be lost sight of. The highly strung, neurotic subject of such lesions suffers under these circumstances more than the less sensitive and phlegmatic.

Hypertrophic Endocardial Angina is met with in valvular disease of the heart with such large hypertrophy of the organ as displaces it from its usual position, and causes the impact of the heart to be not against the soft and yielding intercostal space, but against the hard and resistant ribs. In young subjects of a sensitive type an erethism of the cardiac muscle seems to be engendered which may provoke a frequent and exhausting succession of anginal attacks on which the usual alleviants have little effect. Whether this erethism be provoked directly or indirectly may be arguable, but is not important. That is, whether it be the result of a direct stimulation of the cardiac muscle from costal impact, or whether cardiac impact against the resistant cage and the nervous endowment of its lining and soft parts raise the irritability of the associated segments of the spinal cord.

The following is a case in point which was reported in a paper on 'Thoracostomy in Heart Disease,' in the *Lancet* for July 4, 1908.

A man, nineteen years of age, was admitted to the Great Northern Hospital on December 12, 1907. On admission he exhibited in an exaggerated degree the well-marked phenomena of aortic valvular disease. The heart's apex-

beat was in the fifth and sixth spaces about 4 inches from mid-sternum. The area of cardiac dulness was increased. There was visible pulsation in the intercostal spaces to the left of the sternum, with exaggerated systolic impulse in the carotids and suprasternal space. The cardiac action on palpation was heaving and concussive, with pronounced diastolic thrill over the aortic area. On auscultation a loud and coarse systolic and diastolic bruit was audible at the aortic base, and at the apex a rumbling presystolic murmur. His pulse-rate was 88 to 92; its rhythm was regular, and its character was of the well-known water-hammer type. The blood-pressure, determined by the Riva-Rocci instrument, is noted to have been 125 to 130 millimetres of mercury. Respiration was shallow, but there was no evidence of lung disease. His other organs were normal, and there was no anasarca or effusion into any of the cavities or cellular textures of the body. The urine was free from albumin.

The patient remained in hospital about five weeks, being discharged on January 18, 1908. He was treated by rest and cardiac tonics, taking in the first instance strychnine and belladonna with nitrite of amyl to relieve his anginal pain, and later, as he made little progress, with strychnine, strophanthus, and digitalis, which increased the force of systole without relieving his pain. His systolic blood-pressure is noted on January 3, 1908, as having been 160, and his attacks of pain varied in frequency and intensity, but were apparently severe at times. The pain was retro-sternal and across the chest, but did not radiate into the arms. There was considerable mobility of the heart's apex-beat, which is noted by me as having been 9½ centimetres from mid-sternum in the fifth space when lying on the back, 6½ centimetres from the same point on right decubitus, and 11 centimetres from the tip of the xiphoid process to its beat in the sixth space when lying on the left side. From this I considered it improbable that he had any costo-pericardial adhesion. His condition appeared to be stationary. The attacks of pain, which frequently occurred at night as well as during the day, kept him awake. Although he was able to walk about gently and take his meals regularly, he

made little progress towards greater activity with comfort, but exhibited none of the phenomena of progressive retrograde stasis. Under these circumstances he was discharged from hospital, advised to take cardiac tonics and rest at home, and to report himself from time to time. Before he left I informed his father that I believed thoracostomy might be of service to him, but in nowise urged the operation, as I could not speak from experience, direct or indirect, as to its effect in a similar case.

Thus the matter rested until April 13, when he was readmitted under my care, having reported himself from time to time without any evidence of recuperation, and suffering more than previously from cardiac pain, over which the inhalations of nitrite of amyl appeared to be losing their

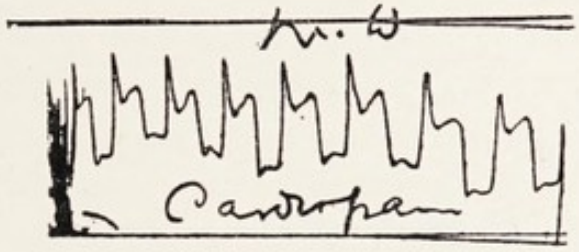

FIG. 16.—CARDIOGRAM (APRIL 23).

power. He was admitted on the understanding that, if he showed no satisfactory improvement, the operation I had suggested should take place. On the 18th I noted that the area of cardiac dulness measured 5½ by 5½ inches, the apex-beat was in the sixth space 5 inches from mid-sternum, the area of palpable concussion measured 3 by 4 inches and was covered by the fifth and sixth ribs over an extent of 4 inches including their cartilages, while the other physical signs were as before. The anginoid attacks were often severe, but varied in frequency. They occasionally lasted from a quarter to half an hour. On the 24th, some time after an attack had passed off with the use of nitrite of amyl, his pulse-rate is noted as 138, his respirations as 24, and breathing capacity as determined by Lowndes's spirometer as 60 cubic inches. On the 27th it is noted that the patient's 'condition (was) unchanged. Fewer anginoid attacks than usual during the last twenty-four hours. Heart

as before. Blood-pressure 170 millimetres (systolic) and 100 millimetres (diastolic), as determined by the point of maximum oscillation. Urine normal.' His temperature had throughout been normal, and the accompanying tracings (Figs. 16 and 17) show the character of his cardiac and arterial pulsations on April 23. These tracings are given for comparison with those taken at a later date.

Finding that the usual remedies, medicinal, dietetic, and postural, had little effect in ameliorating the patient's condition, I advised a præcordial thoracostomy, with results which I shall relate more fully when I come to deal with the treatment of this variety of cardiac pain.

Vaso-Motor Angina.—That the arterial periphery under the influence of the nervous system varies in calibre, and

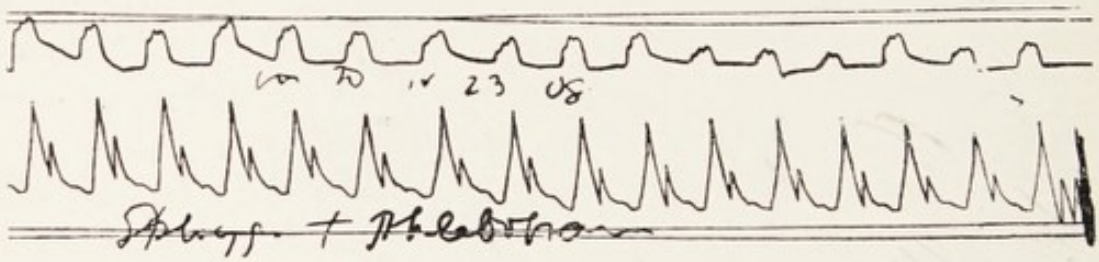

FIG. 17.—CERVICAL PHLEBO-ARTERIOGRAM AND RADIAL SPHYGMOGRAM (APRIL 23).

therefore in permeability, is a fact too well recognized in physiology to be called in question. That this condition, until relieved by vaso-dilatation, raises the general blood-pressure in the body is equally indisputable. That a diminution in size and an increase in tone of the larger peripheral vessels, notably of the radials, have been observed antecedently to, and during attacks of angina pectoris, by physicians of recognized ability and experience establishes beyond cavil the fact that such changes are, if not invariably, certainly in some cases, associated with the painful affection we have been studying. That agents under the influence of which such peripheral arterial tone is reduced likewise relieve the central pain, has been the gratifying experience of many since Sir Lauder Brunton, guided by the physiological experiments of Dr. Arthur Gamgee, first used nitrite of amyl in 1866 for the cardiac pain of a patient in the Edinburgh

Royal Infirmary, who was suffering from aortic valvular disease with angina. That the pulse, however, is not always spastically narrowed in angina is a clinical fact as indisputable, in my opinion, as that it is at times so narrowed. That the blood-pressure at the periphery, even in that form of endocardial angina so often associated with aortic valvular disease, is on occasion one of peripheral depletion from cardiac failure rather than of repletion from peripheral spasm I have endeavoured to maintain in a preceding section. But it may freely be admitted that vaso-motor angina with or, as in Nothnagel's[1] cases, without central lesions may be accepted as a clinical fact. This done, however, are we in all such cases dealing with a pain provoked in the first instance by peripheral arterial resistance, or is the peripheral spasm, if such occur, a consequence in some cases, if not in all, of the centrally experienced pain?

It is well in such a discussion to have a clear conception of the meaning of the terms used. Nothnagel was not the first to coin the term 'angina vaso-motoria.' Landois[2] preceded him, but did not attach the same meaning to the term as Nothnagel did. It is certain that many now describe cases as vaso-motor which have little in common with those which Nothnagel had in view when he wrote his paper. His cases appear to have been a sort of generalized migraine without the headache, in which the prognosis was always favourable, the patients usually quite recovering. In association with very pronounced cardiac pain they are not common, so far as my experience goes. The germ of Nothnagel's interpretation of his anginous cases may be observed in the paper which he published a year earlier in the same journal[3] on 'The Doctrine of Vaso-Motor Neuroses.'

That such agencies as cold, emotional excitement, reflex disturbances from various organs, and certain poisons, which even tobacco may prove to be at times, may commence the vicious cycle by inducing the peripheral spasm which finds expression in angina, we may rationally admit. But that

[1] *Deutsches Archiv für Klinische Medicin*, 1867.
[2] *Correspondenzblatt für Psychiatrie*, 1866.
[3] *Deutsches Archiv*, Band ii., p. 173.

such peripheral conditions, even in these circumstances, require the presence at the centre of some state or states in the majority of cases, which aid the peripheral resistance to find expression in pain, is, I imagine, in view of all the facts we have investigated, quite indisputable. Given a weak spot at the centre, capable of registering a rise of pressure in the vascular system, that rise may be registered as pain, however the increase of pressure be brought about, whether by central propulsion or peripheral resistance. But that a patient with dissecting aneurism of the coronary artery, with neuritic processes active in the aorta and coronary system, and with a delicate and minute innervation of every fibre in the cardiac muscle, should invariably wait upon the remote periphery for the signal of distress is simply unthinkable, and may even be regarded as an opinion no longer tenable. In speaking thus strongly for the central origin of angina in many cases, apart from considerations of peripheral blood-pressure, I am glad to have the support of Sir T. Clifford Allbutt. If a stimulus arising in the gastro-intestinal tract can alter the conditions of peripheral blood-pressure; if the emotional distress caused by such a stimulus may aggravate, alter, or vary such peripheral manifestations of disturbance, may not a pain, among the most agonizing of those which the body can experience, and associated with more emotional disturbance than most of them, play the major rôle in altering the state of the peripheral circulation? Surely the question has only to be asked to be answered in the affirmative. 'May not this rise,' asks Sir T. Clifford Allbutt,[1] 'be rather a consequence than a cause of angina?' I think we may now unhesitatingly answer 'Yes' to this question in the majority of cases.

Compound Angina Pectoris.—We have now examined, with sufficient fulness, the various elements capable of playing a part in the complex clinical picture of painful angina pectoris. But this very complexity argues the participation of more factors than one in many cases. As was maintained in the introductory pages, the functional unit is not single but at least triple, and in the case of the heart and vascular system

[1] Lane Lectures, p. 109.

consists of muscle cell, blood, and nerves. These factors, playing as they do an associated part in the physiology of the organ, frequently play likewise a combined part in the pathology of the organ and evolve by their co-operation the clinical entity compound angina pectoris, into which category most cases of the disorder must of necessity fall. 'I urge, then,' writes Sir T. Clifford Allbutt, 'that, although often an epiphenomenon of aortic valvular disease, there is in angina pectoris a *tertium quid* which marks it as something apart from the crowd of heart diseases and from mere spasmodic neurosis. Structural disease of the heart itself, in a conspicuous sense, may not be an essential part of angina; it may be a contingent, but not an indispensable condition.' He then refers[1] to 'a grave and mortal case' reported by myself to support this contention, and is good enough to regard it as 'described minutely.' Such minuteness of description, however, as the case received when I first published it was insufficient, for after the lapse of seven years I decalcified the right coronary artery from this case and found in it both the internal aneurism and the inflamed and degenerated nervous structures which I have already described, conditions which, under the influence of varying pressure in the circulation, appear to me to supply, at least in *this* case, the *tertium quid* for which we are in search.

The major rôle in compound angina may be taken by one or other of the factors named in different cases. The cardiac muscle may hold in unrelaxing grip the sensitive nerve endings which wander among its fibres—a statement which on theoretical grounds would be disputed by those who deny the possibility of a localized tetanic contraction in the myocardium, and by those who do not recognize the existence of sensory visceral nerve fibres in the heart. The character of the blood and its quantity and impulse may influence the vitality of muscle and nerves alike, and irritation or inflammation of the nervous mechanism may, directly or indirectly, disturb the other partners in the functional action of the organ. The determination of the point as to which of these factors plays the leading part in any given case belongs to

[1] Lane Lectures, p. 98.

the diagnosis of one kind of angina pectoris from another—a task always of the highest interest to attempt to determine and likewise at times most difficult. Before essaying this task, however imperfectly, it is necessary to consider shortly that condition which is usually regarded as closely allied to Heberden's disease, and to which Sir William Gairdner gave the name of 'angina sine dolore' (anguish without pain).

Angina Sine Dolore.—In dealing with painless angina Sir (then Dr.) William Gairdner[1] threw a wide net, and enclosed a large series of cases, syncopal in nature, and associated with a distressing and apprehensive mental state. He included cases of aortic regurgitation with 'anxiety' and 'cardiac oppression,' but without pain, and cases not necessarily associated with valvular disease in which the apprehensive fears of the patient find expression in restless movements and even in cries of distress, but in which there is also no physical pain. Wont and use have made us acquainted with the clinical significance of many terms, and, as I have already stated, the first step in the determination of the nature of a disease is necessarily its recognition from the symptoms manifested, and its designation very frequently by some term denoting those symptoms. The nature of angina sine dolore, however, appears, with the exception of the mental anguish associated with it, to be in most cases, though not in all, the very antithesis of angina cum dolore—Heberden's angina pectoris. In the latter the flaccid syncopal heart found post mortem is the final event in a series; in the former the syncopal state appears to be the essence of the situation. In Heberden's disease the syncope is largely inhibitory, and only follows an agony which would blanch the face and stop the pulse did it occur in the foot instead of in the heart or its vicinity. In angina sine dolore the visceral sensory nerves are free from stimulation; there is no pain, but the patient, even if a brave man and one who can look the King of Terrors in the face with composure, feels instinctively, though free from pain, that he is on the brink of dissolution. This is a point at which the two categories of cases most nearly approach one

[1] *Op. cit.*, p. 565 *et seq.*

another. The whole phenomena, however, of angina sine dolore are relaxant—syncopal. The *tertium quid* which determines the presence of *pain* is absent or in abeyance. No theory of cramp could ever have been suggested to explain the phenomena of the majority of cases of angina sine dolore.

The anatomical and other conditions which underlie the two states may, in many cases, have much in common. The age, sex, and circumstances of the patients may be much the same. The cases may touch each other in the matter of atheromatous and calcareous states of the aorta, coronary arteries, and valvular apparatus of the heart, but these resemblances cannot bridge the gulf which lies between them—the gulf of pain in the one case and of no pain in the other. Cases, no doubt, are met with which have evinced the pain of angina at one time or another, and ultimately die in a syncopal manner without such manifestation. These must be placed in the category of Heberden's disease. They possess the underlying condition which provokes the *tertium quid* of pain. This cases of pure angina sine dolore presumably do not. Indeed, had not so revered a teacher as the clinician who coined the term 'angina sine dolore' placed these words in currency, one would have felt inclined to suggest some such term as 'syncope trepidosa,' as expressing more clearly the nature of most, though not perhaps of all, cases of the disorder.

In his lecture upon the 'Allied and Associated Conditions of Angina Pectoris' Sir William Osler[1] includes Parry's syncope anginosa, the Adams-Stokes syndrome, angina sine dolore, and cardiac asthma. The term used by Parry accurately conveys the idea of pain and fainting, but as Osler points out, syncope in this connection is chiefly applicable to the fatal paroxysm, and I would add to the last phase of the fatal paroxysm in non-endocardial cases, which I regard as the syncope following an inhibitory action of the vagus. The phenomena of cardiac asthma, as emphasized when dealing with endocardial angina, may be associated with a syncopal state of the ventricle, and with depletion, not

[1] *Op. cit.*, p. 67 *et seq.*

repletion, of the periphery, but they may also be associated with the highest degree of pain, thoracic and brachio-thoracic. These two classes of cases, then, I should feel disposed to separate sharply from the category of painless angina. The Adams-Stokes syndrome, on the other hand, which is essentially syncopal in nature, appears to me to belong to the same category as the condition now under consideration. The anatomical basis and circumstances of many of these cases closely resemble those associated with cases of angina pectoris.

Of the varieties of angina sine dolore there seem to be three chief classes: (1) Those which evince alike the fears of impending dissolution, and reveal the fact of syncope in the action of the pulse and of the heart. (2) Those which exhibit the mental phenomena in a very marked degree, but which reveal no sign of imminent syncope to physical examination. (3) Those which manifest the persistently slow, and it may be intermittent, pulse of the clinical picture first painted by Adams and Stokes, but in which the syncopal element is more pronounced in acute cases. The chronic bradycardial case may ultimately manifest angina sine dolore, but he may faint into oblivion without either mental distress or pain, and in the absence of both pain and distress must be excluded from the cases we are discussing. Such cases are more appropriately to be considered among disorders of cardiac motion. It is of interest that the revered originator of the term 'angina sine dolore' died of this affection, as chronicled by his friend, Dr. G. A. Gibson, all too soon deceased. In his case, however, the fatal possibilities of the situation were regarded with a patient calmness which destroyed the applicability of the term in his instance.

Those cases which may be placed in the first of these classes are of comparatively common occurrence. Thus I was consulted by a gentleman, sixty years of age, short, stout, a careful liver, and retired from business, but who at one time suffered a great deal from gout. He complained of occasional attacks of what he termed 'breathlessness,' especially if exerting himself in any way, or excited. These

attacks were associated with a sense of weakness and a cold perspiration of the face, and caused him alarm. He stated that he suffered from palpitation during the attack, but experienced no pain. His pulse when I saw him was 78, and regular but feeble; his heart's action also was regular, but the sounds were distant and not forcible. There was no cardiac bruit. The other organs were normal. I prescribed a cardiac stimulant, small doses of calomel and rhubarb, and trinitrin for the attacks, advising him also to drink good and expensive tea as a stimulant rather than alcohol with his meals. He consulted me again a fortnight later, and informed me that his attacks of 'breathlessness' were of shorter duration and of less frequent occurrence; he stated that he felt in every way better, and thanked me especially for advising him to drink the expensive tea which I had named, speaking enthusiastically of it as 'life to him.' He had a house in the country, and left town to reside there. About a month after the date of his last consultation with me I received a note from his medical attendant, who informed me that he had been hastily summoned to him during the supervention of one of his attacks of breathlessness and faintness associated with cold perspiration and a failing pulse, and that, although pain was not complained of, the patient died after having been about two hours in the condition described.

Of that variety of angina sine dolore in which no evidence of syncopal failure can be detected on physical examination, I have never myself met with an example; nor, indeed, have I ever met with angina of any description in which some variation of pulse from its normal condition could not be detected by the finger or registered by the sphygmograph. Heberden probably did not intend to apply absolutely his statement, 'Arteriæ eorum qui in hoc dolore sunt, naturaliter prorsus moventur.' That it cannot be so applied in painful angina we now know full well, but this anomalous condition appears to occur on rare occasions in angina pectoris. It is, I should imagine, equally rare, or nearly equally rare, in angina sine dolore, for trepidation is present here, though no physical pain. Sir T. Clifford Allbutt has well described

one such case in his lectures already mentioned (p. 113). It was that of a gentleman advanced in years, who had the usual signs of senile heart and thickened arteries. 'All at once,' he writes, 'when my examination was nearly over, the patient said in a hollow voice, "It is coming," and his nurse, familiar with the cry, ran forward with restoratives—with brandy or sal volatile. The face was then ashen and terror-stricken, and he was frozen into an attitude of stillness; he did not even disengage his wrist from my finger, which, fortunately, was then resting on his pulse. In spite of smelling-salts and other restoratives of the ordinary kind, he was thus held in deadly apprehension for some minutes; as the seizure began to pass off he whispered to me, "It will kill me." During all this time his pulse, already somewhat hard, never faltered or underwent any change whatever. There were no vaso-motor phenomena. . . . The patient assured me that he had never had the least pain during any of these seizures.'

We know that there are cases of 'cardial' as distinguished from 'extracardial bradycardia,' to use Dehio's expression,[1] cases now regarded as affected by 'heart-block,' which cannot be accelerated either by stimulants or exertion, and it is conceivable that in an analogous condition circumstances which, under more normal conditions, would provoke variation, fail to do so here. But the immobility of pulse in such a case is something apart from its associated angina, whether painful or painless. With these also we shall be more immediately occupied when considering the disorders of cardiac motion.

Of the third class of angina sine dolore—that, namely, in which cardiac failure occurs with a slow pulse which intermits at intervals and is varied by short periods of acceleration only to be followed by another intermission more or less profound—the clinical picture may be a very striking one. It is essentially an acute syncopal ventricular bradycardia.

Some years ago I met with a well-marked instance of this affection. The patient was a tall, stout man, seventy-one years of age, wealthy, and a voluptuary. As a younger man

[1] *St. Petersburger Medicinische Wochenschrift*, 1892, No. 1.

he had been very powerful and a good amateur pugilist. He had eaten and drunk to excess over a considerable portion of his life, but he flattered himself that he had acted rather prudently than otherwise, as he invariably only consumed 'the best,' as he termed it. Although he weighed 22 stones at the time of his death, his arteries at the wrist were soft for his age, their rate and rhythm normal prior to the seizure to be described, and his heart exhibited no abnormal signs. His kidneys were healthy. The attack I am about to record occurred in the month of January. The weather was intensely cold at the time, and the patient had had a mild bronchial attack, but being much depressed by the death of his only son, he had gone to a theatre with his comparatively young second wife to escape from his worry. He had a syncopal attack during the play and was brought home in a cab. I saw him at midnight. He was seated in a chair in his dining-room, quite conscious, with a pallid face and cold and clammy surface. His pulse-rate was 30 and full, and his respiration-rate about the same. I directed him to be laid on the floor and supported by pillows, for the conveyance of 22-stone-weight in a syncopal condition to bed was difficult. At short intervals his pulse failed entirely, and with the complete failure of the pulse at the wrist the patient shouted loudly, feeling his consciousness about to leave him. He never, however, at this stage completely lost consciousness except when he slept for a few minutes. During one such short interval of sleep his pulse suddenly failed, and the patient awoke shouting in alarm as he did in the same circumstances when awake. The pulse during this time behaved as follows: Immediately after complete failure it slowly rose to 24 a minute, remained for a time at 30 a minute, then rose to 42, 54, and even 72 and 90. Every such acceleration was followed by complete failure and the alarmed shouting I have already described. It was a veritable syncope trepidosa. At no time had the patient any cardiac pain. It was an angina sine dolore. I regret that I have no sphygmograms of this case. Under the influence of hypodermic injections of strychnine and strophanthus, and other remedies, including tincture of belladonna, coupled

with the absolute quiescence of recumbency, the syncopal attacks became less frequent, but when they occurred, as they did at intervals, they were associated with the phenomena already described. The patient was under my continuous observation throughout the night, and I left him for an hour at six in the morning. I again saw him at seven o'clock, when I found that he had had no return of syncope during my absence. His pulse-rate was then 36 and regular, and his respiration-rate was 30. Only the first sound of his heart could be heard, which was distant and muffled and associated with a murmurish vibration, but without actual bruit. Between this time and one o'clock in the afternoon of the same day the patient improved considerably, and at the hour mentioned his pulse-rate was 54 and regular, and its impulse so strong to palpation that I felt inclined to advise him to abandon recumbency for a sitting posture, but decided not to do so. He expressed himself as feeling much better. At five in the afternoon he supposed he was out of danger, but I found that his pulse-force was less than at one o'clock. Soon afterwards he had a syncopal attack associated with epileptiform signs, in which he became cyanosed and rigid. This attack passed off, and the patient again became comparatively comfortable, and I left him. Shortly afterwards syncopal failure again took place, and the patient, I was informed, died without any struggle. There was unfortunately no post-mortem examination of the body.

The conclusions which I draw from this study of the anatomy, physiology, pathology, and clinical history of angina pectoris is that, notwithstanding the very able defence of an untenable position, as I regard it, by M. Henri Huchard and those who agree with him, the exclusively arterial view of the malady cannot be maintained. The rôle of the bloodvessels *regarded as hæmaducts has been exaggerated.* The bloodvessel is more than a *hæmaduct.* It is, like the heart itself, a muscular organ, innervated, irrigated, mobile, and capable, like the greater organ, of being disabled in one or more of its triple constituents—its muscle, its blood-supply, and its nerves. It is capable of being the seat of pain chiefly through such destructive processes or

irritations as influence or lay bare its sensitive structures —its nerves—and by the agony of such pain influence indirectly the heart itself. But what is true of the blood-vessels of the heart is likewise true of extracardiac blood-vessels of the aorta, and even of distant organs only in general connection through the nervous system and blood with the heart. Indeed, hepatic colic may very closely resemble angina and kill indirectly, just as neuritic angina from whatever cause may. What is true of the bloodvessel is true also of this muscle of the heart, which has no monopoly n initiating cardiac anguish by the insufficiency of any property assigned to it. Angina pectoris, I therefore maintain, is not one *except in suffering*, but essentially three-fold—muscular, neural, and hæmic. As regards etiology, I hold that it is not a rise of blood-pressure as such which is causative of angina, as a rule, but blood-pressure in many cases, whether propulsive or obstructive, exercising its influence upon local anatomical lesions or disordered physiological states of the heart or its immediate neighbourhood.

CHAPTER V

THE DIAGNOSIS AND PROGNOSIS OF CARDIAC PAIN

Diagnosis.—The diagnosis, in its widest sense, of angina pectoris includes not only the discrimination of varieties of that malady from one another, but that of the syndrome of angina from other conditions which in some measure resemble it, yet affect quite other structures than those involved in Heberden's disease. For this general diagnosis three chief points have to be borne in mind and usually serve to exclude ailments which can only be mistaken for angina when their indications are imperfectly regarded. These guiding points are—(1) The situation of the pain; (2) the character of the consequences of the pain; and (3) the fact as to whether or not the act of respiration, especially in its voluntarily exaggerated execution, is in any way impeded or tends to influence the degree of pain. The site of pain in all varieties of angina pectoris is, in almost all instances, chiefly in the anatomical situation with reference to the surface of those structures, lesions of which we have found to be associated with Heberden's disease. That site is essentially sternal or retro-sternal, and most commonly some point between the level of the third rib cartilages and the ensiform process. Cardiac pain is practically central and in the situation indicated. It is more rarely epigastric or over the manubrium sterni. The patient in an attack when asked to place his hand over the seat of pain lays it over the centre of the sternum, and only in the second place describes the brachio-thoracic or other direction of radiation, if such be present. This fact serves to distinguish angina

almost always from subdiaphragmatic pain, whether of a functional or organic character, in the higher abdominal viscera. As I have already stated, a very plausible argument may be advanced in favour of an œsophageal or gastric origin of Heberden's disease (p. 56). But clinical history, anatomical examination, and the results obtained by remedial agents indicate a cardiac origin for Heberden's angina pectoris, which might more unequivocally be termed 'angina cordis.'

With the character of the consequences of cardiac pain we have already dealt at length in discussing the clinical history of the disease. While the general collapse which is observed as a consequence of hepatic, renal, or other severe pain may bear some resemblance to that of the later stages of an anginal attack, the characteristic radiation of the pain in cardiac cases usually serves to indicate on well-known anatomical lines the source or seat of the original stimulation. Collapse due to non-cardiac pain, moreover, is usually of later supervention than when due to angina pectoris. The only exception to this rule is, as we have seen, in the case of thrombotic coronary angina, the conditions of which bear a close analogy to renal and hepatic colic.

The last point—namely, the freedom of respiration and especially the unimpeded character of forced or voluntary respiration—serves to distinguish between cardiac and other thoracic pains, whether direct or reflex, which, by affecting the intercostal, diaphragmatic, or other muscles exercised in forced respiration, indicate the structures involved. 'From the disease itself,' as Heberden remarks, 'those who are affected experience no difficulty in breathing, by which circumstance this breast-pang is chiefly distinguished.'[1] The patient's respiration, like his pulse, may, indeed, be temporarily arrested while in the agony of angina, but if he be asked to take a deep breath he can do so without impediment to respiratory movement or increase of pain. These considerations, borne well in mind, will usually serve to guide us aright in the diagnosis of angina pectoris from pain affecting structures other than the heart. Mistakes

[1] 'Commentarii,' p. 309.

will doubtless occur and exceptional and perplexing cases be met with even when these points are remembered, but such exceptions only serve to prove the value of what is a practical and safe clinical guide in this important matter.

By the more particular diagnosis of angina pectoris I mean, not only the discrimination of so-called 'true' from 'false' angina, but the distinguishing of varieties of true angina from one another. If the first task be at times difficult, and even when every precaution to come to a right decision has been taken results occasionally in error, how much more frequently must this be the case when we essay the second! There are cases of 'true' angina to witness the phenomena of which is to have them indelibly fixed in memory, and on the same conditions presenting themselves again they lead to the recognition of that state with promptitude. The clinical histories of the varieties of angina which I have already related contain examples of such. But there are other cases which so closely, in some particulars, resemble those I have mentioned, that it is only by a close consideration of details that we can detect where the resemblance ceases. Even after every care has been taken, however, and we have formed, it may be, a comparatively favourable prognosis, error may occur. As I have already contended, 'true' and 'false' are unfortunate terms to apply to angina unless we have a clear notion of what we imply by their use. If we mean by the former to designate cases which always threaten, and, in most cases, sooner or later destroy life, while by the latter we denote cases which, whatever the degree of distress they exhibit, very rarely or never kill, we may be justified for convenience' sake in making this distinction. As, however, the pain of a fatal angina may not to all appearance be so excruciating as that in cases which recur frequently and disappear apparently for lengthened periods or altogether—although the fact of suffering, of angina, is indisputable and real in both cases—its mere degree cannot be taken as a measure of its seriousness.

The criteria which distinguish cases which are dangerous from those which are not are only to be gathered from a comprehensive view of the circumstances of each case—a

view which comprises the whole of the clinical picture and properly appraises the diagnostic value of special points or details. A margin for error must be left even then, for there are probably cases of which we may say with the poet, 'There is no name for that of which she died.' It is, however, probable that the *sex* of the deceased to whom the late Poet Laureate applied these words in his Human Tragedy would exclude angina pectoris. I say this is probable, for a comparatively small number of women undoubtedly die from an angina which may long have been regarded as a mere manifestation of a troublesome dyspepsia. Sex, however, is an important factor in this relation, and of two patients, one a man and the other a woman, who manifest symptoms of angina, the former will with much greater frequency be found to harbour the fatal variety of the affection.

Although cases of fatal angina in early life have been recorded, and even atheroma has been demonstrated on occasion in the 'teens' or earlier, if we exclude the consequences of syphilis and the general debauchery to which man is at times addicted in earlier life, Heberden's indication of the fiftieth year, the average period of the commencement of atheromatous wear and tear of the vessels, may be regarded as a valuable element in the diagnosis of cases which manifest cardiac pain. At a still later period, say between sixty and seventy years of age, to pronounce a man sound at heart and to prescribe gymnastics, wood-cutting, and so forth, because of a well-preserved softness of radial pulse and the absence of detectable cardiac lesion is, in many cases, to court the disaster which overtook one of the cases the history of which I have related. To be over fifty years of age, then, and to evince angina is to suggest, though not to prove, the presence of the dangerous kind.

Heredity, again, has a certain influence and has to be reckoned as a factor in diagnosis. The father of my patient who died from musculo-spastic angina died similarly suddenly when about to leave his house to conduct a religious service, for he was a clergyman. The anginous family history of the Arnolds is common knowledge, and I have met with a fatal case, the father and several brothers of whom I was

informed had likewise died anginous. When the neurotic disposition is unassociated with a proneness to vascular degeneration, in the production of which gout is a frequent factor, angina is more likely to be less dangerous and more transient than when these dispositions are combined, as they frequently are in cultured families and in those who exercise intellectual professions. Woman, whose frequently much-enduring but more refined constitution than that of man makes her more often than the latter the subject of the less dangerous variety, is also an illustration of this fact. In the death-grip of true angina, moreover, the patient is usually stilled into quietude by his agony, and feels that his summons to pass into the unknown has come. In the agony of so-called 'false' angina he frequently, indeed, fears that death is imminent; but he manifests more excitement, and energetically endeavours to break loose from the grasp that threatens to detain him. Yet it must be confessed that these general impressions, although they have a certain value, are not altogether trustworthy, and the man who off-hand, from such considerations, pronounces definitely for or against the presence of the dangerous or innocuous variety, as the case may be, may find himself sooner or later admonished by the event that wisdom lies in exercising the greatest caution in expressing too positive an opinion. Given a comparatively young subject with normal radial arteries, be they tense or otherwise, free from local heart affection or other organic disease, and without the history of that ubiquitous agent for vascular evil—syphilis—we shall in most cases be justified in looking for a transient cause of angina and the disappearance of the effects of such. On the other hand, given a man (or it may be a woman) past the meridian of life, or with the evidence of local disease, or having an indubitable specific history, we may be fairly certain that Death has not only beckoned the patient, but will in many cases return at no distant date and insist upon his following him.

Admitting, however, that we are in certain cases concerned with angina vera or the lethal type, is it possible to determine in any way the particular variety of the fatal

malady with which we have to deal? For example, is the case before us one of coronary calcification with neuritis, or it may be aneurism, or is it one of musculo-spastic angina? In considering the point, I am aware that I am treading upon altogether disputable ground, and desire to avoid any appearance of a presumptuous dogmatism; but, nevertheless, I wish to point out some considerations which appear to have weight. The age of two such patients may not help us very much, but it is a point not to be dismissed hastily. Taking fifty years as the average age for commencing atheroma in normal circumstances—the period at which the normal wear and tear of life begin to tell on the blood-vessels—it is unquestionable that those cases which ultimately reveal calcification of the coronaries may exhibit the first evidence of angina considerably earlier. It is unnecessary to refer at length to the classical case of John Hunter, the clinical details of which have come down to us from Sir Everard Home, and have been studied with care and rendered still more current knowledge by Sir William Gairdner. Born in 1729, he had the first attack of gout in 1769, his first angina in 1773, his second attack in 1776, and a more continuous status anginosus was established in 1785, which ended in death in an attack in 1793, when he was sixty-four years of age. My own patient, whose case I have related, died at the age of fifty-four years, after having suffered from angina for seven years, but with progressive frequency in the latter portion of that period, and also died during a paroxysm. During the ten years prior to his death he had four times suffered from gout in the great toe. Both these cases, therefore, must early have manifested coronary atheroma, and probably, likewise early, the calcareous change in the vessels. In the case of my own patient I explain the earlier symptoms by neuritis, and the later and more continuous by neuritis and aneurism. Time alone can show whether this conclusion, which is probably correct for the case in question, is of more general application.

The sufferer from musculo-spastic angina may quite possibly be affected as early as the patient from coronary neuritic angina; but in all probability, in such a case, some

sufficient cause of muscular exhaustion will be found in the events immediately antecedent to the attacks which may point to the muscular factor playing the major rôle in the tragedy. The arthritic history may likewise be absent, although not necessarily. But, questionable as these criteria may be, I think there is a less doubtful value to be attached to the general type of the sufferer. The canvas of Sir Joshua Reynolds, painted in 1787, and Sharp's fine engraving made at the same date, after repeated attacks of the malady had probably borne in upon the mind of the great student the true significance of his pain, have brought down to us the keen, alert, and cogitative face of Hunter, associated with a body naturally powerful and, although short, certainly not stout. Those, on the other hand, who possess the heart most prone to musculo-spastic angina—the fatty heart—usually exhibit a general *embonpoint*. They are usually plump of face and stout in body—a stoutness in which the internal organs participate. Till now we have had to be content with such general impressions as these for the purposes of diagnosis. But are we to-day at the end of our resources when we have considered these? The fortunate accident which threw the shadow of the bones of Roentgen's hand on to the photographic plate—and how frequently the enlightening incident is an accident—seems to point to a possible solution of the difficulty on more reliable lines.

In the heart containing calcified coronaries after its removal from the body radiography easily demonstrates this to be present in the course of the affected vessels; but in a mobile organ such as the heart the detection of this condition by this means during life is at present impossible, even by such an arrangement of the apparatus as to avoid, so far as possible, the intervention of opaque structures. But a radiographic examination of other vessels, such as the tibial or any other arteries easily accessible to the rays, seems desirable in cases in which a calcification of the coronaries is suspected. The inference from such vessels to those of the heart is not positive, any more than a coronary calcification need necessarily be associated with a similar state in other vessels, but the detection of a peripheral or

aortic calcification would render the presence of the same state in the coronaries probable. Just as we examine the tension and character of the pulse by palpation for diagnostic purposes, so also by skiagraphy we may determine whether or not the general arterial change in a patient is associated with calcareous deposition. Thus, by an argument from the general to the particular, we may attain to a reasonable estimate of the condition of the coronary arteries of the heart, even though our conclusions may not rise to absolute certainty. Dr. Williams[1] also states that both by radioscopy and radiography calcification of portions of the ascending aorta may be detected.

The symptoms of *acute aortitis*, as we have learned from the clinical history of the condition, are sufficiently diagnostic, and, taken in connection with the comparative youth of the sufferers and the frequent history of specific infection, enable us to distinguish with some confidence this form of angina from others. Even in its more chronic phase, youth, syphilis and the locality of pain serve to guide us in coming to a conclusion. In those cases of angina associated with aneurism of the aorta, when other well-known criteria of this condition fail us, Roentgen radiography is invaluable. Indeed, in no sphere in which it has been employed has its value been more generally acknowledged than in this.

Wassermann's blood-test, reliably made, for the detection of syphilis, may be helpful. By this means I have found positive evidence of the syphilitic state twenty years after the original infection in a case of angina pectoris.

Neuritis being a concomitant of other conditions, its recognition will be regulated by the diagnosis of those states with which it is associated, but the determination of its intra- or extra-vascular seat must at present be left to the pathologist.

The guide to the discovery of endocardial angina is the presence of valvular lesions, and intracardiac pressure is only one of the processes which plays a part in these cases. That it plays a part is probable, for the reasons already given, but its influence has been too exclusively considered.

[1] 'The Roentgen Rays in Medicine and Surgery,' p. 385. New York, 1901.

Even in the dextral valvular case I have mentioned there was an endarteritis at the commencement of the pulmonary artery which cannot be ignored as a factor in the production of pain, under the flapping stroke of the large vegetations which were attached to the valves. That the pulmonary artery, like every other artery in the body, is innervated, I satisfied myself when preparing a course of lectures on the relation of the nervous system to disease and disorder in the viscera.[1]

The syndrome of angina, coupled with various forms of peripheral dysæsthesia, such as coldness of the hands and feet, disturbances of sensibility generally, and at times of motion, unassociated with valvular disease and without evidence in cardiac muscle or in bloodvessel of any of those persistent changes which we have seen to be associated with the graver varieties of the complaint, is in all probability purely vaso-motor and of favourable augury. But a peripheral hypertonus, to use a term employed by Dr. William Russell,[2] due to toxic causes, intestinal or other, and associated with a persistently high pressure with evidence of cardiac hypertrophy, and, it may be, albuminuria, in such cases, even when the pectoral discomfort is not great, will indicate a guarded prognosis.

That most cases of angina pectoris are of *compound*, not simple, origin has been already argued, and the discrimination of one variety from another is largely merely a matter of emphasizing what may appear to us to be the 'predominant partner' among several factors. The absence of pain and the syncopal signs in a large number of cases of angina sine dolore will serve to distinguish this state from Heberden's disease. The erethism engendered by the hypermyotic heart or cor bovinum is, I have shown on occasion, a leading factor in the provocation of angina in aortic valvular disease, and the suprasigmoid distension of the aorta on the lines of Sir T. Clifford Allbutt's suggestion must not be lost sight of as an etiological factor.

Prognosis. — If we regard angina pectoris as one and indivisible, our prognostication of the probable issue of any

[1] Edinburgh: Pentland, 1899. [2] 'Arterial Hypertonus,' etc., 1907.

particular case will be based on such a mass of dissimilar data that our prophecy, always uncertain in angina vera, to use the old collective term, must of necessity lack even a rational degree of precision. As I have endeavoured in the preceding pages to classify provisionally the varieties of angina, both organic and functional—to employ terms having a conventional meaning—I shall consider as shortly as possible the prognosis of angina on those lines, even although the future to which we must all bow should pass a sponge over the whole slate which I have scored.

If we have reason to suppose that a given case is the subject of coronary vascular angina, neuritic or aneurismal, or both, how can we hold out hope of recovery from it or any permanent amelioration of its symptoms? The process we are dealing with we know to be chronic and progressive. There is a curious rapidity in malign progress in the later phases of the disorder, as though a fresh aggravating element had been introduced. This may or may not be aneurismal in character. That it probably sometimes is will not, I suppose, be questioned in view of the facts I have related. In the earlier and less urgent phase of this variety, then, we may guardedly, and viewing the patient as a whole, and not as a coronary artery, hold out the hope, under judicious management, of a considerable length of life, even though we cannot remove the sword of Damocles to a point where it may harmlessly snap the thread which suspends it. But when the progressive degeneration of the vessel has called into painful activity the slumbering sensibility of the coronary vascular nervous system, it will require little foresight to predict a fatal issue in the near future, whether the patient himself be made the participant of this belief or no. It is rarely that the physician is so fortunate as Dr. Bucknill was, who had as a patient Thomas Arnold of Rugby. 'He next asked,' writes Dr. Bucknill, 'if the disease was generally fatal. I said generally (for those who knew him were aware that it was impossible not to tell him the truth).' Thomas Arnold, however, had not, as we know, calcification of the coronaries, but probably fatty degeneration of the heart, for its external surface is reported to have

been healthy, its walls thin, and the colour of the muscle pale and brown. He died in his first attack of what, according to the classification I have attempted, would be called 'musculo-spastic angina.'

What is the prognosis in this variety? A man, unconscious of any failure of power, strenuously occupied bodily and mentally, is suddenly attacked by angina, and the heart, overwhelmed by pain, ceases to beat, and life is extinct. Is there any reason why, if a patient recover from the actual paroxysm of such an attack, the heart-muscle, which did its work so well the day prior to his death, should not continue to do it equally well if he survive the attack, provided the danger-signal raised by such an event be borne in mind? We know that death in a first attack of angina is not common, whatever the underlying organic cause of the affection, but fulminant cases are *usually* associated with a state of cardiac muscle which precludes what Latham describes as the 'strenuous exercise' to which 'T. A.' was addicted, and the continued life of such a patient must be contingent, if he survive the paroxysm, upon the abandonment of such. Any prophecy as to its duration must depend upon the impression made upon the physician of the degree of underlying cardiac degeneration which the attack has revealed. There is more hope for the degenerated heart of a fat man, speaking generally, than for the degenerated heart of a thin man. In the case of the former we shall with greater probability have to deal with fatty infiltration and a possibly healthier state of the muscular fibres of the heart than in the latter, in whom the muscle itself is more probably affected with fatty degeneration. In one such case of apparently true angina in which the aura was brachial and the cardiac pain severe, diet and rest for a time with cardiac tonics so relieved a hunting man forty-eight years of age that he resumed his violent equestrian exercise without any return of symptoms. When I last accidentally met him, he was carrying a heavy portmanteau at a railway-station with comfort.

The prognosis of acute syphilitic aortitis must be dependent upon the extent of the affection. An extensive inflammation

of the aorta associated with angina may kill, whatever its cause. The acute phase of this condition may be regarded in some sense as we do the paroxysm of angina in the cases we have already considered. Once safely over the acute stage of such an aortitis, the patient, especially if his condition be rationally referable to syphilitic infection, frequently responds well to specific treatment. The angina, which has been the plague of the patient and the despair of the physician, often ceases to trouble, to the gratification of both, when antisyphilitic treatment is prescribed, and a favourable prognosis is accordingly justified in such a case. We must also remember that extensive specific atheroma of the aorta may be met with in which there is no history of persistent angina, and in which the angina, when present, has not been the fatal factor. The prognosis of aortic aneurismal angina is the prognosis of aneurism, but as regards the anginous element in acute aneurismal cases I have already given reasons for believing that pain is more characteristic of the intra-arterial than of the extra-arterial stage of its growth. The prognosis of neuritic angina has also already been considered. It is the prognosis of the probable effects of the neuritis in the situation in which it occurs, be it coronary, aortic, or extravascular. Inasmuch as the diagnosis of neuralgic as distinguished from neuritic angina is frequently established by the recovery of the patient, it is scarcely necessary to add that its prognosis is favourable. This is also true of so-called vaso-motor angina and of anginæ reasonably referable to introduced agents, such as poisons, tobacco or other. 'Cessante causâ cessat et effectus.' As I have stated, however, the presence of secondary cardio-vascular changes in vaso-motor angina will modify prognosis.

The severe cardiac pain associated with valvular lesions, and particularly as has been stated with aortic lesions, being probably due to the disordered action of the organ in a condition of non-compensation or of lost compensation, or disordered tone and contractility, whichever phrase be most acceptable, and more precisely to a sudden systolic distension of the base of the aorta and of the coronary vessels, or to a

sudden distension of the chambers, it is to be expected that on the attainment or restoration of compensation the anginal symptoms will diminish or pass off. This, the history of many of these cases justifies us in predicting. In using the term 'distension' in this relation I do not mean spasm, like Heberden, if he be correctly believed to have used *distentio* in this sense, but pain from stretching. In this connection it is of interest to note that while valvular heart disease is, as we know, very common in children, the vaso-motor phenomena associated with these lesions which we so frequently see in older subjects are among children very rare. That they occasionally occur is probable, but I confess I have never witnessed a case, say, in children under twelve or fourteen years of age. Heberden states that he met with angina in a child aged twelve years. This was probably an endocarditic case. The comparative immunity of children from this symptom seems to argue their escape as being due to the greater elasticity of young tissues, and is thus an argument in favour of the view I have expressed as to its rationale in older subjects. The prognosis as to pain, then, in most of these cases is probably to be guided by the prospect of the establishment or restoration of a due balance in the circulation by improved compensatory growth of the organ. In hypertrophic cases, as I shall show, surgical procedure by thoracostomy may be beneficial. The prognosis in compound cases, a class which probably includes the majority, must be governed by the prospects of that factor which is assumed or believed to play the major rôle. Angina sine dolore being essentially due to cardiac muscular failure, the prognosis in this type of the affection is of the gravest. It is a musculo-paretic, not a musculo-spastic, condition; hence, possibly, the absence of pain. The patient is less at the mercy of an accident such as pain, but the cardiac muscle itself is near the termination of its vital possibilities.

We have now discussed the nature and causes of cardiac pain with the imperfection which is, in the present state of our knowledge, unavoidable. I fear, indeed, that I may have appeared to speak on some points with an emphasis unwarranted by the facts before us, but my action has been

dictated by the belief that it is by seeking an organic basis, not of one but of several kinds, that we are likely to obtain precise knowledge of a subject which has been largely dealt with as an appalling symptom, and for the explanation of which some authors offer us only one key. We shall find in considering the treatment of angina pectoris that we possess no panacea. It is, I believe, equally true that for the explanation of the malady we possess no single key; but that there are groups of cases the nature of which, however imperfectly explicable at present, will in time by patient, systematic, histological investigation be explained on more than one hypothesis; for pathological histology, at present unduly depreciated in some quarters, is the anatomy of structural disease.

CHAPTER VI

TREATMENT OF CARDIAC PAIN

If the life be more than meat and the body than raiment, it is likewise true that the possession of life and its employment are more than a knowledge of its mechanism. Notwithstanding instances of disaster to individuals, it is on the whole well that caution and prudence, as regards the preservation and management of life, have not characterized mankind as a race. Many a heroic deed, many a strenuous life lost in the endeavour to reach a high ideal, would have been left unrecorded if the hero, possibly an invalid, had known and endeavoured to obey the dictates of physical prudence which such wisdom would have inculcated, and had not with both hands thrown away life for something better than life. John Hunter, who knew the serious nature of his malady, did much of his best work after angina pectoris had marked him for its own. But with such general considerations, which may be safely left to take care of themselves, like the rising and the setting of the sun, for men of that mould *must* do their work, the physician who has before him a patient who has, or may have, angina pectoris, is in no way concerned. The bond between him and his patient is not the potential heroism of the latter, but the possibility of his demise, if the dictates of physical wisdom be not obeyed, and in many cases of angina, unfortunately, whether they be obeyed or disregarded.

Let us consider first the case of the patient who *may* develop angina pectoris. He is probably a man past the meridian of life, either spare, wiry, and it may be with a history of gout, or stout, aging, and perhaps with some

history of occasional circulatory disturbance such as giddiness or breathlessness on exertion. They have both probably consulted us about some quite other matter—dyspepsia or what not—and they have both, also probably, a degree of arterial atheroma even though the radials at the wrists or the more accessible arteries should not afford an indication to this effect on palpation, digital or mechanical. To auscultation their hearts may be perfectly sound. Let us beware in such circumstances, when prescribing rules for the general conduct of their lives, dietetically or otherwise, not to allow them to go away with the impression that there is no particular need for caution in the general expenditure of their energy. The man fifty years of age who wishes to reach seventy years without the possible introduction of an angina into his life has to adopt an old Scottish motto, 'Gang warily.' The failure to inculcate this advice as regards physical exercise, especially if unwonted restrictions be placed upon the patient's diet at the same time, is, I believe, a fruitful cause of some of the fatal attacks of angina we meet with. As a corollary to this conclusion it may be added that caution in the resumption of activity, especially in those of atheromatous age, during convalescence from debilitating ailments, influenza and other, is of the first importance in this connection. This is especially necessary if such patients should ever have manifested any indication either of angina cum dolore or of angina sine dolore. A little more patience and caution exercised in this respect, and many a valuable life might have been prolonged for a shorter or longer period, to the advantage alike of individuals and of those dependent upon them. If we inculcate such precautions in the case of one who appears to us to be a candidate for angina, how much more will it be our duty to do so in the case of those who have actually experienced an attack? We must in such cases not only indicate a suitable dietary and inexorably limit the exertion of the patient to such an amount as his own experience teaches him is safe, but also point out to him the danger of emotional excitement and the comparative safety of a calm and equable state of mind. The desirability of the attainment of this calm may be

indicated by the physician; the world is too old for it to be necessary to add that every man must build that inexpugnable citadel of his existence for himself.

Into the details of the dietary of these cases I need not enter fully, because they must be regulated according to the type of angina we have to deal with. We may have to advise one man to become a vegetarian, and another to eat meat; but a strict moderation as regards the quantity both of solids and fluids must be prescribed, as we are dealing with a condition very sensitive to fluctuation in blood-pressure and to the work imposed upon the heart. For this reason, also, the clothing and climatic surroundings of the patient have to be regulated. Those who in their physical misfortune have the consolations of wealth should avoid the rigours and changes of an unsettled climate by living during the more severe weather in more favoured portions of the globe. Those who cannot do so must, so far as possible, by loose, warm clothing, and especially sufficient warmth in bed, endeavour to create that summer in their surroundings the attainment of which elsewhere their circumstances do not permit them to seek.

As a transitional stage to the consideration of medical and surgical methods, invaluable in some phases of the disease and useful in others, let us shortly deal with the question of baths and exercises. In connection with angina pectoris, the differences in the operation of these two processes must be borne clearly in mind. Long known and universally employed as has been medical bathing in this connection, it is to Nauheim that attention has been chiefly directed since Beneke wrote on the effects of hot salt-water bathing on chronic heart disease. His writings on the subject appeared at intervals between 1859 and 1875, and they are those of a cautious and candid observer. He states, in a pamphlet published in 1872,[1] that while many of his cases improved after a course of treatment, there was little immediate effect observed upon the action of the heart and on the character of the pulse. This observation by Beneke agrees with

1 'Zur Therapie des Gelenk-Rheumatismus und der mit ihm verbundenen Herzkrankheiten'

my own experience as regards cautiously employed warm baths.

It is reported of Lord Beaconsfield that, on being asked by an ambassador of his party, who had received an invitation to dine with a political club of the Opposition, whether he might accept the invitation, the Premier answered: 'Why not? A man must dine somewhere.' To us sons of the Norse water-dogs, who still smell the brine dashed from the prows of their warships in the faces of our Viking ancestors, it is at least as desirable that a man should bathe somewhere. Whether he dine or bathe, so long as he do so prudently, the effect is calculated to be beneficial, provided he be not too much exhausted either to eat or to wash. If, when sufficiently able to do either, he do both regularly and systematically for a month or so, and thereafter rusticate in a pleasant atmosphere at a moderate altitude, and take gentle exercise for an additional period far from the madding crowd and the harass of his calling, it would require a very pessimistic person to prognosticate that he would not return refreshed from his holiday to face the duties of life. Such a course of treatment is not calculated to be detrimental to any circulatory disturbance whatever, or to any other form of physical debility. In a case of the type in which sclerotic angina frequently occurs, and which I examined at Nauheim in 1896, as may be read in my book on 'Cardiac Failure and its Treatment,'[1] I found that the vascular changes induced by recumbency in a Nauheim bath differed little from those induced by recumbency on a Nauheim sofa, for the patient visited me at my hotel on the same day upon which I examined him in his bath. I do not wish to imply by this statement that we cannot, by manipulating the temperature and contents of a medicinal bath, powerfully influence the circulation, but merely that a bath of moderate temperature and of not too irritating constitution, such as is usually employed, is rarely detrimental to any kind of circulatory disorder. This conclusion on this point is upheld by the practice at Nauheim to-day, for whereas at one time practitioners there considered that the baths were not suited to

[1] Rebman, London, 1897, p. 129.

arterio-sclerotic conditions, they now believe that with circumspection they may in such cases be used without injury to patients. In short, given the presence of organic angina pectoris, the bath which will not injure the patient may be harmless, but cannot be rationally expected to be beneficial. On the other hand, in some temporarily enfeebled myocardia associated with angina, the continuous use of saline and effervescent baths, with attention to general hygiene and carefully regulated exercises, may prove useful.

But exercises, whether mechanically or manually administered, and which are at times of much service in appropriate cases of cardiac debility or disordered compensation in valvular diseases of the heart, must be specially considered in relation to cases of organic angina pectoris. Of these methods there can be little doubt that the Swedish resisted movements, systematized and regulated in their application to cardiac cases by the late Dr. August Schott, are the safest and best. Be they, however, never so safe and never so good in suitable cases, the increase in the force of cardiac systole which results from their employment places them entirely out of court in the treatment of any but the most innocuous forms of angina pectoris. Remembering the possibility of the occurrence in coronary angina of such an aneurism as I have demonstrated, I ask whether any rational being would advise any resistance movements in such cases with the knowledge that such a lesion may underlie the manifestation of pain. I place no mark of interrogation after this sentence, for it requires no answer. An occasional anginous attack in one probably the subject of fatty heart may not perhaps be capable of being placed in the same category as those I have referred to, but the advisability of either baths or exercises even in such a case, provided there be evidence of the attacks having been indicative of an organic basis to the symptom, is highly questionable.

If, on account of a possible aneurism in connection with angina, exercises of the kind mentioned be contra-indicated, what is to be said of complete rest? We know that angina may occasionally be a troublesome symptom in aneurism of the aorta; we know also that absolute rest, especially if

associated with a reduction of the quantity of the food taken, on the lines practised by Tufnell of Dublin, has resulted in amelioration of the symptoms due to aortic aneurism. Would these results indicate the employment of a similar line of treatment in cases of coronary angina, even if we had the means of determining positively that the physical basis of the disease was aneurismal? There is no doubt that complete rest is less frequently associated with attacks of the pain than activity. The conditions of the blood-pressure, therefore, in this state are more equable than during movement. On the other hand, the patient, possibly a gouty man, and but for his attack a healthy man, condemned to his couch for an indefinite period, would be quite likely in his enforced inactivity to manifest signs of his constitutional taint. If we grant that with careful supervision, and a restriction of food to the necessities of his body, such a disturbance as an attack of gout be avoidable, could an attempt to induce coagulation in the very small sac of an internal coronary aneurism be brought about without the greater evil of a coronary thrombosis? In discussing the prognosis of angina pectoris, Sir William Osler[1] states that 'recovery is quite possible, and there are instances in which the attacks disappear entirely'; but, unfortunately, the instance he mentions was but an angina symptomatic of lost compensation in aortic valvular disease. That such and other cases having an enfeebled myocardium without valvular disease recover with the restoration of cardiac power we know. I am not aware, however, that there is any record of the recovery of a case of coronary angina, even though it may have been slow in development and compatible with life for a lengthened period. It is theoretically possible that a small and even dissecting internal coronary aneurism might so thicken and shrink as to become practically obliterated, with or without blood-coagulation in its interior; but such a case has to be found before this supposition can be substantiated. That it has not been found may perhaps be due to its not having hitherto been sought for or suspected to exist. I have urged these theoretical objections against complete

[1] *Op. cit.*, p. 139.

rest in coronary angina that I might not appear to lose sight of the collateral evils which may attend such a course of treatment. But were the dangers of coronary thrombosis in the circumstances very great, they might be equally urged against the treatment of aortic or of any other aneurism by this method, for coronary disease might be coincident with these, and the occurrence of coronary thrombosis would probably bring about the death of the patient. If, therefore, much judgment would be required in instituting treatment by complete rest in the early stages of a coronary angina, there need be no hesitation in asserting that in its later phases, as indicated by an increase in the frequency of the paroxysms, rest is the only rational treatment of the disease. It is calculated to postpone as long as may be the final scene, and it is not impossible that by some fortunate accident it might induce greater defensive powers in the artery which might cure the patient. The possibility of such a cure we may admit: the prospect of it we are not at present justified in holding out to our patient. The fear of inducing thrombosis by recumbency in the coronary arteries need not, however, deter us from instituting such a treatment; for, as Dr. G. W. Balfour has pointed out, the coagulation of blood, even in aneurismal sacs of large size, is not easily brought about during life, and improvement, when it occurs under rest and the use of iodide of potassium, appears to be induced rather by a thickening and shrinking of the walls of the aneurism than from coagulation within it.[1] In the internal coronary aneurism I have shown the blood-coagulation in the lumen of the artery and that in the sac were evidently both of about the same age, and probably both occurred either in the act of death or very shortly before it.

The Drug Treatment of Angina Pectoris is divisible into (1) the management of the paroxysm of cardiac pain, and (2) the treatment of the patient in the intervals between attacks.

1. **The Treatment of the Paroxysm.**—The mine once laid, be it in coronary, cardiac, or aortic lesion, the spark necessary to its explosion is, in a large number of cases,

[1] 'Lectures on Diseases of the Heart,' p. 467.

exaggerated blood-pressure, whether direct and propulsive from the heart, or indirect and obstructive from the peripheral arterial system. This is probably the case in all save the musculo-spastic and musculo-paretic or syncopal cases. It follows that in most cases the regulation and moderation of blood-pressure are the primary objects which the physician has in view when he prescribes a medicinal agent to remove the injurious effects of excessive vascular pressure. Concurrently with these it may be also necessary on occasion that he should employ more direct analgesics, and in the final stage, if need be, one form or another of artificial respiration or cardiac stimulation, to which reference may now be made. Cardiac failure in angina pectoris being primary or due to vagal inhibition, as has been maintained, the employment of Laborde's method of rhythmical tongue traction[1] in combination with Silvester's method of artificial respiration is indicated. In the profound and prolonged syncope observed in the status epilepticus I have repeatedly found Laborde's method apparently very efficacious. Laborde's own explanation of the *rationale* of this procedure seems sufficient. He attributes its efficacy in great measure to the stimulation by tongue traction of the lingual fibres of the superior laryngeal branch of the vagus, and to the cardiac and respiratory reflexes thus provoked. The combination of artificial respiration with tongue traction, which might be termed the 'Silvester-Laborde' method, and for which the co-operation of two persons is necessary, would most completely fulfil the indications in anginal syncope. But the direct heart-stab of angina pectoris which has reached the final stage of cardiac inhibition will probably prove more hopeless of recovery than syncope from other causes. There is no valid reason, however, why perseverance should not occasionally be rewarded in these circumstances as well as in others in which such persistent efforts to restore animation have been successful.

The most powerful influence in counteracting the paroxysm of angina pectoris must be ascribed to the *nitrites*. Nitrite

[1] 'Les Tractions Rhythmées de la Langue,' etc., par J. V. Laborde. Paris, 1894.

of amyl, the vaso-motor influence of which was originally determined by Guthrie in 1859,[1] was first used in the treatment of this disorder by Lauder Brunton in 1866. Whatever difference of opinion may be entertained regarding the physiological mechanism of angina pectoris, there can be none concerning the frequent efficacy of the nitrites in relieving cardiac pain. In 1879 Murrell[2] enriched our therapeutic armoury by using nitro-glycerine in the treatment of angina pectoris, and Matthew Hay in 1883 used nitrite of sodium for the same purpose. One of the best general accounts of 'the pharmacological action and therapeutic uses of the nitrites and allied compounds' which has appeared in the English language is that given in his Croonian Lectures by the late Dr. D. J. Leech,[3] of Manchester. The scope of my present remarks precludes a digression into the very interesting pharmacological and physiological phenomena revealed by a study of the effects upon the circulation of the nitrite group of medicinal agents, and I cannot do better than refer those who wish to peruse a masterly treatment of the whole subject to the lectures of the late Dr. Leech, an observer whom medicine could ill spare. There are, however, some points in connection with the comparative celerity with which these drugs act and the length of time for which their effects last which are of practical moment in the treatment of angina pectoris. These points have been well summarized by Dr. Leech[4] in the following words: 'Changes in arterial tension are not often indicated by subjective phenomena, and their extent and duration can only be measured by the sphygmograph. With this instrument I have tried by means of tracings taken frequently to estimate the time at which it reaches its lowest point and its duration. The influence of amyl nitrite on the pulse commences a few seconds after inhalation (Fig. 18).

[1] *Quarterly Journal of the Chemical Society*, London, 1859.

[2] *Lancet*, January 18 (p. 80), January 25 (p. 113), February 1 (p. 151), and February 15 (p. 225), 1879; and 'Nitro-glycerine in the Treatment of Angina Pectoris,' London, 1882.

[3] *Ibid.*, June 24 (p. 1499), July 1 (p. 3), July 8 (p. 76), July 15 (p. 123), and July 22 (p. 177), 1893.

[4] *Ibid.*, July 1, 1893, p. 3.

The tension is reduced to its lowest point in from fifty to sixty seconds, and remains extremely low for thirty or forty seconds, the pulse-waves being reduced in size and sometimes irregular. Then it rises again, occasionally suddenly, and in from a minute and a half to two minutes the pulse is only a little lower in tension than it was before the inhalation. The slight lowering may continue for several minutes. Isobutyl and isopropyl nitrites when inhaled act almost like amyl nitrite. Sir B. W. Richardson, to whom we are so much indebted for knowledge concerning amyl nitrite, has drawn attention to the fact that when it is taken internally its effects are much slighter, though more prolonged, than when inhaled. Instead of the tension falling at once and recovering in two minutes, it falls gradually for usually twenty to twenty-five minutes, remains low a short time, and then rises to its normal height an hour to an hour and a half after the dose has been taken. Because amyl nitrite is such a powerful agent when inhaled, 1 drop is regarded as the proper dose, but this is a mistake; from 3 to 5 minims may be required to produce about the same effect as that which follows 2 grains of sodium nitrite. A small dose of sodium nitrite (2 grains) distinctly affects the pulse in two or three minutes; the point of lowest tension is usually reached in from eight to forty minutes, and distinct influence on tension ceases in one to three hours. Ethyl nitrite has much the same effect, and on the whole lasts as short a time.' The sphygmograms which I have copied from Dr. Leech's work on 'The Nitrites,' etc., 1902, illustrate some of these points (Fig. 18). The tracings which I have shown from the pulse of the patient suffering from angina whose case I have related illustrates the effect on the pulse of the nitrites (1) in an interval between attacks, and (2) during the attack (Fig. 18). They were published in *Treatment* of October 14, 1897. A shows the character of the pulse when the patient was free from pain, and not under the influence of trinitrin. A dose of trinitrin was then administered to him with the results shown in B. The upper of the two tracings in this figure shows the acceleration of pulse and depression of pressure characteristic of the action of the

nitrite on a pulse not under the irritative influence of pain, and its gradual rise of pressure as the accelerant influence of the drug passes off. C shows the character of the pulse taken after an attack had just passed off, and during which no trinitrin was taken. In D the lower of the two tracings exhibits the quick, small, hard pulse characteristic of some cases of angina which may be instructively compared with the unirritated yet accelerated pulse due to the action of trinitrin in the absence of pain, as shown by the upper

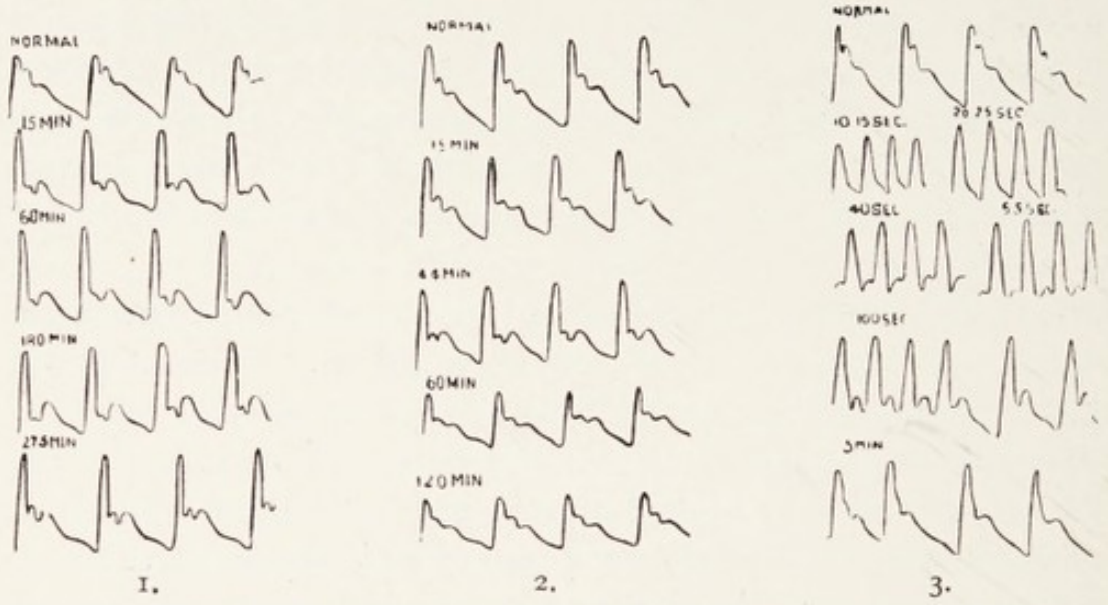

FIG. 18.—TRACINGS SHOWING THE RELATIVE EFFECTS OF (1) THE INHALATION AND (2) THE INTERNAL ADMINISTRATION OF AMYL NITRITE, AND (3) OF THE INTERNAL USE OF AMYL NITRATE.

The upper tracings are the normal, the lowest after the lapse of a considerable period (two and a half to four hours), and the intermediate of the effects of the drugs within those periods. The amyl nitrate effects reproduced occurred within three minutes. (Leech.)

tracing in B. The upper of the two tracings in D is characteristic of the pulse when an attack of angina, combated by the use of the nitrite, is passing off. With these tracings from a classical case of Heberden's disease we may also profitably compare those already given when discussing the rationale of endocardial angina, and which are from a case of regurgitant disease of the aortic valves. They show the small, accelerated, low-tension pulse of some cases, and the gradual rise of pressure in the pulse as pain passes off, and the artery is filled under the influence of the nitrite (Fig. 15).

As a rapid action of the drug is desirable during an attack

of pain, the inhalation of the nitrite or the internal administration of trinitrin in a fluid form is indicated. Of these, the method by inhalation is more prompt in action, and is to be preferred in the circumstances. Nor need we fear the use of more rather than less than the conventional dose of 5 minims of nitrite of amyl, for it has, accidentally and intentionally, been demonstrated over and over again that, although a very small quantity of this drug has a physiological and therapeutic effect, very large quantities have been inhaled and used without persistently ill-effect. Even the 1 per cent. solution of nitro-glycerine may be taken by some patients in considerable doses, though others can only tolerate small doses. Dr. Murrell mentions a case which took 100 minims of the solution as a dose, while another suffered severely from ½ a minim.[1] I generally use 2 to 5 minims, and the only inconvenient consequence I have noted has been some degree of headache. The dioxidation of the blood under the influence of the nitrites may be disregarded for therapeutic purposes, as the effect of these drugs is very evanescent. But gratifying as is the result of the use of the nitrites in many cases of angina, they do not by any means always succeed in relieving the patient. The agony may continue notwithstanding their free use.

In these circumstances, placed as we are between the possibility of witnessing a fatal inhibition or syncope, and our own fears lest we should use too powerful an analgesic, we must run some risks, if necessary, to arrest an almost certain catastrophe if the pain should continue. Our sheet-anchors, then, are chloroform, with or without the subsequent or combined use of ether and the hypodermic injection of morphia. On confronting such a situation Dr. G. W. Balfour writes as follows:[2] 'In a few cases the relief obtained [by the use of nitrites] is sudden and complete; more often the relief that follows is gradual and doubtfully due to the treatment, while in a few cases no relief seems to follow even the most prodigal use of these drugs. Then we are forced to have recourse to the free administration of chloroform, and

[1] *Op. cit.*, p. 71.

[2] 'Lectures on Disease of the Heart,' third edition, London, 1898, p. 331.

this must be given so freely as to narcotize the patient rapidly and completely; given in this way I have not seen any case that has not been relieved, though I have seen several in which the relief has not been permanent enough to restore the patient to comfort. In these cases the chloroform has had to be supplemented by the hypodermic injection of morphine, and of this I have never hesitated to give a sufficient dose, generally from half a grain to a whole grain; such a dose as this has kept the patient asleep for some hours, and he woke free from pain, but exhausted.' In view of the opinion which I strongly hold, that in the fatal issue of painful angina inhibition of the heart exercised by way of the pneumogastric nerve plays an important part, it is desirable that the use of atropine should be combined with the employment both of the nitrites and opium and other remedies—indeed, that it should always be used in these cases. For we know that, experimentally, the inhibitory function of the vagus is placed in abeyance by the use of atropine, and its influence in small doses upon the heart is at once to accelerate its rate and augment its force. I should add that Dr. Leech disapproved of the use of morphia. Even in the later stages of angina he states that increased doses of liquor trinitrini up to 20 minims is a safer proceedure.[1] The method, degree, and rapidity of anæsthetization must, however, be gauged in each case. It may safely be stated that the experience of no one man will include a very large number of cases of organic angina pectoris (other than those connected with valvular disease) requiring such extreme measures. Personally I have only met with a few such. Stokes, one of the founders of cardiology, confessed, as I have stated, that he had never seen even a mild case of angina other than that kind of cardiac pain associated with valvular lesion, and this will rarely require such vaporous anæsthetics as chloroform, although it may often require the hypodermic injection of morphia in addition to the use of the nitrites. This combination has also been found useful by Huchard. In short, cardiac failure, threatened by cardiac pain, is to be treated

[1] *Lancet*, July 15, 1893, p. 124.

on the same lines as cardiac failure threatened by hepatic, renal, or any other form of physical suffering. It must be remembered, moreover, that the angina associated with valvular disease (usually aortic) is frequently in direct ratio to the degree of lost tone and contractility in the ventricle, and that production or restoration of these is the only road to a permanent removal of the angina. While, therefore, the important but engrafted symptom of cardiac pain must receive attention, the underlying cause must not be lost sight of. To treat this well in cases of aortic regurgitation requires much judgment and experience.

2. **Medicinal Treatment in the Interval between Attacks.**—Because the depression of blood-pressure by the nitrites relieves the pain of angina, owing in great measure to their vaso-dilator effects, it is rational to believe that had we some agent which could for a longer period maintain such a vascular state, a greater immunity from the recurrence of such attacks might be secured. Professor J. B. Bradbury, of Cambridge, reasoned thus when he gave his Bradshaw Lectures in 1895.[1] That we may stave off an attack of angina by a timely dose of nitrite when a patient is about to perform some act likely by a rise of blood-pressure to induce one, may be admitted, but before regularly employing vaso-dilators which are less evanescent in their action than the nitrites, we have to consider whether high arterial pressure as such is the cause of angina. That a habitually high-pressure pulse may be one of the causes of the atheroma and its consequences, of which angina may be the expression, can scarcely be doubted; but the actual cause of anginous attacks, the mine, as I have said, or local lesion once existent, seems to be less an equably high blood-pressure than an irregular or intermittent one. To quote Heberden again, 'Those who are attacked by this disease are wont to be seized with the most severe breast-pang while walking. . . . As soon, however, as the pace is arrested all distress is quieted in a moment (*totus angor momento conquiescit*).' Heberden's words perhaps convey the impression of a more rapid disappearance of pain on the cessation of those actions

[1] *Lancet*, November 16, 1895, p. 1205.

which induce them than always takes place, but they also establish the fact that it is the sudden or intermittent rise of blood-pressure—it is, so to speak, the *blood-stroke* rather than the blood-pressure—which induces the pain, for the average blood-pressure, even if it be high, is doubtless maintained in some such cases after the attack has passed off. When relief is observed in some cases of angina pectoris on the establishment of an apical bruit, indicating incompetency of the mitral valve, it is probably due not only to diminished blood-pressure at the periphery, but also to lessened systolic impulse and perhaps to slightly diminished ventricular output. I am aware that in arguing thus I impugn in such cases the correctness of the theory of cardiac ischæmia. If, however, my argument be correct, what we have to arrive at is securing an equable pressure, so far as possible, not necessarily a low pressure. The constant normal variation of pressure renders this object only partially attainable, and this fact adds to the pity with which sufferers from angina must ever be regarded, for they carry as a condition of very life an enemy in their bosoms whose onslaught may be delivered at any moment and whose stroke cannot altogether be avoided even by the greatest watchfulness.

The higher nitrates, chiefly those of erythrol and mannitol, which Professor Bradbury recommends as more persistent vaso-dilators than the nitrites, have not come into general use, and it is perhaps questionable whether they are likely to do so. Sir Lauder Brunton's explanation of the *modus operandi* of the nitrites—namely, that they have a paretic influence upon vascular muscle—has been very generally adopted, and Dr. Leech also satisfactorily showed in his Croonian lectures that the final, if not initial, action of these drugs upon the heart itself was of the same character. The nitrates, although not acting so powerfully as the nitrites, act in the same way, and the continuous employment of a cardio-vascular depressant is not calculated, on theoretical grounds, to be permanently beneficial. The late Dr. G. A. Gibson, however, writes:[1] 'From the few observations

[1] 'Diseases of the Heart and Aorta,' p. 782.

which have been allowed me since the drug (erythrol tetranitrate) was brought forward, no doubt has been left in my mind as to its value. Although somewhat less rapid in its effects it is much more persistent' (than the nitrites). Indeed, in some cases of failing heart with low tension and angina we are compelled circumspectly to use strophanthus or digitalis. My own practice when this is necessary is to combine it with aromatic spirit of ammonia and spirit of nitrous ether. The ammonia, as Dr. Leech states,[1] retards the decomposition of the nitrous ether, and the nitrous ether tends to control the action of the cardiac tonic. This theory may be erroneous, but I have found the combination at times to act well in practice. Digitalis given alone is frequently found to aggravate matters in sclerotic cases manifesting angina.

To secure, so far as possible, an equable blood-pressure we must avoid as well as employ certain drugs. In this sense tobacco is a drug calculated to raise blood-pressure, and should theoretically be altogether avoided. The physician will, however, have to judge of the advisability of recommending this step by the character and circumstances of individual cases. To deprive a man long used to the solace of tobacco of his 'smoke,' if he be not a stoic, may be to precipitate rather than to obviate angina. But in any case permission to smoke must be inexorably limited to that minimum which will secure mental as well as vascular equability. We must also endeavour to remove indirect causes of vascular tension.

We have been taught by physiologists the value of the play of neuro-vascular action in the splanchnic area as a safety-valve to threatened excess of vascular pressure elsewhere, and the use of certain agents which combat fermentation and promote the flow into the *primæ viæ* of the natural secretions which secure a normal blandness of intestinal content is of the first importance. Among such agents I should place in the first rank the milder mercurials—calomel and blue pill. I have cardio-vascular patients who have taken from half a grain to one grain of calomel once or twice

[1] *Lancet*, July 22, 1893, p. 178

a week regularly for a twelvemonth at a time, with unquestionable benefit in maintaining an equable blood-pressure. This practice on my part is no recent procedure, but I did not derive it from my *alma mater*, the University of Edinburgh, for when I was in my professional infancy my clinical teacher, one of the ablest who ever felt a pulse—the late Professor Hughes Bennett—had, perhaps, among some dislikes, one prime aversion—namely, mercury in every form. So that I began practice fully convinced that, did I prescribe that drug to my patients, I should shortly have around me a tremor-struck and gibbering public execrating me as the mercurialized prisoners in a quicksilver mine might their gaoler! From this delusion I was freed many years ago by the late Sir William Broadbent. How mercurials act in these circumstances, whether as eliminants, or as antiseptics, or as more direct vaso-dilators, it is not necessary for practical purposes to inquire. Their action is, without doubt, frequently beneficial in maintaining a certain softness and pliancy of pulse for long periods. General gastro-intestinal medication by alkalies or acids as required, or by the use of saline or other aperients, is indicated for the same reason—namely, to remove or to correct possible causes of intermittent blood-pressure of an injurious character.

It is possible that some of the repute enjoyed by iodide of potassium in cardio-vascular sclerosis may be due to its efficacy in cases of specific origin, but such a supposition does not account for its general use by observant physicians. Although the drug had some vogue in such conditions before the advocacy by Dr. Balfour of its usefulness, there can be no doubt that his emphatic and authoritative utterances in its favour have done much to secure its general adoption as a cardio-vascular remedy or agent in treatment. He compares its action in one place to that of the nitrites[1] and in another refers its therapeutic effects in aortic aneurism to the reduction by it of intra-arterial blood-pressure.[2] The dose he recommends for continuous use as a vaso-dilator is 2 grains every twelve hours. There is one objection to its employment which has, perhaps, more weight in cases of

[1] *Op. cit.*, p. 379. [2] *Op. cit.*, p. 467.

angina pectoris than in cases not associated with these paroxysms—namely, its tendency at times to cause gastro-intestinal irritation and thus to provoke those reflexes which induce irregularities in vascular pressure. But the same objection applies to mercurials, and if the administration of both these useful agents be coupled with a scrupulous care as to the blandness and sufficiency of diet and the avoidance of condiments such as vinegar, ketchups, and effervescent wines and beverages, this objection is in no way comparable to the advantage which is constantly derived from their use in the intervals between attacks of angina. Concerning the *modus operandi* of the iodides in inducing a more equable vascular tone, there is room for legitimate differences of opinion, for much obscurity attends our present knowledge of the subject. We must be content, meanwhile, to act empirically so long as our action is beneficial to the patient, and wait for that enlightenment which science sooner or later affords those who industriously look for it and patiently expect its advent.

Drugs other than powerful analgesics and vaso-motor agents may be of service in some cases of angina. If the malady be associated with anæmia due to some specific cause such as malaria, or be attributable, as appears to be the case in some instances, to lead-poisoning, or be aggravated by defective blood-states of the kind more commonly met with, the treatment of the accidental state, *secundum artem*, by such agents as arsenic or iron or iodide of potassium, as the case may be, is calculated to be of benefit. In functional or inorganic cases they may be all that is necessary to effect a cure when coupled with attention to general hygiene.

What has been said as regards the treatment of angina pectoris applies in the main to the treatment of angina sine dolore or syncope trepidosa. As Dr. Leech has remarked, although the ultimate influence of the nitrites on the cardiac muscle is depressant, it is probable that by inducing a larger number of smaller contractions the nitrites help to unburden an overladen heart, even if the individual contractions be less powerful than normal (*op. cit.*). But in the

syncopal variety of the complaint, if we still classify it with Heberden's disease, hypodermic injections of strychnine find appropriate employment as powerfully raising the reflex irritability of the spinal cord, and toning the adjuvant mechanism of voluntary muscular respiratory effort. M. Huchard has well remarked (*op. cit.*) that in some severe cases of painful angina the pain itself is the best safeguard againt an overdose of a direct analgesic like morphia. So, also, it may be equally cogently maintained that in painless or syncopal angina the syncopal state is the best antidote against the injurious influence of a somewhat bold use of strychnine. With this powerful agent we may usefully combine, as in the case I have related, the tincture of strophanthus, which acts as powerfully on the heart as the preparations of digitalis, and is less likely than the latter to raise a peripheral obstacle to the circulation, as it acts less on the distant arterioles. Cushny has pointed out that the direct injection of strophanthus into the blood by way of a vein most rapidly secures its effect, and thus also strychnine may be used in combination with that drug, notwithstanding its more recently asserted inertness by some in cardiac failure. Such a combination may be to syncopal angina what opium, chloroform, and atropine are to painful angina of a severe type; but the fuller consideration of the treatment of this type of angina, as also of other modes of cardiac failure and aberrant action, will be more appropriate when discussing the treatment of disorders of cardiac motion.

The Surgical Treatment of Angina Pectoris.—While the condition which has been termed in these pages 'hypertrophic endocardial angina' is associated with an exaggerated action of the heart, and might as such be classed with disorders of cardiac motion, and considered both as regards its nature and treatment in that portion of this work dealing with these conditions, it will on the whole be more convenient to consider the treatment of this state now, as the operation I am about to describe was undertaken chiefly on account of the distressing pain experienced by the patient.

With the general subject of cardiolysis I shall deal later. I have already given some particulars of the case which

prompted me to advise præcordial thoracostomy for its relief (p. 107). It was the first, and so far as I know is the only case which has been thus dealt with, although the relief afforded appears to justify its repetition under suitable circumstances.

My previous experience of such cases had been that relief from cardiac pain was only obtainable by increasing doses of trinitrin, while short intervals of improved cardiac action were not always to be expected, and even if they occurred, they did not long postpone the deteriorating signs which heralded the ultimate failure of the heart. I therefore determined to afford the enlarged heart more room to act in, while its power was still good, and asked Mr. Stabb to secure free space for cardiac systole without the incarcerating barrier of hard rib, in the area of thoracic concussion. This he very skilfully did on May 1, 1908, in the following manner: A U-shaped incision, with its descending limb in the centre of the sternum, commenced at the level of the sternal attachment of the third rib. Sweeping round at the level of the seventh rib, the ascending limb of the incision was carried to a point corresponding with its commencement. A large flap was thus secured and raised, consisting of superficial textures and muscle down to the ribs. This was turned back and spread between warm, moist towels. The fourth and fifth intercostal spaces were next cleared down to pleura, and the fifth rib, covered by its periosteum and perichondrium, carefully separated from the underlying pleura, and $4\frac{1}{2}$ inches of its length from the sternum outwards were removed in three pieces. The sixth rib was similarly cleared, and $5\frac{1}{2}$ inches of it were removed in one piece, its greater arching allowing more easy detachment without injury to pleura. The internal mammary artery required ligature. A small puncture was made in the pleura while the last portion of the fifth rib was being removed, air being aspirated through it during respiration. The puncture was stitched, but was not rendered quite airtight thereby. The raised flap was then replaced and accurately applied by alternate fishing-gut and horsehair sutures, a drainage-tube being inserted at the lower external angle of

the wound and left in position for twenty-four hours, when it was renewed. After removal of the ribs and display of the underlying movements of the heart, the latter appeared to be smooth, rolling, and without adhesion to the extraneous structures exposed. Mr. C. H. C. Visick administered the anæsthetic (chloroform) with the Vernon-Harcourt apparatus, the patient taking it well. Towards the end of the operation strychnine was injected hypodermically, rather as a precautionary than necessary measure. The wound healed by first intention, except at the point of issue of the drainage-tube, where a little leaking continued for a short time.

The temperature was slightly raised (99·2° F.) on the day following the operation, and swung between that point and subnormal till May 9, when it again remained normal or subnormal. The pulse was slightly quickened, running from 108 to 126 until the 9th, when it dropped to 92 to 100, the average pulse before operation having been 80 to 96. The rate of respiration was most altered, its highest rate being 60 on May 4, and averaging about 40 until the 10th, when it dropped to an average 32 till the 17th, and later to 24. The highest respiratory-rate before operation noted was 36, and the lowest 20. The higher rate recorded was apparently due to shallowness of respiration from pain in the side due to the wound.

The effect of the operation objectively and subjectively was most satisfactory. The space previously occupied by the fifth and sixth ribs consisted of a soft, unresisting cushion formed by the fourth, fifth, and sixth intercostal spaces and the sites of the removed ribs. The palpating hand found little thoracic concussion but the powerful throb of cardiac systole into the yielding covering of the heart. The other physical signs remained as before, but the exaggeration of carotid and suprasternal pulsation was less marked, and the blood-pressure registered was from 120 to 130, and at times as little as 115. The apical presystolic rumble was not to be heard. The only palpable thrill was basic (aortic) and diastolic in time. The skin on the præcordia to the left of the sternum, below the level of the fourth rib, was decidedly diminished in sensibility, doubtless

from severance of the fifth and sixth intercostal nerves, but it later recovered sensibility somewhat. The area thus

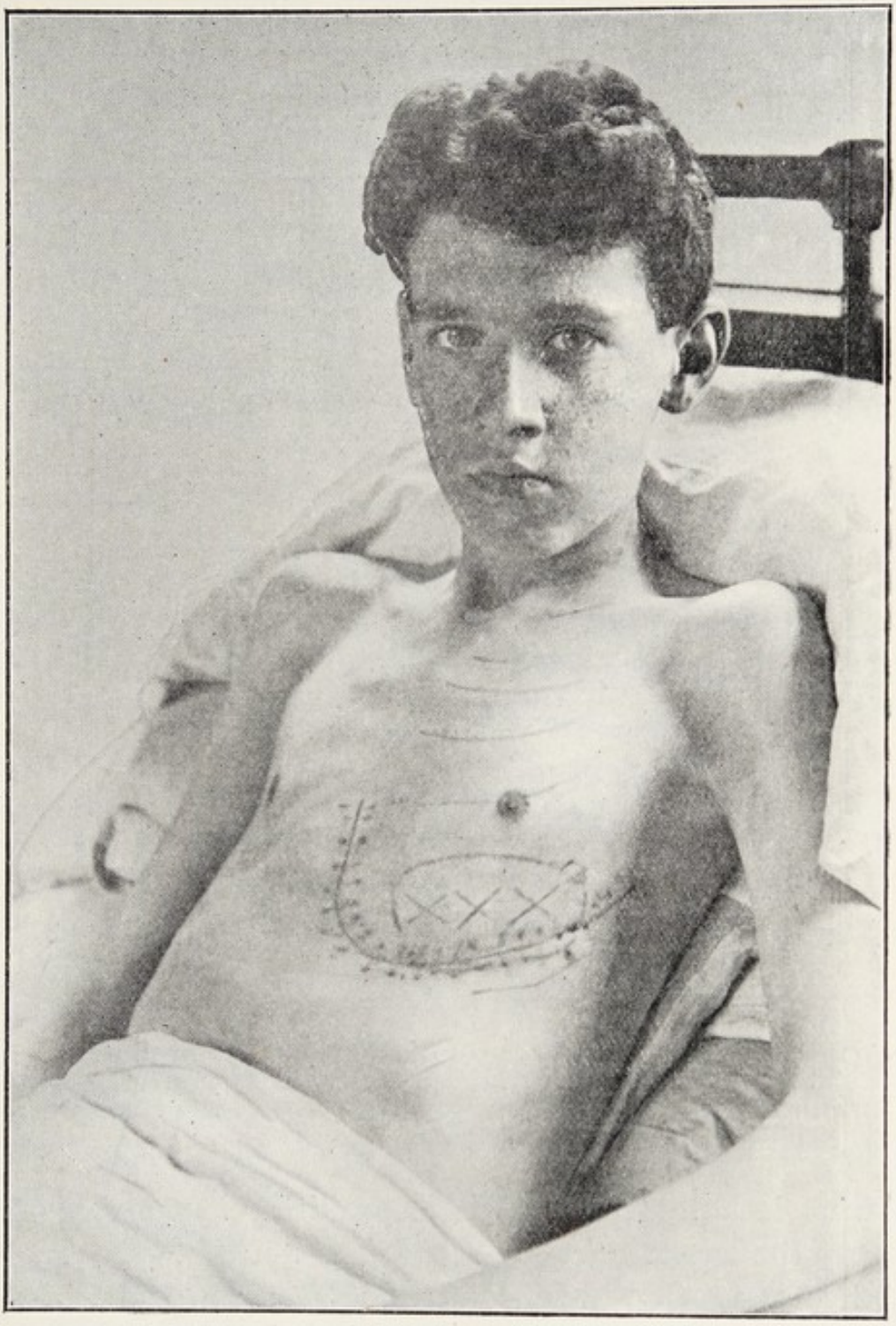

FIG. 19.—PRÆCORDIAL THORACOSTOMY.
The crosses show the enlarged area of the apex-beat, and the markings show its situation.

robbed for a time of all sensation (tactile, painful, and temperature) measured about 2 by 4 inches. Finally, before

operation the chest capacity as registered by Lowndes's spirometer was 60 cubic inches, and on May 18 it was 63. The capacity of two other men, presumably normal, was 120 and 130 cubic inches respectively. Subjectively, the most gratifying result has been that the patient has had a greatly diminished experience of angina, and even when

FIG. 20.—CERVICAL PHLEBO-ARTERIOGRAM, SHOWING RESPIRATORY UNDULATION (MAY 11).

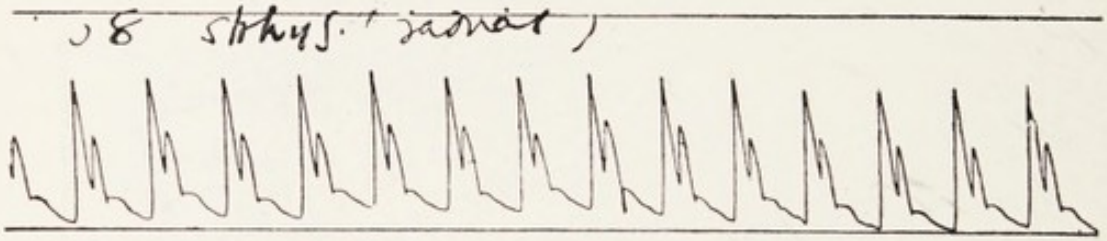

FIG. 21.—RADIAL SPHYGMOGRAM (MAY 11).

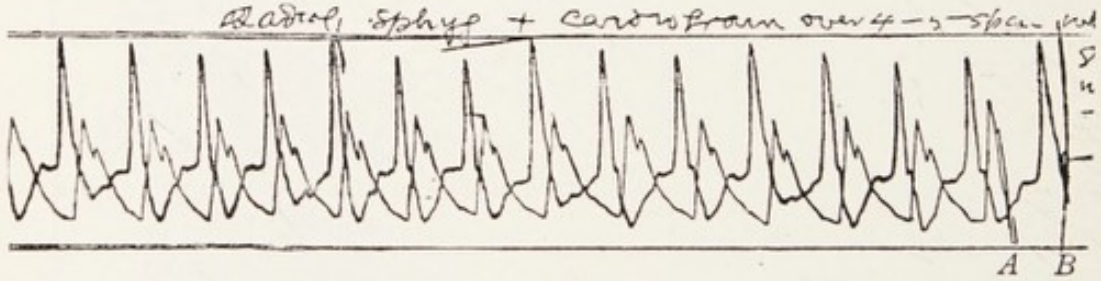

FIG. 22.—CARDIOGRAM FROM STERNAL END OF ENLARGED APEX-BEAT INTERLACED WITH THE RADIAL SPHYGMOGRAM, ON ACCOUNT OF THE NARROWNESS OF THE STAGE. THE ARRESTS AT *A* AND *B* SHOW THE CORRESPONDING PHASES OF MOVEMENT (MAY 18).

suffering it he affirms that it is decidedly less severe than prior to the operation.

On May 12 Dr. James Mackenzie saw the case with me and asked the patient whether in his opinion the degree of relief he had experienced justified his having undergone the suffering of the operation. This question he emphatically and without hesitation answered in the affirmative. The nurse in charge of the ward during the night also informed me that the quietude and uninterruptedness of the patient's

sleep after operation was in striking contrast to his broken rest before that event.

He was dismissed from hospital in all respects more comfortable than before operation and perfectly satisfied with his progress on June 6. Before leaving he was provided with a poroplastic shield to protect the more exposed

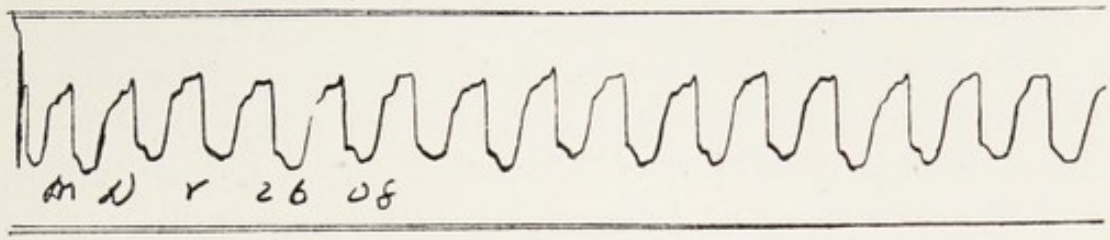

FIG. 23.—CARDIOGRAM FROM STERNAL END OF ENLARGED APEX-BEAT (MAY 26).

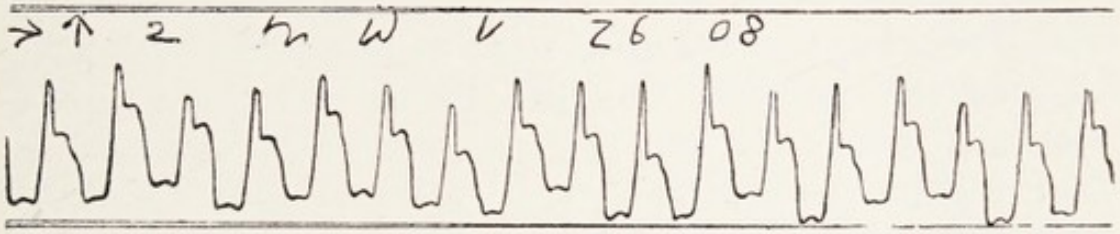

FIG. 24.—CHARACTER OF CARDIOGRAM AT MIDDLE PORTION OF ENLARGED APEX-BEAT. SHOWS RESPIRATORY UNDULATION (MAY 26).

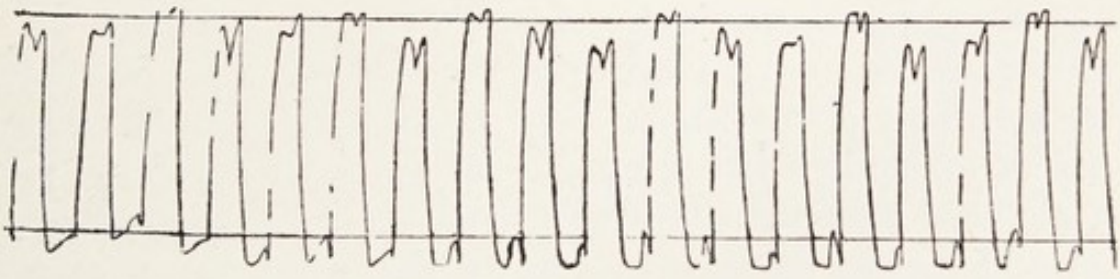

FIG. 25.—CARDIOGRAM OF OUTERMOST PORTION OF APEX-BEAT. SHOWS LARGE EXCURSIONS AND ALSO RESPIRATORY UNDULATIONS (MAY 26).

heart from accidental injury, in imitation of the costal bulging adopted by Nature in the young. This was later replaced by one made of aluminium and covered with leather. It transpired in the discussion on Brauer's cases (*loc. cit.*) that the enlarged interspaces tended to shrink apparently without undoing the benefit of the operation. This did not occur in this instance.

The accompanying tracings exhibit the character of the cardio-vascular movements at different points and at different dates (Figs. 20 to 25) at this period of the case.

Eighteen months after operation I reported in the *Lancet* of November 20, 1909, the progress of the case. Since leaving hospital on June 6, 1908, there had been no occasion to readmit the patient. He had reported himself from time to time, stating that he continued comparatively free from discomfort, but that he still had occasional attacks of pain, usually at night, which were relieved by inhaling nitrite of amyl. These attacks were, however, according to the patient, of much less frequent occurrence than prior to the operation. He had been taught the light trade of a hatshape-maker, and was endeavouring to earn his livelihood by this means. On reporting himself at the hospital on July 5, 1909, the following note was made of his condition: He stated that he had recently had a little more pain during the night when lying down, but that the great relief to his discomfort which followed the operation was still maintained. He looked well, and weighed 8 stones 7 pounds, which was heavier than any previous weight except one—on June 25, 1908, when he had just returned from a convalescent home. The pulse-rate was 96 to 114, and its rhythm regular. The instrumental determination of the blood-pressure was omitted on this occasion, but a few days later was found to be 140 millimetres of mercury with the Riva-Rocci instrument for the systolic and 80 millimetres for the diastolic pressure. The ribless interval in the præcordia measured 3 by 4 inches, as it did soon after the operation. The heart pulsated powerfully in it, and the hypertrophy of the organ appeared to have increased, the systolic impulse impinging rather more on the seventh rib than it did a year previously. There was a systolic and diastolic bruit most audible in the right second interspace, and a systolic bruit of high pitch at the apex conducted towards the axilla and quite distinct from the aortic bruit. In the situation of the fifth left interspace there was still slightly diminished cutaneous sensibility. It will be remembered that this was very marked after the operation over an area of 2 by 4 inches. The respiratory rate was 30, and the lungs normal. The patient was otherwise healthy.

The accompanying cardiograms and sphygmogram, which

were taken on a wider field than those previously given, show the nature of the pulsations. It will be observed that they are vigorous and modified in shape according to the site of application of the receiver, the larger and more powerful excursions of the lever being taken from a portion of the

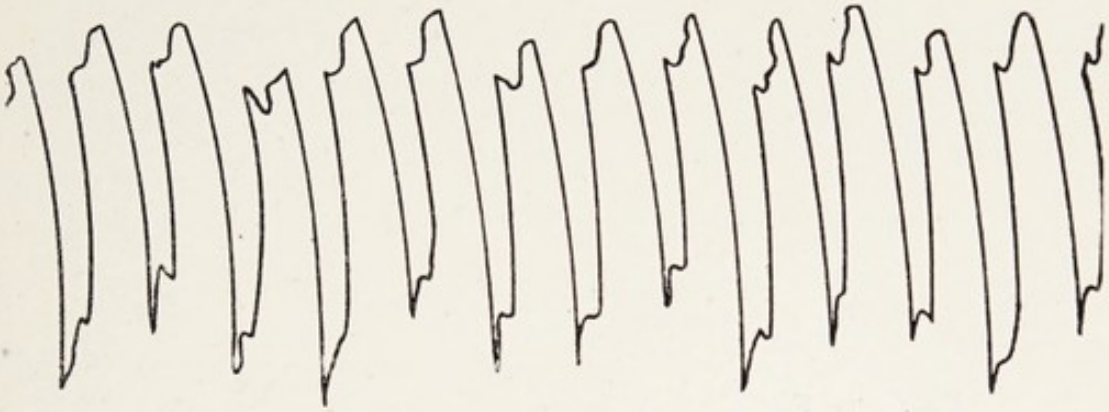

FIG. 26.—CARDIOGRAM OF THE OUTER CARDIAC APEX.

FIG. 27.—CARDIOGRAM OF THE INNER CARDIAC APEX.

Both cardiograms show respiratory undulations.

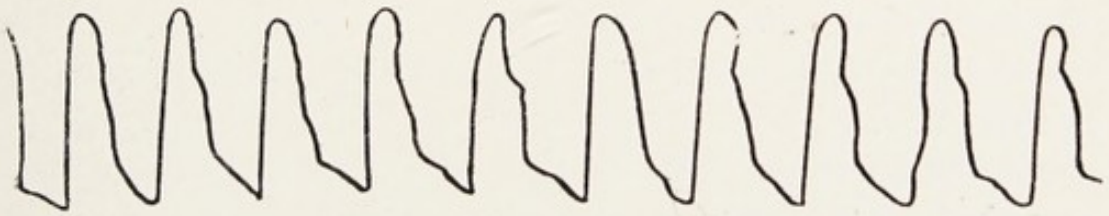

FIG. 28.—RADIAL SPHYGMOGRAM.

heart which received better the left ventricular thrust. The act of inspiration diminishes tactile impulse, as may be seen in the tracings.

For the next three years the patient's condition remained much the same. The following cardiogram, taken on April 17, 1911, on one of his visits to the hospital to report

himself, shows a sustained vigour of impulse and the same characters as he had had three years previously:

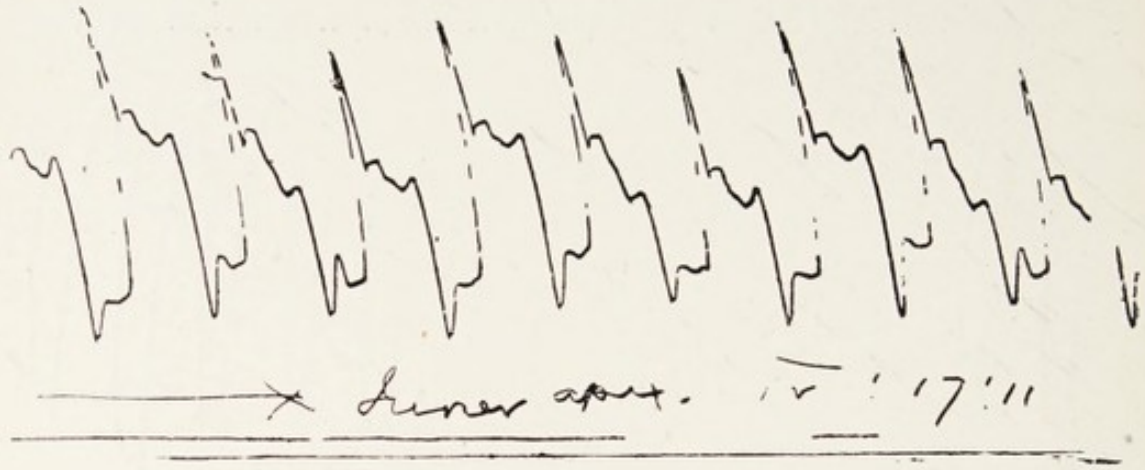

FIG. 29.—CARDIOGRAM OF THE INNER CARDIAC APEX.

The normal relation of the auriculo-ventricular sequence will be observed.

In August, 1912, being apparently in his usual health and comparatively free from anginous attacks, although still suffering from them, and obtaining relief from the inhalation of nitrite of amyl, the patient had an opportunity of going to a convalescent home, and was accepted as an inmate. He remained there altogether nine weeks. I subsequently learned that he occupied a room the immediately preceding tenant of which had died there of infective endocarditis. In the sixth week of my patient's stay at the home, and while out on the seashore, he suddenly lost some power in his right arm, and his speech became difficult. As later events showed, he must then have had ambulatory infective endocarditis. Three weeks after this attack he improved somewhat in these respects, and, returning to London, presented himself at the hospital, into which he was readmitted on September 12, 1912. He looked emaciated, had a temperature, showed the physical signs he had previously done, and although two attempts to grow a culture from his blood failed, was diagnosed to be suffering from infective endocarditis. After a stay of four weeks in hospital, progressively losing strength, he died on October 10, 1912. During this fatal illness the impulse of his heart was diminished, he complained of no cardiac pain, and had no

orthopnœa. As the cardiac action and pulse-rate of the patient showed no variation from its previous state, being as a rule regular and of a rate proportionate to his temperature, no graphic record of his cardio-vascular movements was made, a circumstance to be regretted in view of the condition observed after death.

On examination after death the outer surface of the pericardium in the area of the præcordial opening surgically made was totally adherent to the pleura and the latter to the subcutaneous textures, but there were no viscero-pericardial adhesions and little pericardial fluid in the sac.

The heart was much enlarged, weighing 1 pound 15½ ounces with, and 1 pound 12 ounces without, the contained blood. The right auricle was dilated, the tricuspid valves normal, and the foramen ovale closed.

The right ventricle was hypertrophied, and the pulmonary artery and its valves normal.

The mitral valves were thickened, but neither cusps nor the left auricle showed any vegetations. The left ventricle was much hypertrophied and dilated; the walls at their thickest measuring ⅞ inch. The papillary muscles were much hypertrophied. The edges of the aortic valves were thickened, but the chief pathological conditions affected the non-coronary cusp of the aortic valve, which was covered with recent large vegetations which extended down on to the ventricular surface of the aortic cusp of the mitral valve.

On dividing the non-coronary aortic cusp and its vegetations vertically and removing a wedge of underlying texture to examine the condition of Valsalva's sac and the bundle of His, the floor of the right auricle from the coronary sinus to the pars membranacea septi was observed to have large extravasations of blood.

On microscopic examination the aorta behind the affected cusp, the pars membranacea septi above the bundle, and the lower part of the auricular muscle, showed inflammation in various stages up to abscess formation.

The upper portion of the bundle near its commencement, in its right half and right limb, were seen to be infiltrated with leucocytes, and an abscess abutted upon the right

limb. Muscular fibres in the upper part of the bundle near its commencement, where active inflammation was present, had undergone a peculiar blanched swelling with distinctness of the nuclei, which resembled large Purkinjean cells, but was evidently a result of the inflammatory process.

The spleen contained numerous, and the kidneys some, infarcts of various ages.

No bacteriological examination was made.

I have related this case fully, because it is the first to have been treated surgically for the relief of cardiac pain. That the patient was immediately and for rather more than four years much relieved by the procedure is indisputable. That his relief and the general force of his heart might have been maintained for considerably longer had he not unfortunately and accidentally acquired a fatal infective endocarditis, can as little be questioned.

PART III

DISORDERS OF CARDIAC MOTION

CHAPTER I

INTRODUCTORY

In the whole range of physiological observation there is no more venerable study than that of the movements of the heart and bloodvessels. The length of art and the shortness of life, the fleeting nature of opportunity, the possibility of misreading experience, and the difficulty of forming a judgment which were borne in upon the Father of Medicine, have in no sphere been more exemplified than in the field of cardio-vascular observation. But in the collective life of man knowledge of the science of the circulation and of the art dependent upon it has encouragingly increased, and may in the future be expected to expand more and more until some approximation to the whole truth has been attained, provided it be sought in the modest spirit expressed in the immortal aphorism quoted.

The vital activity manifested by cardio-vascular muscle lends itself well to observation. It can be felt, seen, and heard. Neural and hæmic activity can only be studied indirectly, and although essential factors in the functional unit, they are less obtrusive than the other member of the triology, which, from the very grossness of its manifestations, has tended at times to relegate to a subordinate place both its fellows.

By the exercise of touch, sight, and hearing, naked and aided by ingenious instruments, certain facts have been gathered and registered which enable us to discriminate

between normal and abnormal modes of cardiac action. The hand, the stethoscope, the radiograph and the various graphic methods, including the sphygmograph, the phono-cardiograph, and the electro-cardiograph, have supplemented one another in eliciting phenomena during life, the interpretation of which in the light of anatomical, physiological, and pathological research has helped to reveal in some measure the nature of cardiac motion, normal and disordered.

Normal cardiac action has a certain regular sequence of pre-auricular, auricular, and ventricular contraction and relaxation, and an average and corresponding rate and force, influenced under normal circumstances by posture, respiration, emotion, and external agencies acting by absorption, inhalation, and application. Under abnormal circumstances these standards of the normal are departed from, and it is the task of the physician to determine how and why such abnormalities arise, and how a return to the normal may be promoted, when this is possible.

Pre-auricular motion is vascular rather than cardiac—although, as we have seen (p. 13), the coronary sinus is a very complete chamber in itself—and of scientific rather than clinical interest. Disorder of cardiac motion may therefore, for the physician, be said to begin with the auricle, and to be limited to that chamber or shared by it and the ventricle. The power to discriminate clinically between the independent movements of these chambers we owe to the more recent graphic methods of observation with which the names of Marey, Chauveau, Mackenzie, Wenckebach, Einthoven, Gibson, Lewis, and many others are associated.

Before considering more particularly the character and nature of special forms of disorder of cardiac motion, it is advisable to draw a distinction between departures from the average normal of cardiac action and *diseases* of the heart in which this average normal of movements is departed from. The point will be found to be of value when we come to consider the prognosis of cardiac motor disorder. For a man may be dying whose heart shows little departure from the normal average in cardiac motion, and very striking departures from the average may be transient, or, if more

persistent, have little influence in curtailing the life of the patient or subject who presents the phenomena in question. Bearing this distinction in mind, we may attempt a classification of disorders of cardiac motion preliminary to their further consideration.

The Classification of Disorders of Cardiac Motion.

In attempting to classify the sensory disorders of the heart it was remarked that the process was largely one of the classification of symptoms, and a division was roughly made into those with and without pain, while a provisional association of these signs with certain anatomical conditions was indicated.

The classification of the disorders of cardiac motion must also be as largely one of the phenomena of cardiac action—quick, slow, irregular, strong, or weak, as the case may be; that is, of the rate, rhythm, and force of the heart's action.

In a sense, any departure from a standard rate, rhythm, and force, whether it be over-quick or over-slow, regular or irregular, or whether it vary from a standard average of force as plus or minus, may be regarded as an arhythmia. But there is a clinical convenience in separating the regular from the irregular variations of cardiac action, and in applying the term 'arhythmia' to manifestly arhythmical or irregular actions.

The essence of arhythmia is, however, the plus or minus character, as the case may be, of the incidents which compose the heart's beat. As we have seen in Part I., these fall into the three phases of that beat described by Foster, as already quoted (p. 13)—namely, the actions of the sinal structures and those of the auricles and ventricles.

Of the movements of the heart, the first and principal is *contraction*, the contraction of the sinus, the auricles, and the ventricles—the systoles of the heart.

The cessation of these actions and the return to positions and conditions of rest may also be regarded as movement; but the diastole is in a very limited degree active, although the mechanical effect of auricular and ventricular diastole is not unimportant.

The average systole, then, as regards rate, rhythm, and force, forms the standard, departures from which constitute the disorders of cardiac action.

It would be possible, indeed, to classify accelerated actions in rate as tachysystoles, retarded as bradysystoles; irregular actions in rhythm as anisosystoles; excessive or erethitic actions in force as hypersystoles, and insufficient as hyposystoles. But the field is already occupied by certain terms which have a conventional meaning attached to them, and are sufficiently descriptive for recognition.

The term 'arhythmia,' however, while frequently used to denote irregularity of action, might suitably be displaced by one which has occupied much attention of recent years, and which, as Vaquez points out,[1] is the underlying feature of many irregularities, however dissimilar superficially—namely, the extra or premature contraction, the extrasystole.

For 'arhythmia,' as denoting irregularity of action, 'extrasystolia' might without disadvantage be substituted.

With this preface on the principles enunciated in this book, the classification given in the following table fulfils the conditions of convenience:

- Disorder of—
  - Rate
    - Tachycardia
      - Sinal (unimportant).
      - Auricular (Syn., auricular flutter; auricular extrasystolia).
        - Temporary.
        - Persistent.
      - Auriculo-ventricular
        - Thyroidal.
        - Paroxysmal (?).
        - Fibrillational (?).
    - Bradycardia
      - Sinal (unrecorded).
      - Auricular (unrecorded).
      - Auriculo-ventricular.
      - Ventricular.
  - Rhythm
    - Extrasystolia
      - Sinal (unimportant)
        - Non-tachycardial.
        - Tachycardial (Syn., flutter).
      - Auricular
        - Rhythmic
          - Non-tachycardial (Syn., allorythmia).
          - Tachycardial (Syn., paroxysmal tachycardia).
        - Arhythmic
          - Non-tachycardial (Syn., irregular heart).
          - Tachycardial (Syn., tachy-arythmia; auricular fibrillation).
      - Ventricular
        - Rhythmic
          - Non-tachycardial (Syn., allorythmia).
          - Tachycardial (Syn., paroxysmal tachycardia).
        - Arhythmic
          - Non-tachycardial (Syn., irregular heart).
          - Tachycardial (Syn., tachy-arythmia; auricular fibrillation).
  - Force
    - Hypertonia
      - Hypertrophic.
      - Non-hypertrophic.
    - Hypotonia
      - Hypertrophic.
      - Non-hypertrophic.

1 'Les Arythmies,' Paris, 1911, p. 182.

CHAPTER II

AURICULAR TACHYCARDIA; AURICULO-VENTRICULAR TACHYCARDIA; AURICULAR FIBRILLATION

TACHYCARDIA of the sinal structures of the heart—namely, the venous entrances into both auricles and the coronary sinus—is not a condition that can be discussed clinically, although as a consequence of disorders of the auricle, sinal conditions may reasonably be regarded as disturbed in action. Their clinical importance as disorders of cardiac motion cannot be great, with the possible exception of that of the coronary sinus, the stasis or impotence of which may be disadvantageous to easy circulation in the important coronary venous circulation. As I have said, clinical disorders of cardiac motion commence with those of the auricle.

By those who regard the sinu-auricular node and other scattered portions (which I have been unable to detect) of nodal texture in this neighbourhood as the true remains of the embryonic sinus and the representatives in the mammal of the well-developed sinus of the amphibia, the interpretation of the term 'sinus' here used is not shared.

Auricular Tachycardia, or **Flutter,** if not rare, as it probably is not, has until recently been seldom detected or studied. So far as I know, a case met with by myself in 1903, and published in 1908[1] under the title of 'Jugular Embryocardia,' was the first recorded. I also referred to it in the course of a paper on 'Cardiac Motion as revealed by the Vivisection of Disease,'[2] in which I gave the jugular and radial tracings from the case. The patient was a woman,

[1] *Lancet*, January 2, 1909. [2] *Lancet*, January 9, 1909.

forty years of age, who was under my care at the Great Northern Central Hospital from March 7, 1903, till July 17 in the same year. She had had rheumatic fever ten years previously, and was admitted with double mitral valvular disease, some aortic obstruction, and a tricuspid reflux bruit. The ventricular rate was 114 to the minute, and its rhythm regular, while the auricular rate was 240. My attention was

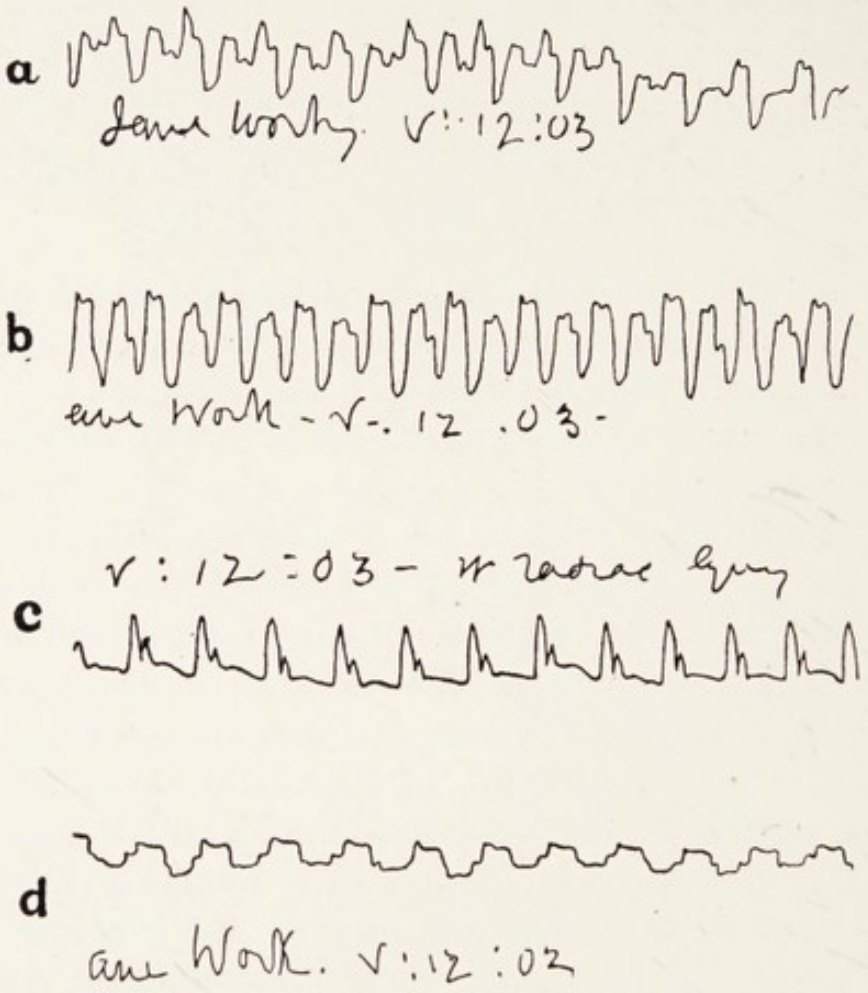

FIG. 30.

a and *b*, jugular tracings ; *c*, radial sphygmogram ; *d*, cardiogram.

drawn to the phenomenon on auscultating the right supra-clavicular region, when sounds exactly resembling those of the fœtal heart at the seventh or eighth month of pregnancy were heard, and the clinical polygraph reproduced auricular pulsations at the rate indicated. Hence the name I gave the sign (Fig. 30). Although the patient when admitted had large anasarca and considerable pulmonary engorgement, and was in great measure relieved of these conditions when

discharged, there was no cessation of the audible auricular tachycardia throughout her stay in hospital, and I was informed by Dr. Purdie, of Haverstock Hill, under whose care she came afterwards, and whose attention was drawn to the auricular phenomenon, that the sign persisted until the death of the patient some time afterwards.

Since detecting this condition by jugular auscultation, I have frequently auscultated the supraclavicular triangle containing the internal jugular termination and the valves of the vein, and at times have heard a series of sounds evidently due to phases of the cardiac cycle. When heard with clearness in cases of cardiac failure, I have regarded them as evidence of auricular repletion, but they have not appeared to me to be sufficiently constant or easily appreciable to be a very useful aid in clinical diagnosis.

Dr. O. Josué,[1] however, carefully describes these phenomena, correlates them with the incidents of the phlebo-arterial sphygmogram, gives directions for their detection, and considers 'auscultation of the venous pulse' a convenient and valuable means of diagnosing the relations of the phases of the cardiac cycle in the absence of the more ponderous implements of cardiac graphology.

Soon after the publication of my case another was related by Drs. Herz and Gordon Goodhart,[2] in which there was an auricular rate of 236 and a ventricular rate of 72 to 120. It was that of a woman, thirty-nine years of age, with a rheumatic history, the subject of mitral valvular disease and hemiplegia. In this case, as in mine, the auricular condition persisted for a lengthened period; but, unlike my case, this one exhibited irregular ventricular action, and the observers made the valuable note that while atropine administered increased the ventricular rate, it had no effect in this respect upon the auricle, which had apparently attained its maximum speed. This effect of atropine in the case serves to exclude it from the category of so-called 'heart-block,' or at any rate from that series to which cases exhibiting the

[1] Report, Section I., International Medical Congress, 1913, and Soc. Méd. des Hôp., February 14, 1913, p. 401.

[2] *Quarterly Journal of Medicine*, January, 1909.

Adams-Stokes syndrome belong. Herz and Goodhart, correctly as I believe, regarded the case as an auricular neurosis. Too few of these cases have so far been observed to determine the full significance of the condition. Thirty cases in all have now been noted,[1] and the flutter has been found at times to pass into a fibrillation. The indifference of the ventricle as a rule to this mode of auricular stimulation, without the evidence of any organic 'block,' is very striking, and argues a neuro-muscular movement of the atrium independently of the ventricle, and necessarily also the independent action of the ventricle. The ventricle in my case pursued the even tenor of its way, undisturbed and apparently uninfluenced by the pace of the auricle.

Several cases of this condition have been published by Dr. Thomas Lewis,[2] in which he was enabled to satisfy himself by electro-cardiographic examination that the ventricular rate could in these cases be reduced without any fall in the rate of the auricular tachycardia. This he explains, as might be anticipated, by the action of digitalis upon the auriculo-ventricular bundle, blocking it against the transmission of the auricular impulses showered upon it.

It will be remembered that in the first part I referred to Erlanger's statement that the inhibitory action of the vagus was abolished by interruption to the bundle, from which one was led to the belief that inhibition of the ventricle was considered to depend upon a prior inhibition of the auricle. This explanation of ventricular inhibition, if regarded as constant, will, however, scarcely be maintained in view of the clinical experience of these cases of auricular tachycardia, in which, with a reduced ventricular rate under digitalis, the auricular rate continued unchanged. For Lewis the 'true heart-rate' is the rate of that portion of the heart in which the rhythmical action is supposed to start.[3] For some that is still only a fraction of the heart-rate—namely, that of the auricle.

Dr. W. T. Ritchie, of Edinburgh, showed me in 1912 the

[1] Ritchie, *Quarterly Journal of Medicine*, October, 1913.
[2] *British Medical Journal*, July 29, 1912, p. 1481.
[3] *Loc. cit.*, p. 1482.

electro-cardiogram of a case of ventricular acceleration with some irregularity of action, in which there was well-marked disproportionate auricular tachycardia, and in which ventricular inhibition with retardation could be induced by pressure on the pneumogastric nerve, without in any way influencing the rate of the auricular tachycardia. This result could certainly not be attributed to any but a neural cause, which must therefore have acted either on the bundle of His or on the ventricle directly.

Later in the same year Dr. Ritchie published his valuable paper on 'The Action of the Vagus on the Human Heart,'[1] recording the results obtained by manual compression of the nerve. He found that compression of the nerve on the right was more effectual in inducing inhibition than manipulation of the left; that fibrillation and 'flutter' (for the latter of which terms I have used the expression 'auricular tachycardia') were not affected by vagal compression even when the ventricle was inhibited; that 'the depression of the ventricular rate was usually proportionate to the degree of auricular retardation'; and that cases of ventricular bradycardia ('complete auriculo-ventricular dissociation') were not apparently affected.

In view of the anatomical considerations mentioned in Part I., these results are what one would have expected; but they do not necessarily imply that ventricular depression, though proportionate to auricular retardation, was a consequence of the retarded muscular action of the auricle. For if—and it has not yet been proved—the left vagus has more to do with the auriculo-ventricular node than the right, the latter also innervates that structure, and, doing so, may at the same time affect both the upper and lower chambers without ventricular depression being a result of auricular inhibition.

Auriculo-Ventricular Tachycardia. — A well-recognized condition, and one of more frequent occurrence, or which has been more frequently observed, is that in which auricle and ventricle are alike and equally accelerated, if we may consider the jugular tracing under these circumstances as

[1] *Quarterly Journal of Medicine*, vol. vi., No. 21.

still giving evidence of jugular activity. It may be temporary or persist, but is frequently paroxysmal, and arises in different conditions and at various ages. As we shall learn, it is, however, questionable whether paroxysmal tachycardia can be regarded as an auricular state. Temporary auriculo-ventricular tachycardia, while it may prove fatal, cannot strictly be regarded as a disorder of cardiac motion. It is the normal acceleration of action witnessed under conditions of excitement and exertion, the whole heart—sinal, auricular, and ventricular—quickening in order. When it proves fatal, as it occasionally does even in the athlete insufficient preparation for such exertion, or some unsus-

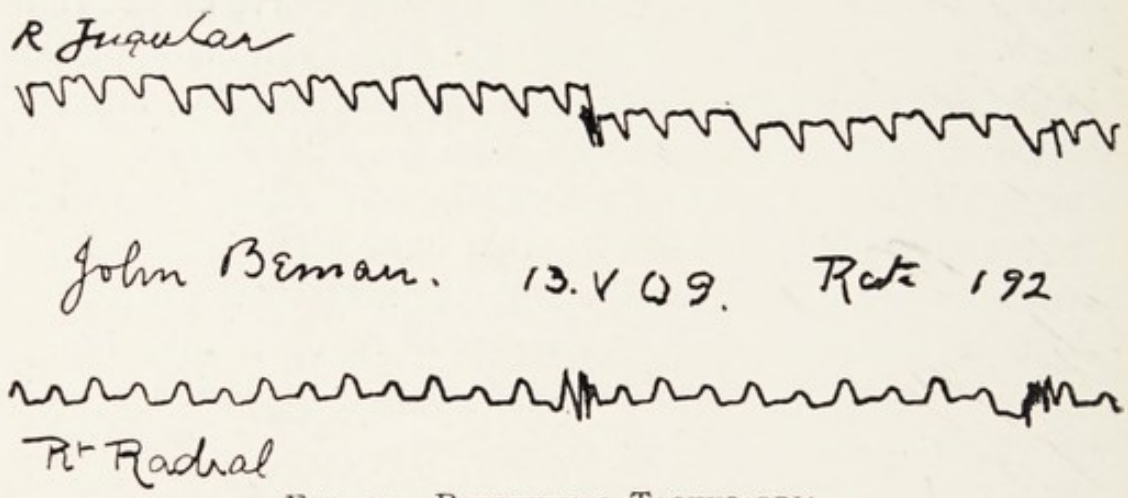

FIG. 31.—PAROXYSMAL TACHYCARDIA.

pected myocardial degeneration determines the issue. To the mechanism of such failure I shall again have occasion to refer when considering the prognosis of tachycardial conditions.

Whether one be justified in applying the term 'persistent' to a type of case in which the pulse-rate is for a lengthened period above the average normal, may at present be questioned. Our knowledge of auriculo-ventricular tachycardia is chiefly derived from a study of the paroxysmal variety. One has seen persistent cases from time to time. They are, perhaps, more frequently met with than persistent bradycardia, of the organic nature of which we now know more than we do of that of these cases. But observers have noted such a condition to persist for years, if we may assume them to have been of this character in the absence

of auricular tracings. A patient, however, is not continuously under observation, and it may be that there are periods of comparative slowness of the heart's action which escape notice, and the occurrence of which would invalidate the use of the qualifying adjective. The fact remains, however, as Balfour has remarked,[1] that cases occur in which 'the heart-hurry may last for years' and the patient attend to his usual avocations with little subjective sense of discomfort. We know that in Graves's disease an auriculo-ventricular tachycardia of high rate may endure for a very considerable period and justify its title to be regarded as persistent, even though it may ultimately assume a phase of irregularity or extrasystolia, pass into normal action, or recovery, or, again, as sometimes happens, into a moderate bradycardia, associated with a degree of myxœdema.

The varying organic circumstances under which the condition arises, and the imperfect pathological examination of such cases as have been investigated after death, do not justify the expression of a positive opinion as to the essential cause or causes of the state. This lacuna in observation will, doubtless, gradually be filled, and even now we have reason to believe that such attacks, without discoverable physical basis in the first instance, tend to induce organic change, as I shall again indicate when discussing the nature of so-called auricular fibrillation. Hoffmann's interpretation of paroxysmal tachycardia given by Vaquez[2] may apply to these cases also—namely, that the condition represents an extrasystolic state.

Carefully distinguishing them from this class, we must place among tachycardiæ the persistently accelerated heart of *Graves's disease*. In such a commencing thyroidism, with its characteristic concomitants of thyroid enlargement, exophthalmos and muscular tremor undeveloped or imperfectly developed, may easily be mistaken for the persistent tachycardia of which I have just spoken, its real nature only transpiring later. This auriculo-ventricular tachycardia is usually regarded as a result of an excess of thyroid secretion

[1] 'Clinical Lectures on Diseases of the Heart,' p. 279.

[2] *Op. cit.*, p. 277.

entering the blood, and acting upon the heart-muscle directly, or through the agency of the accelerant nerves. That hyperthyroidism, if not the first, is *an early* factor in the production of this form of tachycardia, is probable, and there is some evidence that a diminished output of the secretion is associated with a fall in the heart's rate. With the atrophy of the thyroid gland, which at times succeeds the thyroid enlargement of Graves's disease, a bradycardia may take the place of tachycardia, and the patient manifest, as I have said, some degree of myxœdema. The underlying cause of thyroidal tachycardia—namely, hyperthyroidism—probably acts *per vias nervosas* on the cardio-motor nervous system.

In dealing with *paroxysmal* auriculo-ventricular tachycardia we are on surer ground than with the non-paroxysmal variety. The condition is of frequent occurrence and its clinical study more advanced.

Paroxysmal tachycardia may be observed in hearts which present no evidence of organic disease, in those in which the myocardium is degenerated and in valvular disease of the organ.

It arises suddenly, may last for hours or days, and subsides at times into normal action as suddenly as it arose. It is frequently observed to be recurrent, the same patient at intervals presenting the phenomena characteristic of the state. It is met with among men with much the same frequency as among women, and has been observed in the young as well as in the old. The pulse-rate is frequently very high, and is best counted by auscultating the heart or by a graphic reproduction of the movements. Pulse-rates of 150, 190, 200, or more in the minute, are frequently observed. The counting of high rates can only be satisfactorily done with a stop-watch having a large seconds hand, and by multiplying fractions accurately counted. The power to do so probably varies with individuals.

The waves registered by the sphygmograph during tachycardia of high rate (Fig. 31) show the incidents of the cardiac cycle to be so abbreviated that the blood-flow through the chambers appears to be continuous rather than intermittent, systole being imperfect and diastole not more than indicated,

while the cervical phlebo-arteriogram loses its features and becomes, as it is termed, of the ventricular type. In other words, the contractile power of the auricle is impaired chiefly by repletion. Hoffmann's view that the condition is due to a regular succession of additional sinal systoles propagated, is interesting but not convincing.[1]

There was much to be said for James Mackenzie's conception of a nodal rhythm in explanation of these cases. Mackenzie, however, considered both ventricle and auricle to be simultaneously stimulated by the abnormal excitability of the node of Tawara to excessive action. The term was, however, only provisionally used by him,[2] and was, I think, too completely abandoned on the advent of 'auricular fibrillation.' The stimulation of the auricle by the node of Tawara is not, as we have seen, anatomically probable, but on the grounds stated in these pages, the conception of tachycardia as a condition of massed ventricular extrasystole, as Vaquez suggests,[3] has, for the reasons given in Part I., much to recommend it.

In the lower rates the incidents are naturally better marked. This continuous flow of blood through the chambers and its imperfect propulsion appears to be indicated by the increase in the area of cardiac dulness in many cases during persistence of the attack, which has been noted by various observers, though denied by some,[4] but was first fully described by Martius.[5]

It is a remarkable feature of this condition, especially when it arises in hearts which are otherwise normal in action and structure, that the subjective discomfort of the patient, who might more appropriately at times be regarded as a subject rather than a patient, is very slight. A case affected with valvular disease may even show a degree of cyanosis without having much subjective discomfort. It is no less remarkable that this lightning flicker of cardiac action should at times, and that frequently and even as a rule, suddenly pass into a perfect quietude of normal rate

[1] *Deutsch. Archiv f. Klin. Med.*, vol. 78, p. 64.
[2] 'Diseases of the Heart,' 1908.
[3] *Op. cit.*, p. 237.
[4] Hoffmann (*loc. cit.*, p. 57).
[5] *Die Tachycardie*, 1895.

and rhythm. Some patients describe the subjective sensation of an intrathoracic jerk or stop when the tachycardia passes into a normal rate. We shall, however, learn, when dealing with the prognosis of the regular and irregular tachycardiæ, that the condition may under certain circumstances prove fatal, and that there may be an interesting difference in the apparent mechanism of such fatality.

Knowing, as we now do, the complete innervation of all varieties of cardiac muscular fibre which are functionally active, including the tracts of so-called primitive but better-named nodal muscle, it appears to be more reasonable to regard these sudden explosions, with undue acceleration and as sudden resumptions of normal action, as caused rather by variation in nerve control than by a sudden access and abolition of an intrinsic muscular excitability having no relation to the nerves, which may be demonstrated clinging to the affected structures.

Auricular Fibrillation.—I have never myself observed, nor am I aware that anyone else has met with, a case in which auricular bradycardia was unassociated with ventricular bradycardia; and with auriculo-ventricular, or what some term 'sinus,' bradycardia we shall deal in its own place. We have seen that there may be an auricular tachycardia of high rate without apparent proportionate acceleration of ventricular action, and in the absence apparently of such conditions of 'block' as prevail in certain cases of ventricular bradycardia. It is therefore not unreasonable to expect that experience may yet produce an auricular bradycardia without associated slowing of the ventricle.

In the meantime this gap in the regular sequence of varieties of auricular action left by the absence of such a bradycardia, may be occupied by an interesting paretic or distended state of the auricle, which Lewis[1] has described under the term 'auricular fibrillation,' and which has been regarded as the greatest discovery in cardiology of modern times.[2]

[1] *Heart*, 1909-10, vol. i., p. 306.

[2] Mackenzie, 'Oliver Sharpey Lecture.'

This state is characterized by a condition of contractile impotence, in which, however, there is considered to be a fine mural stimulative activity of the muscle elements. In the electro-cardiogram the condition appears as a fine tremor of the auricle.

This impotent distension of the auricle is always found associated with a well-marked irregularity of ventricular action, of which it is regarded as the direct cause, a shower of auricular impulses falling upon the ventricles by way of the auriculo-ventricular bundle, and causing those chambers to act with an irregularity often of high rate. The ventricles may indeed, under these circumstances, beat irregularly with considerable force, the force of the contractions varying, although the chamber sending the stimulative impulses is in a condition of mural tremor and cavital impotence. This is explained as a rule on the 'all or none' principle of cardiac muscular contraction, and by the assertion that the resulting ventricular contraction bears no relation to the amount or degree of auricular stimulation, but probably to the period in the post-refractory phase of ventricle which the stimulus happens to hit.

It is assumed by some that as faradization of the auricle in experiments on animals causes auricular paresis, with fine tremor of its walls, and at the same time irregular action of the ventricle, these conditions so induced and the auricular fibrillation with irregular ventricular action seen clinically are identical states. For the truth of this assumption Lewis believes he has obtained proof, under non-experimental circumstances, in the fibrillating auricle and jactitating ventricle of a horse which had shown evidence of cardiac dyspnœa during life, and the heart of which was exposed as soon as possible after it had been killed, by being shot through the brain.[1]

On phlebograms auricular fibrillation is regarded as represented by a series of minute wavelets in the diastolic period.

Although the condition may be met with when there is

[1] 'Mechanism of the Heart-Beat, p. 230.

no valvular disease of the heart, and at times when the aortic or arterial orifice is valvularly disabled, the majority

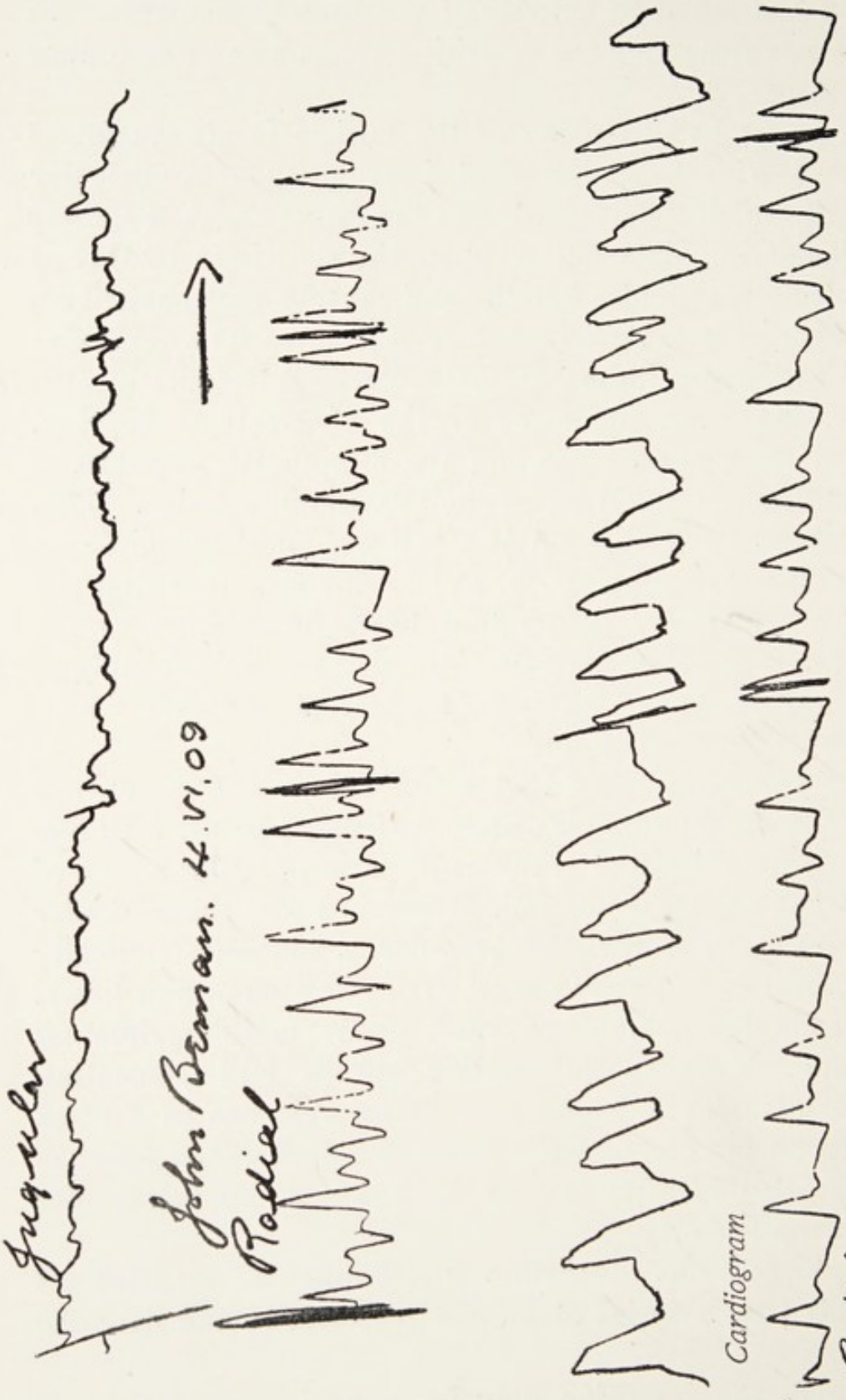

FIG. 32.—VENTRICULAR TACHY-ARHYTHMIA.

of cases occur in connection with disease of the auriculo-ventricular orifice, and among these most frequently when

that orifice is contracted—that is, in mitral stenosis. Tricuspid stenosis alone is too rare a lesion to afford clinical material for comparison. Indeed, the same case may at one time exhibit a regular auriculo-ventricular tachycardia, at another the irregular ventricular action characteristic of 'auricular fibrillation,' at still another an auriculo-ventricular bradycardia with the cantering rhythm, and finally normal and undisturbed action in rate and rhythm of both auricles and ventricles.

Lewis believes also that the electro-cardiogram may show a partial tremor of the auricle, which presages a fibrillation. But whether this be so or not, it is certain that the distended auricle of auricular fibrillation is only met with in the presence of great irregularity of the ventricle, in which the chief feature is the abbreviation or elision of the diastolic interval, relieved at times by a prolongation of diastole and ventricular systole, large in proportion to the blood-content of the chamber (Fig. 32)—in short, in connection with a condition of irregular and tachycardial ventricular extrasystolia.

This largeness of occasional ventricular contraction following abbreviated events is regarded as a compensatory pause in post-extrasystole, during which a larger production of stimulus material is rendered possible. Whether this supposition holds good or not, it is certain that there is more blood in the chamber for projection at the same time, and that it is represented by the larger wave registered by whatever instrument the registration be made, whether finger, eye, or ear. It is likewise certain that the murally active and cavitally impotent auricle is filled with residual blood resulting from the stagnation attending the diminished diastole and imperfect systole of the ventricle.

It is no doubt heterodox to suggest—for orthodoxy is at times of rapid growth—that there is a possibility or even probability of the ventricle taking the initiative in the production of that condition of the auricle in which, vainly trying to perform its function, it collapses in fibrillation. Even Mackenzie's nodal rhythm, which had a good deal to be said for it at one time, is not now mentioned in this con-

nection among the instructed, although some considerations set forth in Part I. rather support such a possibility. It is to be noted, however, that the monopoly not long ago enjoyed by the sinu-auricular node as initiating the cardiac cycle is now regarded as not invariable. We have also seen that a tachycardia of the auricle alone may exist for a lengthened period by which the ventricle may be little if at all influenced, although the response of the latter chamber to the accelerant action of atropine shows that it can respond normally to stimuli. There seems no sufficient reason, therefore, why the ventricle should not also manifest an independent rhythm, as in some cases is undisputed, and even influence indirectly, if not directly, the contractility of the auricle. Indeed, the extrasystolic theory of paroxysmal tachycardia supports the view that irregular tachycardial ventricular extrasystolia may be the active and precedent cause of auricular fibrillation.

But there should be no difficulty, on the part of those who accept the anatomical and functional continuity of the auriculo-ventricular node and bundle with both auricle and ventricle, in accepting also a reversed conduction of ventricular riot to auricle, the thin-walled and distended condition of which would be more calculated to pass into paretic tremor than to be roused to more vigorous action by the ventricular stimulus. Experimentally, we know, Gaskell considered he had demonstrated the reversibility of muscular conduction.

Retrograde stasis in the chambers of the heart, which is not at present recognized by some myogenists, has, however, we shall find, something to be said for it still, and may quite probably again find a place in the explanation of cardiac failure in some cases.

On the argument developed in these pages, and founded on the view of the innervation of the heart discussed and the anatomy of the nodal structures, there is no straining of reason in regarding the irregularity of ventricular action as preceding the distension and tremulous impotence of the auricle on the lines of a massed and irregular ventricular extrasystolia somewhat resembling Mackenzie's theory of

'nodal rhythm,' which was, I think, too precipitately abandoned for Lewis's assertion of auricular fibrillation as the effective cause of ventricular riot. Nor is it unreasonable to attribute the disappearance of this condition to a restoration of more effective and regular ventricular action, which relieves the distension of the auricle and permits it to recover a normal tone and rhythm. In this connection reference may be made to a paper of Professor Cushny's on 'The Action of Digitalis in Heart Disease.'[1] While he eliminated vagal inhibition by the use of atropine, in the cardiac slowing induced by digitalis in auricular fibrillation, he also found that there was no blockage of auricular impulses in the auriculo-ventricular bundle. He suggests that improved nutrition of the ventricle by better contraction may render it less responsive to auricular stimulation. In any case, the ventricle slows in spite of the auricular fibrillation, and the latter disappears with the slowing of the ventricle. Asked by a participant in the debate which followed whether in this matter we should have to 'go back to the beginning' in trying to account for the action of digitalis, Professor Cushny advised 'a clean slate,' the old explanation of blockage of impulses having 'proved to be incorrect.' Under these circumstances the effect of a showering of auricular impulses by the tremulous cavity may also be called in question.

One feature to be noted in connection with that irregularity in mitral stenosis, which is regarded as dependent upon the mural activity and cavital impotence of the auricle, is that a presystolic bruit existent prior to the fibrillation may be observed at times to persist during that phase, even when the bruit is of the rolling or crescendo type.[2] If the auricle under these circumstances be impotent, the bruit must be dependent upon ventricular aspiration and systole. The *rate* of irregularity—the amount of ventricular extrasystole—has, however, probably an influence upon the retention or loss of bruit, as with high rates of even *regular* auriculo-ventricular

[1] Proceedings of the Royal Society of Medicine, vol. v., No. 9, p. 200, Therapeutic Section.

[2] *Lancet*, October 8, 1910.

tachycardia—that is, in the absence of auricular fibrillation—the bruit is abolished in mitral stenosis[1] (Fig. 31).[2]

In both rhythmic tachycardial ventricular extrasystolia (auriculo-ventricular tachycardia) and in arhythmic tachycardial ventricular extrasystolia (auricular fibrillation, tachy-arhythmia) the auricle is impotent and dilated, but ventricular irregularity appears to be necessary for the presence of so-called 'auricular fibrillation.'

[1] *Lancet*, October 8, 1910.

[2] These tracings were taken by my then House Physician, Dr. A. E. Gow. The radial shows the alternating ventricular action so frequently observed in these cases—an alternation which is not always so regular as in this instance, but at times has an extra-systolic irregularity approaching that of ventricular tachy-arhythmia.

CHAPTER III

AURICULO-VENTRICULAR BRADYCARDIA; VENTRICULAR BRADYCARDIA

Auriculo-Ventricular Bradycardia.—Abnormally slow pulsation limited to the sinus or auricle has not been recorded, although quite possible of occurrence. The next condition to be considered is, therefore, auriculo-ventricular bradycardia, which is termed by some 'sinus bradycardia' (bradycardie sinusale).

In a certain number of cases a habitually slow pulse, normal to the individual, is probably one in which there is an equal slowness of auricular and ventricular systole. The majority of cases, however, of auriculo-ventricular, or what has also been termed 'true' bradycardia, are those observed after acute diseases, such as pneumonia, or after the motor perturbations attending auriculo-ventricular tachycardia or 'auricular fibrillation,' in cases of valvular disease of the heart. Of the latter, the following is an example which exhibits the equal retardation of auricular and ventricular systole, and a proportionate lengthening of the interval between the waves denoting auricular systole and the carotid pulse—the so-called 'a.c. interval' (Fig. 33). The condition is usually transitory, and is soon replaced by a normal rate and rhythm of cardiac action. Fever, emotion, exertion, and various chemical and animal stimuli such as alcohol and the secretion of the thyroid gland, which act as I believe through the nervous mechanism of the heart, normally produce an accelerated action of both chambers. Their failure to do so quite as validly argues a want of

response in a portion of the nervous as in a part of the muscular mechanism.

As I have said, auriculo-ventricular bradycardia is usually transient, but not invariably so. In a case of Graves's disease with exophthalmos, tachycardia, mitral regurgitation,

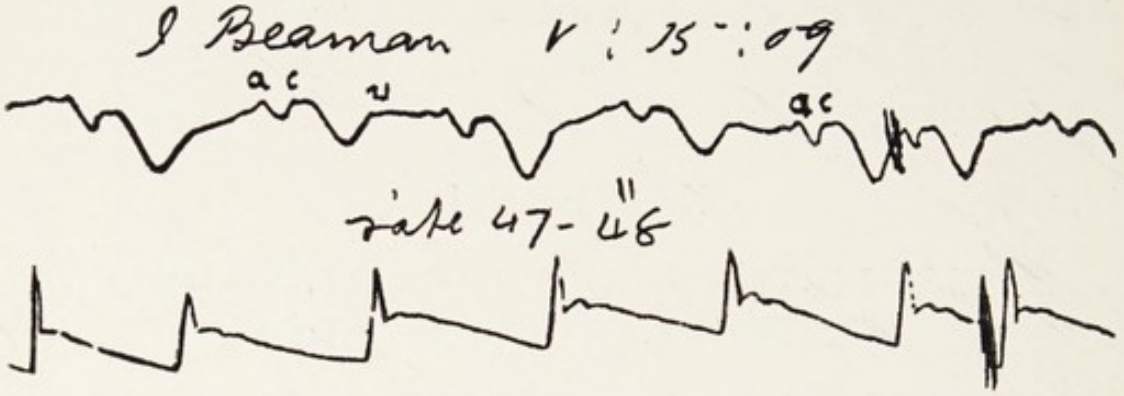

FIG. 33.—JUGULO-CAROTID AND RADIAL TRACINGS OF AURICULO-VENTRICULAR BRADYCARDIA WITH APICAL TRIPLE RHYTHM.

and muscular tremor which ultimately passed into one of thyroid atrophy, disappearance of exophthalmos, and the establishment of a slow pulse, I have known the latter to continue, with a pulse-rate of 48 to 60, to last for years, and on examination by the clinical polygraph to show a correspondence of auricular and ventricular action as the follow-

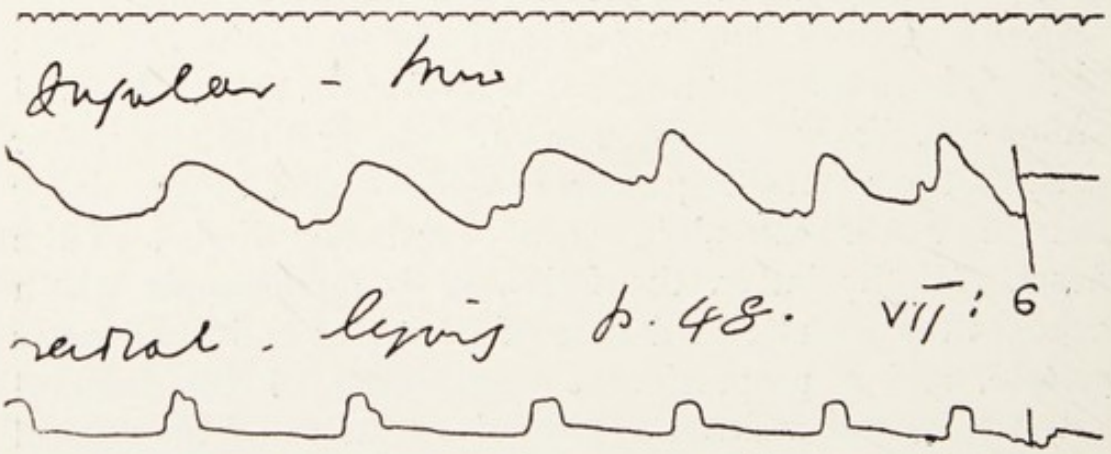

FIG. 34.—PERSISTENT BRADYCARDIA SUBSEQUENT TO THYROIDAL TACHYCARDIA.

ing tracing shows (Fig. 34). The tracing was made with a non-stationary instrument and is not very good, but sufficiently shows the bradycardia to be auriculo-ventricular. The a.c. interval is not materially increased, and corresponds in proportionate duration to the systole of auricle and ventricle. In this case there is no auriculo-ventricular blockage, for on

one occasion the subject preceded me up a long staircase, at the top of which I seized the opportunity of counting her pulse, which had risen to 78, and after a short interval fell again to her now usual rate of 48-54-60.

Ventricular Bradycardia.—By this term I mean to denote a retarded action of the ventricle of a more persistent type limited to that chamber, just as by the use of the term 'auricular tachycardia' an accelerated action limited to the auricle was implied. The term, moreover, involves no theory of causation, but is purely descriptive. This cannot be said of its synonym 'heart-block,' while the only recommendation of its other equivalent, the 'Adams-Stokes syndrome,' is that it perpetuates the memory of two honoured names in medicine, but is in no way descriptive of the condition.

For the now general acceptance of the fact, that in these cases the retardation of action is limited to the ventricle, we have to thank the graphic method, and especially the use of the clinical polygraph of and by Dr. James Mackenzie. The condition, however, was fully appreciated and described by Chauveau so long ago as 1892.[1] Skiascopy has also confirmed the results of sphygmography, and the electrocardiograph supports both.

Before, however, the fact of independent auricular and ventricular action was generally accepted, a distinction of clinical value had been made into accelerable and non-accelerable bradycardia. Dehio,[2] in 1892, drew a distinction between cardial and extracardial cases, the former being assumed to depend upon a cause in the heart itself and the latter upon an introduced cause. While cardial cases were found to be uninfluenced by circumstances normally inducing an acceleration of the heart's action, such as exercise, alcohol, and atropine, extracardial cases responded to these.

A ventricular bradycardia which ultimately becomes constant and invariable may have periods of accelerated action, and in some cases, while the usual condition of the heart is that of bradycardia, it may not, up to the demise of the

[1] *Revue de Médecine*, vol. v., p. 161.

[2] *St. Petersburg. Med. Wochenschrift*, No. 1, 1892.

patient, evince a *constant* retardation. Such cases are said to exhibit incomplete or partial 'block'—are cases of variable ventricular bradycardia.

A variable ventricular bradycardia may, moreover, be an acute mode of cardiac failure and associated with the mental phenomena of a sense or fear of impending death, like those cases of cardiac failure without pain, to which Sir William Gairdner applied the term 'angina sine dolore,' but in which there is the sense of impending death, without necessarily any retardation of ventricular action. Of this condition, which I termed 'syncopal bradycardia' I have given an instance in some detail when dealing with sensory disorders (p. 118).

Persistent ventricular bradycardia, or 'heart-block,' has since then received much study in view of the anatomical and physiological researches dealt with in Part I. A considerable number of cases have been watched during life and examined after death, and more or less disease—interstitial, neoplastic, acutely inflammatory and aneurismal—has been discovered, which has been considered to interfere with the conduction of impulses from the auricles to the ventricles by way of the auriculo-ventricular tract or bundle usually associated with the name of W. His, junior.

That the cases examined fully after death have usually been those in which ventricular retardation was observed during life is only natural, and that bradycardia observed during life should have been referred to those conditions is equally natural, in view of the results obtained by physiological experiments on the lower animals. in which the bundle, among other textures, has been involved.

But we have also learned that the same results have not been obtained by all experimenters, and it is admitted that pathological conditions which have in some cases been associated with 'block' have in others not been so associated.

It is desirable that more frequent examination should be made of the structures in question in cases which have *not* presented evidence during life of so-called heart-block. That cases occur in which there is well-marked disease of

the bundle of His without ventricular retardation is indisputable. In such cases it is usually held that there must have been a sufficient escape of its fibres from disease to permit conduction. Precisely how much disease of the bundle is necessary, or at what point it should be situated to cause the ventricle to take on its own action has not been determined; but when ventricular bradycardia persists, the interruption of the bundle has been assumed to be complete at some point, however much or little disease has been discovered, and where bradycardia has been variable, such interruption has been considered to have been incomplete.

Whether the auriculo-ventricular node with its branches, which is now generally regarded as a neuro-muscular organ, be looked upon as a bridge between auricle and ventricle for the conduction of muscular impulse from one chamber to the other, or, as has been done in Part I., as an organ dominating ventricular action, there can now be no reasonable doubt that disease affecting it may materially interfere with its function, which may also, however, be interrupted when gross lesion of the structure is not to be detected. The latter circumstance, however, in the case of a neuro-muscular organ is not remarkable.

To display the clinical history of ventricular bradycardia I shall follow the method pursued in Part II. in giving the history of cases of sensory disorder, and relate one out of several cases I have seen in consultation and in hospital practice, because of the more continuous opportunity it afforded for clinical observation during life and for anatomical investigation after death. Persistent ventricular bradycardia is not often met with.

A man, thirty-one years of age, was admitted under my care to the Great Northern Central Hospital on November 23, 1906, and discharged 'improved' on January 9, 1907. He had suffered from rheumatic fever when fifteen years of age, and on four subsequent occasions. He denied having had syphilis, and the Wassermann test was negative. Eighteen months prior to admission he began to have attacks, in which he fell down and became unconscious for

a short time, but he had no dyspnœa or other sign of cardiac failure such as anasarca.

When I first examined him at the hospital the heart's apex-beat was in the fifth space $3\frac{1}{2}$ inches from mid-sternum, the area of cardiac dulness measured $4\frac{1}{2}$ by $5\frac{1}{2}$ inches, there was a systolic bruit at the apex with a faint diastolic bruit, and a systolic bruit at the cartilage of the fourth left rib and in the fourth space. At the aortic base there was a systolic bruit. The pulse-rate was 66 and the other systems normal. He had, in other words, obstructive disease at the aortic, and regurgitation through the mitral, orifice. On November 28 it was noted that the pulse-rate was 30, and that the patient had experienced 'half-fainting fits.' Sphygmograms taken on December 4 show that the pulse varied in character, being at times regular and within a few minutes, exhibiting groups of pulsations separated by a bradycardial pause (Fig. 35, *a*, *b*). The tracings taken on December 6 show that the patient had well-marked dissociation of auricular and ventricular contraction. The pulse-rate was 26, and during diastole several faintly audible added sounds could be heard (Fig. 35, *c*).

On December 11 the patient had an attack of pronounced syncopal bradycardia, and my house physician at that time (Dr. Mackenzie Gunn) made an interesting note and some instructive tracings. The note runs: 'To-night the patient had one of his so-called fits. It lasted from 6.30 to 7.30. He complained of a feeling of faintness and numbness all over his body. His pulse ranged from 30 to 40. It was very irregular and intermittent. It came in two, three, four or more beats, followed by a long pause lasting two to four seconds. Now and then, at irregular intervals, the pulse completely stopped for about ten to twenty seconds. There was no difference between the beats at the radial pulse and at the apex-beat. Listening during the long pauses, a number of very weak beats could be indistinctly heard. They got quicker towards the end of the pause. Blood-pressure went up from 100 to 190 and came down to 105 when he got better. The pulse got much more regular as the fit passed off and the pauses disappeared. Breathing

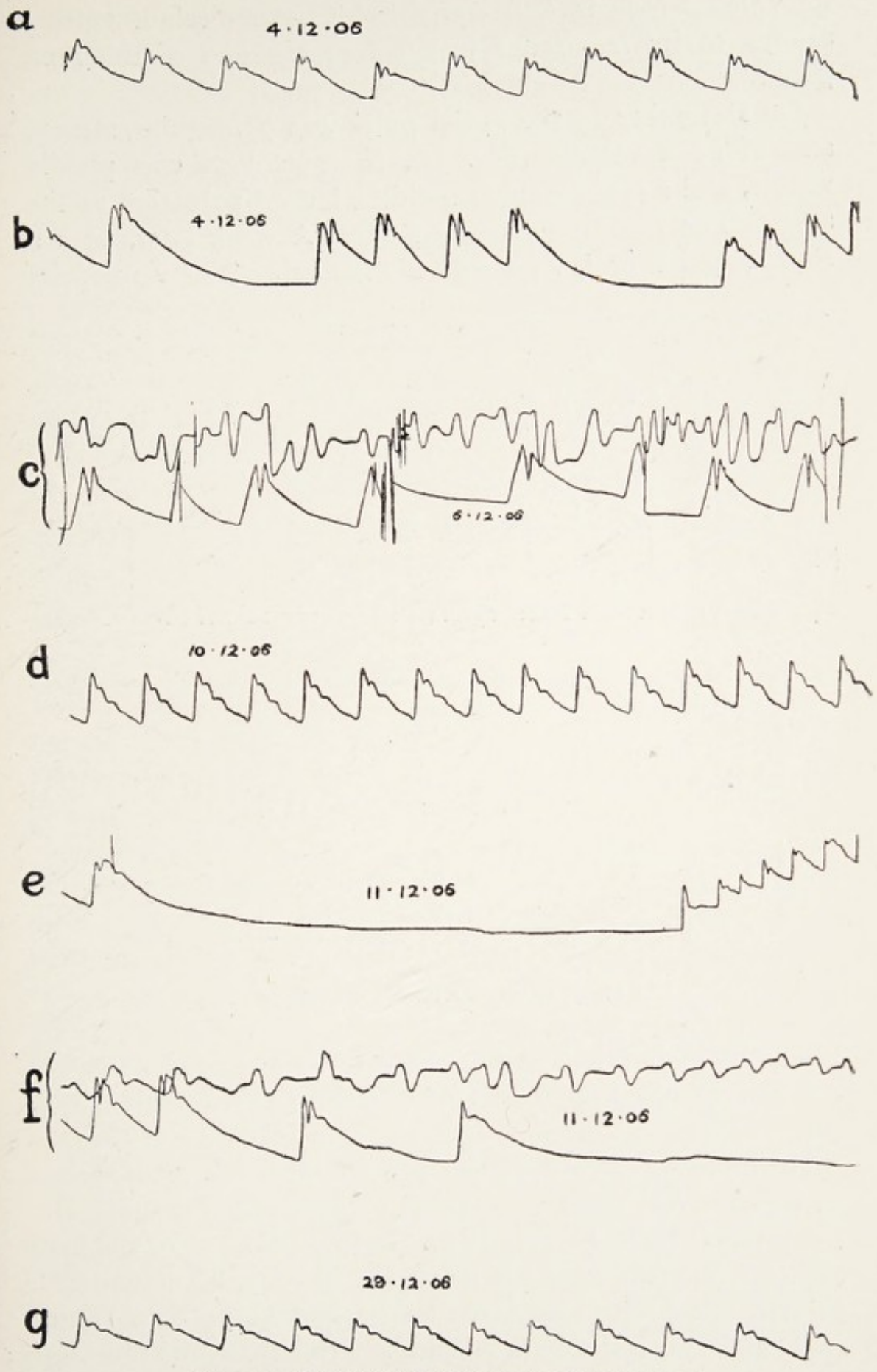

FIG. 35.—VARIABLE VENTRICULAR BRADYCARDIA.

was also irregular and somewhat Cheyne-Stokes, getting always to its maximum just before the end of the long pause.'

On December 29 the radial pulse was 75, regular, strong, and of good volume (Fig. 35), the apex-beat was plainly visible, and a systolic thrill could be felt on palpation at the apex. The blood-pressure was 170. A week later, with a regular pulse of 50, he was discharged. The ventricular

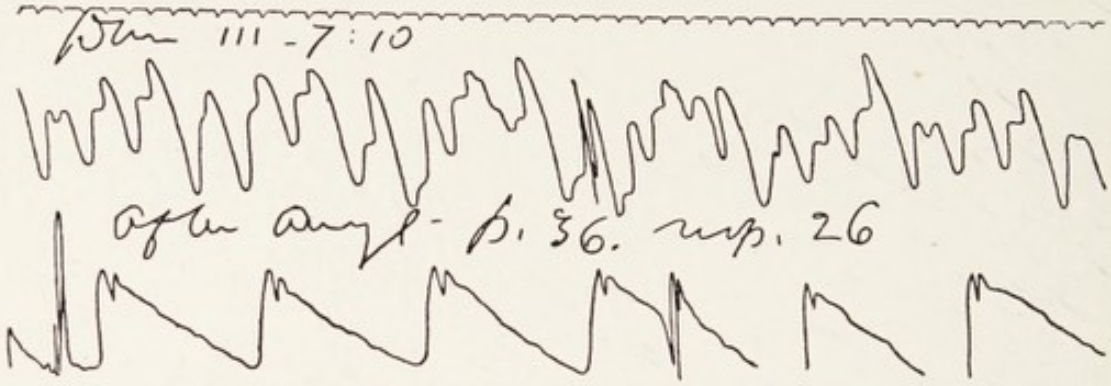

FIG. 36.—VENTRICULAR BRADYCARDIA UNAFFECTED BY NITRITE OF AMYL.

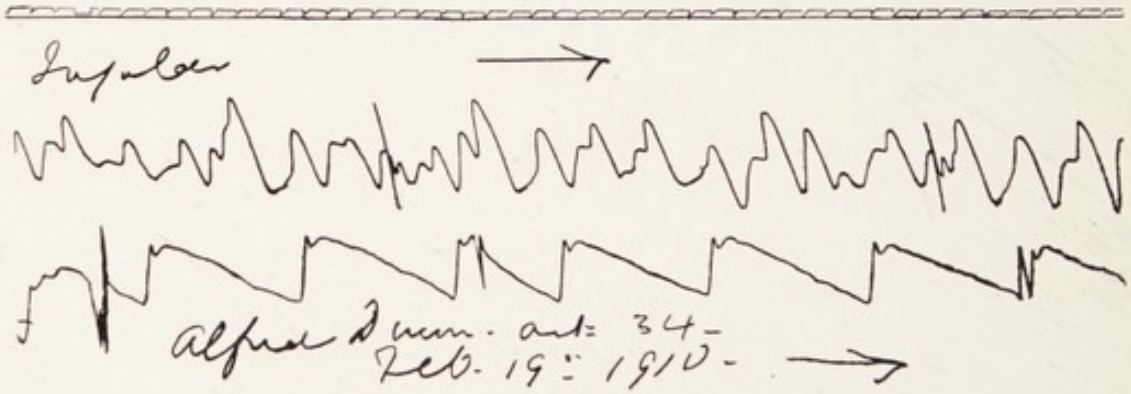

FIG. 37.—VENTRICULAR BRADYCARDIA.

bradycardia, in other words, was variable at this stage of the case, or to use the current expression, the heart-block was incomplete.

No more was heard of the patient until his readmission to the hospital three years later, on February 14, 1910. In the interval he had worked as a 'beer-bottler.' For the first eighteen months after his discharge in 1907, he stated that he had felt pretty well, but that he had never been 'quite well' since his last stay in hospital. After that time fainting-fits recurred, but he is noted as having been 'pretty fit' until

five weeks prior to readmission, when the fainting-fits, headache, and pain over his heart became much worse and more frequent. In these fits he fell down and lost consciousness. The patient was a careful liver, a total abstainer, and non-smoker.

On February 19, when I visited the hospital, the following note was made: Pulse 42, regular. Apex-beat sixth space, $5\frac{1}{2}$ inches from mid-sternum. Area of cardiac dulness 5 by $6\frac{1}{2}$ inches. Bruit systolic, loudest at apex and at the third left cartilage. Right cardiac dulness about mid-sternum. Bruit traceable to the left, becoming fainter towards the mid-axillary line and traceable to the right for about the same distance, but less distinctly. It was also audible in the intervertebral grooves, but loudest on the left. Liver dulness from sixth rib to three finger-breadths below the right costal margin. Some crepitation at the base of the left lung posteriorly. His urine on admission had a trace of albumin, but with the microscope showed nothing abnormal.

On the 29th his pulse-rate was 36, respiration 24, temperature normal, and in the evening slightly subnormal. While the physical signs were the same, a systolic bruit in the aortic area was more pronounced and was transmitted into the carotids. The aortic second sound could not be heard, and there was no reduplication audible. In short, since the previous stay of the patient in hospital his heart had become more hypertrophied and its rate had sunk to a lower level, where it remained more persistently, although there is a note of his pulse having risen to 60 on one occasion during his stay. The ventricular bradycardia had become less variable, the heart-block more complete.

During his stay in hospital on this occasion there is no note of his having had any attacks of syncopal bradycardia, and at his own request he was discharged on April 6.

A tracing taken on February 19 (Fig. 37) shows a 2 : 1 auriculo-ventricular rhythm, while one made on March 7, after the inhalation of nitrite of amyl, shows an unaffected radial pulse of 36, and no alteration of auricular rhythm (Fig. 36).

Soon after his return home the patient again began to

suffer from pain in the abdomen, heart, and left shoulder, and on April 11 he had a fit similar to those previously experienced. He was therefore again admitted to hospital on April 14, 1910. The physical signs were unchanged. During his stay in hospital of nearly four weeks, the average pulse-rate was on this occasion rather lower than formerly, never exceeding 40, except on one occasion when it reached 48, and that only the day before death, while the respiratory-rate was rather higher. Tracings taken on May 9 and 10 show a less pronounced auricular action, and one taken after the patient had had four doses of $\frac{1}{100}$ grain of atropine and $\frac{1}{60}$ of strychnine showed little acceleration of the heart's action. The rate, however, did rise after the exhibition of these drugs from 40 to 48.

He died syncopal at 3 a.m. on May 12, having had several severe attacks between April 29 and the date of his death.

The body was examined twelve hours after death, and it is unnecessary in this place to give full details of the whole. Suffice it to say that the organs generally showed no disease other than the congestion attending stasis in the circulation, and that the brain and spinal cord were healthy to the naked eye.

The heart *in situ* was greatly enlarged and exposed. There were some costo-pericardial adhesions, but none between the heart itself and the sac. The heart was placed in formalin solution for subsequent examination, which was neither necessary nor wise, as the shrinkage of texture involved rendered it difficult to estimate the capacity of the chambers and orifices of the organ. This, however, in this case was a minor matter. The aortic orifice was narrowed by fusion of the cusps, and at the base of the right coronary cusp there was a portion of calcified texture (Fig. 38). The mitral orifice was contracted chiefly by formalin, but the mitral curtains were thickened and had undoubtedly been incompetent during life. There was slight atheroma of little importance in the aorta, and the coronary orifices were patent.

For *microscopic examination* I handed the heart in the first instance to Professor Keith, who was good enough to inves-

tigate the condition of the sinu-auricular node, the auriculo-ventricular node, and a portion of the auriculo-ventricular bundle. I personally completed the examination of the bundle and of other parts of the heart.

The arteries in the *sinu-auricular node* are thickened and the interstitial textures hypernucleolated. The muscular fibres of the node seem normal, as also do the fibres themselves of

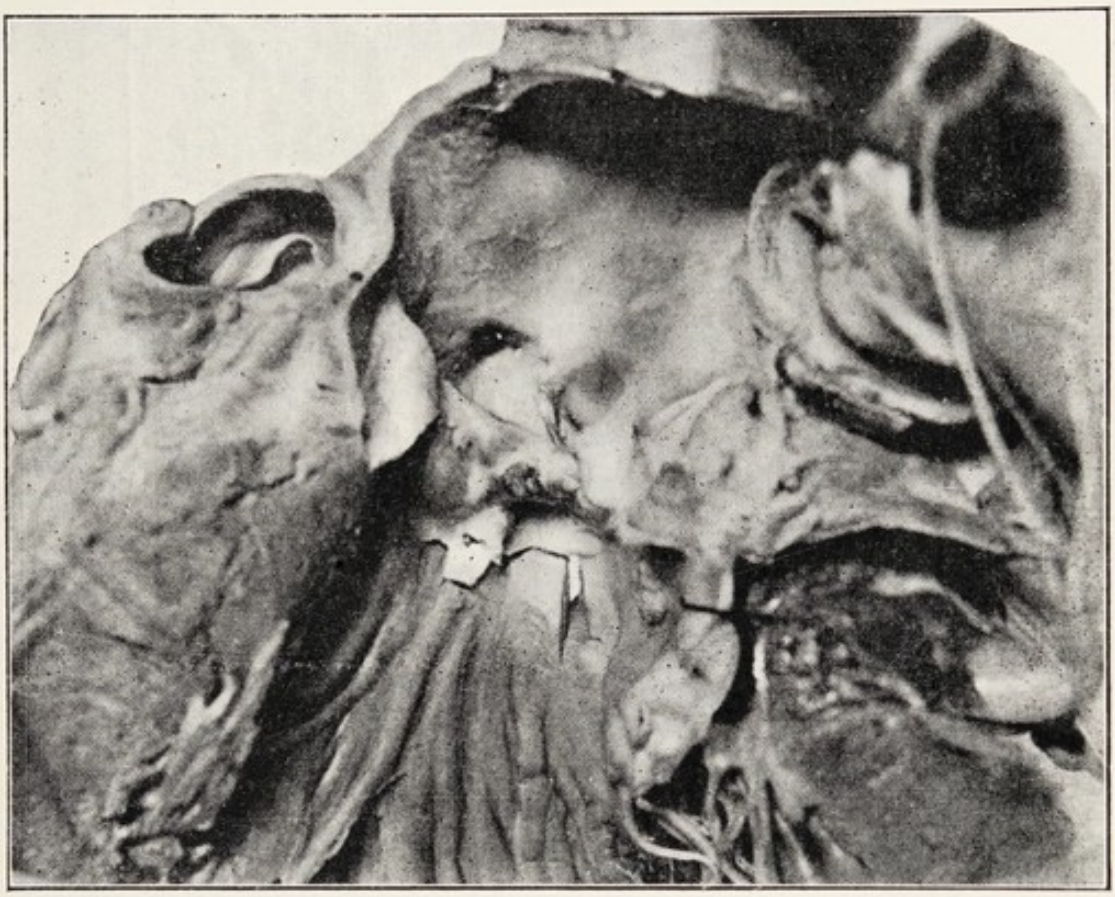

FIG. 38.—LEFT VENTRICLE, AORTIC CUSPS, AND AORTA: PARS MEMBRANACEA SHOWN.

Below it a space marks a portion in which the bundle was normal, and to the right a larger space at which the calcification was situated. In the interval between these the bundle was increasingly altered as it neared the calcification.

the auriculo-ventricular node. The auricular muscle generally is normal, as are ganglion cells abutting upon the sinu-auricular node. The auriculo-ventricular tract (bundle of His) is normal at its commencement beyond the node, or shows only a slight increase of interstitial tissue. As sections proceed towards the right, they show increasing interstitial tissue, until, at a point to the *left* of the calcification at the base of the right coronary cusp, the muscular texture of the

bundle appears strangled by the increased fibrous tissue, and the arteries of the part are greatly thickened and some of them obliterated. The calcification itself appears to be beyond and to the right of the bundle muscle (Fig. 38).

This case sufficiently illustrates the rise, progress, and result in ventricular bradycardia. The condition, variable in its earlier stages, when the syncopal attacks of an inhibitory character, followed by accelerated action (Fig. 35, *e*, *f*), were more numerous, passed into the sluggish, slow pulsation incapable of being influenced by any external excitant such as emotion, movement, or medicinal agents, which through the nervous system produce, under normal circumstances, quickened pulsation (Figs. 36 and 37).

Clinical and pathological evidence supported the assertion of the patient that he had not suffered from syphilis, and there was every reason to regard the causal lesions as of rheumatic origin.

The ventricular bradycardia of earlier and middle life is in a considerable proportion of cases associated with the consequences of syphilitic infection, or of new growth when not attributable to other acute septic states. In later life the condition is more frequently connected with nutritional changes of a cardio-vascular sclerotic type with fibrotic invasion of the auriculo-ventricular nodal organ.

In diagnosis the determination of the fact that the bradycardia is more or less persistently limited to the ventricle is of the first importance, and the prognosis in such cases is always grave.

CHAPTER IV

EXTRASYSTOLIA

THE usual explanation of the ventricular extrasystole is that in the period of the relative recovery of the muscle in the refractory phase, an abnormal excitant induces a contraction or contractions, the normal excitant being considered to be the auricular impulse. This principle, without having auricular impulse as the normal excitant to contraction, is considered to apply to cardiac muscle in general. That is to say, that during the period when the stimulus material is being reproduced, it explodes in a certain section of the heart, sinus, auricle, or ventricle, as the case may be, before its proper time; in a word, prematurely.

This premature explosion may recur rhythmically or arhythmically, and slowly or rapidly. The sinal extrasystole appears to have no, and the auricular extrasystole not much, clinical importance. Vaquez suggests that the determination of the site of the extrasystole, whether, that is, it be in auricle or ventricle, may be desirable, as it may be the first evidence of organic change. The diagnosis of organic change is, however, scarcely likely to be made by the determination of auricular extrasystoles. The auricular extrasystole he also asserts is necessarily propagated to the ventricle.[1] If this be so it differs from the tachycardial auricular systole, which I regard as an extrasystolic condition; for, as we have seen when considering auricular tachycardia, these auricular contractions need not affect ventricular action, even when blockage at the auriculo-ventricular junc-

[1] *Op. cit.*, p. 201.

tion may be supposed to be absent as shown by the atropine test (p. 171).

So wide is the range and so protean the form which the extrasystole of cardiac muscle may assume, that it would be difficult to prove that auricular tachycardia or flutter is not, like paroxysmal ventricular tachycardia, an extrasystolic but more persistent phenomenon—in short, an extrasystolia of high rate. If it be such, it is certainly not necessarily conducted to the ventricle.

The clinically important extrasystole is, however, the ventricular extrasystole. What is the mechanism or cause or provocative of extrasystole? It has, of course, long been

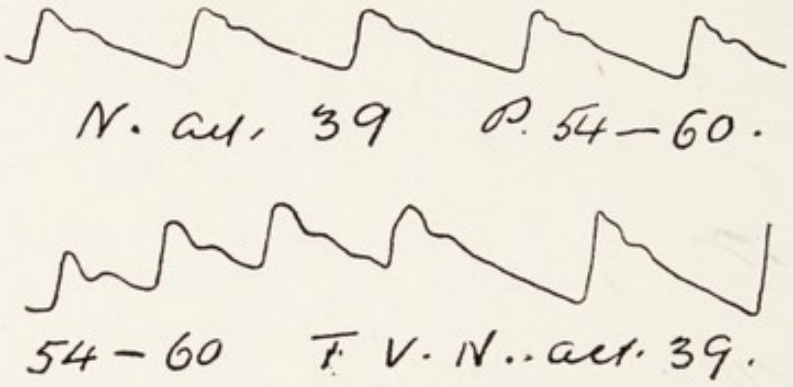

FIG. 39.—THE RADIAL PULSE BEFORE AND DURING FORCED RESPIRATION.

known that movements of the body, emotions of the mind, and changes in respiration affect the rate and rhythm of the heart. If the normal heart-rate be slow, the effect of respiration is more easily observed, as may be seen in the following sphygmogram taken from a paper I published in 1896[1] (Fig. 39). The increased rate on the rising plane of inspiration will be noted, and the retardation at the commencement of expiration. These changes are admittedly the result of control of the cardio-vascular mechanism by the nervous system.

But without exertion, emotion, or exaggeration of respiration, the heart may exhibit departures from the average normal in action, both of a regularly and irregularly recurrent character, and these may be observed in some instances to be modified by voluntary movements. Thus, a cantering

[1] *British Medical Journal*, March 14.

or triple rhythm (an extrasystolic phenomenon) which is audible on recumbent repose may be abolished by causing the patient to sit up in bed. The heart showing a bigeminal pulse and quadruple grouping of sounds (likewise an extrasystolic sign) while the patient is seated (Fig. 40, A, B) may pass into a canter with the disappearance of the bigeminal pulse on his assuming the erect position (Fig. 40, C), and return to bigemination and quadruplication on his resuming recumbency or sitting down (Fig. 40, A, B). The alteration is due to the acceleration or retardation of pulse according as the patient stands or sits. The extrasystole may also rhythmically coincide for a length of time with auricular systole. In some of

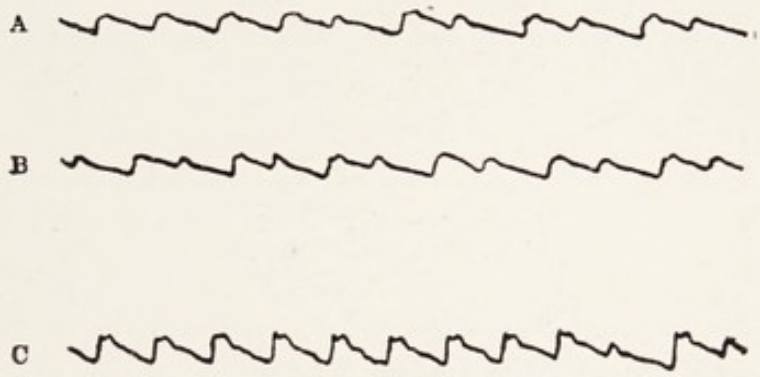

FIG. 40.—SPHYGMOGRAMS OF QUADRUPLE, TRIPLE, AND MIXED RHYTHM FROM A CASE OF DOUBLE MITRAL DISEASE.

these cases, if the patient be thin and the structures at the root of the neck consequently not unduly covered with fat, it may be observed that at the moment when there is an intermission in the radial pulse, a wave of some size is flashed into the neck in the position of the internal jugular vein. If a simultaneous tracing be made of the jugular and radial pulses, it will be found that at the moment of radial intermission the pulse-wave at the wrist is small and in the jugular large. The small radial (ventricular) wave or extrasystole occurs at the same moment as the auricular systole, and is in no way dependent upon the latter. The following tracing, taken from a boy of ten who was under my care at the Paddington Green Children's Hospital in 1906, shows this phenomenon (Fig. 41). It persisted for a length of time, occasionally disappearing and again recurring, always with the same rhythm. In this case it will be seen that the

extrasystole occurred at every third beat. The bigeminal pulse may also, it is well known, be produced by the use of digitalis in both mitral and aortic valvular disease (Fig. 43).

The sustained rhythmicity of some of these extrasystolic cases, their modification by postural change, and their production by an introduced agent, seem to indicate a neural factor in their genesis—that is, that the stimulus which anticipates the normal recurrence of the heart-beat, acts through the nervous system as on the principles enunciated in this book does the stimulus on which depends the heart's beat itself. But the extrasystole may lack regularity of appearance, and

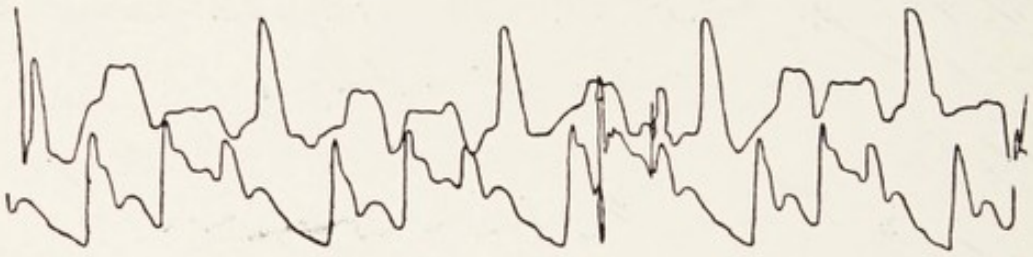

FIG. 41.—VENTRICULAR EXTRASYSTOLE COINCIDING WITH LARGE AURICULAR WAVE.

arise at irregular intervals. The rate, moreover, of the occurrence of the phenomenon may be average or slow, or it may be rapid or tachycardial. Regular ventricular tachycardial extrasystolia, we have already seen, appears to be a reasonable explanation of cases of paroxysmal tachycardia (p. 177), while irregular ventricular tachycardial extrasystolia is synonymous, it is maintained, with the term 'auricular fibrillation' (p. 182). If there be a certain indefiniteness and imperfect explanation of the term 'neurosis' as applied to these phenomena, the objection applies no less to the term 'inosis,' or muscle-state. The considerations I have mentioned, however, I submit, point to an active participation of the nervous system in the genesis of both systole and extrasystole—in the production alike of the normal and abnormal forms of cardiac motion which have already been more fully considered.

CHAPTER V

HYPERTONIA AND HYPOTONIA

REFERENCE has now been made to disorders of cardiac rate and rhythm. It remains to mention those of cardiac power or force. Hypertonia and hypotonia are rather barbarous words, but their meaning is apparent—excess and insufficiency of tonic energy.

It will be remembered that in Part I. three kinds of cardiac muscle were described—vascular, nodal, and tonic—and a vaso-motor, cardio-motor, and cardio-tonic nervous system regarded as existent. Indeed, the 'tonic nerves of the heart' from the sympathetic outflow is a sufficiently accepted expression, and an accessory-vagal tone may also be considered to exist. Tone or tonicity is also the fifth point in the myogenic interpretation of the nature of cardiac motion.

While in the somatic musculature tonic contraction may be produced at will, and sustained somatic tone be regarded as dependent upon the central nervous system, this tone may involuntarily and locally pass into tetanic exaggeration or spasm. Although increased tonic action in cardiac muscle is beyond the control of will, yet it may, by way of the augmentor nerves, be roused by the emotions. Localized tetanic cardiac spasm is, however, as already stated, disputed by physiologists.

Inflammatory processes, moreover, such as those attending pericarditis, by direct stimulation of the nerves penetrating the myocardium and by the rise of temperature associated with inflammation, may likewise provoke increased tonic cardiac action for a time.

Hypertrophic increase in the *bulk* of visceral muscle increases or magnifies its contractile power. This is demonstrable in the case of the pregnant uterus, in which we know the power of tonic contraction is directly as the bulk of the visceral muscle. In the case of the uterus, as I have shown elsewhere,[1] while the muscle of the organ is enormously increased in bulk, the nervous system is little if at all enlarged, the natural provision of tortuosity of the uterine nerves allowing full innervation by *uncoiling*, without any increase in the nerve elements, which may be shown to be absent by the measurement of individual fibres. This coiling and uncoiling of the uterine nerves is a very remarkable provision, laid down as the nerves are for a function to be exercised in the future.

In the heart, as Kölliker pointed out, there does not appear to be any *multiplication* of muscle elements in hypertrophy, but an increase in their size; while the nervous system, as in the uterus, and as might have been supposed *a priori*, remains unchanged. But, as in the case of the uterus, increased muscular bulk is associated with an increase also in the power of tonic contraction.

As was pointed out in Part II., when discussing sensory disorders of the heart, this hypertrophic increase may reach proportions which, by the direct stimulus of impact against the resistant osseous thorax, may induce an erethism or exaggerated tonic contraction of the organ akin to that associated with the stimulation of the augmentor nerves by inflammatory processes, to which reference has been made. Such is the condition to which I have applied the term 'hypertrophic hypertonia' to secure uniformity with the other qualified nouns employed in the semeiological nomenclature adopted.

Non-hypertrophic hypertonia is an erethitic condition which may be a transient or remittent state dependent upon neural stimulation usually of a sustained emotional character; or it may be the initial stage of a condition which, both with and without mechanical embarrassment of the

[1] 'On the Relation of the Nervous System to Disease and Disorder in the Viscera,' 1899, p. 57 *et seq.*

heart from defective valves, proceeds to hypertrophy. This non-valvular hypertonia of the heart is the result of vascular conditions frequently of toxic origin, and associated with a high pressure which renders the work of the heart more difficult. To this arterial 'hypertonus' Dr. William Russell has drawn attention in his work on 'Arterial Hypertonus, Sclerosis, and Blood-Pressure.'

Like all excessive actions which cannot be indefinitely sustained or maintained, exaggerated tonic cardiac action tends to be succeeded by, or to induce, a minus quantity. Hypertonia necessarily yields to hypotonia, expenditure is followed by loss.

The hypertonic capacity of cardiac muscle is in proportion to its nutritive power. Protracted difficulty induces increased effort, the active structures requiring increased nutrition, and, the factors underlying this process being normal, hypertrophy is induced with increased power and tone. The nutritive factors failing, this increased power wanes and hypotonia gradually takes the place of normal or increased tone.

The factors involved in nutrition are those concerned with organic action—namely, the blood, the innervation, and the specific cell. The pathology of cardiac failure is imperfectly known, but the grosser manifestations of defective nutrition are perceptible. The muscle cell may be in a condition of fatty degeneration or atrophic from the pressure of increased interstitial textures. The bloodvessels themselves may be involved in an obliterative endarteritis, which is frequently but not always of syphilitic origin, and the nervous elements may likewise be hypernucleolated, shrunken, or pigmented. Although the significance of pigmentary degeneration is doubtful, all these abnormalities indicate defective nutrition, which, regarded in its widest sense, is the cause of cardiotonic failure. The atrophy of nervous structures so frequently met with in syphilitic diseases of the heart appears to be secondary to obliterative endarteritis of the smaller vessels.

As tone and hypertonia may be induced by stimulating emotions passing in all probability chiefly by the tonic

sympathetic nerves to the heart, depressant emotions may also directly and indirectly induce an enfeebled or atonic condition of cardiac motion. This factor, the injurious influence of which directly upon cardiac action and indirectly upon it through its effect upon nutrition and rest, is not always sufficiently recognized.

The nervous channels for depressed or diminished tone probably course chiefly in the vagal or higher accessory-vagal outflow, which appears to have a special relation to the cardio-motor inhibitory nervous system, and endows the nodal or cardio-motor structures. But the more exact knowledge of the functional relation of the nervous system to the nodal structures is still in its infancy. It is not long since its anatomy was altogether unknown.

Encroachments upon the auriculo-ventricular nodal structures, interferences with its nutrition, or grosser solutions of its continuity, we have seen may interfere with ventricular response in such a manner as greatly to prolong ventricular diastole. This allows a repletion of the chambers with blood, which, the nutrition of the heart being otherwise good, induces in some cases considerable hypertrophy of the ventricles. When there is, in addition, valvular disease of the heart, it may be difficult to determine how much of the hypertrophy of the chambers is due to compensatory growth intended to deal with circulatory embarrassment caused by the valvular lesion, and how much to the effort involved in dealing with the surplus blood-weight in prolonged diastole. That, apart from such lesion, bradycardia induces ventricular hypertrophy is frequently demonstrable—that is to say, the cardio-tonic musculature contains all the factors for hypertrophic nutrition and growth even when the cardio-motor reflex is abolished. Before the stage of its absolute abolition is reached, the heart is more prone to exhibit severe inhibitory syncopal accidents, as shown in Fig. 35 (p. 191).

Hypotonia may result from the local overwork of a mechanically disabled organ, or from the general exhaustion of the organism by a more generally enfeebling cause having one or another form of anæmia in its train, or it may be from these conditions in combination.

The following case, an account of which was given in a paper on 'The Treatment of the Muscular, Hæmic, and Neural Factors in Heart Disease,' in the *Edinburgh Medical Journal* for October and November, 1904, illustrates the hypotonic collapse of an over-hypertonic or bovine and for a time compensated and sufficient heart. It also shows the rhythmical extrasystolia induced by digitalis, as also the progressive failure of the heart after a temporary rally under the bold use of the drug:

A man, thirty-six years of age, was admitted into the Great Northern Central Hospital on September 8, 1903, suffering from aortic valvular disease with dyspnœa and anasarca. He was treated by rest in bed and general care. Small doses of digitalis were prescribed and given irregularly. I first saw the patient on October 8, after he had been a month in hospital, when he was orthopnœic and had anasarca of the legs and œdema of the abdominal wall. The area of cardiac dulness was increased; there was a double basic bruit, best heard to the left of the sternum, and a systolic bruit at the apex.

A mixture containing 12 minims of tincture of strophanthus, 30 minims of the liquor of the perchloride of mercury, 5 minims of the liquor of strychnine, and 20 minims of the spirit of nitrous ether, was prescribed and given three times a day. This did little good. The anasarca increased, and the lungs became more engorged. The patient slept little, was orthopnœic, and, in short, presented all the well-marked features of cardiac failure in its last stages.

Southey's tubes were now inserted into his legs as a preliminary to the bolder use of digitalis, and in the three days, from October 13th to 16th, 18½ pints of fluid were thus drained away, reducing the bloated appearance of the patient and abolishing, on the day after their use, the jugular evidence of auricular tension. The pulse on the 14th was small and paradoxical from dyspnœa, as may be seen in Fig. 42, the breathing shallow, the cardiac bruits faint in sound, and the urine contained one-sixth of albumin. On the 14th—that is, on the day following the drainage of the anasarca—I prescribed a mixture containing 15 minims of

tincture of strophanthus, 10 minims of tincture of digitalis, 5 minims of the liquor of strychnine, 15 minims of aromatic spirit of ammonia, and 30 minims of spirit of nitrous ether in each dose. One dose was to be given every four hours. I again saw the patient at the hospital on the afternoon of the 16th, when he had taken twelve doses of this mixture—that is, 300 minims of the combined tinctures of strophanthus and digitalis, 60 minims of the liquor of strychnine, and 6 drachms of the spirit of nitrous ether. I found him lying peacefully in bed, with a pulse, as will be seen in Fig. 43, of 48 to 54 at the wrist, with an intermediate abortive

FIG. 42.

FIG. 43.

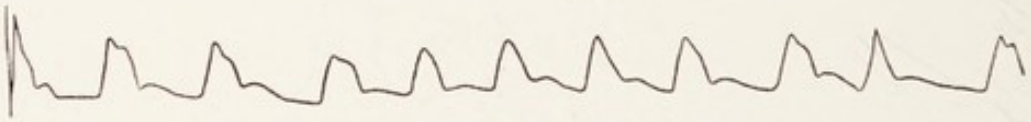

FIG. 44.

systole, as shown by the tracing. The patient complained of feeling a little sick, but also stated that he felt much better. The cardiac area, so far as could be determined by percussion, measured 4½ by 5 inches; the basic bruits were loud, rough, and slowly uttered, and the apical bruit had assumed a musical pitch. The ventricle, in other words, was contracting better, the heart acting with greatly increased force, and the chambers of the organ emptying in proportion. The use of digitalis and strophanthus, in view of these circumstances, was discontinued, but their effects for a time persisted, as may be seen in Fig. 44, which I took on the 17th. Here the pulse-wave is still full, although irregular in size, but is not strictly a pulsus alternans, and the abortive systole has been replaced by a complete systole. The house physician's notes of the case for this day state that 'the patient's sleep

[was] good; [his] pulse slow, about 72, [and] fairly steady.' A distressing cough, too, had diminished; but, on auscultation, ægophony could be heard at the base of the right lung posteriorly, where there was also some dulness. This was the signal of further trouble, which turned the scale against the patient, for fluid began to collect somewhat rapidly in the right pleural cavity. The breathing again became embarrassed, and the anasarca of the legs increased. The pulse quickened, and for a time remained of good tension, as may be noted in Fig. 45, but became hypotonic and feeble, as shown in Figs. 46 and 47. Southey's tubes were again inserted

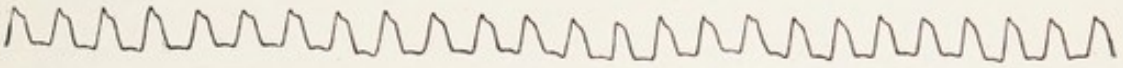

FIG. 45.

FIG. 46.

FIG. 47.

into the legs, and drained fairly, but not so freely as before. Sixteen ounces of serous fluid were aspirated from the right pleural cavity, and the full doses of digitalis and strophanthus resumed, but without benefit, the patient dying rather suddenly during the night of October 27. The hypotonic heart, regarded as a jaded horse, had pluckily jumped its last ditch under stimulation a fortnight previously, and now, in spite of spur and whip, floundered exhausted at the last attempt to leap.

The necropsy next day revealed a bovine heart, as the illustration shows, weighing 36 ounces, with contained clots, dilated and hypertrophied in all its chambers, which also all contained blood, and with a well-marked aortic lesion (Fig. 48). One of the cusps is transformed into a calcified dendritic mass. There were no pericardial adhesions, but

the right pleural cavity contained 3 pints of serous fluid. The aortic lesion impeded in a measure the escape of blood from, and allowed its regurgitation into, the left ventricle.

The hypotonic heart which has not known hypertrophy, and has become dilated and weak, may, like the transiently and intermittently hypertonic, be of neural and emotional origin, or, when permanent, a consequence of disease of the coronary vascular system.

We have in Part I. noted how much restriction of the coronary vessels may occur without the induction of cardiac

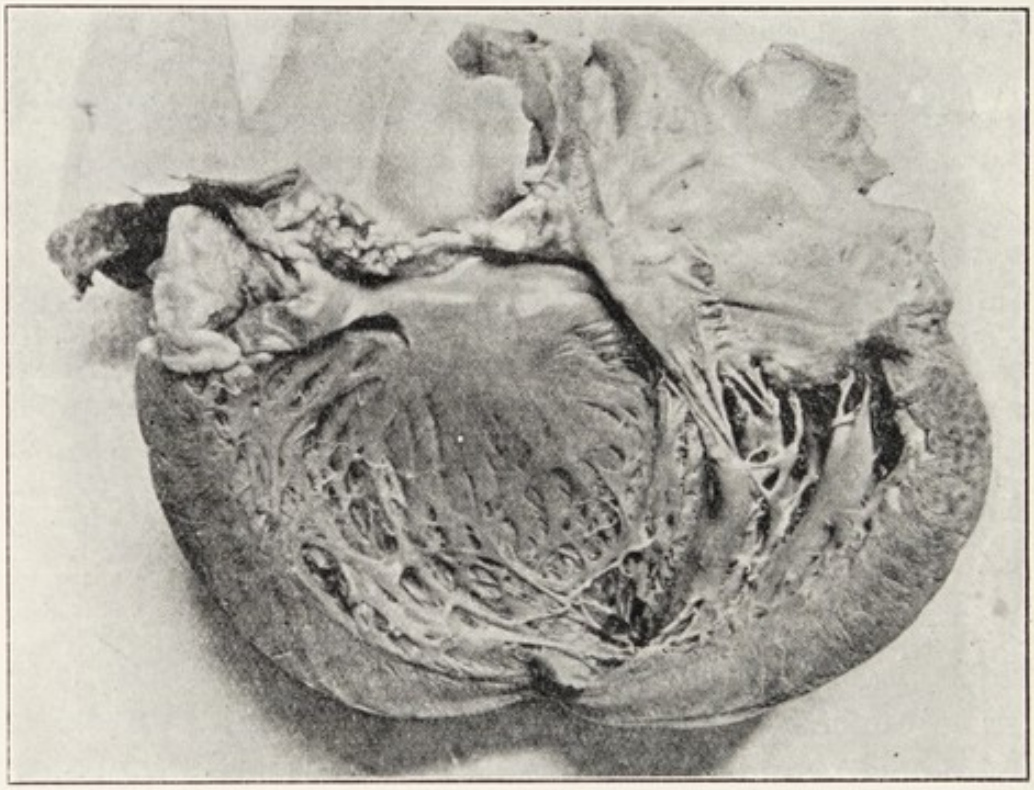

FIG. 48.—HYPERTROPHIED AND DILATED LEFT VENTRICLE WITH DISEASE OF AORTIC VALVES.

muscular degeneration, owing to the freedom of vascular anastomosis. But a persistent limitation of the arterial flood of freshly oxygenated blood in the case of so perpetually active an organ as the heart is rarely unassociated with more extensive and general vascular disease than that of the coronary vessels.

As one constantly observes, general arterio-sclerosis *without* kidney disease, and with free coronary channels, leads to hypertrophy of the heart, and at times to large hypertrophy.

But the arterio-sclerotic with obstructed coronaries frequently has a small heart—small without concentric hypertrophy. These are the cases of persistent non-hypertrophic hypotonia in which cardiac insufficiency with and without cardiac pain is the rule. Even without the gross evidences of muscular degeneration, such a heart is insufficiently nourished to act vigorously—it is hypotonic.

CHAPTER VI

THE PROGNOSIS OF DISORDERS OF CARDIAC MOTION

THE pathology of disorders of cardiac motion in cases showing persistent extrasystolia has not been investigated with sufficient care to allow conclusions to be drawn as to the organic bases of the condition in many cases: variable and transient arhythmiæ are probably due to stimuli which require no gross lesion to explain their presence. Since the discovery of the nodal system, suggestions have from time to time been made, by exponents of myogenic views of cardiac disorder, that transient lesions of that structure probably account for some abnormal phases of cardiac action met with in the course of more general diseases.[1] These, however, are merely surmises. But it may be affirmed with some confidence and in general terms, not as absolutely applicable, that mechanical lesions — organic diseases — affecting the mitral orifice are more frequently associated with irregular types of extrasystolia, while lesions of the aortic orifice are usually accompanied by cadented or regular arhythmiæ.

Nevertheless the prognosis of disorders of cardiac motion —and these, of course, are not *all* extrasystolic—depends upon the conclusion arrived at as to the situation and nature of the textures involved in, or influencing, the movements of the heart.

We must also remember that with little disturbance of rate and rhythm of movement, or noisy manifestation of

[1] Lewis, *British Medical Journal*, vol. i., 1913, p. 484; Mackenzie, Proceedings of Royal Society of Medicine.

disease, a heart may be failing seriously, while striking departures from the normal may be transient and negligible. For example, a man of fine physique, sixty-seven years of age, was under my care in hospital in 1911, who presented a slightly accelerated rate and perfectly regular rhythm of cardiac action. The sphygmomanometer gave a low reading, and the sphygmograph showed quite regular waves of low tension, as evidenced by the depressed dicrotic notch and featureless lines of the tracing; while the waves were also of slightly unequal size, but without an alternating inequality. The jugular phlebogram exhibited the exaggerated inspiratory and expiratory undulations of dyspnœa, and the auricular wave, though imperfectly marked, had a normal time relation to the carotid wave of one-fifth of a second (Fig. 49). The

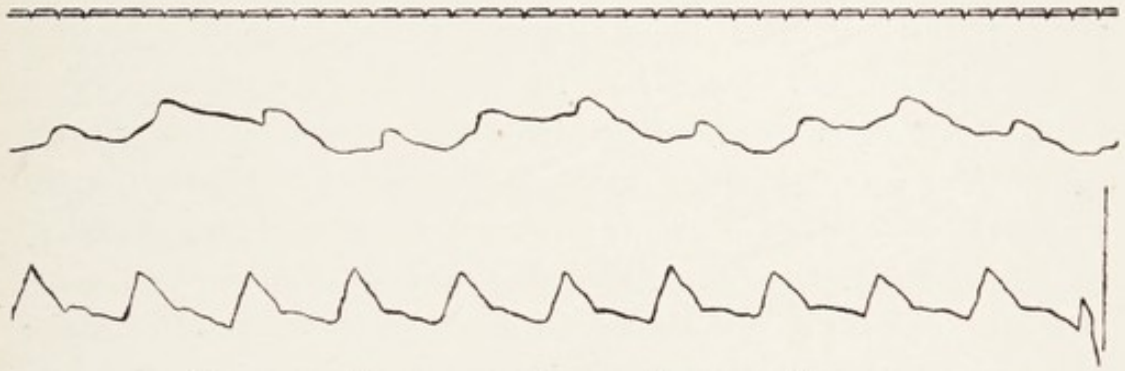

FIG. 49.—RADIAL AND JUGULO-CAROTID TRACING.

patient, however, showed a sign to which considerable importance attaches as evidence of grave cardiac failure—namely, what I termed a 'reversible pulmonary œdema' in a note sent to the *Lancet* of May 6, 1911, the seesaw œdema, or *œdème à bascule* of the French—that is, an œdema of the pulmonary bases which a single revolution of the patient on his own axis in bed served to transfer from one lung to the other. The patient died, and I found on examination fatty degeneration of the ventricular muscle in the recent specimen, and after section in paraffin considerable interstitial sclerosis of the ventricle, with atrophy of strangled muscle and a perfectly normal auriculo-ventricular node and bundle. The patient died from disablement of his cardio-tonic, not his cardio-motor, musculature.

With these preliminary observations, we may proceed to

consider *seriatim* the prognosis of the varieties of disordered cardiac motion already mentioned.

The **cantering rhythm** may be a transient phenomenon in valvular disease of the heart, disappearing with more forcible action of the organ. Occasionally it may persist for a lengthened period without apparent detriment to the patient. It may, however, as in renal disease, be significant of cardiac failure, and its prognosis, in view of the serious organic condition involved, grave in proportion.

Except in cases in which, from mechanical defect, the auricle has to play the part in greater or less degree of the ventricle, as in some cases of congenital malformation, and to a less extent in stenosis of the auriculo-ventricular orifices, a man may be said to live on his ventricle. So long as the latter chamber responds sufficiently normally, in force and tone, to stimuli, the circulation is maintained, and with it life. The auricle which has to play the part of a ventricle in malformation of the heart seldom does so for any length of time, a fact which is in itself sufficient evidence of the importance of the *mechanical* factor in the maintenance of cardiac action. In auriculo-ventricular stenotic cases, even if we accept a myogenic theory of cardiac action, the stasis of auricular fibrillation or other atrial vagary does not kill, except in so far as it influences ventricular action.

Disorders of cardiac motion, therefore, limited to the auricle, do not appear to have any grave prognostic significance. A **tachycardia of the auricle** ought, on a myogenic hypothesis, by exhausting that chamber, likewise exhaust the ventricle; but while, in the case I have recorded of this condition, the ventricle was failing to cope with the labour involved in driving the blood through a heart valvularly disabled, the contractions of the ventricle itself were regular in rhythm, and apparently uninfluenced in rate by the abnormally accelerated auricle, a condition of 'flutter' which is most probably a mode of auricular extrasystolia. Ritchie has noted flutter to continue for seven years.[1]

But although the exhaustion of the ventricle by tachycardia of the auricle as such may be questioned, it is possible that

[1] *Quarterly Journal of Medicine*, October, 1913.

a persistently accelerated auricle may indirectly affect the underlying ventricle by emphasizing its normal repletion with blood, and thus throw more work upon it. Such a handicap may yield to digitalis acting on the ventricle when it has lost its power to affect the auricle. It must, however, be remembered that such a propulsive effect upon the ventricle of this auricular tachycardial extrasystolia, or flutter, must be periodically limited or checked by the closure of the tricuspid and mitral valves during ventricular systole.

Auriculo-ventricular tachycardia, when persistent, tends to shorten life. This conclusion is warranted by such investigation of these cases as has been made by Herringham and others, but further examination of the subject is necessary before dogmatic statements can be made on the prognosis of this mode of cardiac motor disorder.

A paroxysmal tachycardia, which, as we have seen, is more probably a massed ventricular extrasystolia, may arise during grave illness, and suddenly cease, leaving the patient comparatively comfortable with a regular heart beating at a normal rate, but be succeeded later by further evidence of cardiac failure, which may prove fatal without any return of accelerated action. I have had experience of this in the case of a patient whom I had known for many years, whose heart was sound, but who developed a paroxysmal tachycardia during her last illness (bronchitis), which suddenly ceased with relief after lasting twenty-four hours. Next day the ventricle became regularly extrasystolic. This, too, passed off. Two days later the patient showed a loud mitral regurgitant bruit, transmitted to the left and audible in the back, which persisted, and on the following day the heart suddenly ceased to act in syncope. Here there was progressive disturbance from regular tachycardial extrasystolia, through cadented extrasystolia to hypotonia and syncope.

But the mechanism of fatal paroxysmal tachycardia may be quite otherwise.

One of the conceptions of cardiac failure discredited by the new cardiology is that of a retrograde dilatation and stasis in the chambers behind the left ventricle—that is, in

the left auricle, and later in the lungs, right ventricle, and right auricle. But this new view is contradicted by frequent experience, and one of the modes of death in paroxysmal tachycardia, when the condition proves fatal, supports the older view of the mechanism of failure to which I have referred.

For example, F. M., a female child of seven years, was admitted into the Great Northern Central Hospital on August 8, 1902, under the care of my colleague, Dr. Clifford Beale. She had slight fever with a mitral regurgitant lesion, and had had rheumatic fever two years previously. The case was discharged convalescent on the 20th of the same month. Her progress was stated to have been satisfactory until she presented herself on September 26 at the hospital, when she had well-marked tachycardia, the pulse being uncountable at the wrist, and the heart-rate, determined by auscultation, 200 in the minute. The child was at once admitted under my care.

The attack of tachycardia, which was associated with very little fever or respiratory distress, lasted for a week. The heart-sounds were distinct, the systolic bruit loud, and the area of cardiac dulness much extended. The urine was free from albumin. On October 2 the pulse-rate fell from 200 to 100 in the minute, and thereafter varied from 98 to 108, until October 12, when it rose to 160, and is stated to have fallen in the evening to 100; the child died the same night. The liver was enlarged, and the urine had contained albumin for some days prior to death. On October 8 there was œdema of the face and of the feet, and that in the former position became more pronounced than in the lower extremities. Impaired resonance was also now detectable in he right interscapular region, and a drowsiness which had begun to manifest itself became more marked, but without any evidence of abnormal loss of power in the limbs. That is, there were no signs of paralysis from embolism or other cause. On the 11th the child was evidently sinking, and died, as I have stated, during the night of the 12th. The post-mortem examination was made on October 14th by myself. There was no trace of recent inflammation of the

endocardium, or of the pericardium, to the naked eye, although the latter contained 3 ounces of clear serous fluid. There were no pericardial adhesions. The left ventricle was hypertrophied, but not markedly, if at all, dilated, and contained little blood. The anterior cusp of the mitral valve was thickened, and the auriculo-ventricular orifice admitted three fingers conewise—that is, two passed from below and one from above. The left auricle was much dilated and firmly packed with dark clot. The aorta and the aortic valves, together with the coronary arteries, were normal. The right ventricle was dilated, and filled with clot, part of which was decolorized. The pulmonary valves were normal, as also was the tricuspid. The tricuspid orifice, like the mitral, admitted three fingers conewise. The right auricle was much dilated, and filled with blood-clot. The myocardium on section was apparently normal. On microscopic examination, the transverse striæ of the muscular fibres were less visible in some sections than in others, stained by another method, but on the whole there was no condition of muscle which could be positively asserted to be pathological. There was, however, distinct evidence in some sections of active interstitial hyperplasia, and of this in immediate relation to the trunks of cardiac nerves in the neighbourhood of the right coronary artery. The cells of the left inferior cervical ganglion, which was examined as one of the motor outposts of cardiac innervation, showed little if any pathological change. The only possible difference between these and cells from a normal ganglion was that they appeared in greater numbers to be pigmented than in normal circumstances. There was no marked hyperæmia of the ganglion. As regards the clinical phenomena of the case, a glance at the chart of the pulse, respiration and temperature of this patient, taken during the two periods of her residence in hospital, showed the remarkable disparity between the pulse-rate and the respiration and temperature during her fatal illness.

This, then, appears to have been a case of paroxysmal tachycardia or massed regular ventricular extrasystolia in which the tachycardial left ventricle, hypertrophied, undi-

lated, and voiding its contents more rapidly than it could be filled, acted practically as an obstacle inducing a retention of blood in the after-coming chambers, and a depletion of the coronary and central nervous systems, with cessation of cardiac action in consequence. The intensity of the venous plethora was shown by the marked facial œdema, the enlarged liver, and the non-inflammatory pericardial effusion.

The mode of death exemplified in the case of massed ventricular extrasystolia just narrated may be observed in cases in which the heart hurry may be assumed to have been one originally shared equally by sinus, auricle, and ventricle. Death during emotional tachycardia or the tachycardia of emotion and exertion combined, or of strenuous exertion without emotion, affords examples of this. In such cases the ventricles outrun the higher chambers and remain active after the stuffed auricles have ceased to be effective propellers.

Moreover, notwithstanding the fact which may be accepted —namely, that normally the auricles act simultaneously, as do the ventricles—it is demonstrable that the most powerful chamber in the heart—the left ventricle—*can* act vigorously even though its fellow on the right be fixed into immobility. The proof of this lies rather with the clinician and pathologist than with the physiologist. It was well shown in the case of sarcoma of the dextral chambers of the heart to which I have already referred (p. 20), and of which, as revealing the fact under discussion, I now add an illustration (Fig. 50).

The fact in question is not unimportant, as it enables us to understand the mode of death in many cases both of hurried ventricular extrasystole and systole.

Thus, I gave some particulars, in a lecture on Modes of Death in Heart Disease,[1] of the case of a man, thirty-five years of age, whose heart I was asked to examine after death, by Dr. Frank Rushworth, of Hampstead. Before breakfast on August 15, 1904, he went to the Hampstead Swimming Bath, and there swam a short race of two lengths of the bath with a friend. At the end of the race he was observed to

[1] *Clinical Journal*, vol. xxvi., No. 11, p. 161.

drop his face on the water, make a few feeble efforts to swim, and then sink. It was not realized at first that he had sunk never to rise again. The situation was, however, soon grasped, and he was quickly drawn out of the water. Dr. Rushworth was summoned, and arrived in about ten minutes, when he found the man dead.

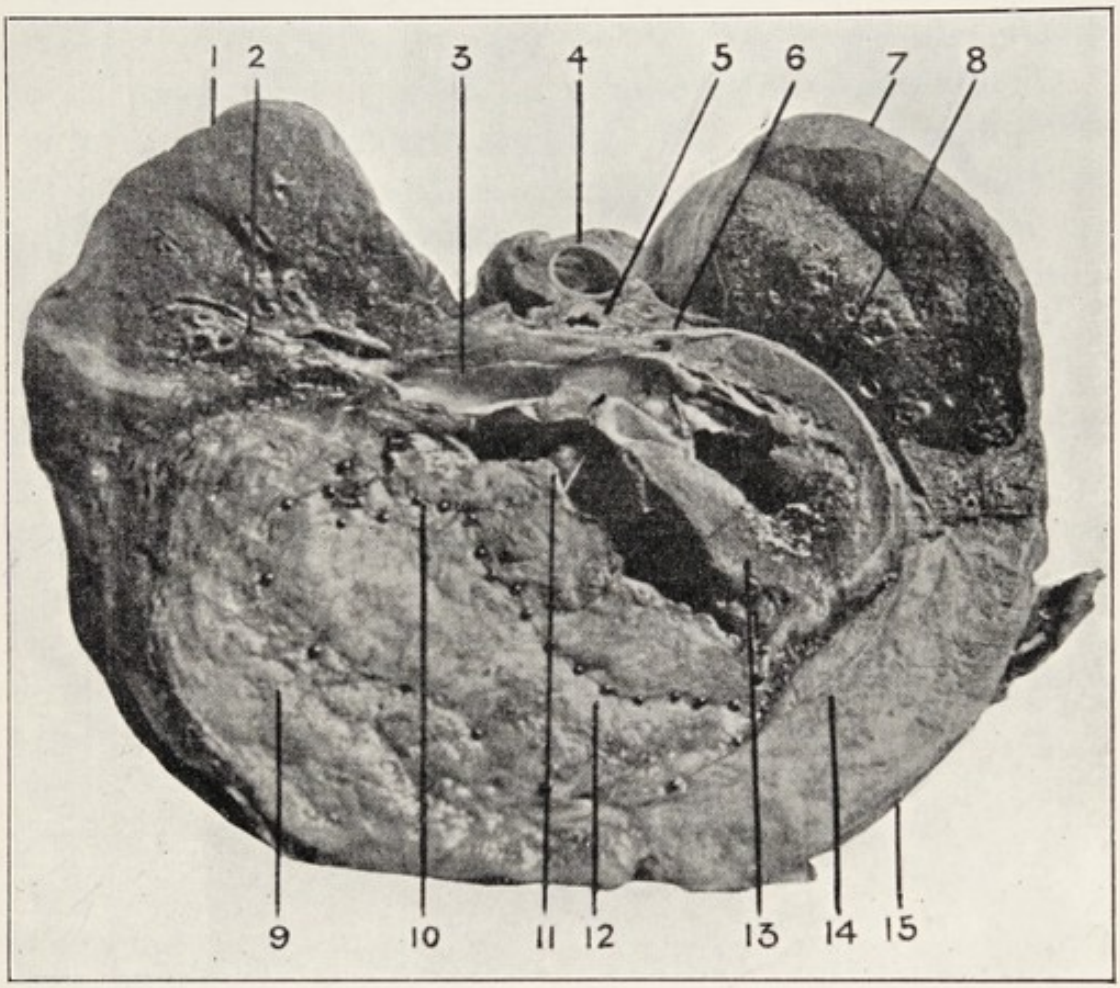

FIG. 50.—TRANSVERSE SECTION THROUGH SARCOMATOUS HEART AND LUNG.

1. Right lung. 2. Bronchi. 3. Pulmonary vein leading into left auricle. 4. Descending aorta. 5. Œsophagus. 6. Left coronary artery. 7. Left lung. 8. Left ventricle. 9. Mediastinal sarcoma. 10. Sarcomatous right auricle. 11. Sarcomatous nodule at mouth of coronary sinus. 12. Sarcomatous right ventricle 13. Septum ventriculorum. 14. Sarcomatous pericardium. 15. Outer surface of sarcoma.

We examined the body thirty-six hours after death. The corpse was that of a well-developed, well-nourished male, somewhat early grey-haired, with a fair amount of fat, but not obese. Rigor mortis was present in moderate degree.

On opening the chest, the lungs were found retracted, exposing the pericardium rather more than usual. The

pericardium was normal, and contained about 2 drachms of serous fluid.

There was a considerable deposit of fat on the heart. The right auricle was seen to be much distended with blood, and on grasping the heart the left ventricle was felt to be firmly contracted. The right ventricle was flaccid or soft, but wrapped round the left ventricle. The left auricle was *not* distended.

On dividing the inferior vena cava, a large quantity of dark, fluid blood escaped; this was the case also on dividing the pulmonary veins. There were no coagula. *After the evacuation of the blood the right auricle shrank well*, and the right ventricle was undilated and normal.

All the valves and orifices of the heart were normal. The left ventricle was neither hypertrophied nor dilated, and was in a condition of tonic contraction and empty. There was slight atheroma at the root of the aorta, but the coronary arteries were normal to the naked eye, although one of them had a cribriform entrance.

Muscular fibres taken from the left ventricle showed well-marked striation, and many a fine pigmentary degeneration. This I originally described (*loc. cit.*) as a granular fatty degeneration, but stated (p. 164) that it 'was in the centre of the fibre, and approached more or less closely both poles of the nucleus,' which is more characteristic of pigmentation than of fatty degeneration.

The lungs were healthy and crepitated generally, but showed recent engorgement. The stomach, liver, spleen, kidneys, and brain, were normal. The brain had very few *puncta cruenta*.

The man thus appears to have died from what we are in the habit of calling 'cardiac failure,' with *repletion* of the dextral chambers and pulmonary veins, and depletion with contraction of the left ventricle.

These are some of the chief anatomical conditions found in asphyxia, and, as regards the left ventricle, in fatal hæmorrhage; but when the body was removed from the bath there was, I was informed, no evidence of much water in the respiratory passages, and there was certainly none found at the necropsy.

The patient had died suddenly with arrest both of respiration and circulation, and sank—one of the modes of so-called 'cramp,' a condition to be met with, not only in tne water, but under other circumstances in which the relatively powerful left ventricle outpaces the supply chambers, and fails to receive from them the quantity of blood necessary to irrigate the coronary, vascular, and central nervous systems. There is thus failure of all three factors in the organic functional unit.

I have related these particulars of this case in some detail, as they show very clearly a mechanism of cardiac failure long known and appreciated, which at the moment is rather apt to be obscured by some current and, as I believe, erroneous conceptions drawn from cardiac graphology and experimental physiology.

To return to the prognosis of paroxysmal tachycardia, it is a common experience that the condition, whether regarded as a sinu-auriculo-ventricular state or as a massed ventricular extrasystolia lasting for longer or shorter periods, recurring occasionally or frequently, arising suddenly and as suddenly ceasing, may come and go in many cases without leaving any permanent consequences detrimental to activity or life. But while present it cannot be regarded as devoid of serious possibilities, and more especially so if there be any more permanent mechanical or degenerative disability of the heart associated with it.

The **tachycardia of thyroidism**, we know, may eventuate in the recovery of a normal rate and rhythm of the heart's action. But cases of long duration may develop a persistent extrasystolia, and a comparatively small number, in which there is ultimately thyroid atrophy and a more or less marked condition of myxœdema, may even become bradycardial.

The subject of hyperthyroidism with tachycardia may, we know, die exhausted. Death, however, in such case is to be attributed rather to the effects of the endogenous poison than to the tachycardia. But thyroidal tachycardia engrafted on a mechanically disabled heart from rheumatism or other cause may precipitate a fatal issue, just as tachycardial ventricular arhythmia (auricular fibrillation) may do.

As I have stated, there is no clinical evidence of a **true auricular bradycardia**, and the **auriculo-ventricular bradycardia** arising after acute disease is usually a transient phenomenon. I have, however, mentioned a case (p. 182) of post-thyroidal auriculo-ventricular bradycardia which has lasted for years without apparent disablement of the patient, and this is probably not a solitary instance of persistent auriculo-ventricular or sinal bradycardia.

With **ventricular bradycardia** the question of prognosis is different. Although those exhibiting this syndrome may live for years, death usually occurs suddenly in syncope, and the condition must be regarded as one in which life is always jeopardized and in most cases shortened. Rapidly fatal ventricular bradycardia in advanced life is usually a more or less acute mode of cardiac failure, as also is the fatal bradycardia of diphtheria in children. The earlier in life a persistent ventricular bradycardia is established, the graver as a rule is the prognosis, because the condition is probably dependent upon gross lesion by specific disease, new growth, or inflammation with calcification of the ventricular nodal textures.

All irregularities of ventricular action, in hearts which are sluggish in response to the normal accelerants of emotion, exertion, or such drugs as alcohol, atropine, or the nitrites, have a degree of ventricular bradycardia. Dehio's test for the diagnosis of 'cardial' from 'extracardial' cases serves as a rule to establish the fact as to the presence or absence of a normal response of the heart to alterations of intra-cardiac blood-pressure—that is, it reveals the presence or absence of a normal ventricular response. The various graphic methods may also be used to ascertain the same point, which by these is determined according as a prolongation of the interval between auricular and ventricular systole is or is not declared, or the relation of auricular to ventricular systole is or is not dislocated. Partaking as such cases do of the nature of ventricular bradycardia, they demand, like it, care and a guarded prognosis, and are in many instances significant of impaired ventricular power associated with interstitial sclerosis, vascular insufficiency, or degenerated muscle fibre.

Cadented or **regular extrasystoliæ** normally accelerable, even when recurrent and persisting for considerable periods, ultimately disappear as a rule, and do not seem directly to threaten life; the extrasystolic tends to give place to a normal rhythm. It may be a transient phase in a persistent *irregular* arhythmia.

If the **pulsus alternans** be regarded as a regular arhythmia, it is of evil omen, occurring as it usually does under circumstances of obviously exhausted contractile power of the heart—in a word, of hypotonia. The sign, which, as Vaquez remarks,[1] is not frequently observed, has, it is believed, prognostic importance when noted; but innumerable cases of cardiac failure carefully observed progress to a fatal termination without exhibiting at any time the alternating pulse. Collateral evidence of cardiac failure, or the absence of such, should suffice to determine the gravity or otherwise of the situation.

The **irregularly occurring ventricular extrasystole**, shown by an intermission in the pulse at the wrist, may, like the regularly recurrent extrasystole, pass off and leave not a trace behind, or it may later be replaced by a more frequent and persistent complete irregularity of ventricular action. This was so in the case of a patient who was under my care for some years. He had glycosuria, a condition which, as also his cardiac irregularity, he dated from a severe emotional shock. He was a neurotic man in middle life, and the confidential friend and secretary of a man in an important social position, who committed suicide, and directed him to supervise the cremation of his body and to be present on the occasion. These injunctions, with much repugnance, he faithfully obeyed. From being a man of considerable weight avoirdupois, he shortly afterwards became thin, showed glycosuria, and later an irregular intermission of the pulse, which passed into complete persistent irregularity without valvular incompetency. He lived for some years, and when he died, did so, apparently, exhausted by his glycosuria rather than by definite cardiac failure, although the irregularity remained to the last.

[1] *Op. cit.*

The *paroxysmal irregularity*, or **tachy-arhythmia, of the ventricle**, met with most frequently in connection with mitral stenosis, and associated with **auricular fibrillation**, of which it is now usually regarded as the consequence, but of which I have suggested it may be the cause, may completely disappear, leaving a normal rhythm and rate of cardiac action. On the other hand, it may occur as a *paroxysmal incident* in a case in which the irregularity has become chronic and persistent, and may jeopardize life, and even prove fatal. Such a case which was under my care at the St. Marylebone General Dispensary, had an interesting history, and was under my observation for some years. His condition had originally been attributed to rupture of the mitral valve, from the sudden onset of his symptoms while in apparently good health, but after excitement attending the quenching of a conflagration in a house. The state was, however, one of disordered cardiac motion in an old stenotic mitral heart, a stenosis due in all probability to a choreic endocarditis of early life, which was admitted on interrogation, but had been forgotten.

When I first saw him his cardiac irregularity had become chronic, and he had while under my care several attacks of irregular ventricular tachycardia, presumably associated with auricular fibrillation, although no auricular tracings were made, and which he survived. In one such, however, he died some distance from London, and as I was informed of the event, and was allowed to examine the body, I found the cardiac lesion to be an old funnel-shaped mitral stenosis. The heart generally was hypertrophied and dilated, but not hampered in movement by extraneous tethering. On microscopic examination, there was seen to be a non-syphilitic endarteritis obliterans in the upper part of the septum ventriculorum and lower part of the auricular septum, with sclerosis of the auricular muscle in its lower part, sclerosis of the bundle of His, and extensive sclerosis of the adjoining ventricular muscle.

Disorders of Cardiac Force.—In considering the prognosis of disorders of cardiac force — of hypertonia and hypotonia—an aspect of the subjects we are discussing of

the first practical importance, we are guided by the evidence afforded by any given case of the condition of the cardio-motor (which is here regarded as synonymous with the neural), of the cardio-tonic, and the cardio-vascular factors involved, by the etiology of the disorder, and by the temperament, habits, and circumstances, of the bearer of the affected heart.

Such prognosis may have to be formed while cardiac force is sufficient, when the subject frequently presents himself for life insurance; or during periods of hypotonic failure, when the question of the preservation of life, or the more prolonged restoration of efficient action, has to be considered.

It has an important bearing on prognosis whether the necessity for increased effort on the part of the heart has been imposed upon it suddenly or gradually. The sudden imposition of such a necessity—for example, by the rupture of a valve—may be rapidly disastrous, and the more rapidly so when the accident occurs to the valves guarding the comparatively thin-walled right as compared with the left ventricle. In 1895 Dr. Hall of Warrington[1] reported a case of rupture of the pulmonary valve in a healthy young man, some further particulars of which he wrote me later, which proved fatal in forty-five minutes. But the principle of greater difficulty, in proportion to the unpreparedness of the chambers of the heart to sustain a sudden task imposed upon them, is not limited to the consequences of traumatic valvular rupture. As Wenckebach has pointed out,[2] it may also be seen to be operative in the overthrow of a heart in which the right chambers have been protected by competent sinistral valves, when, by the failure of these, pulmonary difficulty has to be faced by unhypertrophied dextral chambers. This recognition, however, of the influence of back pressure is, surely, scarcely orthodox on the part of so distinguished a neo-cardiologist as Professor Wenckebach!

The gradual imposition of the necessity for increased effort on the part of the heart allows time for nutritional

[1] *British Medical Journal*, January 19.

[2] International Medical Congress, 1913. Report, Section VI.

change and adaptation, with increase of tonic power (hypertonia) in the heart.

When sufficiency of the heart has given place to insufficiency, or threatens to do so, the necessity for increased effort must be diminished so far as possible, and with a rapidity proportionate to the suddenness of the task imposed upon the organ, to allow of its repair by increased nutrition. Sudden embarrassment, for example, may require free venesection, a subject which we shall consider later.

Prognosis in disorders of cardiac force will also be influenced by the situation and character of mechanical disability or impediment. Cases of valvular disease of long standing are met with, in spite of which the subjects of such disease have led strenuous lives in good health even to hoar hairs. But such cases, so far as I know, have without exception been those of mitral regurgitation, and cases, probably, in which a noisy reflux has been less regurgitant than noisy. So long as the aortic valves are normal in such cases, and the ventricles unimpeded in action by viscero-costal adhesions or compression, the effort of maintaining the circulation is little greater than under normal circumstances. With freedom from inherited or acquired vaso-degenerative dyscrasia, it is therefore not surprising when a septuagenarian occasionally informs us that he has had his heart affection from childhood, and has worked hard in the interval.

But a mechanical defect elsewhere—for example, an aortic defect inducing large ventricular hypertrophy, or a mitral constriction leading to auricular difficulty, hypertrophy, or impotence, with restricted ventricular repletion, or ventricles impeded by viscero-costal adhesions—so far as I am aware have never the same tale to tell. In these hypertonia yields to hypotonia sooner or later, but never late, although there may be considerable periods of well-balanced cardiac action —that is, of effective contractility and tonicity—a condition which has quite intelligibly been termed 'cardiac compensation.'

It is impossible to diagnose which of the aortic cusps is defective in aortic valvular disease, but it is probable that

lesions of the coronary cusps, by allowing regurgitation at the moment when, normally, a closed aorta gives diastolic aid to the coronary circulation, conduce to impaired cardiac nutrition, and therewith to earlier failure than would otherwise be the case.

Lesions of the dextral valves are not sufficiently numerous to afford material for judging of their effect upon longevity, but the majority of recorded cases have not attained old age. Cardiac malformations again shorten life in proportion to the departure of such hearts from the normal type. We have already seen (p. 214) that an important cause of acute cardiac failure is depletion of the coronary vascular system by imperfect transmission of blood from the right to the left heart. It is probable that in a more gradual manner this also occurs in dextral valvular disease and cardiac malformation, and to a less extent when there is a considerable degree of mitral stenosis. The nutrition of the heart being thus directly interfered with, it is not remarkable that the period of its activity should be curtailed.

In forming a prognosis in all cases of disorder of cardiac force we must also, as I have said, take into consideration the condition of the general arterial system, the state of the lungs and kidneys, the condition of the general nutrition of the patient, his sex, age, temperament, habits, and circumstances, the probable consequences of associated disease in other organs, and the liability of old mechanical lesions (valvular diseases) to favour the growth of organisms causing infective endocarditis.

Whether in actual cardiac failure (hypotonia) the diminution of the work of the heart will be followed by the re-establishment of cardiac sufficiency, or not so followed, will depend upon the degree of change which has been effected by time in the factors underlying nutrition on which the tonic power of the heart depends, and whether the reflex mechanism (cardio-motor system) be involved or no. If, for example, obliterative endarteritis and sclerotic change, affecting the factors underlying tonic organic action, have permanently impaired the power of the muscle to recover tone, any rally which the patient may show will be of short

duration, if it occur at all. Given, however, essential integrity of the cardio-motor, cardio-tonic, and cardio-vascular mechanism, it is surprising, in many cases, how much cardiac failure may exist, manifesting all the evidences of defective cardiac aspiration and propulsion, and yet yield to suitable treatment by postural and physiological rest, and later to appropriate tonic treatment by drugs and exercises provocative of the factors which underlie cardiac nutrition, with the result that recovery compatible with an activity of life takes place, which astonishes not only the patient, but also those in professional contact with him.

CHAPTER VII

THE TREATMENT OF DISORDERS OF CARDIAC MOTION

NORMAL or orderly cardiac motion has an average rate, rhythm, and force, varying also normally with the various circumstances of emotion, exertion, food, and exposure to external stimuli, which are the natural environment of living man on this planet.

The addition of changes in the cardiac mechanism to which the heart has adapted itself, or of generally acting agencies in the blood, such as those which produce fever, induce conditions of cardiac action which are normal in view of the exciting causes imported, and these do not call for special treatment of the heart itself unless and until it evinces departure in action from that which is normal to the situation.

Cardiac force, as expressed by ventricular systole, may, as we have seen, be in excess of the normal. To determine the probability of our having, in any given case, to deal with such a condition, is necessarily the first step in our procedure, and I am aware that I am dealing here with a difficult subject. But, given a normal quality of bloodvessel, as determined by palpation of the arteries, and a definite rise above the average in systolic blood-pressure, or, in the case of an hypertrophied heart, in its movement of its containing cage, the thorax, we may justifiably assume that we are dealing with an excess of the output of ventricular energy—in a word, with hypertonia. Such excess may or may not demand modification by treatment.

Searching for the cause of this condition, we may find that

it is emotional, physical, or provoked as a reflex by some imbibed or inhaled agent. Or it may result from a combination of such causes.

The emotional factor in all cardio-vascular disorders must never be lost sight of. It may not be in our power to influence it greatly, and it may not be our place to be too inquisitive as to its source, but as man is not an insensitive hydraulic machine, his emotional nature, and the recognized influence on the involuntary organs of the emotions, whether stimulative or depressant, should not be overlooked in the general estimate of the causes of abnormal action. Huchard has very interestingly dealt with this point in cardiac failure [1] and relates the tragedy of several cases which collapsed under one form or another of chagrin and disappointment, while the experience of all could adduce instances of such failure.

The bromides, belladonna, the nitrites, and rest *in its widest sense* with the securing of sleep, are indicated under these circumstances. Such rest in this widest sense, it may not be in our power to inspire (for it is the peace—the εἰρήνη —which denotes a triumph over circumstances, the true 'conquest of the world'), but without it the labouring heart, whatever the recuperative power of its *muscle*, can often be little assisted by the physician. With it, patients with disorder of an organ so much influenced by the emotions as is the heart,

ὡς λυπούμενοι, ἀεὶ δὲ χαίροντες·
ὡς πτωχοί, πολλοὺς δὲ πλουτίζοντες·
ὡς μηδὲν ἔχοντες, καὶ πάντα κατέχοντες,

attain a calm which is anabolic in effect, adjuvant of every therapeutic measure, and itself therapeutic in an important degree, because, among other beneficial effects, it is conducive to recuperative sleep.

The question of securing sufficient sleep is important in this connection. As in the treatment of sleeplessness generally, we have to determine the dominant or effective cause in any particular case—that is, whether pain, dyspnœa, or any other active discomfort in connection with disease of the

1 'Maladies du Cœur (Arterio-Scleroses),' 1910.

heart is the chief cause of restlessness; or whether, alone or in connection with mechanical distress, the disturbing factor is psychical. In the first instance, the particular means effective in relieving or removing the physical distress, of which we shall learn more as we consider further the subject of the treatment of heart disease, will be of the first importance in treating the sleeplessness of the cardiac patient.

When the psychical factor is dominant, as it often is, from the apprehensiveness of the cardiopath, his sleeplessness must be treated on the rational lines found most effective in such circumstances apart from any local affection, care being taken to avoid a quantity or quality of drug which enfeebles the heart.

What are these rational lines, and what the best hypnotics for use in heart disease? It would be out of place to enter here at length into the nature and management of insomnia, but an appreciation of the chief factors in the causation and treatment of sleeplessness in general is necessary to perceive the rational lines on which the condition is to be combated in particular instances.

As stated in a lecture on 'Sleep and Sleeplessness,'[1] I have been in the habit of dividing cases of insomnia into those of (1) cellular or cerebral origin, (2) into cases of neural, and (3) into those of hæmic insomnia, according as the psychical, the sensory, or the hæmic factors play the leading rôle in any given case. That these factors are frequently allied and conjointly operative in bringing about sleeplessness is frequently demonstrable, and this is so in no sphere more frequently than in that of heart disease.

Regarding cellular as synonymous with psychical insomnia, neural as including cases in which sleep is disturbed by afferent impulses of a more strictly physical kind causing discomfort, and hæmic as dependent upon abnormalities in the distribution and quality of the blood, it will readily be perceived how frequently these conjoint causes may be operative in disorders of the cardio-vascular system.

The rational lines, therefore, on which sleeplessness in

[1] *Lancet*, February 8, 1908.

heart disease has to be treated will recognize these avenues for predominant influence, and more success will frequently attend the use of a *combination* of hypnotics than the employment of a single drug acting especially on the centre or periphery, as the case may be.

The production of hypnotics by competitive commercial laboratories has of late years been very active, but few are without objectionable effects, which render great care in their employment necessary, and many, from their depressant action on the heart, cannot be recommended at all in hypotonic states of that organ. A very useful and discriminating paper on the subject was read by Dr. W. H. Willcox at the last meeting of the British Medical Association at Brighton,[1] in which, with brevity and perspicacity, he set forth the advantages and disadvantages of the motley series of hypnotic drugs now at the service of the prescriber.

The hæmic factor in cardiac insomnia is usually, under routine treatment, in the management of disordered conditions of the heart, and need not, therefore, be specially met by an ingredient in the hypnotic of the cardiopath; while the bromides, chloralamide, or small doses of chloral, with a moderate dose of opium or one or other of its extracted alkaloids, form the most useful combination to combat the cellular and neural factors in the insomnia of the cardiac patient.

When a **hypertrophied heart**, tethered by viscero-pericardio-costal adhesions, or not so tethered, is expending its energy or has a hypertonic erethism called forth by having to strike ribs and to lift them as well as blood, it may be desirable, as in the case I have already related as one of hypertrophic endocardial angina (p. 107), to relieve the organ by removing some of the weight it has to carry—namely, by surgical means.

In a paper published in the *Lancet* of July 28, 1906, and read before the Æsculapian Society of London on April 30 in the same year, I considered the circumstances of the normal growth and action of the heart, and the manner in which hypertrophy of the organ is influenced by its adhesion

[1] *British Medical Journal*, September 13, 1913.

to the pericardial sac and extraneous structures. I remarked in conclusion: 'To return to our embryo, the growing organ requires a surplus of room to grow in; the overgrown organ requires more room to work in, and it may be that, tethered by extraneous adhesion, or not so restricted, the hypermyotic heart may in the future, and in some cases, be provided with such by the genius and courage of some surgeon bold enough to undertake the task.'[1]

I was not aware, until I read Professor Wenckebach's paper on 'Some Points in the Pathology and Treatment of Adherent Pericardium,' in the *British Medical Journal* of January 12, 1907, that Professor Brauer, at that time of Heidelberg, had induced Professor Petersen and Dr. Simon to perform an operation for the relief of cardiac motion in such cases with success so long ago as 1903,[2] and had termed his operation 'cardiolysis.' Dr. William Mackenzie, of Melbourne, unaware, like myself, of Brauer's work in cardiolysis, gave very definite instructions for a proposed operation for freeing the heart from costo-pericardial adhesions in the *Intercolonial Medical Journal* of September 20, 1906. Dr. Mackenzie even advocated removal of a portion of sternum as well as the ribs, a procedure which Dr. Simon adopted in two of Brauer's cases.

This is a subject which has long interested me, and in my book on 'Cardiac Failure,' published in 1897, I suggested the severing of pericardial adhesions for the relief of cor bovinum, which I stated would be 'an ideal triumph of modern surgery.'[3] Since then there has, however, been a growing conviction in my mind that it was less the tethering of the heart than its *bulk* and force of systole which were the determining factors in the situation, and that operation to afford room adequate for the free action of the enlarged organ was the primary consideration, whether the organ itself were tethered or not. One has so often observed post mortem a large, muscular heart usually associated with aortic valvular disease, and without extraneous adhesion, succumb to the mechanical difficulty, while possessing

[1] P. 212. [2] *Archiv für Klinische Chirurgie*, vol. lxxi.
[3] P. 88.

apparently an amount of wholesome muscle which one would, *a priori*, have imagined should have contended with the obstacle in the circulation for a much longer period had it not been exhausted by some other cause, and this without any evidence of involvement of the cardio-motor nodal system.

When the cor bovinum is without viscero-pericardio-costal adhesion, its power tends to be exhausted by the stimulus of cardio-costal impact and the movement of resisting structures by the heart. When a similarly hypertrophied heart has adhesions to the sac and ribs which impede effective systole, there is, in addition, the larger blood-content of a dilated heart to induce muscular exhaustion. The heart may be hypertonic in virtue of its drag on its moorings, although complete systole may be prevented by such adhesions. In both cases the prime necessity is more room in which to act, and this afforded, the viscero-pericardio-costal drag is at the same time relieved and more complete systole favoured. It has been suggested[1] that venous inflow into the right heart is impeded in adherent cases. This in itself, however, is not an unmixed evil, for a limitation of venous *inflow* is under the circumstances rather an advantage, and allows the heart to rid itself more easily of blood-content. The accumulation of blood behind and outside the heart is less immediately dangerous than its collection *in* the heart.

Brauer's suggestion that the adhesions in adhesive pericarditis should be left alone, and the incarcerated heart freed from bony encasement, together with the successful issue of the operations consequent on his suggestion, pointed the way to a wider application of his method. Whether the term 'cardiolysis' is the most appropriate one to designate an operation which has to do, not with freeing the heart from adhesion, but with providing the overgrown organ with more room, and saving it the shock of impact against bone, may be questioned. Thoracostomy is perhaps too general a term to indicate its special application to the relief of intrathoracic pressure exerted upon the heart, or induced by

[1] Simon, *British Medical Journal*, December 14, 1912.

a large and powerfully pulsating organ confined within the thorax, because, as we know, thoracostomy may be employed for various conditions not directly due to cardiac causes. But, qualified by words denoting its special application in particular cases, there seems no insuperable objection to the substantive use of a more general term. Turnbull has suggested the term 'præcordial thoracostomy' for such an operation, and this is sufficiently descriptive of the procedure.

In a paper published in the *Lancet* of August 24, 1907, which, like that on 'Pericarditis,' had also been previously read before the Æsculapian Society, I further emphasized the principle of operative interference to relieve intra-thoracic pressure, both when the mere bulk of the organ was the chief feature in the case, and when, as in irremovable mediastinal tumour, the heart was secondarily involved in the neoplastic growth. I referred in that paper, but without giving details, to a case of the latter kind then under my care, in which thoracostomy with this object had been performed with benefit, on my suggestion, by my colleague Mr. Ewen C. Stabb. I had not, however, at that time had an opportunity of applying the principle, as I contemplated doing, to the much more important, because more frequently occurring, cases and those more amenable to treatment, in which *size* of heart, with or without adhesions, was the principal feature of the case, but closed the paper with the following words: 'The question arises whether mere bulk is not a disadvantage in these circumstances, and whether, therefore, the justification does not arise for attempting to relieve pressure by the removal of a portion of the ribs and cartilages in the præcordia, in obedience to the indications given by the præcordial bulging of children, even when it may be assumed that pericardial tethering is absent.'

That a correct diagnosis should be arrived at, if possible, as to the presence of pericardial tethering is doubtless important, as it would naturally guide the surgeon as to the method he adopted in operating, and more especially as to whether a portion of the sternum as well as the ribs should be resected. William Mackenzie's suggestion that a portion of the fifth

rib-cartilage should first be removed to afford an exploratory aperture seems valuable. 'This removal of the fifth,' he remarks, 'I regard as the keystone of the operation. If the intrapleural area is free, we have settled in a few minutes the diagnosis of adherent pericardium, and on that ground alone I consider the operation is justifiable. If we are unable to feel the intrapleural area, we know there are adhesions.'[1] The presence of such costo-pericardial adhesions, with which in the case of enlarged heart it may be assumed in many cases that viscero-pericardial adhesions are probably associated, may indicate the removal of a portion of the sternum in addition to the ribs, as practised by Simon in two of Brauer's cases. But if the clinical phenomena can exclude such adhesion, which, it will be admitted, is often a difficult matter, and especially in aortic cases, in which the character and direction of hypertrophy usually displace the volume of the left ventricle downwards and to the left, it will probably be found that the best guide in most cases to the amount of thoracostomy necessary will be the area of forcible cardiac impulse, and that the removal of ribs and cartilages without interference with the sternum will fulfil every purpose.

When Ernst Venus wrote his article on 'The Surgical Treatment of Pericarditis and Chronic Adhesive Mediastino-Pericarditis (Cardiolysis)'[2] in 1908, seventeen cases had been operated upon with more or less benefit, and without any fatality directly due to the operation. Since then, and excluding my own case already mentioned, a case has been reported by Poynton and Trotter,[3] and another by Sir Robert Simon,[4] both of which survived operation and derived some benefit; and there have also been one or two others. But the measure is not without danger, immediate and more remote, and I regret to state that my own experience furnishes this warning note.

Of two young subjects, one with cor bovinum with little

[1] *Loc. cit.*

[2] *Centralb. d. f. Grenz. Geb. d. Med. u. Chir.*, ii., p. 536.

[3] *Lancet*, June 19, 1909.

[4] *British Medical Journal*, December 14, 1912.

adhesion, and one with much, the former died three months after operation from secondary pericarditis with effusion, and the latter, on whom the operation was very expeditiously and skilfully performed by Mr. E. C. Stabb, within twenty-four hours from pulmonary embolism. The third case, that of a woman about thirty-eight years of age, the subject of mitral disease with viscero-pericardio-costal adhesion, succumbed to the anæsthetic before the operation was begun.

The conclusion I have arrived at from my own experience —and it appears to be that which has impressed itself upon others also—is that most favourable results are to be expected when the operation is undertaken while considerable cardiac power still remains.

As Venus states (*loc. cit.*), 'The usual consequences of cardiolysis are retrogression of the signs of lost compensation on the part of the heart, increased diuresis, the disappearance of albumin from the urine, and lessening of ascites, and of enlargement of the liver and spleen. Patients who before operation were bedridden and incapable of work are restored to health—may, indeed, as one case of Brauer's proves, in spite of an improvident manner of life, for years pursue severe bodily labour with comparative comfort.' The latter is, however, an exceptional case. The results of the operation, when successful, are the disappearance of many evidences of cardiac disablement and the restoration of comfort to the patient, but life has to be conducted on a lower plane of activity, as a rule, than was the case in the instance above quoted from Brauer's experience.

Hypertrophy and hypertonia, we must remember, are expressions of the *vis medicatrix naturæ*, and only require treatment when by excess in growth or impediment to conservative action they tend to embarrass the circulation rather than aid in its maintenance. Even under these circumstances the rational management of hypertonia does not necessarily involve surgical operation, although in carefully selected cases dextrously operated upon, the results are such as to constitute the procedure the only efficient and rational line of treatment.

When circumstances permit of sufficient care being taken

of the patient, much may be done by a regulation of the ingesta, postural treatment, supervision of exercise, and care of the abdomen to render life tolerable, to avert circulatory accident, and to postpone the inevitable hypotonic period.

The vast majority of the cases of abnormal cardiac action with which we have to deal are due to *insufficient* ventricular force, not to an excess of it—in other words, to one or another degree or mode of cardiac failure or **hypotonia**—and the determination of this condition is more easy and less questionable than of the opposite state.

Assuming, again, a normal condition of bloodvessel, or reasonably appraising its value, and by *striking the average for the individual* of his blood-pressure, we can find by this means a lessened ventricular energy which suffices for the practical instinct if it does not satisfy scientific precision. In many cases we have also, as I have stated, the unquestionable collateral evidences of cardiac failure in lung and limb and other extracardiac organ.

Under these circumstances we must employ means which stimulate the ventricle directly or indirectly to more energetic and effective action, and among such agents that 'opium of the heart'—digitalis and the digitalis group—stands in the first rank. I have said that these agents act directly or indirectly, and these terms raise interesting questions. That digitalis acts on the central nervous system in the medulla oblongata is not denied.[1] That it likewise acts by increasing the tone of the cardiac muscle is maintained.[2] In any case, its therapeutic or practical effect is the opposite to that of belladonna and the nitrites, and results, when it acts beneficially, in reducing the rate, improving the rhythm when disordered, and raising the force of ventricular contraction. Those who regard ventricular tachy-arhythmia as due to auricular fibrillation, consider that by producing more or less blockage in the auriculo-ventricular bundle, it rests the irregularly beating ventricle, and also allows it more leisurely to produce its invaluable 'stimulus material.'

[1] Cushny, 'Pharmacology and Therapeutics,' fifth edition, p. 360.

[2] Cushny, *loc. cit.*, p. 362.

This hypothesis does not, of course, account for its having the same effect when a like retardation results from its use in the absence of auricular fibrillation as in the aortic case to which I have referred (p. 205). But the action of digitalis by blockage of the bundle has been called in question by Professor Cushny.[1] He has shown that the a.c. interval may be normal, the inhibitory action of the vagus abolished or assumed to be abolished by atropine, and yet that the ventricle is quieted by the use of digitalis.

A practical consideration of moment in the use of digitalis in cardiac failure is its sufficient dosage. There are those who prefer Withering's method of giving large doses from the first until a digitalis effect has been produced. In the presence of urgent failure this may be advisable, or even as Cushny has suggested, the intravenous injection of strophanthus when time is an important consideration. But in the majority of cases the drug will be found quite as effective and more safe when the higher dose is reached by gradual augmentation, and frequently the desired result may be obtained without approaching the maximum and without producing any of the signs of a surfeit of digitalis as expressed by nausea. What applies to digitalis in particular applies to the whole digitalis group.

But the successful employment of the digitalis group in cardiac failure frequently requires a calculated boldness in its use. 'A patient is before us with the evidences of progressive cardiac failure. He is dyspnœic in various degree, his cardiac area of percussion dulness is increased, he shows the evidences in the heart—and it may be also in other organs—of the effects of a residual accumulation of blood within the chambers of the heart, right or left, or both right and left. We put him to bed, unload his primæ viæ, take into consideration his age and the general state of his vessels and other organs, as well as the particular condition of his heart endocardially, myocardially, and pericardially.

'Finding, as we unfortunately do in many cases, that rest, dietetics, and general care have, unaided, little effect on his state, we try the influence of small doses of the infusion, or

[1] Proceedings of the Royal Society of Medicine, vol. v., p. 200.

tincture, or powder of the well-known pharmacological antagonists to cardiac dilatation and imperfect systole, or combine these under certain circumstances with mercurials, neural stimulants, or more powerful vaso-motor agents such as the nitrites. Fortunately, in many instances, these measures are soon successful. Not every abdominal case comes to laparotomy, nor does every cardiac case require a bolder method of control.

'But, on the other hand, many cases unfortunately do require such treatment if they are not to be allowed to drift into hopeless failure. The danger then lies, as in abdominal cases, in delay, with merely palliative treatment, and the Micawber-like expectancy of something beneficial resulting from a timid use of agents and a plaintive hope in the recuperative powers of Nature.

'A large majority of the cases of which I speak are met with in people either young or in the prime of life. The vessels are pliant, the reserve force of the patient is commensurate with his development and age, but the golden bowl is breaking, and the silver cord is in imminent peril of being loosed, unless we can effectively come to the aid of the labouring heart and surcharged venous system. How are we to do this? How is the patient to be prepared for the action of an agent which, held by the physician in a vial, is as powerful for good and for evil as the knife which the surgeon holds in his hand?

'The vascular system of the patient must be relieved of a certain quantity of blood if necessary, varying from 4 ounces to ½ pint by venesection; his anasarcous limbs must be drained by Southey's tubes or by incision; fluid effusions hampering respiration in chest or abdomen must be removed so far as possible—and all this, not with any great expectation of these measures being successful in themselves, although this is not impossible, but to prepare the medical patient, as the surgeon prepares the surgical patient, for the use of the powerful agents which the physician has at his command.

'These are digitalis or its congeners in action, and of these digitalis itself and strophanthus are the most useful, as

they are also the most powerful, and the tinctures of these, which contain all the active principles of the plants the most efficient as they are also the least irritating preparations of the drug in cases suitable for the method I am about to describe.

'Small doses, then, in the majority of these cases have previously been tried in vain; so far as we can determine, the vessels in the case before us are sufficiently normal, and the heart itself is untethered by firm extraneous adhesion. The drug is then given in 15, 20, 25, 30 minim doses to the adult, at intervals of four hours, until a definite effect is produced upon the action, force, and capacity of the dilated and failing heart. This may require the continuous administration in such doses of the combined tinctures of strophanthus and digitalis until 200 to 300 minims have been taken.

'The patient may feel a little nausea at the end of the course, but his labouring heart may be acting slowly, smoothly, and with increased force, and the patient himself experience a relief to which he has been a stranger for weeks. This effect may be permanent or transient. The physician is not always successful any more than the surgeon. But both may have worked for and merited a success which they cannot always command. In laying emphasis on this principle of boldness in the use of the digitalis group under appropriate circumstances, I am quite aware that I am not inculcating precisely a *new* doctrine, for it is as old as Withering, who first tried digitalis with success in certain dropsies, of the organic cause of which he was ignorant, and it has been taught with no uncertain voice by others, and notably in recent times by the late Dr. George Balfour, of Edinburgh.' These are the words I used in a paper 'On Boldness in the Treatment of Heart Disease,' read before the West London Medico-Chirurgical Society in 1903, and published in its Transactions. Now, ten years later, I see no necessity for adding to or altering them.

A good deal of discussion has arisen lately as to the utility or otherwise of using *strychnine* in disorders of cardiac

motion. Dr. F. W. Price has examined the matter carefully, given large doses of strychnine, and measured the blood-pressure at short intervals, and on several occasions in the same individual, and under like circumstances. As he has found no evidence of increased blood-pressure with the hæmodynamometer he concludes that strychnine has no effect upon the heart. This does not, however, as it appears to me, touch the question of the utility or otherwise of employing strychnine in cardiac failure. Under these circumstances we have to aid the adjuvant mechanism of respiration, and strychnine, by its effect on the respiratory centre, and by raising the tone of the skeletal muscles of respiration, fulfils an indication in the treatment of cardiac failure which none can afford to neglect. The accentuation, moreover, of the reflex sensibility of the cord cannot but affect the spinal connections of the cardio-tonic nervous system referred to in Part I., of the existence of which there is much clinical and experimental evidence.

While the 'opium of the heart'—digitalis—may afford rest to that organ in the way indicated, the value of the *drug opium* itself and its alkaloids, as agents beneficially affecting the disordered heart, may be frequently observed. *Sleep* induced by opium, by its removal of the emotional factor, and by its conservative, anabolic, or building-up influence, as well as in some measure by the stimulation of the inhibitory process in the medulla oblongata by slowing respiration, may be observed to increase the force of ventricular systole. Other hypnotics have a like effect in so far as they induce sleep, but the certainty with which opium and its alkaloids insure it constitutes them the most valuable of agents acting upon the higher centres of the nervous system in cardiac cases, provided the emunctories be fairly healthy. Of its use in particular modes of motor disorder I shall speak later. That it may with advantage in many cases be combined with the bromides and small doses of chloral as an hypnotic I have already indicated (p. 230).

In discussing the principles of the management of the *blood-weight* in the treatment of disorders of cardiac motion, I shall have to remark upon the conditions indicating the

withdrawal of blood from the system. But I may here refer to the employment of certain agents which increase the stimulating and respiratory value of the blood upon which both the muscular and neural factors depend for their activity.

In the opinion of the late G. A. Gibson, Parkes Weber, and some others, the increased percentage of red blood-corpuscles in the blood, especially noted in cases of cardiac malformation, is due to an absolute and compensatory increase in the production of red blood-cells rather than to stagnation, and is called forth by the defective oxygenation of the blood by the lungs, which is met with in such cases. The cardiac debilities also witnessed in pronounced anæmia likewise show the importance of a normal quality of blood in such cases, while their improvement under hæmatinics, such as iron and arsenic, is frequently observed.

The red cells we know act chiefly as oxygen-carriers, and experience teaches us that there is a distinct value in the treatment of cardiac failure by the direct inhalation of oxygen gas. Surgeons, more frequently than physicians, have an opportunity of witnessing the effect of the inhalation of oxygen on the character of the blood. I am told by my friend, Mr. Lockwood, that the cyanotic patient with dark blood escaping from the vessels of a wound, soon after the inhalation of oxygen, shows a definite arterialization in the colour of the blood escaping from the local issue.

CHAPTER VIII

THE TREATMENT OF DISORDERS OF CARDIAC MOTION *(Continued)*

THE **physical treatment** of disorders of cardiac motion includes the drainage of effusions, blood-letting, posture, dietetics, the use of baths, active and regulated exercises, climatic treatment, and the applications of general hygiene.

Cardiac failure has reached a grave degree when the returning circulation has become so sluggish as to relieve itself into the cellular textures of the extremities, and is still more embarrassed when the overflow is into the serous cavities below and above the diaphragm. While large anasarcas and effusions into the peritoneal cavity may disappear as the patient is resting and using cardiac stimulants, effusions into the thorax are less amenable to medicinal treatment. In all these situations, however, the direct removal of effusion may be advisable and necessary, by means of drainage by Southey's tubes, or paracentesis by the larger trocar, as I have already indicated while considering the preparation of the patient for the bolder use of digitalis.

Serous effusion is, indeed, a mode of natural bleeding, which indicates a principle which may guide us in the employment of phlebotomy or leeching. The inflow into the right side of the heart may have to be momentarily checked, to enable the aspirative and propulsive force in lung and heart and artery to absorb effusion, promote more vigorous cardiac motion, and evacuate overflow by the emunctories.

The utility of **leeching** and **venesection** in some phases of

cardiac failure, in which experience has appeared to some to justify their employment, has been questioned by others. That the *pain* attending the enlarged liver of retrograde stasis, and the added restlessness of the patient attendant upon it, is greatly relieved by the application of a few leeches to the right hypochondrium, is so often witnessed, that the utility of leeching for *this* purpose cannot be doubted. Were the scepticism as regards the influence of blood-letting in relieving a distended right heart limited to that considered at times to be effected by *leeching*, one might hesitate to disagree with this view. But such consent need not apply to the question of the utility of *venesection* in this state under certain circumstances. The difference is due, I believe, to the greater rapidity with which blood going to the right heart is withdrawn by venesection, as compared with leeching.

I have been in the habit for many years of illustrating the principle, which underlies the employment of venesection and leeching, by a simple pneumo-hydraulic apparatus represented in the annexed illustration, which was published, with a paper I wrote 'On the Efficient Treatment of Cardiac Failure,' in the *Lancet* for January 30, 1904.

L.H. is a cistern containing water and representing the left heart; *S.A.* is the systemic arterial circulation; the Y-tube *S.C.* represents the systemic capillaries; while *S.V.* is the systemic venous system and *s'.v'.* a vein capable of representing a venesected vessel *v.s.* and the perforation *l.*, at its distal end, a leech-bite. *R.H.* is the right heart to which is attached a manometer *M.* containing a column of mercury *Hg.* From *R.H.* issues the pulmonary artery *P.A.*, passing to the bottle *L* capable of being exhausted of air by aspiration and representing the lungs. Water aspirated into it (*p.c.*) represents the pulmonary capillaries, the fluid in which may find issue by a neck at the bottom of the bottle and be returned by tubing representing the pulmonary veins (*p.v.*) to the left heart. *G.* is a glass U-tube which prevents collapse of the tubing if the lung-bottle be placed at a lower level than the 'heart,' as it may be, with equal effect. If, when the apparatus is filled with water and the mercury

stands at a certain level, the 'vein' *s'.v'.* be left open at its lower end *v.s.* (venesection), the mercury rapidly falls in the

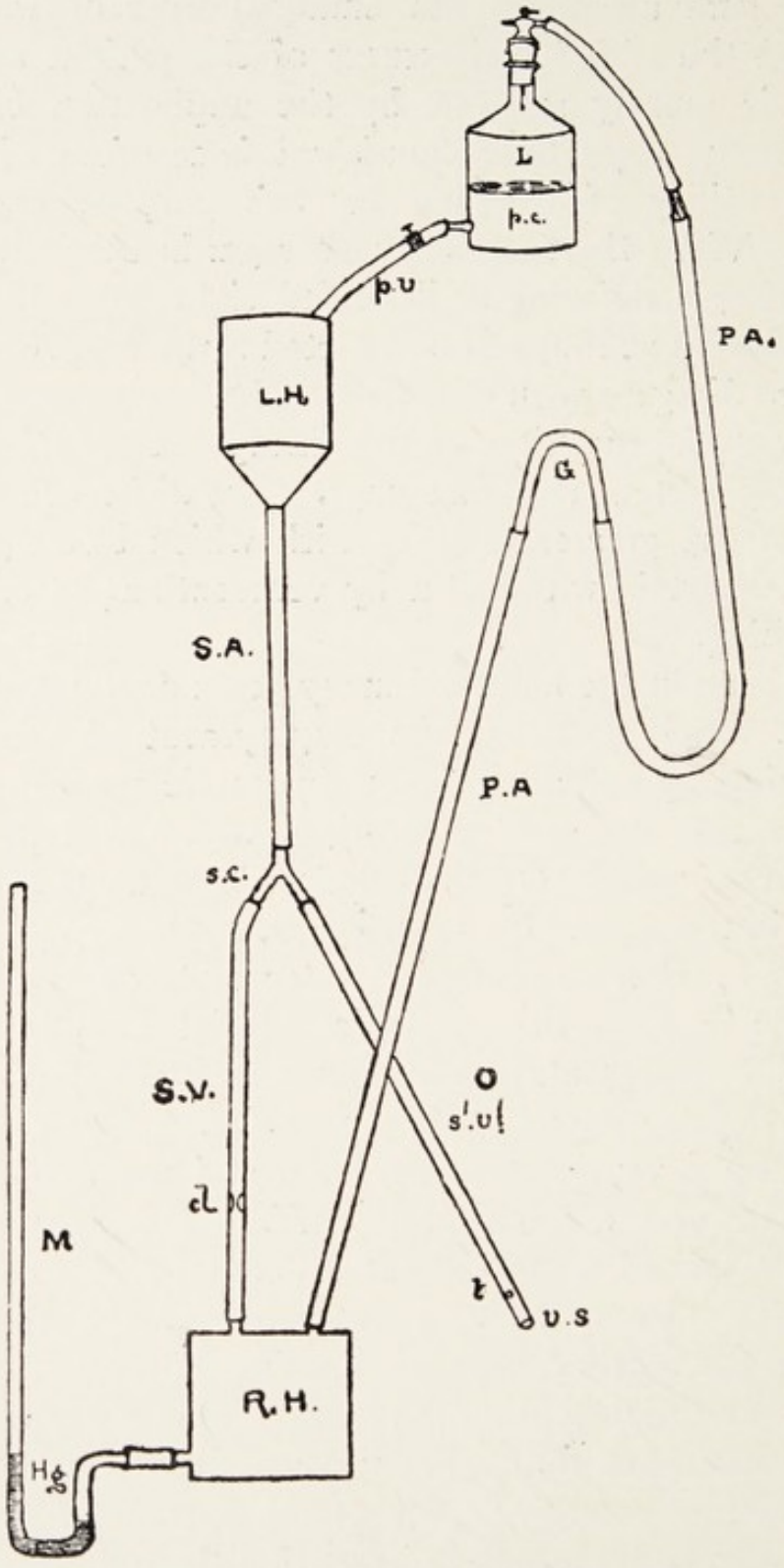

FIG. 51.—SCHEME OF THE SYSTEMIC AND PULMONARY CIRCULATION.

manometer. If the end of *s.v.* be held, but water be allowed to escape from the small hole, *l* (leeching), the fall in the manometer is slow and little. If, with *s'.v'.* closed and the

aspirated bottle in action, the continuity of *S.V.* be momentarily interrupted at *c.l.*, the mercury at once rapidly falls in the manometer just as it does when venesection is represented. If *S.V.* be left uninterrupted, the full aspiration of the 'lung' *L* has little effect upon the manometer.

The conclusion I draw from this simple experiment is that venesection is equivalent to interrupted inflow, and that both enable the right ventricle and lungs to act more easily than when the former is over-distended, while the *slow* reduction of the total quantity of blood by leeching effects this purpose in a proportionately small degree.

Could one indeed for a short time compress even the inferior vena cava without at the same time narrowing the aorta, the manœuvre would be equal to a large venesection, and the retention of blood in the lower extremity by the old boot-vacuum might also be a measure to be reintroduced with benefit.

There is, indeed, a certain analogy between the processes of filling and emptying the venous system. Intracellular transfusion may be compared with leeching, and intravenous with venesection. But intracellular transfusion is, on the whole, more potent in accomplishing the object for which it is undertaken than is leeching in that at which it aims. Venesection is a temporary measure, calculated to relieve the over-distended heart, just as catheterism relieves the over-distended bladder, and permits it to recover effective action. Even post mortem we may observe the elastic shrinking of the chambers of the heart, notably of the right auricle when the contained blood is evacuated, and we know that the hazardous operation of puncturing the distended right auricle during life appears to have been beneficial.

The operation of blood-letting itself, although a simple one, is one in which our predecessors in a past generation were experts, and we, from the disuse of bleeding, are not. Their method, with the addition of the aseptic precautions of which we now know the value, was preferable to the more elaborate technique often witnessed in the present day.

Bind the arm and *see* if possible the vein you propose opening. If the median cephalic or median basilic be

invisible or too deeply covered by subcutaneous fat to be easily accessible, select any other vein of moderate size which is visible, divide the skin longitudinally over it and the vein obliquely. After blood has been drawn, stitch the skin if necessary, and after cleansing apply a sterilized pad over the incision, securing it with a bandage. There is no necessity for freeing the vein, passing ligatures beneath it, and dividing the vessel after phlebotomy, as is often now done.

The quantity of blood, it need scarcely be added, may also be reduced and regulated by a reduction and regulation of the quantity of **food** and **drink**, and by the posture of the body, as regards accumulation in the right side of the heart. The orthopnœic position assumed by the embarrassed cardiac patient is an instinctively taken attitude, which at once limits the venous inflow into the heart, and increases the aid lent by the lungs in conducting the circulation. Judicious **purgation** may also serve a like purpose. Graduated feeding and due evacuation are as important as *graduated exercises* when the latter are indicated.

As regards **graduated exercise** and **exercises**, it has already been mentioned how the rhythm of the heart may be altered by movement of any kind. This fact has been utilized by the introduction of a series of graduated resistance or gymnastic movements, to provoke alternating phases of increased and lessened muscular effort on the part of disabled and hypotonic hearts, by means of which more vigorous action may in suitable cases be restored, and the blood distributed better in the circulation.

If, during the progress of a gymnastic movement, the stethoscope be placed over the heart, and a *bruit* be present, the pitch of the bruit will be observed to rise until a certain highest note is reached, and on cessation of movement, the pitch will be found to fall to the original. The heart may at the same time be observed to quicken, and on cessation of movement to become slower. When the movements made exceed a certain severity, and the general force of the subject is good, a fall in the frequency of the heart's action may in some cases be observed when the movement is at its

acme. But the rule is, acceleration in proportion to exertion, unless some degree of ventricular bradycardia be present, in which case the employment of exercises is contra-indicated. The cause of the raised pitch of the bruit under these circumstances, associated as it is with increased rapidity of heart's action, can only be ascribed to increased velocity of blood passing through the orifice at which the bruit is generated, unless we assume a diminution of the orifice by increased contraction of the heart during the movement.

Inasmuch as the force of a moving body is in proportion to its velocity, it follows that its progressive and retrogressive impulse must alike be increased under these circumstances. We can, therefore, easily understand that dilatations of the heart, especially when *unassociated* with conditions favouring the regurgitation of blood, rapidly rid themselves by increased propulsion of blood accumulated in the chambers. A system of therapeutic gymnastics to utilize such a provocation of cardiac movement was first elaborated in Sweden, and again brought prominently into notice with certain modifications in Germany by Dr. August Schott, of Nauheim, who combined with the gymnastic the balneological use of the gaseous and saline waters of that spa.

These thermal, saline, and gaseous waters have the effect of stimulating the entire cutaneous surface of the body, which is reddened by their warmth and chemical composition, and thus an easier peripheral circulation is promoted, and in suitable cases, and in those not too gravely exhausted, prove beneficial in restoring a more vigorous cardiac action.

Exercise as distinct from graduated exercises in hypotonic conditions of the heart was advocated by William Stokes of Dublin so long ago as 1854,[1] and Oertel of Munich on the same lines elaborated a system of graduated hill-climbing, with a restriction of fluid dietary, in 1884.[2] Cautiously employed, exercise stimulates a hypotonic heart to more vigorous action, and distributes more fully the peripheral blood, while the adjuvant mechanism of the respiratory

[1] 'Disease of the Heart and Aorta,' p. 357.

[2] Ziemssen's 'Handbook of General Therapeutics,' vol. vii.

system is at the same time called into more vigorous action, and the emunctories generally in skin and kidney caused to act more fully.

We have now dealt in general terms with the treatment of disorders of cardiac motion—with hypertonia and hypotonia—and it only remains to consider that of particular forms or phases of abnormal movement.

Auricular Tachycardia, or tachy-extrasystolia, has not yet been observed with sufficient frequency for the formulation of a system of treatment. As an isolated phenomenon (which it has not been in my experience), it would call for little attention other than that on the recognized lines on which neural disturbances are dealt with—namely, the promotion of emotional and physical rest, the removal, so far as possible, of causes of reflex stimulation, and the management of the graver condition with which it is usually associated.

As stated on p. 172, Dr. Thomas Lewis found that reduction of the ventricular rate by digitalis in these cases was not accompanied by a proportionate fall in the auricular tachycardia, and Dr. W. T. Ritchie's observations are to the same effect.

The **Auriculo-Ventricular Tachycardia**, more frequently met with, when paroxysmal (ventricular tachy-extrasystolia), frequently subsides without treatment in a shorter or longer time. But, especially when occurring in cases of, or associated with, gross cardiac lesions, it requires care, and may be influenced for good by complete rest, the application of cold by ice-bags or Leiter's tubing to the head, and above all, by the use of opium.

Some years ago I saw a case, in consultation, in which mitral regurgitation existed with such a tachycardia of high rate which had not rapidly subsided. I advised an opiate, of which six doses were prescribed. The nurse, in ignorance or inadvertently, administered the six doses in one, which were equivalent to about 6 grains of opium. The patient had a profound and rather lengthy sleep, and awoke free from tachycardia. In another case, which was subject to attacks of tachycardia and other forms of disturbed cardiac motion in connection with cardio-vascular sclerosis, for

whom I had prescribed and used hypodermic injections of morphia in hospital, I found his friends, not knowing what treatment he was undergoing, surreptitiously brought him opium for secret consumption, having doubtless empirically found it useful. Judiciously administered, this agent is also more useful than any other in the more persistent varieties of tachycardia, not excluding that associated with thyroidism.

In connection with the treatment of thyroidal tachycardia, or Graves's disease, there are interesting questions which can only be briefly referred to in this place. There have been accounts of its operative treatment published abroad, which have not coincided with the experience of such methods in this country. Some years ago section of both sympathetics, and even avulsion of accessible cervical ganglia, were reported to have been successful in some hands, and on authority which was calculated to inspire confidence. The procedure even found tentative imitation in this country, but does not do so now. At that period I met on one occasion a friend, a distinguished surgeon, who told me he was on his way to cut both the sympathetics in a case of Graves's disease. I told him he would probably kill his patient, which he thought was a very lugubrious prophecy in view of the brilliant results obtained abroad. The prophecy was, however, unfortunately fulfilled with unexpected rapidity after the operation.

Partial thyroidectomy and ligation of thyroid arteries are not so hazardous to life, but a considerable experience of the procedure in this country has not justified the laudation of the measures resting on the experience of some Continental surgeons, especially Kocher of Berne. Cases undoubtedly occur in which thyroidal tachycardia subsides after such operations, sometimes less, sometimes more; but inasmuch as such subsidence is frequently observed without the employment of any such measure, the relation of cause and effect is doubtful. Nevertheless, in a progressive case threatening the life or prolonging the inactivity of the patient, a partial thyroidectomy or arterial ligation are quite justifiable means to adopt. Finally, the atrophic influence on glandular structures of exposure to X-rays has been employed with apparent benefit in some cases. Personally,

I have not employed this measure often so far, but I have seen apparent benefit in one case; while another, probably as a mere coincidence, developed mental aberration, and, I was informed, soon after leaving hospital died insane.

I have already mentioned the clinical observation that Graves's disease at times gives place to a more or less marked myxœdema with slow pulse. This fact has been utilized by the production of sera and other preparations from the blood of animals in which an artificial myxœdema has been induced. These have been administered for considerable periods in cases of thyroidism, and occasionally with apparent benefit, but more frequently with none. Personally, I have had so little confidence in their utility that I have scarcely ever used them without at the same time employing opium, and it is to the latter that I feel disposed to ascribe the benefit accruing as regards a lessening of the tachycardia. On the other hand, in the sluggish pulse resulting from the myxœdematous state following Graves's disease I have found the use of thyroid extract useful.

Auriculo-Ventricular Bradycardia being, as it usually is, a transient condition, post-febrile or post-tachycardial, calls for little treatment other than the general care of the convalescent by rest, warmth, moderate stimulation, and a bland and simple dietary.

There is an acute type of *ventricular bradycardia* which, in the absence of definite evidence of the simultaneous condition of the auricle, I am unable to place positively in the purely ventricular class, in which life is immediately threatened, but which is of an inhibitory—that is, unquestionably neural—type, which may be rescued from death by anti-inhibitants. In 1894[1] I published such a case, in which cardiac action was reduced to 12 in the minute in an infant, the respiration slowed and the pupils contracted, and in whom a large dose of belladonna injected *per rectum* and given by the mouth (90 ♏) was followed by a simultaneous dilatation of the pupils and sudden tachycardia of the bradycardial heart—so much so, indeed, that the child's life was threat-

[1] Med. Soc. Transactions, vol. xvii., p. 48.

ened by the tachycardia, which was reduced by applications of ice. The child recovered, and she is now a strong woman.

I have already mentioned (p. 118) a case of acutely developed bradycardia in an old man, whose heart-rate I knew to be ordinarily normal, and who responded to anti-inhibitant treatment to such an extent as to justify hopes of recovery, but who nevertheless died within twelve hours of the brady-cardial seizure.

Ventricular Bradycardia, when persistent, so-called 'heart-block,' is little if at all influenced by accelerant or other drugs. In a considerable number of these cases, however, the history of a specific infection may be obtained, and the treatment of syphilis instituted. The reputation which mercury and the iodides have acquired in the treatment of cardio-sclerosis generally may in a measure be dependent on its effect on a syphilitic element in some such cases. In combination with the iodides, mercury undoubtedly proves beneficial in cardiac sclerosis, as I shall mention when discussing the treatment of the arhythmiæ; but a persistent ventricular bradycardia uninfluenced by exertion, emotion, or agents which normally increase the heart-rate, shows, as I have said, little or no response to drug treatment. Knowing the liability of these cases to terminate by syncope, the care of the adjuvant mechanism of the circulation—respiration, thoracic and abdominal—is imperative. Strychnine as a respiratory and spinal stimulant, and securing by diet and suitable evacuation good diaphragmatic movement, is indicated.

While emotion and stimulation have no *accelerant* effect upon the ventricle in such cases, alcohol in small quantities is not contra-indicated. Inasmuch, however, as cardiac accelerants, emotion, and exertion may have an influence upon the supraventricular heart in these cases, the increased activity of which may tend to render still more replete the ventricle already too full from its lengthened diastolic pause, these agents, as well as emotional excitement and over-exertion, have, the one to be given with care, and the others guarded against so far as possible. It is thus also, as I

have stated, that an auricular tachycardia may overtax the underlying ventricle and provoke disturbed action in it, *per vias nervosas*, and, as we have seen, digitalis may slow the ventricle, without affecting auricular action.

Rhythmic Ventricular Extrasystolia, even when recurrent, usually passes off with attention to general hygiene. In the event of any probable dominant cause playing a part in the production of the phenomenon, such as protracted emotional unrest, gastro-intestinal disturbance or tobacco, such factors must be combated on rational lines. This is true more often of the pipe than of the emotion. The latter, however, cannot permanently be maintained at the same level, and psychical disturbance in a normal nervous system tends to become tolerated or ignored, even if the disturbing cause be irremovable. I have, however, known emotional disturbance to be so deep as to perpetuate and aggravate, if not to initiate, a cardiac irregularity, which ultimately proved fatal. There was, however, in the case in question, a mitral stenosis—ultimately, no doubt, with cardio-sclerosis. The neural factor was, nevertheless, the active disturbing cause in this case. It was that of an affectionate mother, who was inadvertently responsible for the death of her child. She had had rheumatic fever, and was known to have valvular disease of the heart, but her cardiac failure commenced with the distress attending the unfortunate accident in question.

Arhythmic Ventricular Extrasystolia.—The rhythmical extrasystoliæ are not as a rule persistent, although they may last for a considerable period. They may, as I have said, constitute a transient phase, even in cases which have for long manifested an arhythmical extrasystolia. The most persistent type is arhythmical ventricular extrasystolia. It is rarely met with in young subjects in the absence of cardiac valvular disease, and this, as I have also stated, is much more frequently a mitral than an aortic lesion. In young subjects, also, or in those under middle age, this persistent irregular extrasystolia is usually a late event in connection with valvular disease.

Ventricular tachy-arhythmia (auricular fibrillation), as

manifested by rapid ventricular irregularity, and especially in connection with mitral valvular disease, frequently yields readily to the use of the digitalis group. In non-valvular cases, which are usually met with in more advanced life, these agents are less frequently of service. The condition is under these circumstances usually persistent.

Persistent arhythmical ventricular extrasystolia, in the absence of valvular disease, most frequently occurs, as stated, among those in middle or later life. It is a common manifestation of the 'senile heart.' The pathological condition usually associated with it is an interstitial sclerosis of the auriculo-ventricular textures. This condition may, from its situation, involve the auriculo-ventricular bundle without inducing bradycardia.

When persistent and with a cardio-sclerotic basis, it is not found to respond to drug treatment in the measure in which paroxysmal cases do, which are reasonably assumed not to be sclerosed. The irregularity may, however, have paroxysmal exaggeration even in chronic cases, and in this phase sufficient doses of digitalis may modify the condition, so that a tachycardial irregularity may subside into a condition which remains irregular but is not tachycardial.

The drugs which effect this change are the digitalis group and opium, and these in combination with mercury and the iodides. Matthew Baillie's pill, frequently known as the 'Guy's pill,' although Baillie had no connection with Guy's Hospital, often acts efficiently in this mode of cardiac motor disorder, even when the diagnosis of cardiac sclerosis may be justifiably made. Under its influence the tachycardial element in the irregularity may be notably reduced, and anasarca and other evidence of cardiac failure disappear. The water-logged patient, after a few days of its persistent use, may evacuate such large quantities of urine (for example, a hundred ounces a day) that this combination of digitalis, squill, mercury, and hyoscyamus occurs in some hospital pharmacopœias as, *par excellence*, the 'Pilula diuretica,' notwithstanding the competition of such more recent diuretics as theocin sodium acetate, diuretin—*et hoc genus omne*.

In an acute phase, and associated with such evidences of

cardiac failure as have been mentioned, drugs are naturally combined with physical rest, and a bland dietary calculated to suit a gastro-intestinal state, in which the defective circulation of the blood has its local manifestations.

In the absence of general evidence of cardiac failure, when the circumstances of the patient admit of his leading a rational life of limited exertion, care in dietary, both as to its quantity and quality, and the avoidance or careful use of tobacco and alcohol, are to be inculcated. When the circumstances of the patient do not admit of such care, but life has to be supported by more or less strenuous exertion, this must doubtless be undertaken, but experience shows that the heart, with increasing frequency, collapses under it, until it attains the permanent rest of everlasting diastole in death.

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