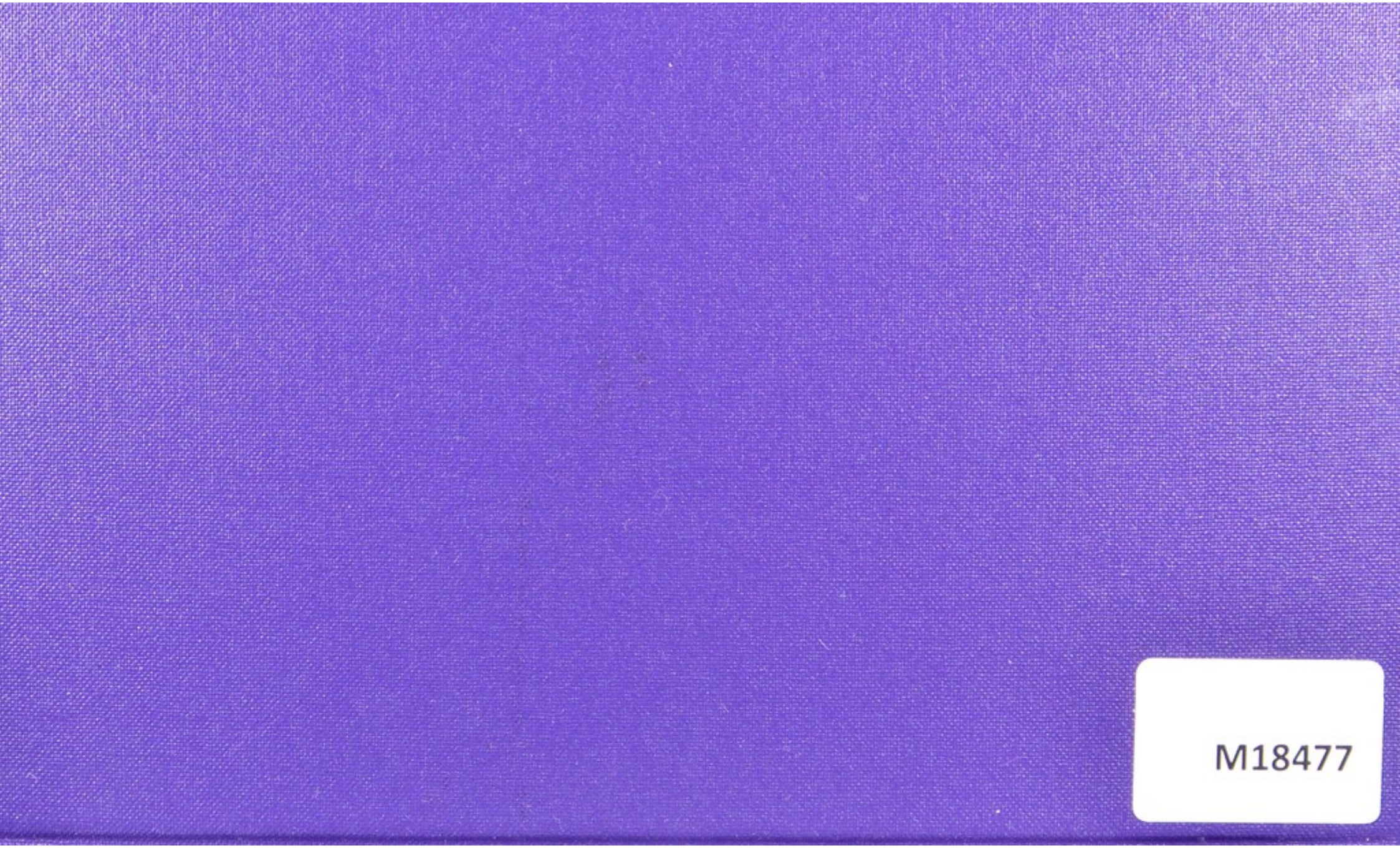
Notes and observaation on diseases of the heart and of the lungs in connexion therewith / by Thomas Shapter.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NOTES AND OBSERVATIONS

ON

DISEASES OF THE HEART

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ON

DISEASES OF THE HEART

AND OF

THE LUNGS IN CONNEXION THEREWITH

BY

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TO

JOHN HUTTON BALFOUR, M.D.,

FELLOW OF THE ROYAL SOCIETIES OF LONDON AND EDINBURGH,

PROFESSOR OF MEDICINE AND BOTANY,

AND DEAN OF MEDICAL FACULTY TO THE UNIVERSITY OF EDINBURGH,

ETC.,

These Pages are Dedicated

AS A MEMORIAL OF STUDENT DAYS NOW LONG GONE BY,

AND

IN TESTIMONY OF AN UNINTERRUPTED FRIENDSHIP

OF NEARLY FIFTY YEARS.

PREFACE.

THE following pages, having reference to the more ordinarily-occurring diseases of the heart, represent the mode, used in their investigation, which an extended experience has shown to be useful for practical guidance.

During the last forty-seven years my opportunities for observation have been extensive. In 1827, in conjunction with the late Mr. Robert Spittal, author of "A Treatise on Auscultation, 1830," my attention was specially directed to the study of pectoral diseases. We were then fellow-students and clinical clerks in the wards of the Royal Infirmary of Edinburgh, under the late Doctors Home, Graham, and Alison, the then very eminent Professors of Clinical Medicine to the University. In 1830 I enjoyed the advantage of further prosecuting this study in the wards of the Hôpital de la Pitié, in Paris, under the immediate guidance of the late Baron Louis, one amongst the many very illustrious physicians that France has produced. Since then, besides an extensive acquaintance with the occurrence of disease in private life, I have occupied the position of

Physician to the Exeter Dispensary, and to the Devon and Exeter Hospital. This latter position I still retain.

These several spheres of observation have afforded me opportunity of accumulating the records of a large number of cases.

In the course of the following pages reference to some few of these cases has been specially, but briefly, made. As a general rule, it has appeared to me to be more useful not to enter into any minute detail and transcript of cases, but to state solely the conclusions to be drawn from them.

It had been my wish, and my intention, to have endeavoured to determine with precision some of these conclusions, by referring the cases, with *post-mortem* examinations, to the test of the numerical method. But this mode of investigation I was obliged to abandon, for I soon found there was an absence of so many elements, necessary for the attainment of accurate and reliable results, as to render deductions thus made, unsatisfactory, and perhaps misleading.

The details of *post-mortem* examinations may be, in themselves, sufficiently trustworthy, but before the physician can venture to deduce from them any positive or scientific conclusions, it is especially necessary for him to know the relative amount which the cases thus examined bear to those not examined under similar, as well as different, circumstances of antecedent disease; and also to those in which the presence of these diseases, though existing, may not have been ascertained; then—many diseases, that have been latent for years, suddenly burst into activity, and it is difficult, after death, to

decide to what period of the disease the origin of the morbid conditions belongs; so also, during life, the physician often fails in ascertaining the origin of disease both as regards time and cause, in fixing and appreciating the true sequence of events, and in separating the complications of one lesion from another. All these difficulties serve to make deductions from a limited amount of observations, by the numerical method, deficient in necessary precision; and imperfect and unreliable statistics offer but unsatisfactory and dreary reading.

Many examples of rare and curious heart disease have passed beneath my observation. The consideration of these I have not ventured upon:—seeing that they yielded no certain nor reliable signs of their existence, the enumeration of their details would have failed in practical results.

Some apology is due for the order and mode in which the matters of the following pages have been treated. I am aware they have the aspect of being somewhat fragmentary, and deficient in scientific completeness. Still I hope they may prove of some small service, by way of suggestion, to my younger professional brethren, in which case I shall consider myself amply repaid for the trouble, and the expense of time, the preparing them for the press may have cost me.

The Barnfield, Exeter,

1*st June*, 1874.

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DISEASES OF THE HEART.

CHAPTER I.

CIRCULATION OF THE BLOOD.

An extended practical acquaintance with diseases of the heart cannot fail to have exhibited to the careful medical observer the very remarkable fact that analogous, if not absolutely similar, departures from the normal physical conditions of this organ may exhibit in different people very different results; in some cases they prove to be associated with fatal tendencies, in others to be consistent with a good average state of health and prolonged life; whilst in other cases a fatal termination may unexpectedly occur, though yielding but few, or even no indications of disease, to the most anxious physical investigation.

It may be assumed that those more obvious physical phenomena which the heart reveals, whether to touch, to careful percussion, or to the attentive ear, as differing from what is usual in health, are due to departures, perchance temporary only, from the normal condition of this organ; but it may also be concluded, where these phenomena are found to be persistent, that they are due to alterations, as the case may be, in its structure or bulk, or to disarrangements of its valvular completeness.

These structural alterations generally entail, sooner or later by themselves, or by their disturbance of the animal economy, a fatal termination. Nevertheless, conspicuous and very remarkable exceptions occasionally present themselves, and the issues of life or death do not appear to be influenced by the inferred condition of the heart; while, on the other hand, accidental or temporary departures from the normal condition may present themselves as very dangerous, and perhaps immediately fatal, affections. Again, fatal cases are occasionally met with, and which appear to be due to structural alterations scarcely, if at all, appreciable by careful examination, or, at any rate, to such as neither alter the size of the heart nor impede its blood-flow.

It is proposed, by reference to the notes of a considerable number of cases, to review some of the affections belonging to these several categories, and chiefly with the object of considering and determining their relative fatal tendencies. But, before doing this, it is proposed to say some few words respecting the action of the heart, in its relations to the circulation of the blood, and of the sources of the normal and abnormal sounds emitted thereby, as also respecting its influence on the pulse.

As regards the heart, it has appeared to me that the view of its action has been unnecessarily—I would even say erroneously—complicated by the general statement that the function of the auricle is that of the "propeller" of the blood into the ventricle. The simpler view that I would hazard is, that the ventricle is essentially the organ that, on the one hand, by a gradual screw-like expansion sucks in the blood, and, on the other, by a gradual screw-like contraction expels it; that the proper function of the auricle is, on the one hand, to contain a ready supply of blood for the expanding ventricle, and, on the other, a safety-pouch to counteract or equalize the backward strain caused by the closure of the auriculo-ventricular valves.

I have elsewhere (*Medical Gazette*, Dec. 5th, 1857) ventured

to state that, as regards the heart, the circulation of the blood is more simply effected than is usually considered to be the case; that, in fact, the heart proper is ventricular and ventricular only; and that the circulation of the blood through the heart mainly depends upon, firstly, its ventricular exhaustion or suction power; and then, secondly, on its ventricular contraction, whereby it is indued with the function of propulsion.

The auricle, on the other hand, is rather to be considered as an appendage proper to the venous system, and not as appertaining or necessary to the ventricular heart,* and that, therefore (without ignoring the influence, as propellers of the blood, of the capillaries and of the veins generally, feeble though this influence may be, as also of the auricle, as portion of the venous system), auricular contraction is not an active agent in the movement of the blood.

The above conclusions are grounded on the fact, that the distension, or greatest size, of the auricles is not induced by a sudden influx of blood from the cavæ or pulmonary veins, but on, and by, the final act of contraction of the ventricles, when there arises a retrograde pressure against the ever onward flow of blood from the venous system. This retrograde pressure is caused primarily, on the closure of the auriculo-ventricular apertures, by the floating up of the tricuspid and mitral valves, and subsequently by these being propelled beyond the auriculo-ventricular axes into the auricles; hence the blood then contained within the auricles is ponded back upon that flowing into them, and their greater distension effected. The relaxing of the valves on the expansion or diastole of the ventricles, whereby the exhaustion or suction

* This is borne out by the anatomical fact stated by Pettigrew (*Edinburgh Medical Journal*, April, 1873): "The auriculo-ventricular fibrous rings have been variously described, the majority of investigators regarding them as strongly pronounced structures, which afford attachment, not only to the valves, but to all the muscular fibres of the auricles and ventricles. A careful examination of these rings in boiled hearts has led me to a different conclusion. They afford attachment to the muscular fibres of the auricles, and to the valves, *but to almost none of the muscular fibres of the ventricles*."

power of the heart is exercised immediately, relieves the auricles of this amount of tension, and they partially collapse as the blood is first drawn from them into the ventricles, on the gradual uncoiling and screw-like expandings of these latter immediately after their final, sudden, and energetic act of contraction has been concluded. Their normal condition of filled but not distended vessels is then restored. In this state, by a kind of vermicular or peristaltic movement, they continue to receive blood and to deliver it into the uncoiling ventricles, till these latter, being again filled, again contract upon their contents, and so again raise the opposing valves.

Probably, during these several processes, the fluid supplied to the auricles and ventricles is so evenly balanced that the pericardium contains, at no one time, a differing amount of contents.

Though not entirely ignoring some muscular consent in the auricles, their normal quality appears to be that of elastic tubes or vessels, capable, within certain limits, of exercising the functions of expansion and contraction. In accordance, therefore, with the varying amounts of fluid supplied to them, they exemplify the phenomena of dilatation and contraction by elasticity rather than by dynamic muscular contractility; while the ventricles distinctly exemplify this latter. It should be here borne in mind that the auricles are furnished with a large relative amount of the elastic layers of the endocardium, and that the musculi-pectinati are fewer and weaker in the left than in the right auricle. Moreover, the thin and fragile structure of the semilunar, as compared with the tricuspid, and mitral, valves, strengthened as these latter are by the musculi-papillares, columnæ carneæ, and chordæ tendineæ, and their sole function being that of preventing regurgitation from the aorta, show them to be fitted, neither by position nor in structure, to resist the strain of a violent propulsion of fluid from the auricles into the ventricles; while the suction power of these latter, during their systole, is precisely such as calls the special function of these semilunar valves into active usefulness.

The very careful and valuable experiments of Dr. Halford,

and, previously, of Pechlin and others, fully confirm the above conclusions; for the ventricles were seen to expand as well as to contract, though no blood was furnished to them. Expansion, and hence a suction-power, being, therefore, a function as normal to them as the recognised one of contraction, and by the aid of which it is universally allowed they are endowed with the power of propulsion.

Supposing the above view of the function of the auricles to be correct, it is then probable that in addition to the support afforded by the strong and unyielding pericardium, the musculi-pectinati act firstly in preventing undue distension; and secondly, after distension has been effected, in aiding towards restoring the auricles to their normal dimensions.

The above, doubtless, is not in accordance with the ordinary explanation of the function of the auricles, which is, that the auricles contract on the blood contained within them, and thus propel it into the ventricle. There are three cogent arguments against this conclusion. Firstly, it would assume that the auricles contained an amount of blood which should be more than equal to the capacity and requirements of the ventricles on their expansion, and this, seeing they are never entirely emptied, they cannot be said to do; secondly, the absence of a normal and regularly recurring blood wave in the jugular and larger veins; and, thirdly, the absence of any sufficient valvular apparatus at the venous extrances to the auricles, with the exception of the valve of Thebesius, which only prevents regurgitation into the coronary vein, and this is only effected, by the elongation of the membrane forming it, at the moment of the greatest distension of the auricle on the closure of the tricuspid valve. Doubtless in the right auricle there exists the Eustachian valve; and, in the left auricle, the pulmonary veins on their entrance are surrounded by muscular fibres. But these structures do not present the adequate perfection of valvular mechanism; moreover, as age advances, the Eustachian valve becomes conspicuously deficient for all closing purposes.

This valvular deficiency would alone militate against the

assumption that the function of the auricles was essentially and primarily that of dynamic propulsive power; for not only is the absence of any such structural arrangement to such an end contrary to the whole principle of the circulation and the mechanism of its organs, but, in fact, the contraction of the auricles would as equally force the contained blood into the cavæ and the pulmonary veins as into the ventricles.

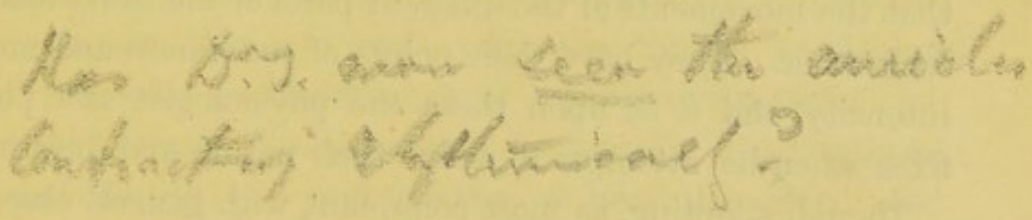
That the suction power of the expanding ventricle is adequate to produce a current in the larger veins, may be assumed by the murmur produced by pressure on the jugulars; while the phenomena observed in certain extreme cases of regurgitation from tricuspid deficiency, as also in those cases where, independently of valvular disease, there occurs an obvious blood movement in the jugular vein, with a varying state of distension of the vessel, indirectly confirm the whole of the above position. For while a true pulse is never to be felt in the vein, an undulatory motion is seen to culminate in its greatest amount of tension, and this greatest amount of tension to be synchronous with the pulsation in the carotid artery.

Again: the ordinary explanation—that the dilatation of the ventricles is passive only, and solely caused by the forcible discharge, by the auricles, of blood into them: or rather that it is divided into two distinct stages; the first due to the simple elasticity of the ventricular walls, and during which stage the auricles are still dilating; the second accompanied by the systole of the auricles, in which the blood is so forcibly propelled as to dilate the ventricles strongly,—is not only inconsistent with the above view, but the structure of the auricle does not warrant such a conclusion: it has neither the general strength nor the character of fibre for the exercise of so large a power, nor does the position of the musculi-pectinati suggest it.

Without discussing what may be the motive power exercised upon the blood in its course through the veins—how much may be due to capillary force, and how much to an elastic or even to a contractile power proper to the veins themselves—I here hazard the opinion that the auricles fulfil

two evident conditions: the one, in connexion with the venæ cavæ and pulmonary veins, of being the ready vessels of supply whence the ventricles, or heart proper, receive that blood which, on their expansion, they require; the other, that of being elastic expanding vessels, and representing those enlargements which invariably accompany the valves of the veins, by which the tension induced by the contracting ventricles, and the sudden closing of the valves consequent on the incoming stream of blood, is compensated for. And I would further hazard the opinion, that any muscular power the auricles may exert on the propulsion of the blood is only secondary.

Whether the closure of the auriculo-ventricular apertures, respectively by the tricuspid and mitral valves, be due to mechanical or vital causes, probably, as it appears to me, to the former, it is not here necessary to discuss. Nor is it necessary to seek for or determine the physiological cause of the rhythmic action of the ventricle. But assuming this simpler view of the heart's action to be correct—viz., that the ventricles act, first by a process of exhaustion, and so as to suck in the blood, and then by contraction to propel it onwards—we will pass on to a consideration of the sounds, normal and diseased, thereby emitted.



CHAPTER II.

SOUNDS OF THE HEART.

It is generally admitted, in accordance with the statement of Dr. C. J. B. Williams, that the series of phenomena which are associated with each complete movement of the heart (as the impulse, sounds, pause, etc.) occupies a cycle of time, divisible into five periods; two of these being occupied by the impulse, together with the first or systolic sound, which is dull and prolonged, the "rub"; one by the diastolic or second sound, sharp, short, and abrupt, the "dub"; and the remaining two by the pause. Some modern observers, however, speak of a third sound, the presystolic* (Vesal, Spring) occupying the latter half of the pause; Pettigrew states that the sounds are really continuous, running into each other by insensible gradations. "The sounds, in fact, merge into each other precisely in the same way that the movements of the different parts of the heart merge into each other. They have their points of maximum and minimum intensity, and it is upon these the physiologist and physician fixes when he attempts to define their nature and duration."

Though adopting, as most consonant with general observation, the above division of the heart's action into five periods, yet, doubtless, very many of the hearts that one examines will warrant a division of the cycle of events into four periods only, and, in which case, the pause is diminished by one half—the first sound occupying two periods, the second sound one period, and the pause one period.

* Spring (*Annales de la Société Medico-Chirurg. de Bruges*, 1861), who refers the sounds to the contraction of the heart and valves, attributes the presystolic sound to the lowering of the auriculo-ventricular valves.

The first and second sounds are now generally, if not universally, admitted to be synchronous with the closure of the valves; the first or systolic sound with the closure of those at the auriculo-ventricular openings, the second or diastolic with the closure of the semilunar valves.

The conclusion as to the direct cause of these sounds is not so unanimous. As many as thirty different sources of the sounds of the heart have been alleged. Some writers attributing them solely and entirely to the sound emitted by the contraction of muscular fibre, and therefore due to the act of contraction in the heart itself; some to the blow of the heart on the parietes of the chest; some to the vibration of the valves; and some to the effect of the heart's contraction on the fluid it circulates; and some regard these sounds as due to a combination of many of these causes. My own conviction is, that the sounds referred to are mainly, if not entirely, due to the natural interference with the even flow of the blood by the various mechanisms of the heart, and that they are to be referred to the ordinary laws of hydraulics, and little, if at all, to be attributed to the contracting act of the muscle of the heart, or to any vibratory motions in the substance of the valves.

This branch of our inquiry presents itself under difficulties, and I only approach it with diffidence, for many of the English school, as Billing, Halford, Fuller, etc., and nearly all the writers belonging to the foreign schools, refer the sounds to the act either of muscular contractions or of valvular vibrations.

Doubtless certain sounds may, under certain circumstances, be emitted during the contractions of muscular fibre; but these sounds are neither identical nor even analogous to the audibly distinct phenomena presented during the heart's action; moreover, the intensity of the sounds emitted by the heart is not, *pari passu*, in accordance with the existing development of the muscular structure of the heart; on the contrary, disease in this organ, whether of hypertrophy or of attenuation, rather shows the opposite fact to be the rule.

The valves of the heart, whether considered in relation to muscular contraction or to vibration, certainly do not

present *per se* those physical conditions adequate to afford a satisfactory explanation of the production of the sounds.

These valves are not, on the one hand, muscular in their structure, nor even closed through the immediate instrumentality of muscular contraction, but solely under the influence of the direct mechanical operation of hydraulic pressure; nor, on the other hand, is their size, under slow vibration, adequate to the production of so loud a sound; nor, supposing there were such adequate rapidity of vibration, is the condition of their floating in so dense a fluid, as is the blood, one thus likely to generate, or, if generated, to transmit sound.

Again, when on the closure of the several valves, the passage of the blood through their aid is not completely effected, though the valves may, and the ventricles must, contract, the former thus enjoying full scope for their vibratile qualities if they ever possessed them, and the latter for those muscular contractions which undoubtedly belong to them, the peculiar sounds said to be referable to their contractions and vibrations are no longer to be heard. It is true sounds are heard, but these are of a quality which cannot be referred to muscular contractions, or to membranous vibrations, though they can be, equally with the normal sounds, accounted for by hydraulic laws. In fact, as is so well known, a very slight disturbance of function, or alteration in structure in these valves, so as to effect a condition either of slight obstruction, or of imperfect closure, may be sufficient to banish entirely the normal sounds, and convert them into others, very different in their character; moreover, both the normal sounds in question are emitted at the precise moment the valves have become closed, and not at the time during which the process takes place, whereby their closure is effected; while any connexion of the second sound with the coiling muscular contraction of the ventricles, or with its final energetic act, is at once set aside by the circumstance of its not being synchronous with it.

Can these sounds be attributed to the heart's impulse, to the blow of the projected apex against the anterior walls of the

thorax. As just stated, this certainly cannot be the cause of the second and louder sound, for this latter takes place at a period of time, sensibly later than that which is occupied by the impulse or blow against the thoracic parietes. The dependence, therefore, of the second sound on this cause, may be at once set aside. It therefore only remains to consider what may be the connexion of the first sound with it. That it is not entirely dependent upon it is obvious from the often repeated experiment of so exposing the heart that it has no chest parietes to strike against. The heart, detached from its normal surroundings, yet carrying on the circulation, emits the sounds. Dr. Markham, in his practical and most excellent sketch of the diseases of the heart, addresses himself to this subject (*vide* Appendix III. "On the Sounds of the Heart"); and advances, as an argument for assuming this sound to be partially influenced by the projection of the apex against the anterior parietes, the commonly observed evidence of the beat of the heart of a nervous, hysterical female. He states this to be conclusively demonstrative of the fact, that a heart can "rap like a hammer against the inside of the thorax." Granting it to be the case that under certain circumstances the heart's impulse against the parietes of the chest is increased both in force and rapidity, no argument can be deduced therefrom, for, concluding the origin of the first or systolic sound to be due to this act, while there still remains the undoubted evidence of the presence of the first sound, though no thoracic wall may exist for the heart to impinge against; and while, as may be too frequently observed, this sound, provided only the valves be imperfect, may be lost and changed into another of a totally different character, notwithstanding the impulse, the *ictus ventriculi* may be more violent and distinct to the feel than natural.

As it is not the purpose of these pages to discuss largely physiological data, but rather to state the practical conclusions arrived at, after many years of observation, I shall not pursue the discussion further, but at once venture here to suggest that as it appears to me, on the one hand, the various theories commonly adduced, in explanation of the normal sounds of the

heart, and their disturbance in disease, are not adequate to explain them, so, on the other hand, that their production finds every explanation in certain of the laws that govern the flow of fluids through tubes; and that, therefore, to the recognised laws of hydraulics we must look for their true, safe, and consistent interpretation.

Having premised this, I will proceed briefly to consider the source of these sounds in relation thereto.*

One of the familiar and well acknowledged laws of hydraulics is, that the momentum of a liquid being as great as that of a solid, anything which opposes this momentum will receive as severe a blow as if from a solid. Dr. Arnott, in stating this law ("Elements of Physics," vol. i. p. 458, Second Edition), gives an example of its application, so analogous to, and happily illustrative of, the whole question under discussion, that I venture to transcribe it here.

"It has long been observed, in household experience and elsewhere, that while water is running through a pipe, if a cock at the extremity be suddenly shut, a shock and noise are produced there. The reason is, that the forward motion of the whole water contained in the pipe, having been instantly arrested, and the momentum of a liquid being as great as a solid, the water strikes the cock with the same force as a bar of metal, or a rod

* Shortly after these observations on the Sounds of the Heart had been published in the *British Medical Journal* (1866), a paper published in the same journal by Dr. Leared, called my attention to the fact that he had previously advanced similar views. His paper and my reply thereto are here appended. I do not, however, consider that our views are altogether identical, certainly not in their details and application. Dr. Leared's paper, which is a very valuable contribution to this subject, is given *in extenso*.

"Under the head of 'Notes and Observations on Diseases of the Heart and Lungs,' in the *Journal* of May 12th, Dr. Shapter has stated in detail his views with regard to the mechanism of the sounds of the heart. After having mentioned other explanations which he considers unsatisfactory, he adds—'My own conviction is, that the sounds referred to are mainly, if not entirely, due to the natural interference with the even flow of the blood by the various mechanisms of the heart; and that they are to be referred to the ordinary laws of hydraulics, and little, if at all, to be attributed to the contracting act of the muscle of the heart, or to any vibratory motions in the substance of the valves.'

"In this passage, Dr. Shapter does not claim the views put forward as his own; but neither does he attribute them to their real source. Possibly he was

of wood, having the same weight, and moving with the same velocity. A leaden pipe, if of great length, is often widened or burst in this experiment. Lately this forward pressure of an arrested stream has been used as a force for raising water, and the simple arrangements of parts contrived to render it

not aware that many years ago I published a paper in which the heart's sounds were held to be caused entirely by the blood itself. I afterwards published the same views in a more developed form, as a thesis read in the University of Dublin. I have endeavoured to show, in this publication, that no other explanation of the sounds which emanate from the circulatory system is so comprehensive or so consistent with known principles. But there is another more important matter. I am prepared to prove that none is so compatible with the various morbid deviations from the normal sounds, or with the production of new sounds. In a practical point of view, the subject is no less interesting than important; and I only regret that other pursuits have interfered with my intention to demonstrate by a mechanical arrangement that these sounds are products of a more or less complete arrest of the blood's motion. I have expended much time and trouble in numerous hydraulic devices and experiments, and the final results are highly encouraging. The *perfect* imitation of the human heart-sounds has yet, however, to be achieved. The difficulties in the way, both as to material, delicacy of finish, want of comprehension and even honesty of purpose on the part of those employed in constructing the necessary apparatus, are great. In a letter of mine published in the *Medical Times and Gazette*, April 7th, 1866, it may be seen how all my pains for the formation of an India-rubber apparatus were frustrated, because the persons to whom its execution was entrusted chose to apply the principle involved to the production of a now popular enema-apparatus!

"In the face of long-established opinions favourable to the valvular theory of the sounds, sustained by names of eminence and the strong bias which writers on the diagnosis of diseases of the heart must inevitably entertain for the foundation upon which their views are based, I have felt that the demonstration in question requires to be complete. Sanguine of success, I hope at no distant day to renew my attempts.

"Although my views have not met the attention which I might have expected, this would be no excuse for apathy in the matter. Speaking with a sincere conviction of their truth, I may be pardoned in stating that this neglect will sooner or later be atoned for. Instead of a bare allusion to my explanations in the last edition of the standard work on *Physiology*, and their complete omission in some recent works on diseases of the heart, I am confident they will one day take the place to which their truth entitles them. I am glad, therefore, to find Dr. Shapter, while he points out reasons for dissatisfaction with other theories, adopting the present one. There are, however, certain conditions requisite for the production and modification of the sounds formed in the circulation, which he has not touched on, and for which I must refer him to my paper. The argument in that paper is strengthened by analogies, than which nothing short of direct proof can be more convincing. Dr. Shapter also not only employs analogy, but uses one identical with one I have given, quoting for the purpose a passage from Dr. Arnott, written without reference to the present subject, which I had not before seen.

available, has been called, on account of the shock, the water ram."

The circumstances attending the circulation of the blood through the heart, will, on consideration, be found to present

DR. ARNOTT.

"'It has long been observed, in household experience and elsewhere, that while water is running through a pipe, if a cock at the extremity be suddenly shut, a shock and noise are produced there. The reason is, that, the forward motion of the whole water contained in the pipe having been instantly arrested, and the momentum of a liquid being as great as of a solid, the water strikes the cock with the same force as a bar of metal, or a rod of wood having the same weight, and moving with the same velocity. A leaden pipe, if of great length, is often widened or burst in this experiment.

"'The circumstances attending the circulation of the blood through the heart will, on consideration, be found to present every condition necessary for the application and illustration of this law; there is the current of blood passing through tubes, and this current suddenly and forcibly arrested by the closure of the valves.'

DR. LEARED.

"'The second sound occurs during diastole, and its mechanism closely resembles the first. The blood having been driven with much force into the aorta and pulmonary artery, a portion of it recoils, but is checked in its rapid descent towards the heart by the semilunar valves. The sound is caused by the concussion thus induced; the force of which is, however, by no means sustained by the valves alone, for they are thoroughly supported by the ventricles and their contents. This is obvious, since there can be no approach to a vacuum in the heart. The valves are to be regarded as separating media, which do not themselves sustain the force of the descending blood. A valve thus supported is known in the arts as an equilibrium valve.

"'An experiment at hand in most houses demonstrates the principle on which the second sound is formed. When a cock, attached to the lower end of a perpendicular pipe of some length, through which water is flowing from a cistern, is suddenly turned, a loud jarring sound is heard. It is caused by a concussion in the water from the sudden arrest of its onward flow. The semilunar valves are here represented by the plug of the cock; and, allowing for the difference between rigid and flexible materials, the conditions are very similar, since the elastic reaction of the vessels effects a pressure on the blood which is effected in case of the water by length of the pipe. If, then, the pipe and cistern are capable of yielding a sound which may be heard at a considerable distance, it cannot be wondered at if the heart and its vessels, on the same principles, give rise to sounds audible through a stethoscope, or by direct contact with the body.

"'If the cock is only turned so as to allow even a small portion of water to pass through, a rushing sound (in this case continuous) results. The change of the normal second sound into a murmur from incompetency of the valves is thus demonstrated.'

"Space will not allow me to place my explanation of the formation of the first sound before the reader. Let it suffice at present to say that it has nothing to do with the vibration of valves; and that, in support of this statement, very strong reasons, derived from pathology, can be adduced. One of the arguments relied on to prove the valvular origin of the first sound, was the great probability that both

every condition necessary for the application and illustration of this law; there is the current of blood passing through tubes, and this current suddenly and forcibly arrested by the closure of the valves.

sounds were formed by the same kind of mechanism; and the dogma that the second was a valve-sound, was held to be unassailable. Whether or not this is the case any longer, I must leave for others to judge, only let the matter be impartially considered. That it is more philosophical to look for a common cause for both sounds, is quite true, and that cause will be found, as already said, in the motions of the blood itself, not in such a vibration of delicate valves as would be necessary for the production of sound in a viscid fluid, and which would imply a degree of strain which, to say the least, is highly unphilosophical.

"The experiments of my late colleague, Professor Halford, have for the time propped the valvular theory of the heart-sounds, and tended to divert attention from every other. But, ingenious and painstaking as these undoubtedly were, there is no one of them of any moment, which, when rightly interpreted, is contradictory of my views. One experiment was supposed by himself and by others to be conclusive as to the truth of the valvular theory. The heart of a living animal having been exposed, he cut off its supply of blood by compressing its afferent veins. No sound was then heard on applying a stethoscope to the heart, because, as he argued, there was no blood to act on the valves. It is curious that I had performed the self-same experiment some time before seeing the printed account of that by Professor Halford, and then wrote to him to that effect. It was intended alike by him as well as by myself to disprove the once generally-accepted muscular theory of the first sound; but it is hardly necessary to say that, according to my explanation of its cause, this sound would be as effectually suppressed by cutting off the ventricular supply of blood, as it would be supposing the sound to be valvular."

My reply to the above communication of Dr. Leared was addressed to the Editor of the *British Medical Journal* (10th August, 1866,) and is as follows:—

"Sir,—In a recent number of this *Journal* (No. 292, Aug. 4th), Dr. Leared directed my attention to the fact that, in a thesis read in the University of Dublin in 1860, on the occasion of his taking his doctor's degree, and subsequently published in 1861, he had propounded the view that the sounds of the heart were due, not to the heart or valvular vibrations, but to the motion of the blood upon itself; and he then quoted at length his statement as regards the second sound. The nature and *rationale*, as thus stated, of the formation of the second sound, appeared to me, as it did to him, to be both identical in principle and in application to that which had been advanced by myself in a paper recently published in this *Journal*, and I at once wrote to Dr. Leared to say so. At the same time, I begged to assure him I had not seen a copy of his thesis, nor was I cognisant of his views set forth therein; that the views advocated in my paper were certainly advanced as a theory of my own, but that, under these circumstances, I at once disclaimed any appropriation to myself of a credit that may properly belong to him; and that I hoped to examine into his views more particularly by direct reference to his thesis.

"Dr. Leared has kindly forwarded to me a copy, and this I have now read attentively. Dr. Leared states generally that 'all sounds formed in connexion with the circulation are produced by and in the blood itself, and their mechanism is virtually the same.' (P. 3.) Having detailed certain experi-

It is the opinion of Dr. Fuller ("Diseases of the Heart and Great Vessels," p. 33) that the impulsion of the blood against the semilunar valves, is not adequate to produce the second sound, because the vessel behind is full; but his reasoning here is not consistent with the law of hydraulics stated in a previous page;

ments performed with fluids of different densities, he goes on to say that 'Two important principles were established by these experiments. First, the sounds may be formed by the motions of fluids only; secondly that the quality of a sound thus formed is materially influenced by the nature of the fluid. The sounds formed by the circulation of the blood are produced on principles similar to those detailed in these experiments. I shall, therefore, for convenience, and as conveying their true nature, designate them as blood-sounds. Blood-sounds are divisible into two classes—sounds which give the impression of a shock; sounds which give the impression of a current. Shock sounds comprise the normal sounds of the heart, and certain abnormal sounds formed in aneurismal sacs. Current sounds are formed in the heart, in aneurismal sacs, and in the large arteries and veins.' (P. 6.)

"The above views are in principle so nearly the same as those advocated by myself, that I at once willingly and cordially yield the merit of their original statement to Dr. Leared. In applying these principles, I entirely agree with Dr. Leared as to the second sound. As regards the first sound, there is considerable difference between us. After reading his statement, I am bound to say I still prefer the explanation given by myself. I also think that Dr. Leared, in setting forth his views, not only inadequately appreciates, but ignores too positively, the vibrating qualities, and perhaps also the conducting power of the various tissues connected with the circulation of the blood, and the relative amount of sound generated and transmitted thereby. Nor do I think he sufficiently defines the differences of the source of the sound produced by the blow of a fluid when its momentum is suddenly arrested—so well set forth by Dr. Arnott—and the sounds emanating from obstruction of current. The sound produced by a drop of water falling from a height is an illustration of the former, and not of the latter. In illustration Dr. Leared fully recognises the difference. These are, however, minor points. Dr. Leared, in his thesis, clearly sets forth the theory that the sounds and murmurs of the heart are due, essentially, to the blood in motion, and my purpose was to do the same.

"To turn to another point. Granted that a shock is communicated to the blood contained within the arteries by the suddenly arresting its backward flow, it has appeared to me that the vibrations thus produced throughout the arterial system offer a satisfactory explanation of the phenomena observable in the pulse-proper. A paper on this subject is prepared for the *Journal*, which I hope may be permitted shortly to appear there. The theory I shall there attempt to support and illustrate is, that the pulse is divisible into three phenomena: 1, the filling of the artery by successive waves, through the agency of the ventricular systole; 2, the vibration in the column of blood throughout the whole arterial system, caused by the sudden closure of the semilunar valves (the pulse); and 3, the contraction and collapse of the arteries during, but not caused by, the ventricular diastole. I am prepared to show that this view of the pulse explains many of the phenomena observable in the pulse during disease."

nor is it borne out by fact. In order to test this, it is only necessary, by way of experiment, to check, by the sudden closure of a stop-cock, the stream of water passing through a filled pipe; the vessel behind is here quite full, and yet the blow takes place. The evidence of this is made sufficiently manifest in the noise thereby produced, and in the obvious vibrations induced in the surrounding materials.

Dr. Leared states* that "two essential circumstances relative to the formation of sound have been overlooked by all investigators—the effect of pressure on the blood in the heart and vessels, and the effect of the consistency of the blood. All sounds formed in connection with the circulation are produced by, and in, the blood itself, and this mechanism is virtually the same. If this statement be true it would be found in practice that a sound of one species would be liable to be changed into a sound of another species. And this is the case, for the normal first sound of the heart is, under certain pathological conditions, converted into a murmur, and on the other hand, a murmur thus produced, may again give place to the natural sound." In following out this theory (*vide* p. 9), Dr. Leared refers the sounds of the heart to the impact between the fluid in motion and that in a state of rest, and says that the absence of obstruction at the outlets of the heart is one of the necessary conditions. My explanation refers the sounds to be due to the sudden interposition of the valves on the moving column of blood, whereby a concussion is produced. The obstructed flow is the hammer, the valves and the surrounding structures the media through which the vibrations are communicated. I very much doubt if the two conditions supposed by Dr. Leared, and necessary to his views, of a fluid in motion and of a fluid in a state of rest, ever exist at the auriculo-ventricular openings, and I feel assured they never do at the pulmonary and aortic openings.

Another law of hydraulics is equally applicable, and found to

* See pamphlet "On the Sounds caused by the Circulation of the Blood: being a Thesis read in the University of Dublin for Degree of M.D. at the Winter Commencement, 1860." Churchill: 1861.

be complete in those cases where interference arises to the normal even flow of the blood, in consequence of the existence of certain departures from a perfect condition of the organs immediately concerned in its circulation.

Hydraulics teach us that if the even flow of fluids in tubes be interfered with, audible indications of this interference are the immediate result. The law here is that sounds are produced by eddies and by obstructions of flow, and are dependent for variety in the proportion of fluid to force and velocity. Hence, when the valves of the heart are diseased, the normal sounds are found to be modified, or even in some cases to be so entirely superseded by other and very different sounds, as to lead to the inference that they scarcely perform the functions of valves. We have in all these instances then presented to us, certain qualities of sound, variously designated as murmurs, bellows-sounds, *bruit de soufflet*, etc., which are also presented to us on the passing of fluid through tubes, under certain circumstances, whereby the flow is interfered with, though these tubes be not furnished with valves. Aneurisms, arteries abnormally pressed upon, etc., offer themselves to the medical observer as frequent illustrations of this law. Sir T. Watson says ("Lectures on the Practice of Physic," vol. ii. p. 235) the whole matter is to be thus briefly expressed:— "The blowing sound may be occasioned by any change which alters the due proportion between the chambers of the heart and their orifices of communication with each other, and with the blood-vessels that respectively enter or leave them; it may also be occasioned by a preternatural velocity in the passage of the blood through a healthy and well-adjusted heart. Dr. Elliotson, I think it is, who has offered this apposite illustration of the phenomenon. If the arches of a bridge have a certain relation to the quantity of water in the river, and to the force of the current, the water passes through them quietly and without any noise. Diminish the size of the arches, and the water begins to go through them with an audible rushing or roaring sound. The very same thing will happen if the arches remain unchanged in size, but the quantity of water in the river, and therefore its velocity and force, be augmented by heavy rains. So it is in the

heart. If one of its orifices—say the aortic orifice—be narrowed by disease of the valves, or in any other way, the blood will not, as before, glide through it smoothly and without noise, but will yield that sound which we call a bellows-sound. So also, if the orifice retain its natural dimensions, but the capacity of the cavity from which the blood is driven be augmented. Nay, the same blowing-sound may be produced though the cavities and orifices are all healthy, and duly proportioned to each other, if the velocity of the circulating blood be increased beyond a certain measure."

The above passage has been quoted as well and forcibly exemplifying the law of murmurs, so frequently to be met with in diseases of the heart, and when it necessarily becomes the office of the physician to pronounce upon the condition of the heart in relation thereto. Given the flow of blood and the existence of a murmur as evidence of its normal relations being interfered with, there is then required to be ascertained the precise cause whereby this interference is effected: whether it be through the means of obstruction in, or by inadequacy of, the conveying-tube; whether there be an increased or a diminished calibre in the chief propelling vessel, or disproportion in the quantity or quality of the transmitted fluid, etc.

Having now set forth these two laws of hydraulics, and assuming their applicability to explain, as the case may be, the normal sounds and the murmurs occurring to the circulation of the blood during its passage into, through, and out of the heart, it will be useful to examine whether as theories they prove, experimentally, consistent with the various phenomena that occur.

It is now proposed to see how far that law of hydraulics which, while enunciating that the momentum of a liquid is as great as that of a solid, further states that anything which receives the momentum will receive as severe a blow as if from a solid, is applicable to the normal valvular sounds emitted by the heart, and is borne out by the rhythmical phenomena observed during the heart's action. In considering the effect of these phenomena we soon find the estimation of other conditions

and elements to be necessary, viz.: the capacity for resonance of the various tissues which are directly influenced by this momentum, and how far the parts immediately surrounding them are media favourable for the conduction of sound; the quality of the blood itself must also be considered. Nevertheless, the modifications caused by these disturbing elements neither add to, nor detract from, the above law.

To proceed. Immediately succeeding the second sound, there is the "pause," occupying two-fifths of the period in which the cycle of the heart's movements are performed. The "pause" is characterized, with the exception of the presystolic sound occasionally heard, and which is probably due to the eddying of the blood as it passes through the auriculo-ventricular openings, by no ostensibly external active phenomena. It is the period in which the ventricles, by active muscular agency, are gradually uncoiling and thus expanding, and therefore drawing or sucking in the blood from the right auricle and cava, or from the left auricle and pulmonary veins, as the case may be. This is done, looking to the supply afforded by the auricles, so gradually, that the auricles are scarcely collapsed thereby, the blood apparently flowing into these latter almost, if not quite, as quickly as it is thus drawn from them. In all this there arises no obstruction to the flow of blood, nor any indications of the formation of eddies, save, as above stated, such as may be indicated by the presystolic sound; the blood passes easily and inaudibly from the auricles into the ventricles.

This period of the "pause" is then succeeded by the first sound. This takes place at the precise moment in which the ventricles, by coiling or screwing up their fibres, start into contraction, and when the mitral and tricuspid valves are thrown back and closed by the blood thus forcibly impinging against them, and when as a necessary consequence, these valves recoil on the onward current of blood proceeding through the auricles. One of the functions of the auricle, and perhaps the chief, is now exemplified; for so sudden and so energetic is this closure of the valve, by the powerful force brought to bear upon it by the pressure of the blood on, and during, the coiling process of

the ventricle, that the obstructed venous current immediately distends the auricles, and were it not for the strength of their elastic fibres, or for the musculi-pectinati or for the support from the pericardium, a constantly recurring over-distension, or even rupture, would ensue. As it is the auricles receive the shock, and by their elasticity neutralize the probably injurious effects of this sudden check of the blood current.

The following observations made by Dr. Carpenter ("Principles of Physiology," p. 557) are so confirmatory of the above, that I venture here to quote them, especially as the facts mentioned therein were recorded entirely independently of the views now sought to be deduced from them. He is referring to the case of a child which came under the notice of Cruveilhier, and where, from the accidental exposure of the heart, ample opportunity was afforded for carefully observing the phenomena, and that by a practised and most competent observer. "The diastole of the heart has the rapidity and energy of an active movement: triumphing over pressure exercised upon the organ, so that the hand closed upon it is opened with violence. This is an observation of great importance; but of the cause to which this active dilatation is due, no definite account can be given. But the dilatation of the auricles appears to be much greater than can be accounted for by any *vis a tergo* (which, as will hereafter appear, is extremely small in the venous system) or by the elasticity of its substance, for it was observed in this case to be so great that the right auricle seemed ready to burst, so great was its distension, and so thin were its walls. Moreover, the large veins near the heart contract simultaneously with the auricular systole, and not with its diastole, so they can have no influence in causing its dilatation."

The first sound, then, is synchronous with this distension of the auricle, and with the simultaneous closure of the auriculo-ventricular valves, and occupies, equally with the preceding "pause" two-fifths of the time consumed in the cycle of the heart's action. Assuming it to be due to the forcible closure of these valves against the stream of blood flowing from the auricle into the ventricle, this dull and prolonged sound may be

accounted for partly by the energy with which the valves are closed, and partly by the comparatively large column of blood thus acted on; but chiefly by this column itself impinging on a large amount of soft membranous material, the sound being thus distributed and rendered less sharp. It may also be that the quality of this first sound is partly determined by the expanding of the auricle itself, and by which the checked momentum of the fluid is modified.

It is probable that all these causes, more or less, combine to make that difference in its duration and quality, from that which is observable in the second sound, and by which it is immediately succeeded.

This second sound, which is comparatively loud and sharp in tone, occupies a duration of time amounting to only one half of that proper to the first sound, and takes place at that moment of time in which the ventricles, after having by their rapid and forcible contraction closed the auriculo-ventricular valves, and after having impelled the blood contained within them, into the arteries, resume by the uncoiling of their fibres their condition of expansion, and, then, consequently recommence their suction power. They now offer the condition of being able to redraw back to themselves the blood they have just discharged; but at the precise instant such a backward stream is commenced, the semilunar valves are forced thereby into action, and here there occurs, as was the case on the closure of the auriculo-ventricular valves, a sudden obstruction to a moving current of fluid. But this obstruction, though prompt and energetic in its action, and effected through the medium of membranes easily acted on by the opposing current, and being, from their thin and dense structure, better conductors of sound, has not to contend with so large an amount of fluid, as is the case with the auriculo-ventricular valves, when their respective closures take place, nor do the fleshy and contracted and consequently empty ventricles offer so fit a medium for the distribution of sound, as the thin and distended auricles. Hence this second sound is short, sharp, and loud, while the first is dull and prolonged.

The cause of these two sounds is primarily due to the inter-

rupted momentum of the moving fluid; their quality and duration being due to the density of the medium of the obstruction that receives the blow of this interrupted momentum together with that of the parts surrounding, they being the means whereby the sound is conducted and rendered appreciable. Hence the metallic ring, occasionally heard with the second sound, is probably due to excited, sudden, and forcible closure of the semilunar valves in the midst of these readily vibrating media; hence, also, arise the modifications of these sounds, in respect to quality, tone, and intensity, when heard from different portions of the chest wall.

Such, then, is the explanation now offered of the normal rhythmical sounds of the heart; but experience shows these may be variously interfered with, especially in their rhythmic action and by their being marked by, or converted into, murmurs, and to the pathologist there then arises an anxious field of inquiry. It therefore becomes necessary to ascertain whence proceed these sources of interference, and to what morbid changes or disordered actions they may be due.

Of the disturbance in rhythm only, nothing need here be said, as it will be the subject of reference subsequently; but we will pass on to a brief consideration of the special physical causes of the substitution, by murmur, of the normal sounds.

In pursuing this inquiry it will be found that the hydraulic law of the production of sound by the eddying of currents satisfactorily explains the phenomena of these murmurs, and that a due appreciation of the bearing of this law will assist greatly towards forming just and satisfactory conclusions in the instances presented to us. In the course of discussing some of these, to be subsequently more particularly referred to, this will be rendered more obvious. For the present it will, by way of illustration, be only necessary to summarize some of the more salient positions induced by disordered action.

If the mitral or tricuspid valves, though efficient as regards their valvular office, present any obstruction to the flow of

blood, there will arise a presystolic murmur, and this will precede and perhaps entirely take the place of the first sound. In the former of these two instances the murmur is concluded by it. More often, however, the murmur entirely masks the natural sound; but in both the murmur is induced by some valvular obstruction or want of proportion between the current of blood and the aperture through which it passes, so that a sonorous eddy is thereby caused. But if there be imperfection in these valves, whereby, on contraction of the ventricles, a regurgitation of blood takes place into the auricles—and it is well to bear in mind that this is not an unfrequent condition of these valves, both in disordered states of the heart, as well in its diseased states—the first sound is never heard, but only a murmur, and that a protracted one, and sometimes even so protracted as to greatly interfere with the audibility of the second sound, or, at any rate, with its easy and correct appreciation.

The explanation of the above is, that the imperfection in the valves, as regards their closing function, does not efficiently offer an obstruction to the momentum of the flowing current, and hence there is wanting the normal first sound, whilst the murmur in place thereof may probably be due to a sonorous eddy in the blood as it enters the ventricle, but certainly to the eddy induced by the regurgitation into the auricle through the limited aperture in the unclosed valve, during the contraction of the ventricle. When the murmur is so protracted as to interfere with the second sound, there is generally abnormal patency, permitting a sonorous regurgitant eddy; and this may succeed, and be continuous with, a sonorous presystolic eddy caused by obstruction to the stream of blood when flowing into the ventricle. The sounds induced by these two eddies are generally so continuous as to render their having two separate and independent sources for their production, to be undistinguishable by the ear.

Under the above circumstances, these phenomena will certainly occur, unless it be in those rarer cases where, with a dilated and enfeebled ventricle, the valves are so patent as to

offer little or no obstruction to the regurgitant blood; then perchance no proper murmur may occur, but only a continuous and tumultuous sound. The explanation of this is to be found partly in the very undue patency of the valves, and partly in the enfeebled and disordered condition of the ventricle inducing a deficient momentum in the current of the blood itself. This failure of murmur, and in its place only indistinct and ill-defined sounds, is a frequent occurrence during the feebleness in protracted cases of heart disease, which heralds failure of life. Under these conditions, though aware of the full extent of the heart disease, one is often unable to recognise a true murmur.

If there be an obstructing imperfection in the semilunar valves of the aorta, uncomplicated with other morbid complication, there will be a murmur, most probably so distinct and pronounced as somewhat to interfere with the audibility of the first sound at the base, and, may be, even at the apex; the first sound is, as it were, absorbed and overpowered by it. The second sound immediately succeeds this murmur. Both the murmur and the second sound are heard most distinctly at the base of the heart, over the region of the aortic area, and in the course of the aorta. This series of phenomena is explained by the sonorous eddying of the blood produced by the imperfection of the valve being synchronous at its commencement with the first sound. For the most part obstruction is the cause of the murmurs in the semilunar valves. More rarely they are produced by imperfections permitting regurgitation; and then, when regurgitation with an attendant murmur does exist, it is almost always preceded by murmurs as the evidence of obstruction.

It is necessary to observe great caution in concluding the presence of a regurgitant murmur in the semilunar valves. It is probable that it is less common than is generally assumed to be the case; and, most certainly, a murmur often appealed to as evidence of an imperfection in these valves permitting regurgitation, has been eventually shown not to be due to this cause; but, possibly, to only very slight obstructions to the systolic flow, or, may be, to some dilatations in the aorta, or even in the pulmonary artery. In investigating and diagnosing the source

of these murmurs—as they are due, not exclusively to regurgitation, but to perturbations in the flow of the blood—it must not be lost sight of that they may equally be produced by aneurisms in these arteries, and where there is no regurgitation, as by that amount of imperfection in the valve which permits it.

To what indications are we, then, to appeal for a correct diagnosis as to the precise seat and origin of a murmur thus situated, so as to decide whether it be, firstly, caused by imperfection in the semilunar valves, or, secondly, whether it be regurgitant or otherwise. Certainly not to the quality of the sound itself—for this alone is not adequate to this end—but, amongst other circumstances, to its position as regards time and place; and the due estimation of this is, more frequently than otherwise, most difficult; for, if these valves present so imperfect a condition as to permit regurgitant murmurs, they likewise are deficient in that perfect condition of structure necessary to offer a complete and successful opposition, or contraction, to the momentum of the obstructed stream of blood.

On looking exclusively to these valves, we might perhaps say, that where a murmur is synchronous with the heart's systole, it is due to obstruction, and to obstruction only; but that where it is synchronous with the diastole, then it is due to regurgitation. In this latter case it should be somewhat prolonged, as continuing during the more lengthened period of the diastole—it being then, and then only, when regurgitation should ensue; and, inasmuch as the diastole is more gradual than the systole, and the arteries, whether aortic or pulmonary, not favourable vessels for supplying, in a retrograde mode, blood to the ventricles, the regurgitant sounds would be comparatively feeble, and for the most part continuous with the systolic murmur, or so in succession to it, as to present what may be termed a double murmur. Regurgitant murmurs in the semilunar valves are, however, comparatively but of rare occurrence.

The two normal sounds of the heart, as has been shown, are due to the closure of the valves against the current of the blood. The murmurs, on the other hand, are, as regards the valves, heard under disordered conditions of these valves, so that their

normal relations to the flow of the blood become disturbed; and hence they may arise whenever there is a current of blood relatively too large for the aperture guarded by these valves, and thus presenting a condition of obstruction to its free flow. Any circumstances, therefore, occurring to these valves whereby the fluid, passing through them, finds a narrowed passage will produce a murmur, and consequently it is immaterial, as a source of sound, whether the blood flows normally onwards or abnormally backwards—the physical condition as regards production of sound is the same.

It may be, in the present day, no very difficult task to set forth what may be the sounds heard as proper to each lesion of the heart; nevertheless, practically, a differential diagnosis is frequently fraught with great difficulty, inasmuch as the sounds and murmurs are often continuous, or one sound may mask or entirely supersede another. Then, again, there is the contiguity of the similar parts of the two hearts, and the synchronisms in their actions and their sounds, to be carefully estimated and duly separated.

The due appreciation of these confusing indications requires much practical skill, and a large necessity for taking into consideration many attendant circumstances. Some few of these constantly recurring difficulties in diagnosis may, with advantage be referred to.

CHAPTER III.

VALVULAR SOUNDS, THEIR DIAGNOSIS AND ITS DIFFICULTIES.

It is here proposed briefly to point out some few of the difficulties which occasionally prevent a ready and correct appreciation of the sounds of the heart. In doing this, though the sounds and murmurs, belonging exclusively to the valves, in their relations to the circulation of the blood are here solely referred to, it must not be inferred that other signs no less important and independent of these are ignored. On the contrary, the value in diagnosis of some of these latter will be in due course not only considered, but perhaps seen to be of paramount importance, so that, without them, prognosis in disorders of the heart will be essentially at fault. The object at present, however, is mainly to illustrate the positions advanced as to the immediate cause of the sounds, and then to show what may be the importance of these sounds, normal or abnormal, by themselves, towards estimating the condition of the heart itself.

In investigating, by auscultation, any of the disorders of the heart, the first and the chief point to be arrived at, in reference to sound, is the accurate ascertainment of the presence or the absence of either of the two normal sounds.

If there be an absence of either of these sounds, it may then be inferred that some other sound has taken its place, and that this other sound is not a normal one—that it is, in fact, a new sound, and manufactured, as it were, by diseased

structure or by disordered action or by an altered condition of the blood.

If the above position be true, it will be at once seen how very important it is to ascertain the existence, or the contrary, of both normal sounds; and that, here, confusion in diagnosis must be sought to be carefully avoided, lest the inferences thence deduced be erroneous.

A careful observer, and one of our best authorities upon diseases of the heart, says that absolute deficiency of either sound, or of a murmur taking its place, has never fallen under his observation; that, in fact, neither systole nor diastole has ever been, in his experience, absolutely noiseless over the entire cardiac region. The above strong and pointed statement is made by Dr. Walshe, after noting that, in cases of extreme weakness, the first sound may be *quasi*-deficient at the left apex; "but it will then be found at the right apex and at the base. So, again, the second sound may be *quasi*-deficient at the base from excessive feebleness, or from being covered by a prolonged systolic sound or systolic murmur; but, in the first case, excitement of the heart, increasing the energy of its contractions, will invigorate the sound, and in the second case, the sound will be heard at the right apex."

Though cases have occasionally presented themselves to my observation in which I could not satisfy myself of the absolute conclusiveness of the above statement, that both the sounds, if not superseded by others, were thus always present, yet the position is so generally a safe one that, in order to arrive at a correct and sure conviction of the presence, or of the absence, of the two normal sounds, it is necessary fully to appreciate those various accidental circumstances which, when the two normal sounds are really present, tend to obscure their being duly recognised.

Doubtless, in estimating these circumstances, those special variations and peculiarities which may occur in the several properties or conditions proper to, and characteristic of, the sounds themselves, such as "intensity, duration, pitch, and quality," must be considered. Each of these may, under the modifying

influences of sex, age, attitude, exertion, excitement, debility, etc., be so altered or intensified as to lead to false inferences; and hence, in the place of normal valvular sound, the presence of a murmur may be erroneously assumed.

A naturally weak heart, or a heart in which the ventricles are hypertrophied, will frequently, on agitation, generate sounds, or so modify the valvular sounds, that these are to a certain extent masked; the sounds become hurried, intensified, and confused, and might, on a hasty examination, be regarded as being murmurs. A little care in examination will generally succeed in detecting that the sound is really a normal valvular one. Sometimes, however, from continued hurry of the ventricular impulses, and from these not following in regular succession, the marked and uniform valvular sound is merged in, or superseded by, that continuance of sound which, in contradistinction to regulated rhythmical sound, is denominated by the term "noise." It then becomes difficult indeed to separate and appreciate the two normal sounds.

In cases of fever, the first or systolic sound is often the subject of considerable modifications. In some cases there is a feebleness, passing into nearly a total absence of this sound; in others, it becomes prolonged, and almost assumes the characteristics of a murmur—perhaps there may even be a murmur; for occasionally, in fever, the muscle of the left ventricle is found to be weakened, and even degenerated in its structure. It is therefore probable, though neither the mitral nor tricuspid valves may be diseased, there may arise some deficiency in the complete action of these valves; so that, practically, there may really exist a certain amount of valvular error.

The above sources of error, more or less, arise from modifications of the valvular sounds themselves; but the main causes of embarrassment, whereby the correct recognition of the presence of the normal valvular sounds becomes obscured, have not their origin so much from any real variations or actual modifications of the valvular sounds, as from causes extrinsic to the valves themselves.

The modifications of the sounds occasionally induced by an

excess of nervous excitement in spanæmia and allied affections have perhaps their cause more exclusively in the disturbed passage of the blood through the irregularly palpitating heart itself. Immediately succeeding the first sound, but still commencing, as it were, the second sound, is a soft murmur, and the second sound itself, which concludes this, is heard, sharp, loud, and accentuated. Both these latter sounds are produced under the influence of nervous excitement during an increased and rapid impulse. The murmur itself has been by some attributed to the action of the heart within the pericardium, whereby a slightly appreciable friction-sound is produced. It is not, however, a friction-sound; for it not only has not its distinctive character, but an equally rapid and augmented impulse without the nervous excitement fails to induce this specific murmur. Considering the character of this murmur, and of the succeeding accentuated valvular sound, it is not unreasonable to conclude them both to be due to the increased rapidity of the flow of blood through the heart agitated and rapidly palpitating under nervous excitement; the murmur being produced by an eddying disturbance in the flow of blood through the ventricle, or may be from an excess of pressure on the unusually rapid flow of blood through the aortic opening, and the accentuated second sound by the sudden jerk of the semilunar valves, whereby the backward flow of the blood is arrested. In these cases of nervous excitement, it is invariably the second sound which is implicated, and from the modifications in which errors in diagnosis may arise.

Though, under these circumstances, there is undoubtedly the presence of a murmur, and though the valvular sound is exaggerated and altered in tone, yet a careful examination can separate the accentuated sound from the murmur, and thus enable it to be recognised as the normal sound, only modified by the excited systolic action of the heart.

The more usual source of confusion arises, however, from various sources extrinsic not only to the valves but to the heart itself. Amongst these the most common are breathing sounds so synchronously occurring with the diastole as to obscure and (as it were) overlay one or other of the normal sounds of the

heart. Dr. Latham (vol. i. p. 65) states this condition of things clearly:—"It has been said that endocardial murmurs are best imitated by modulations of the breathing and by help of the mouth. Hence it is not to be wondered at that there should be an endocardial murmur which nearly resembles the natural murmur of respiration. The commonest of all the endocardial varieties is the bellows-murmur; and the natural murmur of respiration is only a gentle sound of the same kind, but more prolonged. Hence the morbid sound of the heart and the natural sound of the lungs are sometimes so much alike that, if the systole of the ventricles and the act of inspiration kept time with each other, it might not be easy to determine from which of the two organs the murmur came; and in point of fact I have sometimes listened and hesitated, and hesitated and listened again and again, before I could satisfy myself that a murmur which came altogether from the lungs did not in part proceed from the heart also. It has been carried with an impulse into the ear as if it came from the heart." Dr. Latham also adds that "the method of clearing up the doubt is to auscult the heart, while the respiration is suspended for a quarter of a minute."

Doubtless, this is true. Nevertheless, cases constantly occur where the respiratory murmurs are both so prevailing and so protracted as to render it extremely difficult, and even, at times, impossible, to separate the sounds of the heart from them. Perhaps the most embarrassing circumstances exist when the lung contiguous to the heart is, besides being in an emphysematous condition, the seat of bronchial *râles*; the murmur, the resonance of the lung, and the almost invariable feebleness of the sounds of the heart themselves, each offering elements of difficulty. The first sound, especially of the left heart, is, under these circumstances, at times so effectually masked as to render it next to impossible, taking the element of sound only, to diagnose the absence of murmur, and the presence of the valve sound, in the left auriculo-ventricular valve.

Murmurs, generated in neighbouring structures when in certain inflamed and morbid states, not unfrequently appear to

proceed from the heart, and thus interfere with a due appreciation of its existing normal sounds.

The more notable examples of this source of confusion are to be met with when the pericardium becomes the seat of acute disease; for the friction-sounds of a recent case of pericarditis may very frequently be suspected to be a murmur, and thus to be indicative of valvular disease. It may at times be difficult to identify, as such, these friction-sounds, nevertheless there are signs which, if attentively considered, will generally lead to a correct diagnosis. First and foremost, the intrinsic character of the sound must be ascertained and appreciated. A pericardial friction-sound has neither the quality nor the pitch of a valvular murmur; it is deficient in that blowing or whistling character which invariably distinguishes the latter. It is, however, easier to state this than always correctly to appreciate the characteristic differences of the two sounds; but other circumstances come to our aid. The sound is usually increased on pressure; and it is found to occur most commonly both with the systole and the diastole, and always with the systole if with the diastole; and in favourable cases—that is, where the ordinary rhythm of the heart's action is not greatly impeded—careful auscultation will detect the normal valve-sound followed by the pericardial friction-sound, and this latter is usually observed to be a more pronounced sound with the systole than with the diastole. Other circumstances also tend to separate the pericardial from the normal valve-sounds—as the more sudden occurrence of the former, the rapidity with which they shift their seat, the fremitus often communicated to the hand, but more especially their sudden disappearance under treatment.

Occasionally during pericardial disease short clicking sounds are heard accompanying both systole and diastole. The presence of these very materially confuses a due appreciation of the valvular sounds. Both Stokes and Walshe refer them to the inflammatory processes within the pericardium; the former says that want of consonance establishes their true origin; the latter that they are only distinguishable at the time from modifications of the valvular sounds by their non-synchronism with them, and

by the extreme irregularity of their occurrence, and that he has satisfactorily traced them to the pericardium, and further, in all probability to the separation, without attrition, of surfaces glued together with exudation matter.

Various other sources of difficulty, extrinsic to the heart, whereby the normal sounds are on occasion materially interfered with, and even at times effectually overpowered, might be enumerated. Amongst the chief of these are those which have their origin in foreign and abnormal pressure, by contiguous diseased structures, on the great vessels immediately emerging from the heart, or even pressing on the heart itself; so that murmurs are manufactured, if we may so apply the term, in places which, in health, are not the seat of sound; but so near to the seats of normal sounds as to interfere with the true appreciation of these latter.

It is not only necessary to be on our guard as to the existence of murmurs having this foreign origin; but essential, towards a correct diagnosis, that their true cause should be recognised.

Their investigation and study are also interesting in a physiological point of view; for they serve to illustrate and confirm, strongly and clearly, the positions assumed on the formation of murmurs and the cause of the normal sounds. We find these murmurs induced when there is evidence of no other disturbance but that of current; and we find the normal valvular sounds, though the valves be perfect, disturbed and perverted both by modifications of the ventricular force and of the quality of the blood. The normal valvular sounds which should ensue in consequence of the vibrations caused on the sudden valvular arrest of the column of blood, being, by one or other of these means, increased, diminished, or changed in tone.

The friction-sounds produced by a pericarditis, and which very nearly assimilate themselves to valvular murmurs, have been just alluded to; still they are but friction-sounds. The physical consequences of a pericarditis do, however, at times, really cause murmurs in the great vessels. The lymph exuded during a pericarditis may so envelope the two large arteries at their origin, as to bind them down and contract their area;

thus effecting a disturbing pressure adequate to produce a considerable amount of murmur, and even to completely mask the normal second sound.

Dr. Markham extends this observation (p. 35); and says that this murmur may be due to loss of elasticity in the aorta, "or some other alteration of its coats caused by the inflammatory process; or to irregular action in the heart's muscular movements involving those of the columnæ carneæ, whereby the function of the auriculo-ventricular valves is rendered temporarily incomplete; or, again, when the murmur is persistent, it may possibly be ascribed to the pericardial adhesions; these being of such a character as to prevent the walls of the heart, and consequently the columnæ carneæ, from freely contracting, so that the mitral orifice is left partially unclosed during the heart's systole." In this latter case, however, the valvular sound cannot be said to be masked, but is really obliterated by the murmur induced.

It has been stated by some observers that the presence of a serous effusion into the sac of the pericardium may also be the remote cause of a murmur; but it has never been my lot to observe a case in which this has been satisfactorily made clear. It is, however, very certain, that effusions of fluid, in cases of acute pleurisy, into the left pleura, especially when attended with excitement of the heart, will produce a murmur. Dr. Stokes (p. 531) says, "It is distinct from any modification of the friction-sound, and consists in a systolic murmur, often broken into two parts, sometimes intense. This murmur is most evident during inspiration; but it continues in expiration and even when the patient holds his breath."

It is probable that the immediate source of this murmur is due to the eddies caused in the flow of blood through either the aorta or pulmonary artery, or both of them, as these vessels are contracted at their orifices in consequence of being bent and somewhat twisted from the forcible displacement of the heart—a displacement which at times is very considerable.

It is also probable that those murmurs, so often met with in cases where the heart is displaced by deformities in the parietes

of the chest, or by tumours in the abdomen or of the abdominal organs, are, in like manner, due to the bending or twisting of the great vessels as they emerge from the heart. When this takes place in the aorta the murmur may entirely obscure the second sound at the base, and yet it may be heard at the apex. This is probably to be explained by the fact that though at the base both the aortic and pulmonary valve sounds are thus obscured, and at the apex the aortic valve sound also, that of the pulmonary artery not being so obscured, is there heard.

In those chests which have a weakly constructed bone-work, a murmur may sometimes be produced by pressure from without, or, at any rate, some sound so very like it as to excite suspicion of the existence of a valvular murmur. This is especially the case in those who, having weak bone-work, are also pigeon-chested. In some cases, especially in these latter, the murmur is so very easily effected, that the ordinary pressure of a stethoscope, during investigation, may be adequate to produce it, and even occasionally to require the greatest caution that it be not thus produced.

The murmurs induced by tumours within the mediastinum, or by glandular and similar enlargements encroaching on the great vessels, appear to be due to simple pressure only; so also is the murmur induced in the pulmonary artery when pressed upon by an aortic aneurism.

The whole of these artificial murmurs are synchronous with the systole. Nevertheless, they are heard more distinctly at the base of the heart than at the apex; and this is a very important feature of their existence, and to be well considered and appreciated in summing up the diagnostic signs.

Murmurs may also be heard, the valves being perfect and normal in their function, in those cases in which an obstruction to the flow of blood within the ventricle or in the larger arteries takes place in consequence of the accidental formation of clots, or of those remarkable and unaccountable matters, the purulent cysts. I conclude there are no very distinct signs by which the presence of these latter may be inferred. The former may, perhaps, by their more frequent seat being in the right ventricle,

and by the suddenness of their production, and by the nature of the antecedent illness, yield some grounds of suspicion for their existence. The whole subject, however, of these formations, is one of doubt and difficulty.

The valvular sounds occasionally undergo a complication of some passing interest, whereby they become, as it has been termed, reduplicated. The two normal sounds may thus be added to, so as to form three or even four sounds. Dr. Walshe (p. 79) has given an elaborate summary of these sounds, and such as only a very practised ear, exercised in a large field of observation, could hope to recognise and fully to appreciate. This difficulty of accurately appreciating these sounds in all their specified varieties is not to be wondered at, considering the space of time in which they occur, and the very limited field of their generation, and the invariably agitated, or rather irregular, state of the heart's action at the time.

The more simple form of this reduplication is not, however, rare, nor difficult of recognition. It occurs with the second sound, which thus becomes divided in time, and as it were cleft in two. But, whether it occur with the second, or with the first, or with both the sounds, it is heard sometimes similar in tone, and sometimes not, to the sound of which it is the reduplication; but usually the first portion of the divided or cleft sound is the most accentuated. For the most part, a reduplicated sound is met with in cases which present apparently the characteristics of functional disorders of the nervous heart only; but it also occurs in cases of active inflammatory disease; and in these cases it is usually the second sound that is cleft, while there is associated with it a mitral murmur. It rarely or never occurs in chronic diseases of the heart. But, whether it be a nervous or inflamed heart, there is one condition necessary for its development, which is, that the heart should be the subject of an unusual amount of systolic excitement. It is for the most part a passing symptom, varying often, and rarely or never permanent.

Dr. Stokes (p. 119) says, "Its origin is difficult to declare; but that it is to be attributed to valvular, rather than to

muscular action, appears more than probable." Dr. Walshe (p. 80), from the limitation of the phenomena to certain points of the cardiac region, sees difficulty in referring these reduplicated sounds to irregularity in the closure of the valves in respect to time. Seeing, however, that a reduplicated sound is heard over the spot where it is produced, and that the normal sounds themselves are not necessarily very pervading, and are easily obscured, the first one by the second, and *vice versâ*, this limitation does not appear sufficient to overthrow the hypothesis of the valvular formation of a reduplicated sound.

I am disposed to conclude this reduplication of the sounds is due either to irregularity in the systolic action of the two hearts, so that this function does not agree in time; or to a ponding back of pulmonary blood on the pulmonary valves; or to inflammation, or irritability in one set of valves, that hence there arises a want of synchronism in the closure of the valves; and hence also one set, or, may be, both sets of valves of one heart, act more tardily than their congeners in the other heart —thus dividing into two sounds what in health would be, from their synchronism, one sound only.

As the first sound depends on the blow caused by the column of blood on the closure of the auriculo-ventricular valves, it may be inferred that a reduplication of the first sound has its origin in the disturbance of the synchronous closure of the mitral and tricuspid valves; while, as the second sound results from the sudden closure of the semilunar valves in each heart, a reduplication of this sound may be said to have its origin in a want of synchronism in these valves.

This theory of the formation of these reduplicated sounds, agrees entirely with the view proposed in these pages, that the heart's action is essentially ventricular, and that the sounds are caused by the arresting of a column of moving blood on the sudden closure of the valves. The production of two sounds only, in the heart's normal state, from the four valves, is effected by each corresponding set of valves acting in the most perfect synchronism. Any irregularity in this synchronism may hence induce the reduplication in one or both sounds.

If we examine the cases accurately in which these reduplicated sounds occur, they certainly present features which offer fair ground for assuming that there exists that amount of turbulent action which may induce a want of synchronism in the systole of the ventricles, and that hence this may be the cause of these irregularities in the sounds of the heart. The soundness of this view being granted, then the existence of a reduplicated sound is to be regarded as symptomatic of a want of synchronism in the muscular action of the two ventricles.

In nervous affections of the heart, there often are to be observed the elements which might produce this want of unison.

In those cases in which the second sound, being reduplicated, is associated with a mitral murmur, it may be assumed it arises from the too early closing of the aortic valves, in consequence of the spasmodically hurried systole of the left ventricle, under the excitement of recent inflammatory disease, whereby the sound induced by the aortic valves anticipates that caused by those in the pulmonary artery.

Or, on the other hand, any condition of the blood whereby its amount may be unduly forced into, or retained in, one ventricle over the other, probably would, by impeding its free, ready, and synchronous expansion, be adequate to cause this phenomenon. Dr. Cockle adopts this view where it occurs in cases of mitral regurgitation. "The aorta receiving a diminished supply of blood, in consequence of the regurgitant stream into the left auricle, fails of its accustomed stimulus of distension, and as a possible consequence, lags behind the pulmonary artery in its recoil. The closure sound of its sigmoid valves would then, in point of time, be subsequent to the recoil of those of the pulmonary artery, and produce the phenomenon of reduplication. This sign also might both characterize the disease, and, within certain limits, measure the amount of regurgitating blood."

Those cases, where a full inspiration induces a doubling of the second sound, and which is inaudible in ordinary breathing, Dr. Walshe (p. 81) says may be explained by the unduly abrupt rush of blood into the pulmonary artery, whereby the necessity

for closure of its valves to meet the recoiling fluid is felt a little earlier than usual.

Looking at the circumstances that belong to each of these instances, it is probable that reduplications of the first sound originate in want of synchronism of the commencement of the systole; and of the second sound in excitement, whereby its diastole is terminated too quickly.

CHAPTER IV.

ENDOCARDIAL MURMURS: THEIR DIAGNOSIS AND ITS DIFFICULTIES.

In the preceding chapter the presence of the normal sounds, and the non-existence of valvular murmur, were assumed conditions. In this, on the contrary, the actual presence of one or more of the endocardial murmurs will be assumed, together with some of the circumstances which, in practice, occasionally interfere with, and embarrass, their due appreciation.

The practical difficulties that interpose themselves towards ascertaining the presence of a cardiac murmur may be referred chiefly to the following sources: the being overpowered or masked by murmur emanating from other diseased structures; the being confusedly intermingled with other cardiac sounds when there exists a turbulent action in the heart itself; the being thought to be, from other circumstances not what it really is, but a murmur referable to some other source; the being so slight and feeble in tone as to be with the greatest difficulty appreciated as such; or the being entirely ignored from its fleeting and intermitting character.

Of the foreign sounds that interfere with the due appreciation of an existing murmur, the "râles" emanating from a pervading bronchitis are frequent and conspicuous. These are often so similar in their nature, and so pronounced in tone, that, when coexisting, it often becomes a very serious practical difficulty to discriminate, from amidst them, the cardiac murmur. To a certain extent the same may be said of the abnormal respiratory sounds occasionally heard in a pneumonia, and which are found

to offer a not infrequent complication with endocardial murmurs, especially when the heart is, coincidently with a pneumonia, the subject of a rheumatic inflammation, and when it especially generates the soft blowing murmur not altogether unlike that occasionally met with in the respiratory organs. So similar, indeed, at times, are these several murmurs that perhaps the only reliable mode of distinguishing between them is by procuring, for a short space, an arrest in the breathing. (*Vide ante*, p. 32.) The difficulty here is induced by a *quasi* similarity of origin, both the respiratory and cardiac murmurs being due to the interference to the even flow or passage of fluid. In the one case, the fluid being the air, in the other the blood.

Pleuritic and exocardial friction sounds have been shown to interfere with the due appreciation of the normal sounds of the heart, and so, in like manner, their presence may render difficult the easy recognition of an existing cardiac murmur by intermingling with, and thus obscuring, it. In these cases the recognition of the cardiac murmur must be sought not only from its retaining, under all circumstances, its perfect synchronism with the rhythm of the heart, but in the character of the sound itself; and in the presence, may be, of a valvular thrill, independently of the friction fremitus accompanying the exocardial or pleuritic friction sounds; and in being less circumscribed than are these latter, the cardiac murmur being generally appreciable in the course of the great arteries; as also, when the murmur can be ascertained, in the absence of the true valvular sound. Nevertheless it is a diagnosis of great difficulty. It is, therefore, not surprising Dr. Stokes should say (p. 34) that "when it happens, coincident with the attack of pericarditis, a diseased action is set up in the valves, the determination of the latter may be difficult, during the continuance of the true friction murmurs. If the valvular sign be, as it commonly is, a bellows murmur, it may be completely masked by the loudness of the friction sounds, and only become manifest on their cessation."

A true appreciation of a cardiac murmur may, in some few cases, be interfered with by murmurs originating in the large

arteries. Ossific or other deposits, or ulcerations in their coats inducing a roughened state of their surface, or aneurismal dilatations, or any abnormal pressure upon them from surrounding tissues, may each originate murmurs, not only loud enough to mask the presence of a valvular murmur, but so similar in nature as to defy a positive and differential diagnosis.

A cardiac murmur may be at times fully recognised as a murmur, but may nevertheless be thought not to be cardiac in its origin, but indicative of disease elsewhere, as when it originates a suspicion of exocardial, instead of endocardial, disease, or when it so communicates its sound into the great vessels as to induce the conclusion that the disease has its seat in them. Again, the murmur may be so transmitted through large glandular or cancerous masses, or portions of indurated lung, or even through aneurismal dilatations, as to mislead us regarding its true seat and origin.

Difficulty in diagnosis sometimes ensues when there occurs a temporary cessation of the murmur; it may be that there exist the elements for its formation, yet it is only to be heard under certain conditions, and usually those are conditions under which an examination of the heart is not commonly made, namely, after violent or exciting exercise; a period of repose, and there is no murmur to be heard. Sometimes, as disease progresses, the murmur so loses its characteristics as not to be recognised as such; this is especially the case when the powers of life are failing. Occasionally, also, these murmurs, from being doubled in each set of valves, become, when the heart is agitated, so mingled and involved as to offer to the ear nothing but a turbulent sound.

Supposing, however, that the presence of a cardiac murmur be satisfactorily ascertained, other doubts spring up, such as, to which of the valves is its seat to be referred; then whether it be caused by physical or only passing modifications in the heart's structure; or by an alteration in the composition, relative amount, or force in the current, of the blood, irrespective of any alteration in the heart itself.

As regards the ascertaining to which valve a murmur may

owe its origin, the first practical difficulty obviously arises from there being a double set of valves in somewhat close contiguity to each other. In this we have not now the practical difficulty that attended these investigations a few years since, for experience has taught us that disease, and the accompanying murmurs, belong mainly to the left heart, and that murmurs have rarely their seat in the right heart, without a previous and coexisting presence in the left heart. It may, in fact, be assumed, unless other circumstances indicate the existence of disease in the right heart, that the seat of murmur is in the valves of the left heart.

Sometimes the normal valvular sounds of the right heart may be heard and distinguished from an existing murmur of the left heart. Nevertheless, their subjective absence must not be adduced as evidence of the presence of disease of the right heart, it being for the most part found that murmurs in the left heart overpower and mask the sounds of the right heart. Diagnosis in this case will mainly depend on the accompanying physical sounds, on the direction of the murmurs, and on the absence of a fluid oscillation in the large veins, and with no attendant venous hum.

Practically, in the absence of all other indications, we have only, as a general rule, the murmurs arising in the left heart to deal with, viz., diastolic, and systolic regurgitant, murmurs of the mitral valve; and systolic, and diastolic regurgitant, murmurs of the aortic semilunar valves, and to these may be added murmurs induced by roughnesses and clots in the endocardium, or those resulting from a diseased state of the blood itself.

Nevertheless, it is always necessary to bear in mind that the right heart may also be primarily diseased, and, in cases of diseased left heart, often becomes contemporaneously or subsequently so; therefore, when investigating the condition of the heart, it is incumbent not to neglect if possible, the due ascertaining of this.

Premising that the immediate object now is the discrimination of one cardiac murmur from another, independently of all external sources of confusion, and assuming, in the absence of

other signs, that the left heart is the special seat of murmurs, it then becomes necessary, when the existence of a murmur is discovered, to ascertain whence is its precise origin in this heart, and whether it be a murmur of flow, or of regurgitation.

Cases frequently present themselves in which there may be but little difficulty in at once referring a systolic murmur, either to a regurgitation through the mitral valve, or to obstruction to the flow of blood through the aortic semilunar valves, or that disease in these latter valves is the cause both of this, and of a regurgitant murmur.

Nevertheless, difficulties in diagnosing each of these murmurs may occur. For instance, a slightly prolonged soft low murmur at the apex of the heart, synchronous with the diastole and followed by a normal first sound, might reasonably be assumed to be dependent on some obstruction to the even flow through the mitral valve, but without any of those conditions which permit of a regurgitation through this valve. It is, however, more than probable, looking to the results of experience, that such a murmur is not due to this cause, but to a regurgitant murmur in the aortic valves, and which, being produced by the diastole is not only synchronous with it, but is found to descend also towards the left apex. Though the possible case of a mitral flow presystolic murmur, the auriculo-systolic of Gairdner, thus exclusively occurring, is, by way of illustration, here mentioned, I am not aware that I have ever satisfactorily heard it. Doubtless its occurrence as such is very rare, and that it should be so is not surprising when it is borne in mind that in all probability it would depend on some source of obstruction to a free flow of blood through this valve, which is yet so perfect as a valve as not to permit any regurgitation.

Some recent observers, more especially Gairdner, Peacock, and Sutton, recognise a greater frequency of this murmur. The former esteems it easy of recognition, while the two latter appear to acknowledge some difficulty in a ready diagnosis. Supposing, however, that a presystolic mitral murmur do exist, but with the coexisting conditions of other valvular diseases, we might find that its due appreciation may not only be inter-

fered with by the phenomena attendant on the mitral regurgitant murmur, as also by the presence of a regurgitant tricuspid murmur.

The obstructive mitral murmur when it does occur—and it does not always occur, though there be obstruction—is low in pitch and rough in tone, and is, for the most part, associated with a systolic regurgitant murmur. This latter is usually so much more marked and prominent than the diastolic or presystolic murmur as to absorb and mask it, and thus become in great measure the only sound heard. This, at least, has been the result of my own experience, but Skoda defines the two murmurs as distinctly to be heard; the diastolic murmur immediately preceding the systolic. Under certain circumstances, this clearness and definition of the two murmurs has, certainly, appeared to me not to be altogether unappreciable, especially when a rapid and spasmodic action of the heart induces a quick flow of blood through the valve, but under ordinary circumstances the evenness of the flow of blood and the amount of the mitral valvular obstruction are inadequate to produce a murmur sufficiently loud or marked to be heard as a murmur, separate and distinct from the systolic regurgitant murmur of the same valve. The presence, however, of a presystolic mitral murmur may be assumed to exist in connexion with a mitral regurgitant, though there be a difficulty of definition between the two murmurs when the sound emitted commences in the presystolic period, and is continued into the systolic. Dr. Barclay* details a case in which a prolonged harsh grating murmur, under the influence of digitalis, was analyzed into two murmurs, a soft presystolic murmur which terminated before the harsher and systolic murmur commenced.

The *rationale* of this presystolic murmur appears to be that it is caused by the narrowing of a contracted auriculo-ventricular aperture, disturbing the even flow of blood from the auricle into the ventricle by the diastolic and suction action of this latter. Hence the conditions of what is termed the "fluid-vein" are fulfilled; and hence, also, from the relatively common want of

* *Lancet*, 1872, pp. 283, 352, 392.

energy in the afflux, the murmur is at times scarcely appreciable, but, if appreciable, is always essentially presystolic; or at times, by being continuous with, merges into the louder and more pronounced systolic regurgitant murmur generally, or perhaps always, associated with it. The ingenious experiments of Dr. Leared illustrate this position. Dr. Barclay, however, refers this presystolic murmur to a divided systolic action of the ventricle anterior to that which completes the systole. By this the blood is driven back through the patulous valve, causing a murmur anterior in time to the recognised and well-defined systolic murmur. This does not appear to be a likely or in any case tenable position, and is certainly not consistent with the view advocated in these pages. The analogy of the aortic regurgitant murmur, which is unquestionably due to the systole of the ventricle, leads to the conclusion that the mitral flow or presystolic murmur is also due to this and not to a contracting force in the auricle.

The systolic mitral murmur is usually defined and to be heard distinctly at the left apex, offering, for the most part, as its chief characteristic, a gentle blowing sound; or this, with only a slight roughness; and, if it be followed by a distinct valvular second sound, presents but little difficulty in the way of satisfactorily recognising its origin. When, however, the valvular second sound is indistinct, it has always appeared to me by no means easy to refer, with precision, the murmur to the mitral valve, for there may, and not unfrequently do, exist other causes for a murmur, audible at the base, very similar in sound and systolic in time; as also those which are due to rupture or other disorders of the chordæ tendineæ, rheumatic roughnesses of the endocardium, or even clots. Fortunately these several causes of confusion do not often present themselves.

The chief practical difficulties, preventing an easy recognition of this mitral systolic murmur, arise from the coexistence of other valvular lesions, inducing a general confusion of sounds, but more especially the presence of a loud systolic murmur in the semilunar aortic valves. This, from being synchronous with

the mitral regurgitant murmur, is very apt to mask, or so overpower, it as to prevent its easy recognition.

The basic murmurs, which may be four in number, two systolic and two diastolic, are often very difficult to define, and this is not surprising, considering that their origin is within a very narrow compass, and that the two systolic murmurs are synchronous with each other, as are also the two diastolic, and that these two latter so immediately follow the former, as to be often undistinguishable as any other than a continuous murmur.

Experience, however, has taught us that basic murmurs have their origin only very rarely in the valves of the pulmonary artery, and that when they do there exist, murmurs will also be found to exist in the aortic valves; therefore, as a question of diagnosis, when a systolic basic murmur alone is heard, there is rarely much difficulty in deciding its principal seat to be in the aortic valves, and that in these, by some sources of obstruction, there occurs interruption to the free and easy flow of the blood. Nevertheless, if this murmur be soft and low in tone, and there be synchronously with it, a loud systolic regurgitant murmur, and which, it may be assumed, has its origin in some deficiency in the mitral valve, the recognition of the aortic murmur may be attended with very considerable difficulty. The presence, also, of an aortic regurgitant murmur, may, likewise, so mask and overpower a constrictive aortic murmur as to render it not always easy to decide on the presence of the latter. For the most part, however, if the systolic aortic constrictive murmur be loud and well pronounced, its presence can usually be satisfactorily recognised.

The aortic diastolic or regurgitant murmur has been shown above to be sometimes mistaken for the mitral constrictive; on the other hand, this latter, when it is a prominent murmur—and, to a less extent, the same may be said of the constrictive murmur of the tricuspid valve—may mask or prevent a full and due appreciation of the aortic regurgitant. These are not, however, frequent sources of difficulty; moreover, the murmurs induced in the aortic valves are usually, but not always, sharper

and harsher in tone than those of the mitral and tricuspid valves.

The aortic regurgitant murmur is often concluded by a sound somewhat sharp and accentuated. It has been assumed this is the normal valvular sound of the pulmonary semilunar valves. Unless there be a want of ventricular synchronism, this cannot be the cause of the sound, as the systolic pulmonary valve sound is synchronous with the commencement of the aortic regurgitant murmur. It is probably the succeeding first sound, rendered sharper by the spasmodic action of the ventricle in consequence of the regurgitation of the aortic blood, and heard plainer by conduction; or it may be due to the vibrations caused by the forcible influx of the opposing columns of blood. Most probably, however, the above concluding sharp sound is a conducted first sound; as the tendency of the conflux of the opposing columns of blood would rather be to generate murmur, and thus add to, and prolong, the murmur due to aortic regurgitation.

With regard to the murmurs emanating from the semilunar valves of the pulmonary artery, and from the tricuspid valve, little is to be advanced here; occasionally a well-defined and isolated murmur in the former is to be heard; but these are peculiar cases. And perhaps exception may be also made in the case of a regurgitant tricuspid murmur, from there being some capability of tracing it in the course of the larger veins. But then difficulty presents itself, for it so generally happens when there is tricuspid regurgitant murmur, that the veins themselves are the independent seat of murmur. For the most part, independently of the comparative rarity of valvular disease in the right heart, murmurs here are so seldom isolated, that if not obscured by corresponding murmurs in the left heart, they are at any rate so synchronously mixed up with them as to be scarcely distinguishable. The difficulty of diagnosing these murmurs separately from those of the left heart, solely from the position of the murmur or the character of its sound, has hence appeared to me insuperable, and I cannot but endorse the opinion generally entertained in this respect.

The synchronism which makes of the two sets of valvular sounds but two sounds only, acts more energetically in the case of the murmurs from the prolongation of the sound on the one hand, and the much louder and sharper character of the murmurs emanating from the left heart on the other. In order to arrive at conclusions about the state of the valves of the right heart, it is necessary to call to our aid a full consideration of the attendant symptoms. It will be our part, subsequently, to refer more particularly to these; at present, the various sounds and murmurs emanating from the heart are alone taken account of.

Having, as nearly as one can, settled the precise seat whence proceeds the murmur, there then arises the question as to its nature; whether it be due to organic, or only to functional, disturbance, or to an altered condition of the blood, or to the presence of clots in the heart itself. The accurate discrimination of these is really a point of great practical importance.

As a general rule, and perhaps with the sole exception of the mitral regurgitant murmur heard in chorea, epilepsy, and after exhausting masturbation, and when there are clots in the heart, one may exclude from the category of functional murmurs all murmurs heard at the apex, regurgitant murmurs at the base, and all murmurs when there is collateral evidence of a diseased heart. There, therefore, only remains, with the above exceptions, the systolic murmur with which functional murmurs can be confounded. As the seat of these murmurs is more frequently than otherwise in the pulmonary artery, or spreading in the direction of the larger vessels, with both the aortic and pulmonic valvular sounds distinctly audible, their recognition as functional murmurs is not always unsatisfactory.

For the most part they are hæmic murmurs, and discoverable in those persons whose condition of blood is poor, in cases of incipient phthisis, after excessive hæmorrhages, in chlorosis, or when there is menstrual irregularity, and also, occasionally after a sustained and increased action of the heart from over-exertion or from extreme nervous excitement. As a general rule, in cases of chlorosis and spanæmia, the seat of the murmur is in the

pulmonary artery; whilst in anæmia, and after excessive loss of blood, it is in the aorta.

The systolic basic murmur heard occasionally in fevers not typhoid, but rather of the relapsing type, is probably of the same nature and origin. Dr. Stokes, who first called attention to it, says (p. 426): "This murmur, whatever may have been its seat and actual cause, is clearly to be placed in the category of inorganic murmurs, and its frequent development in the relapse, in cases too where enlargement of the spleen was observed, while at the same time the signs of softening of the ventricle were wanting, makes a strong case in favour of its being in some way connected with a depraved state of the blood."

While speaking of the difficulties that surround the recognition of murmurs in the heart, allusion must not be here omitted to those cases in which no murmur exists, or, at any rate, can be recognised, though there may be the elements of murmur, as far as the existence of diseased valves is concerned. As these cases will be particularly referred to subsequently, it will be here sufficient merely to state that this absence of murmur is chiefly met with where there is, at the same time, evidence of a want of force in the current of blood that passes through them—the main, or rather the absolutely necessary, element towards the production of sound or murmurs. Hence this absence of murmur is very frequently met with in those cases in which the current of blood is impeded by an over-distension of the cavities, and also when the powers of life are failing, so that pre-existing murmurs are lost in sounds which are inappreciable as belonging either to the category of normal sounds or murmurs.

CHAPTER V.

THE PULSE: ITS PHYSICAL ORIGIN.

In the preceding pages, the sounds and murmurs emitted by the heart and arteries, viz., the audible effects of the several vibrations induced by the movement of the blood, and by the disturbance and sudden arresting of its even flow, have alone been referred to. It is now proposed briefly to consider those vibrations which more particularly reveal themselves to, and are familiarly appreciated by, the sense of touch, viz., the recurring impulses and palpitations of the heart, and those recurring shocks, therefrom resulting, experienced throughout the arterial system. The impulse or beat of the heart, which, in healthy and fairly nourished persons, under circumstances of repose and in the recumbent position, is inappreciable to the touch, immediately becomes perceptible, on assuming the perpendicular position, in the space between the fifth and sixth ribs about an inch and a half to the left of the sternum. The phenomenon thus developed, though partly due to a slight tilting forward of the apex, is mainly effected by a change of shape in the heart itself, whereby it becomes elongated during the spiral contraction of its fibres. The amount of impulse, as well as the rhythmical action that characterizes it, are liable, under a great variety of circumstances, to considerable, and sometimes immediate, modifications. The almost imperceptible beat may present the characters of an obvious blow or sharp shock, and, instead of being limited to a narrow space, may be diffused over a large amount of the parietes of the left chest. So, likewise, its rhythmical action may be notably accelerated or interrupted.

These variations in the force and rhythm of the heart's pulse are, however, in their general occurrence familiar to the observers of the action of this organ, and, as they will necessarily require much careful consideration in the sequel, when speaking of some of its disordered conditions, they need not be here further referred to. It is now proposed to pass on to a consideration of the vibrations felt throughout the arterial system, and which we familiarly recognise and understand under the designation of the "pulse." When disease, with its several phenomena, is presented to us, we usually separate for special study and notation the modifications in the circulation of the blood, as evidenced in the frequency and character of the pulse. If this can be said of disease in general, it may more emphatically be said of diseases of its moving power, the heart itself; and hence, to a certain extent, the pulse has been regarded as one mode of interpreting the condition and action of this organ.

Before the adoption of the more modern means now employed in the investigation of the diseases of the heart, this was especially the case. Indeed, volumes have been written to show that the true appreciation of the pulse was an infallible means of indicating not only the morbid conditions of the heart, but of discriminating the locality and extent of almost all other diseases. Though we may not, in the present day, thus dogmatize upon the pulse, nor attribute to it that perfect indication of diseased action, which was attempted heretofore, yet we fully appreciate the value and importance of duly observing the departures from its healthy standard, and of considering how far these may be indicative of diseased action, whether this be in the heart or in the system generally.

In considering the phenomena and the *rationale* of the pulse, the physical agencies, whereby the blood is transmitted from heart to heart, have, since the days of Harvey, been often investigated with much assiduity, and various theories in explanation have been advanced. The difficulties attending these investigations may be appreciated by the varying and contradictory conclusions arrived at.

To some of the more prominent of these theories reference

will now be made, It is not, however, necessary to enter into any minute detail of them, especially as they are to be found fully set forth in several elementary and other works. A careful consideration of the bearing of these theories will show that they may be classified under distinct heads.

1. Those that regard the pulse as due to the distension of an elastic artery, whereby its diameter is increased.

2. Those that regard the arteries as inelastic, and the pulse as due to an extension in its length, whereby a displacement or locomotion takes place.

3. Those that refer the pulse to distension of the artery, with an active contractile power superadded, so that it has a systole and a diastole of its own.

It will be observed that the above refer solely to the proximate cause of the pulse; but in referring to the theories themselves, it will be seen they generally comprise within these terms the remote cause also. Thus in one the pulse is stated to be due to the "impulse of the left ventricle being communicated along the arterial tube and the column of blood which it contains." (A. Thompson, "Cyclopædia Anatomy and Physiology," Art. Circulation.) In another the circumstances are stated somewhat more precisely, and the pulse is said "to be due to the alternate dilatation and contraction of the artery, from the impulse and comparative retardation of the wave of blood corresponding with the systole and diastole of the left ventricle of the heart." (Haller, Richerand, Hales.)

In examining these several theories, we first see that the general weight of authority lies with the opinion that the arteries are endued with the property of contractility, and that this property is excited to action by the force of the left ventricle; and then that, with one exception, they set forth that the impulse from the heart communicates itself to the column of blood contained within the arteries; nevertheless they differ materially as to the mode by which this is effected; thus, according to one theory a temporary dilatation of the artery is induced at each recurring influx of blood; according to another, the dilatation is followed by an active state of contrac-

tion; according to others, neither of these conditions is admitted; while one attributes the phenomena of the pulse solely to a locomotion in the artery itself. All the above recognise the moving column of blood as the medium in which are developed the phenomena of the pulse, whilst the artery is only the containing tube acted on by this medium. The theory, which is an exception to this general view, is that which refers the pulse solely to a contractile power in the arteries themselves. These several theories, as before observed, comprise both the proximate and the remote causes of the pulse; the latter being almost universally attributed to the contractile agency of the heart itself, while the former has not only been also referred to this, but likewise to other and very different agencies.

It will be useful to look particularly to these, so as to separate and define them; and in doing so we will first refer to the immediate phenomena of the pulse, and those circumstances which belong to its proximate origin, and then consider separately its remote cause, so as to ascertain the precise value, in reference thereto, of the contractile agency of the heart. In considering the nature of the pulse we may, for the sake of argument, discard all questions about the causes inducing the circulation, and look solely to those peculiar phenomena, to which the term "pulse" has been given, occurring normally and periodically in the arteries; in the capillaries, under conditions of excitement or inflammation; in certain veins normally, and in others under certain forms of disease (*varix*).

Before going further it may be as well to define what, according to the best recognised authorities, the pulse is, and see if the definitions comprise all the phenomena belonging to it. The usual definition is, that it is "the stroke or beat of an artery, as recognised, for the most part, by the finger applied to the integuments lying over it" (Bostock, Allen Thompson). Other and somewhat differing definitions are that it is "a jetting movement of the blood, whereby, by dilatation and elongation (which latter is the greatest) is effected the pulse." (Kirkes, Carpenter); and "a succession of impulses or distensions alternating with the reaction or subsidence of the vessels, and felt

throughout the body wherever the arterial ramifications penetrate" (Dalton).

If we regard the pulse as comprising the whole of the phenomena that take place from beat to beat, the above definitions are found to be neither descriptive nor distinctive, inasmuch as they do not accurately pourtray the whole of the periodically recurring phenomena attendant on the circulation of the blood through the arteries. Nor do they refer solely to the shock. Assuming the correctness of the recognised theory of the cause of this shock, and which also sets forth and accounts for that continuous wave, whereby the progress of the blood is effected throughout the whole system, they are notably incomplete. The definition of such a pulse should include the whole series of phenomena; viz., the undulating wave, the shock or sudden vibration, and the pause. The short sharp stroke or vibration occurring almost simultaneously throughout the whole column of blood is, however, peculiarly that portion of the series to which the term "pulse" is given, and to which doubtless it is not only convenient, but I trust in the sequel will be seen, truthful to restrict it.

In another place * I ventured to advance the theory that the pulse was divisible into three phenomena: 1, the filling of the artery by successive waves, through the agency of the ventricular system; 2, the vibration in the column of blood throughout the whole arterial system, caused by the sudden closure of the semilunar valves (the pulse); and 3, the contraction and collapse of the arteries during, but not caused by, the ventricular diastole.† It will be the purport of the following pages to support and illustrate this view.

* *British Medical Journal*, 1st Sept., 1866.

† By the aid of the sphygmograph Dr. Sanderson believes that the following facts are made out with tolerable certainty and distinctness:—

"1. At the moment that the heart begins to contract, a vibratory movement of the blood contained in the aorta is produced, in which the molecules of liquid are projected forwards in the axis of the vessel. A similar vibratory movement occurs at the moment that the ventricle ceases to contract. These two movements differ only in this respect, that, in the former, the primary shock is directed towards the periphery, in the latter, towards the heart. 2. Each of these movements is propagated in the direction of the stream at a rate of about ninety feet per minute, the one expressing itself at the radial artery by a sudden

The differences which characterize the above several phenomena of the circulation of the blood through the arteries, are in health sufficiently recognisable, while in disease they often become strongly marked, and thus offer indications of the utmost value in diagnosis. Following, as they do, a natural order, they occupy in their relations to each other definite proportions of time. This rhythm of the arterial circulation may be divided into five periods—one being occupied by the wave; one by the shock (pulse); and three by the pause.

As generally confirmatory of this position, I would refer to

expansion of the arterial tube, the other by a sudden collapse. 3. The exact moment at which each movement occurs is indicated by the sphygmograph, the former by a sudden vertical ascent of the lever, the latter by a descent. In either case the primary movement is followed by a succession of smaller movements in alternately opposite directions. By measuring the time which intervenes between the first and second vibration, the exact duration of the systole of the heart may be ascertained; for the one occurs at the instant that the ventricle hardens in contraction, the other at the instant that the aortic valve becomes tense in closing. 4. Both movements manifest themselves with much greater distinctness in some cases than in others. In this difference it seems probable that the respective valves concerned have much to do, and that the systolic vibration is produced by the sudden tightening of the mitral valve, just as the diastolic vibration is due to the sudden tightening of the aortic valve. The precise physical conditions on which the degree of vibration depends have not as yet been ascertained, but observation points to the conclusion that the intensity of the systolic vibration is greatest when the arterial pressure is lowest at the close of the diastolic period. 5. From the moment that the ventricles begin to contract, the fulness of the arterial system and, consequently, the arterial pressure, rapidly increase. As, however, the arteries at first yield readily to the tide of blood, the tension does not attain its maximum until some time after the hardening of the ventricle in contraction. The duration of the interval between the one event and the other, that is, between the closure of the mitral valve, and the moment of the highest pressure in the radial artery, varies. It is longest when the arterial system is full, shortest when it is comparatively empty. Hence the measurement of this interval comes to be of considerable importance. 6. From the moment that the artery attains its greatest distension it begins to collapse. The form of that part of the sphygmographic tracing which corresponds to the period of relaxation, is, as has been recently pointed out by Dr. Divers, parabolic; in the normal pulse the parabolic form is not easily distinguished, the time of descent being nearly rectilinear, but in all those pulses in which the collapse is rapid, it is very obvious, and most of all, in what has been called the monocrotous form, which corresponds to the thready pulse of authors. 7. In certain conditions of the circulation the radial artery, immediately after the distension produced by the contraction of the left ventricle, suddenly collapses, and then as suddenly expands again, the second expansion being sometimes nearly equal in intensity to the first. This constitutes dicrotism."—"Handbook of the Sphygmograph," p. 19.

the figures of arterial action delineated by aid of the sphygmograph (*vide* Foster, Sanderson, etc.). Though the sphygmograph may probably not be so perfect an instrument as the educated finger, it shows, by its written diagrams, sufficiently accurately the increasing tension of the onward wave, occupying one-fifth of the rhythmical time, and which culminates in the sudden vibration or shock; and it also notes the decadence of the arterial tension immediately succeeding the shock. It appears to me that, in the diagrams of the normal pulse, the period, nay the existence of the shock (pulse) is not distinctly marked; no alteration or sudden variation occurring in the gradually increasing tension of the onward wave, till the period of the collapse, which ensues after the tension has attained to its greatest height. The sphygmograph, under such circumstances, really marks less the pulse-shock than the other phenomena of the arterial circulation. In certain instances of disordered circulation this exclusiveness may, however, not be so marked. By reference to these diagrams, where the shock or pulse proper is interfered with, its rhythmical duration can, perhaps, be appreciated; and, as such, these diagrams are so far confirmatory and illustrative of the theory I desire to propound; and they will, in the sequel, be again referred to.

Before the true nature and origin of the phenomena which constitute the pulse can be fully appreciated, it is necessary that the physical qualities of the arteries, and of the moving column of blood, in relation thereto, should be duly estimated, they being the physical media in which this peculiarity of the circulation takes place, and by which it is transmitted. As regards the arteries the questions immediately arise: 1, are they endued with elastic and with contractile powers? and 2, are they liable, where not sufficiently bound down, to displacement by the force with which the contained fluid is moving within them?

It will not be here necessary to revert to the experiments and arguments of the several exponents of each of the above views; it will be sufficient to state generally there are grounds for assuming, that while the arteries may be in certain positions

subject to displacements by the active force of the blood, and likewise endued with elastic and contractile powers, whereby a tension of the vessel is maintained, and thus enjoy the function of "reproducing a force proportional to the degree in which they are extended beyond their natural dimensions" (Young, vol. i. p. 519), yet it cannot be assumed that both or either of these accidents of their existence are sufficient to explain the occurrence of the pulse. The pulse may, in fact, be originated and transmitted irrespective of any such qualities.

Is the onward wave of the blood, then, the cause of the pulse? It is to be borne in mind that this and the previous questions, notwithstanding it be admitted that an active vitality exists both in the arteries and in the blood, are to be decided by the ordinary laws which determine the phenomena of mechanical motions, inasmuch as the vibratile motions of the blood and of the arteries, though taking place within an animal frame, are regulated by the same general laws as the motions of inanimate bodies. Hence, though the blood, in accordance with the properties of fluids, when forced through the arteries by successive waves, exerts an amount of lateral pressure equal to that which rules its onward course, yet, it having been also granted that the artery is endued with the quality of elasticity, there exists precisely that condition, viz., the onward current of a fluid, proceeding by successive waves through an elastic tube, which militates against the wave, of the onward current, being transmitted as a pulse—a condition which not only interferes with the tactile appreciation of the wave of the onward current but really tends to equalize and obliterate it. It represents, in fact, the very mechanical apparatus adapted for effectually counteracting the disturbing effects of an unequal flow, and to which all tubes which are the subject of a supply, *per saltum*, without such elastic quality, would inevitably be liable. Moreover, the velocity of the transmission of the impulse, whereby a fluid is propelled through elastic tubes, is diminished by an increase of tension. Doubtless this law holds good as regards the wave flow of the blood itself, while there is no evidence that the pulse is thereby obscured or retarded; on the contrary,

the greater the tension the more decided and pronounced the pulse. Hence, we may assume that the agency of the wave which effects the onward current of the blood, does not entirely account for all the phenomena observed in the pulse.

It has been assumed, when describing and defining the pulse, that there are evidences of a wavelike expansion and of a sudden vibratory shock or recoil, and then of the temporary, though not entire, subsidence of undulation and vibration. The arteries are, undoubtedly, the means whereby these undulations and vibrations are transmitted and made manifest, and the blood contained within them is as unquestionably the seat of them. Again, so much as the arteries are elastic, or locomotive tubes, so much may they be influenced by the pressure exercised by the fluid they contain; hence, they reveal the varying tension caused by the onward wave of the blood. Supposing this onward wave to have induced that amount of lateral pressure which places the artery in a state of tension, there then exists that condition which qualifies the artery to be the sensitive transmitter of other vibrations, than those of the onward wave, which may occur amongst the particles of its contained fluid, inasmuch as the vibrations of a fluid within a tube will be less interfered with when the containing tube is inelastic; or if elastic, when, by means of extreme tension, it is so completely filled as to represent one that is inelastic.

In the elasticity of the artery there exist the conditions, not only of resisting injury from the sudden pressure of the onward wave, but likewise of facilitating its transmission; while in its full and tense state, when this wave is at its summit, it offers conditions of being the ready transmitter of such vibrations, other than those of the onward wave, which may take place in its contained fluid; and this fluid, as being incompressible and inelastic, is the easy recipient of any vibrations to which it may become subject. It is not improbable that, as regards the artery and the blood (separate from the moving power, the heart), the above comprise the only conditions really necessary for the perfect development and transmission of any vibrations which may take place in the one or be revealed by the other.

The artery then presents the conditions both of an elastic and an inelastic tube—the one, capable of equalizing the sudden propulsions of the wave of blood, and the other, of transmitting vibrations occurring in its contained fluid other than those of its propelled wave; while the blood presents, on the one hand, a wave-flow, and, on the other, enjoys the usual qualities of a fluid, of being the easy recipient of vibrations. We will now briefly examine how far the phenomena, observed to take place during the rhythmical flow of the blood, are referable to either or to both of these conditions.

The obvious properties of the heart are its systole and its diastole, and it is to its systole that the circulation of the blood through the arteries is immediately due. By this function the whole, or, may be, only a certain portion, of the blood contained in the ventricle is expelled into the artery, and it is assumed, the moment at which this takes place corresponds with the pulse in the artery. Be this as it may, the propulsion of the blood through the arteries is associated, either at the moment of the systole of the ventricles, or at some other moment, with certain obvious phenomena to which the term "pulse" has been given.

Doubtless the systole of the ventricle, and the emission of blood thence proceeding, satisfactorily accounts for the circulation, but does it as conclusively account for all the phenomena of the pulse? It has generally been assumed that it does, and Young, in his learned and elaborate paper on the functions of the heart and arteries, seeks to show, from the experiments made by himself, Hales, Poisseuille, and others, that such an assumption may be demonstrated as a mathematical truth. In the course of his observations (p. 519), he says: "The successive transmission of the pulsations of the heart, through the length of the arteries, is so analogous to the motion of the waves on the surface of water, or to that of a sound transmitted through the air, that the same calculations will serve for determining the principal affections of all these kinds of motion; and if the water which is agitated by waves is supposed to flow at the same time in a continued stream, and the air which conveys a

sound to be carried forward also in the form of a wind, the similitude will be still stronger."

It will be observed that, in the above, Dr. Young refers to two sets of phenomena, viz., flow, and vibration superadded thereto, and it is to this distinctness of phenomena taking place throughout the arterial system, during each rhythmical movement of the blood, investigation is now directed. Though the phenomena of the circulation and of the pulse are coexisting, it does not follow, arguing *à priori*, that they are necessarily due to the same cause, or have precisely the same identical origin. The free circulation of a fluid through a tube, whether it be *per saltum* or not, may be effected without being accompanied by the phenomena of a pulse. Hence, the circulation of a fluid is not dependent on the causes that induce the pulse. As regards the cause of the occurrence of this latter, several questions present themselves, the more prominent of which are:—Is the pulse a phenomenon, attendant upon and caused by the onward wave in a moving fluid? or is it a vibration only through its several particles? or is it a vibration superadded to, or distinct from, the onward wave? or, can it be produced without this fluid being a circulating or moving one? It has already been shown, when referring to the physical qualities of the arteries and of the blood, that there exist grounds for concluding that the vibrations of the onward wave do not comprise the whole of the vibrations that take place within the artery. In examining the above questions, it will be necessary to ascertain if this really be the case.

It has generally been considered that the wave of blood discharged at each systole of the ventricle fully accounts for the pulse, and there have been many experiments made and much argument adduced to prove that it is so. Those of Dr. Young are the most comprehensive. "It may be supposed" (he says, p. 214) "that the heart throws out, at each pulsation, that is about seventy-five times in a minute, an ounce and a half of blood; hence, the mean velocity in the aorta becomes eight inches and a half in a second; and the velocity in each of the succeeding segments must, of course, be smaller in proportion,

as the joint areas of all the corresponding sections are larger than the area of the aorta." In another place he says (p. 519) "that the velocity of the pulse must be nearly the same as that of an impulse transmitted through an elastic tube, under the pressure of a column of the same height as that which measures the actual arterial pressure: that is, equal to that which is acquired by a heavy body falling freely through half this height. In man, this velocity becomes about fifteen feet and a half in a second, to which the progressive motion of the blood itself adds about eight inches; and with this velocity, of at least sixteen feet in a second, it may easily happen that the pulse may appear to arrive at the most distant parts of the body, without the intervention of any very perceptible interval of time." In considering the above, we see that two circumstances are detailed: the one, the progressive motion of the blood, which in the aorta is estimated as having a mean velocity of eight inches and a half in a second; and the other, the mean velocity of the pulse, which is estimated at about fifteen feet and a half in a second.

Assuming the correctness of the above, it is obvious that the circulating wave of the heart is not sufficiently rapid to account for the simultaneous pulse exhibited throughout the arterial system. The one, or may be two, ounces of blood forced into the arteries at each contraction of the ventricle, is obviously, *per se*, inadequate to effect so universal and instantaneous a vibration. Nevertheless, it is here necessary to state that there is a great discrepancy between the estimated, and, from experiment, the inferred velocity of the calculation. Hering inserted prussiate of potass in one jugular vein of the horse, and detected it in the other in twenty seconds, and hence inferred the whole circulation of the blood only occupied this short space of time. This experiment, the more especially as the viscidity of the blood exhibits a resistance four times greater than that of water, offers evidence of a most mysterious fact in the ordering of the animal economy, but as regards the physical origin of the pulse, it has little or no bearing. Though the blood may traverse the system in this incredibly short space of time, it would not account for the production of the pulse, synchronous,

or nearly so, as this is throughout the whole course of the arteries.

Moreover, in the history of the circulation, there exists a large number of facts which show the pulse to be a measurer neither of the quantity nor of the speed of the blood; for often, most often indeed, the frequent and throbbing pulse is associated with an enfeebled circulation, and a lessening in the amount of the blood circulated; nor is it an indicator of the *vis a tergo*—of the force of the heart's action—for the pulse may be feeble while the action of the heart is violent and excessive. The position that the pulse is a measurer neither of the speed nor quantity of the blood circulated, nor of the power of the heart, is most evident when, assuming that the ventricle discharges the whole of its blood at each systole, it is considered a pulse at 120 would, over a pulse at 60, exhaust the labour of the heart by the square of the result. That the onward wave of the circulation does not constitute the pulse is evident, from the fact that if a ligature be applied to an artery, the vibration of the pulse is felt both at the proximate and distant ends of the vessel, where so tied, though the blood at these points forms no longer a portion of the moving fluid. The circulation is arrested, but the pulse continues.

The pulse is also felt more extensively in some cases in which the circulation is materially diminished, as where the blood becomes inspissated, or it may be even rendered more obvious and largely diffused, though the onward wave must be, comparatively speaking, disguised, or even lost entirely, as in an aneurismal dilatation. Here, a fibrinous mass, which may scarcely admit a flow of blood through it, communicates on every side a hard and exaggerated pulse. The same may be observed when an artery is ossified, or where overlayed by a dense material, as also in those circumstances where elasticity compensates for, and obliterates, the shock of the onward wave; as occurs in the trunk artery and its larger branches, which offer but little external evidence, by expansion, of the onward wave, but freely communicate the vibrations of the pulse. The vibrations of the pulse are otherwise seen to be independent of the forward wave,

for velocity in this may differ in different arteries, while the pulse retains its synchronous action. Again, where inflammation has induced a state of local congestion, the pulse may be, in the arteries concerned, more markedly felt. The above instances show conclusively, that the peculiar circumstances of pulsation, in whatever they may consist, are, in effect, separate from the blood-wave; and therefore it may be inferred they do not owe their immediate source to that mechanical power or force which originates the circulation of the blood.

The blow which the heart makes against the ribs—the *ictus ventriculi*—and which does not occupy the period of the ventricular contraction, but only its concluding act, is neither a pulse nor the cause of it; not only are they not synchronous, but they bear little or no uniform relation in force to each other; the former may be violent, the latter weak. Moreover, the various observations which have been made on the circumstances of the circulating wave, show it not to be the result of a violent or spasmodic contraction of the originating power, such as might be inferred to be the case if it were the result of an action producing a violent rush of blood, and this so sudden as to be ejected at a single moment, and also to produce the vibration of the pulse. The experiments of Hales may be particularly referred to, in confirmation of the above; as also the absence of recoil in the distant arteries, and the absence of any bursting effect when, in an aneurism, the distant vessel is tied.

After fully considering the whole question, we cannot but conclude that the circulating wave and the pulse are two separate and distinct operations, and therefore, as taking place in the same medium and transmitted by the same tubes, not due to the same cause. Doubtless, the contraction of the ventricle causes the projection of the column of blood, and this column traverses the arterial system by successive waves; while the pulse is a vibration taking place simultaneously through the whole particles of this, as it were, solid column of blood. The vibrations which constitute the pulse, obey the same laws as sound transmitted in bodies, or those vibrations which, when a blow is struck on one end of a log of wood, are felt distinctly by a hand

applied to the other, although there be no visible locomotion. The pulse is, in fact, similar to that tangible shock which "is conveyed through a fluid without any apparent accumulation of it, or change of velocity. Thus, the working of a water pump may be discovered at great distances through iron pipes, or even through elastic pipes of leather, as those of a common fire engine, from which the water is spouting nevertheless in a uniform stream." (Arnott.)

What, then, is the cause of those vibrations which constitute the pulse, as separate from the circulation? In the course of the preceding argument it has been shown that, in the arteries and in the moving column of fluid within them, the physical conditions exist for the ready transmission of vibrations occurring in this latter; and it has also been shown that the mechanism of the heart is well adapted for the production of these vibrations.

Having reference to that general law of hydraulics, which enunciates that the momentum of a liquid in motion is as great as that of a solid, and, hence, that anything which opposes or arrests this momentum, will receive as severe a blow as if from a solid, and cause within it the same disarrangement of particles, we see that, as the blood in circulation is a moving column, and its motion be opposed or arrested, there then exists the conditions for producing those vibrations within the column similar to those which would take place in a solid. It has also been shown (page 15) that, in the sudden closure of the semilunar valves, the mechanism for thus opposing and arresting the circulation exists.

It is probable, considering the elastic properties of the arteries, that were it not for the closure of the semilunar valves during the diastole of the ventricle, whereby the momentum of the column of blood is arrested, the supply *per saltum* would have been equalized in these vessels, so that there would not only have been a pulseless wave, but one rendered tolerably even in its flow. The onward wave is, both in its origin and in its effect, independent of the vibrations which constitute the pulse, as it also is of the cause of these vibrations;

while, on the other hand, these vibrations are, in their origin independent of the cause of the circulating wave. They are, in fact, dependent for their development on that valvular mechanism, whereby the onward wave is suddenly arrested. It has been stated, that the passage of the blood through the arteries is characterized by three phenomena: 1, the filling of the artery by the onward wave; 2, the vibratory pulse shock; and 3, the gradual cessation of vibration (page 56). Looking to what has now been set forth, these phenomena may be thus explained. The moving wave, whereby the circulation of the wave takes place, is accounted for by the systole of the ventricle; then the sudden vibrations taking place simultaneously throughout the whole moving column of blood, and which constitute the shock or pulse, are accounted for by the momentum of this moving column being arrested by the sudden closure of the semilunar valves; and the succeeding pause, as that period during which the momentum of the wave is temporarily arrested, and before it again has re-established its even flow.

As regards the relations of the pulse to the sounds of the heart, in respect of time, it may be stated that the onward wave commences simultaneously with the first sound; the vibrations that constitute the pulse shock commence synchronously with the second sound; and the pause occupies the time between the subsiding of these vibrations and the first sound. The reading of the sphygmograph shows an ascending line; this commences synchronously with the first sound, while the highest point of this depicted line is synchronous with the second sound, and the fall or wave-like decadence from this highest point represents, save during slight continuance of vibrations, the pause or period between the second and first sounds. The explanation of all this is, that the ascending line of the sphygmograph depicts the expanding of the artery by the onward wave of the blood forced into it by the systole of the ventricle. Its highest point, which is synchronous with the pulse vibration, and with the second sound, represents the instant when the momentum of the onward current is arrested; while the fall in its line represents the period between the second and first sounds, and

before the systolic action of the left ventricle has re-established the onward wave-flow. Dr. Sanderson appears to differ from this conclusion when he states "that the intensity of the systolic vibration is greatest when the arterial pressure is lowest at the close of the diastolic period." This appears to be doubtful, for it is more in accordance with the laws of hydraulics that vibrations should be greater in a tube tense with fluid than in one not so; moreover, the closure of the aortic valves is more energetic, and they are better placed, as regards position and time, to induce a vibratory wave than the mitral valve.

Before concluding this chapter, it may not be out of place to observe, that the theory of the pulse above set forth, explains many peculiarities of the circulation observable in disease, and which, without it, are not, as it appears to me, easy of explanation; such as the violent beat of the heart with a feeble pulse; the absence of the true pulse vibrations when aortic valvular insufficiency is confirmed; the undulations, without pulse-shock, of the venous pulse, etc. These will, however, be more particularly referred to when these diseased conditions are the especial subject of remark.

CHAPTER VI.

GENERAL PROGNOSIS FROM SOUNDS, MURMURS AND PULSE.

HAVING now passed in review the *rationale* of the pulse, and the causes of the sounds and murmurs proper to the heart, as also some of the difficulties that may prevent their ready and perfect recognition, we will briefly refer to some of the more certain conclusions which may be deduced from the observation of these phenomena.

First, as regards the valvular sounds. If these be all clear and distinct, and free from any murmur, it may be inferred there is the necessary proportion between them and the current of the blood; that, in fact, the passage of the blood is not relatively interfered with; so that, looking, on the one hand, to the amount and force of the blood, and, on the other, to valvular structure, it may be inferred as regards the flow of blood, the valves are in a passive state, and exert no force whereby its flow is resisted or impeded; and, as a correlative of the above, it may be inferred, when valvular murmurs take the place of the normal valvular sounds, that a disproportion between the current of the blood and the opening it has to pass through, does exist; and that the valvular opening, whatever may be its condition, acts more or less by interfering with the flow of the current. This may be due either to excess of force in the current of the blood itself, or to disorder in the valves.

If valve sounds exist, preceded by a murmur, the prognosis is that the closure of the valve is complete, but that the passage of the blood preceding this closure is interfered with, from one or other of the above causes, so that it has not room to pass through

its channels without an eddy being produced. It may also be inferred, if a valve be sufficiently diseased to produce a regurgitant murmur, that the normal valvular sound from this valve will not be heard, the murmur entirely occupying the place of it. The murmur being due to the return of blood through the limited and diseased opening, while the absence of the normal valvular sound is due either to this latter sound being masked by the murmur or to its entire absence, by reason that the diseased condition of the valve does not afford the necessary check to the onward stream of blood. Nevertheless, a valvular sound may occasionally be heard synchronously with the regurgitant murmur, but then it emanates from the corresponding valve in the other heart.

If the diseased obstruction be in the semilunar valves, the murmur thus resulting will be found to be synchronous with the normal sound of the mitral valve, but when loud and prolonged, may, to a certain extent, so mask it as to prevent its easy recognition; hence, as a general rule, it may be assumed that a murmur which supersedes both sounds may possibly have its origin in disease of the semilunar valve only, while a murmur, followed by a distinct normal second sound, indicates alteration in the mitral valve; if, however, the mitral murmur be a regurgitant one, it may seriously interfere with the easy appreciation of the second sound; generally, however, it can be detected either in the left, or in the right, heart, and in such a case a correct diagnosis can be arrived at.

If the semilunar valves be so diseased as not only to obstruct the systolic flow, but likewise to permit regurgitation, there will either be a very prolonged, or a double murmur. If the necessary conditions exist in the valves of the pulmonary artery, probably the same phenomena might be observed; but as these latter are rarely the seat of independent lesion, the murmurs generated in them are synchronous with those generated in the aortic valves, and not distinguishable from them.

Independently of all other considerations, the presence of a double murmur generally indicates disease of the semilunar valves, a double murmur being more rarely produced by the

mitral valves; for though these be so contracted as to interfere with a free flow, the slower way in which the blood passes through their valvular opening, does not always necessarily produce a presystolic murmur. Under such circumstances, and they are of frequent occurrence, a regurgitant murmur only is heard, as emanating from the mitral valve, while the second murmur is the result of regurgitation through the semilunar valves. Hence, as a general rule, it follows if there be a double murmur, the semilunar valves are diseased, and, may be, the mitral also. In order to decide this, it becomes necessary to ascertain the presence or the absence of the first sound. If present, there no longer remains any difficulty in the diagnosis.

The explanation of the above may be briefly summed up. In those cases in which the valve, by offering obstruction to the flow of so much blood as is presented to it, induces an eddy, and consequently a murmur, yet being perfect as a valve, the murmur is succeeded by a true normal valvular sound. Where there is permanent patency of the valve, but no obstruction to the flow, there is no normal sound, but there is a murmur. There is also no normal valvular sound where there is both obstruction to flow and imperfect valvular closure, but there is a murmur accompanying both the flow of blood and its abnormal regurgitation; this murmur may be one prolonged murmur or a double murmur; for the most part the former.

The quality and the character of the sound of endocardial murmurs vary greatly in intensity. Perhaps the quality of semilunar murmurs is sharper and generally more intense in tone than those of the mitral and tricuspid valves. Little practically, is, however, to be concluded from this; slight and unimportant disease may be accompanied by the louder sounds, and the extremest of disease by little or even no sound at all. Physically (and it is this view only that now occupies us), this may depend on various causes, the chief of which are the relative frequency and force of the circulation, on the one hand, and, on the other, the vibrating power of the parts concerned. As are the force, the amount of fluid, and the obstruction, and the

vibrating power of the structures concerned, so are the intensity and the quality of the murmur.

The indications of the pulse were, in former days, much studied and much relied on as a means of prognosis. Galen in the second century classified the phenomena of the pulse with nice precision; and from his time, till the discovery of Harvey, the views of Galen were adopted. Then a new impetus to the study of the pulse was given, and in the first half of the eighteenth century its phenomena and their indications were set forth with singular minuteness, more especially by Floyer*, Solano†, Nihell‡, De Bordeu§, and Wetsch‖. If we may believe the statements of these observers, their success in prognosis was thus made to be very considerable.

The modern physicians, though always noticing the pulse, have certainly not done so with a precise accuracy, and have thrown aside as useless much that the older observers regarded as important. The sphygmograph has shown that, in some respects, the observations and descriptions of our fathers were not altogether futile. Dr. Sanderson (Handbook of the Sphygmograph) duly sets this forth.

Dr. Arnott, after detailing the phenomena exhibited by the pulse, makes these pertinent observations (vol. i. p. 544, 2nd Ed.):—"The preceding considerations exhibit the pulse as a complex subject, and one on which professional opinions are not yet settled. By showing its close relation to the powers of life, they also prove it to be an object of high importance to the medical practitioner. This last truth has scarcely ever been questioned but by persons singularly deficient in the power of tactile discernment, or utterly uninformed; yet, because no simple and good analysis of the pulse and detail of its relation to morbid states, has appeared, the degrees of skill acquired with respect to it by individual practitioners are very various,

* "Physician's Pulse Watch," by Sir John Floyer, Knt., 1707.
† "Lapis Lydius Apollinis," by Solano de Lugues, 1730.
‡ "Crises of the Pulse," by James Nihell, M.D., 1750.
§ "Varieties of Pulse and the Crisis each Indicates," De Bordeu, 1764.
‖ "Medicina ex Pulsa," J. J. Wetsch, M.D., 1770.

and in a great measure accidental. Some try the pulse merely for form's sake, because patients expect it; many examine it only to count its frequency; but others read in it, with confidence, much of the history and probabilities of the disorder, and decide on the treatment accordingly. Few who have attended to the subject at all can confound the pulses of such diseases as acute rheumatism, gastric inflammation, the fits of ague, etc. The author remembers to have conversed with a Chinese practitioner, who had only the scanty medical information of his countrymen, but who judged by the pulse in a way to surprise."

In the present day we must all feel that there is much truth in the above. That the indications of the pulse have not been accurately studied is perhaps due to our having other and perhaps more certain modes of arriving at a correct diagnosis of disease. The readings of the sphygmograph have certainly during the last few years excited attention to the subject, and perhaps much that is useful and satisfactory may be expected from it; but it cannot do all. It notably fails in indicating the pulse-shock and the varying condition of the afflux wave. The finger is the more accurate instrument, and reveals to the careful observer more than can be done by any mechanical appliances. Moreover it is always ready. Mechanical appliances, on the other hand, are difficult of application,—the sphygmograph especially so,—and by their variations and errors rather complicate and embarrass the data for just conclusions. From my own experience, I entertain the feeling that the notation by the watch disturbs the observation of the more reliable evidence otherwise yielded by the pulse. The facts tested by the watch are not always important, and may be approximately, if not more correctly, ascertained by only a slight amount of careful study and attention, so as to render unnecessary the thus noting the frequency and the irregularities of the pulse-beat; while its habitual adoption leads to the giving an undue importance to the value of these phenomena. Moreover, the compound mental effort of counting the frequency of the pulse simultaneously with the noting and comparing this frequency by the mechanical

aid of the watch often so distracts as to interfere with a correct conclusion as to this fact itself. Hence the very means adopted in order to arrive at a precise and numerically-accurate observation often defeats itself. In my early professional days an old and experienced practitioner remarked to me that no two people told by their watches the beat of the pulse to be the same; and there is much truth in the remark. I have certainly myself found advantage in attending to the general phenomena of the pulse without the distraction of a watch; and I think, too, with a better power of appreciating, not only them but, excepting in special cases, its numerical frequency.

Though the indications to be derived from the pulse, towards the ascertainment of specific disease, may neither be very numerous nor perfect, still there are some. Here reference will only be made to a few of those indications directly bearing on the condition of the heart.

The pulse, as regards rhythm, generally speaking, both in health and disease, indicates the rhythmic action of the heart, and should there be any failure in the concord of these, it may be inferred there is either valvular disease, or that the walls of the heart fail in nervous power, or are degenerated; and the same may be said of the force of the pulse, this depending on the energy with which the blood is propelled by the heart. Hardness in the pulse is a measure of arterial tension, softness of feeble pressure; frequency indicates excited action, infrequency that the action is languid.

If the distending wave be full, loaded, and sluggish, the cavities of the heart are generally congested, and when, with these, the accompanying vibratile shock is unduly sharp, a superadded irritability of the ventricles, with a sudden and energetic closure of the aortic valves, is indicated. If the pause after the energetic vibration be prolonged, it argues slowness in the diastolic action of the ventricle.

An inadequately filled artery indicates a feeble and languid action of the ventricle or valvular insufficiencies.

If the vibratory beat be quick (*pulsus celer*) or, as Dr.

Sanderson proposes to call it, short, a rapid closure of the mitral valve, with quick first sound, is indicated. Instances of this are met with in anæmia, and after large hæmorrhages. If with the *pulsus celer* the pause be almost obliterated, so that to the short the frequent beat is also present, a rapid and irritable ventricular action may be assumed. This form of pulse occurs in acute rheumatism, and in pericarditis.

If the beat be slow (*tardus*) with rhythmic irregularity, there is generally some degeneration and weakness of the ventricular walls; but, should this slowness pass, at times, into syncope, there is probably fatty degeneration, and if all this be accompanied by frequency and feebleness, there is probably ventricular dilatation superadded to the fatty degeneration. If there be a true intermission (not rhythmic irregularity only), there is probably valvular incompleteness, or a failure of power in the systolic action.

An undulation in the systole or filling of the artery indicates failure of systolic power in the heart, or deficiency in the blood supply from regurgitant disease of the mitral valve, or of obstructive disease of the semilunar valves.

If the pulse be irregular, both in force and rhythm, at the same time small, short, and feeble with marked undulations after the beat, it is probable the artery is ill supplied with blood, and that this is due either to mitral regurgitation, or to aortic valve obstruction. Should the force be so varied as to have at times a few occasional strong beats, there is probably also some hypertrophy of the ventricle.

If the pulse be soft and undulating with thrill in the aortic region, it is more than probable aortic obstruction is the diseased condition, and if small and prolonged, this aortic obstruction is accompanied with hypertrophy.

If the pulse be weak during diastole, giving the impression of an emptied vessel, while the act of filling is abrupt, short, jerking, with the undulations of a prolonged wave, and giving the impression of a full vessel, there probably exists an insufficiency in the aortic valve, and this is rendered more evident if the pulse be only a vein wave, and is devoid of vibratory shock.

The prognosis is confirmed if the pulsation of the superficial arteries exhibit an active forward movement in the direction of their axis, but which becomes diverted, where the vessels are curved, into a vermicular or peristaltic action.

The dicrotous, or rebounding pulse, and which the older physicians deemed significant of approaching hæmorrhages, is an exaggeration of the normal elastic moving power of the artery, and is essentially due to disturbed oscillation in the current of blood from want of relation between heart force and arterial elasticity; hence it may occur where there is aortic deficiency, or where the arteries are deficient in vaso-motor power, or where the venous system and capillaries are loaded.

A prolonged, lagging, vibratory pulse, with thrill, indicates feebleness and venous obstruction, with, perhaps, dilated right ventricle. It is not infrequently observed in old persons.

The preceding brief survey of the sounds and murmurs, and of the rhythmical action, of the heart, shadows forth their value in the diagnosis of disease; while the end and aim of the following pages are to ascertain the practical bearing, which these, in conjunction with a general physical examination of the heart and associated circumstances, afford us towards determining not only the nature of its diseases but their fatal tendencies, or the probabilities of cure or alleviation.

The grave difficulty of the imperfectness whether of accurate observation, or of its correct rendering, immediately and prominently, presents itself. Doubtless the sounds and physical indications, which, in modern phraseology, may be termed the objective qualities, are defined and true to themselves, not so the subjective qualities of the observer, for these vary not only in different individuals, but also in each individual at different periods of his professional life. The sounds emanating from the heart may be simple, and must be defined and truthful; but the ear that listens to them may be imperfect. Supposing, however, this difficulty to be surmounted, and the sounds emitted are equally recognised by all, there remains the further difficulty that the lesson to be learned from them is limited and incon-

clusive; and then we yet further learn that correct diagnosis can only be arrived at by a consideration of other elements.

But if this be their limited use in diagnosis, in prognosis it becomes still more limited; here the unaided sounds and murmurs emitted by the heart, are anything but conclusive indicators of results;—there being many diseases of this organ of a most fatal tendency, in which their morbid conditions are only faintly present, or entirely absent; while some, in which they loudly present themselves, are not so. It will be seen that other actions, vital and mechanical, must necessarily be duly considered; so that, while pointing out the diagnostic value of the sounds and murmurs, as regards disease and its consequences, it will also be necessary to show that, in themselves and without the aid of other concomitant symptoms, they cannot be relied on as the sure and certain indicators of the precise condition of diseased structure, or of its relative gravity and fatality.

CHAPTER VII.

ERRORS IN THE IMPULSE OF THE HEART.

THE abnormal conditions of the functions of the heart which prominently offer themselves for observation may be classified as (1) errors of impulse; (2) errors of rhythm; (3) a departure from the heart's usual or normal sounds; and (4) the occurrence of pain. These abnormal conditions may exist separately or collectively, and may be due either to organic disease or to functional disorder; now indicative of fatal tendencies, now of only a passing disturbance in the animal economy. In order to arrive at a correct diagnosis and a safe method of treatment in these cases, it is absolutely necessary to fully, nicely, and accurately appreciate these several and special indications.

It is of the first importance to ascertain if these abnormal symptoms be due to, or in any way associated with, organic lesion. Seeing the book-certainty with which organic disease and its distinctive features are described, it might be assumed that this is a matter of no great difficulty. One of the most experienced observers of diseases of the heart, indeed says:—"The facility of making a correct diagnosis between functional and organic diseases of the heart is not so great as modern writers lead us to believe; and we more often arrive at a just conclusion by instinctive skill, the result of experience and judgment, than by communicable rules of diagnosis."—(Stokes "On Diseases of the Heart," p. 495.)

In the above, as it appears to me, Dr. Stokes begs the whole question of the difficulty of diagnosis in these cases; the accurate judgment of a man of vast experience being made to

supersede the deficiency, to the inexperienced, of definite or even suggestive rules. There really is, however, great difficulty, not only in describing the means towards a correct diagnosis between functional and organic diseases of the heart, but the diagnosis itself, even to the most practised and able observers, is too often a conclusion not easily to be arrived at; and this Dr. Stokes himself, in a preceding page, fully acknowledges.

Supposing, however, we can positively and satisfactorily eliminate all suspicion of organic lesion, it then becomes necessary to ascertain the immediate cause of the functional disturbances exhibited, paying especial attention to such concomitant disorders of the general health as may present themselves. These may not only be the exciting cause of the disordered action, but may have important influences on the future condition of the heart. There remain, in fact, for anxious investigation, the origin and the probable consequences of these functional disorders, with the indications of cure.

In order to arrive at something like rules for our guidance towards the true appreciation and management of these functional disorders, the several prominent symptoms of diseased action, in relation both to organic disease as well as to these latter, must be carefully studied and appreciated.

Amongst the prominent and very frequently occurring indications of disorder of the heart, disturbances in the force of its normal impulse are to be enumerated. Its force may be diminished or increased. Either of these conditions may be associated with organic disease or with functional disorder only.

The circumstances attendant on, and giving importance to, the condition of a diminished impulse, may conveniently be first considered. A diminished impulse, varying from that which is slightly below the ordinary standard to that which is virtually uncommunicable to the observer, whether by eye or hand, is at times met with; and the due importance of this it will be necessary to appreciate, together with those other

symptoms with which it is found to be associated. While we feel that, in many cases, a diminished impulse is consistent with the enjoyment of good health and long life; still it is a condition so often associated with debility in the structure of the heart itself, or with some other important disorder or lesion of the general system, that the study of this symptom, and its several associations and probable consequences, is worthy of the physician.

The circumstances whereby a patient's attention is ordinarily arrested when there is the presence of a diminished impulse, are the occurrence of (1) breathlessness on slight exertion, with (2) a feeling of debility, amounting at times to a realized inability to do that which otherwise would be within the usual compass of his power; and this may be associated with (3) the occurrence of occasional faintness, even to actual fainting. On more minute inquiry, we may find, on examination, that there is a diminished (systolic) impulse; and we naturally first seek to find if the heart itself present any objective physical causes for such weakened condition. Is it diminished in size? Is it enlarged? Are its sounds less distinct or more pronounced? The personal condition of the patient is here to be considered, as, in a chest well clothed with fat, but little impulse may be immediately perceptible, though the heart be normal; while in very thin persons, though the impulse is really diminished, the thinness of the walls so far permits it to be felt as may at first induce the idea that its contractile action is not enfeebled.

Due regard being given to these personal conditions, we may, having ascertained that there is a diminished systolic impulse, find it to be associated with (1) a normal condition of the heart as regards size; or (2) a diminished or (3) an enlarged condition; or it may be associated, in addition to either of the above conditions, with diminished or increased valvular sounds, or irregularities in rhythm, or with other indications of heart affection; or there may be no indications of a structurally altered heart, but disease in con-

tiguous or other organs influencing the just recognition of the impelling power.

Having duly appreciated the existence and the amount of the diminished impulse, and also the objective symptoms presented by the heart itself, it is necessary to weigh how far the former is dependent on the latter. We must also consider such other remote causes as may exist. Before pronouncing a diminished impulse to be due solely to a passing weakness, it is necessary to carefully eliminate each and all of those associated conditions which indicate it to be due to permanent disease. When this is done, and not till then, are we competent to assume the diminished impulse to be functional only, and such as may be so ministered to as to procure ultimate recovery.

It may be assumed that a diminished impulse is immediately due to a deficiency in the contractile agencies of the heart; and that this deficiency may be in the nervous influences solely, or may be owing to permanent physical incapacities. Of the latter, we have illustration in several structural diseases of the heart; of the former, in circumstances, whether passing or permanent, of great physical exhaustion, such as on the occasion of shock, of the depressing influences of certain specific medicines, and of adynamic fevers and other diseases, more especially of the spinal cord and brain-tissue.

Before considering the purely nervous causes of diminished impulse, we will briefly pass in review the circumstances attending this disturbance in the functions of the heart in its connexion with some of the more important structural affections with which this condition is frequently associated, so as the better to appreciate the *rationale* of the influences which produce it, not only in these affections, but in those, also, of a purely nervous origin. The most important structural affections, with which diminished impulse is associated, are characterized by a condition of the heart whereby the dulness on percussion in the præcordial region is increased. The physical conditions of the heart in connexion with this indication may, in general terms, be referable to (1) dilatation;

(2) dilatation with hypertrophy; (3) fatty deposits; (4) fatty degeneration; (5) deposits in the pericardium; (6) deposits in the pleura. After investigating the distinctive features of these conditions, it may be useful to note such of these as may serve to elucidate the *rationale* of the deficiency in impulse exhibited in each; and also the importance of this deficiency in relation to prognosis and treatment.

In the class of cases now to be considered, those which are associated with, or dependent on, valvular disease will be excluded; observation being limited solely to such as have an independent origin. Nevertheless, we may have to consider cases where the acoustic evidence of mitral murmur is present; this being due, not to valvular disease, but to the amount of dilatation, generally without hypertrophy, rendering the valves insufficient, and thus permitting a regurgitant murmur.

When the impulse is feeble, and there is obviously, on examination, an increase in the area occupied by the heart, as indicated by increased dulness, or by the impulse being diffused, we may assume, without going into an elaborate discussion of the various circumstances connected with contiguous disease or displacements by other organs which may modify the diagnosis, that there is either enlargement of the heart itself, or that there exist abnormal deposits in the pericardium or pleura; and, save when the enlargement is due to fatty deposit, or to hypertrophy with softening, general or specific, we may also assume, if the sounds be louder than normal, that the enlargement is due to dilatation and thinning of the walls of the ventricles.

We will now refer more in detail to the heart-symptoms which may characterize each of these conditions.

When, with the dulness on percussion extending abnormally in a lateral direction, the impulse is deficient in force, and at times slightly undulatory in character, yet diffused over an extended area, and this area, like the dulness on percussion, is extended laterally, but is not to be felt either in the back

or below the right shoulder-blade; when, while the apex-beat is apparently seated towards the sternum, its shock is found to be, though diminished and indistinct, to the left of the nipple, and below the fifth interspace, and its shock, at this point, not more marked or appreciable than over the general area of the heart's position, and the rhythm, though sometimes regular, for the most part disturbed, the pulse being, at the same time, weak and intermitting; and when the valvular sounds, especially the systolic, are somewhat sharp, clear, and heightened in pitch,—when this series of phenomena present themselves, we may infer that there is a generally dilated condition of the heart, the muscular structure of which is neither flabby in texture nor softened by infiltration or fatty degeneration; and we may also infer the absence of hypertrophy.

Sometimes, however, the above-named conditions are qualified by a persistent rhythmical irregularity, with the occasional occurrence of the first sound being absorbed in a murmur; we may then infer that dilatation has proceeded so far as to render some of the valves insufficient. The tricuspid, and then the mitral, are the chief seats of this extended dilatation; should, in extreme cases, the aortic valves also become thus inefficient, the rhythmical action of the heart and the irregularities in the pulse become very marked, while indistinguishable murmurs take the place of the valvular sounds. We may perhaps also infer, when the apparent impulse projects unduly towards the sternum, while both sounds are heard sharp and equal, and sufficiently pervading to be appreciated below the right shoulder-blade, and, at the same time, there is no marked percussion-dulness over the sternum, that the left ventricle is unduly dilated; and we may also infer, if, in addition to great rhythmical disturbance, the jugular veins be unduly distended and present the appearance of an undulating movement, that dilatation of the right ventricle exists; and if there be no appreciable abnormal dulness on percussion, we may also conclude that the dilatation is confined to the right ventricle; while if the left ventricle be dilated, we may conclude that the right participates in the like condition.

In all the above cases, we infer the presence of dilatation

without special structural degeneration of the parietes of the ventricles. This dilatation, for the most part, exists in both ventricles; but, as just observed, sometimes the right side is solely so affected. As a general rule, the diffusion of the impulse is according to the ratio of the area of the dilatation; while the sharpness and intensity of the sounds are proportionately increased by the thinning of the parietes.

In addition to the above general indications of a dilated condition of the heart, it may also occasionally be noticed that the dulness is projected more to the left, especially towards the base, and that this dulness is apparently superficial, and to be detected by very slight percussion; at the same time the impulse, though weak, is persistently irregular, and presents the character of an abnormal sharpness with quickness, and is generally reduplicated. It thus gives the impression of a vibration or rapid undulation rather than of a distinct beat. The valvular sounds are also very indistinct, and are thus, though sharp and sounding, very difficult to define and separate. The whole of these phenomena appear to be superficial, and to have their seat immediately beneath the surface of the parietes of the chest. Under these circumstances, we infer the heart to be not only thinned and dilated, but to be, in addition, adherent generally to the pericardium.

We now pass on to the consideration of another series of cases, which, besides being characterized by a weak and enfeebled impulse, and by an abnormal extension of dulness on percussion, presents, as regards the character of the sounds, a somewhat different class of symptoms. Besides the abnormal amount of dulness, the impulse is found to be not only weak, dull, and generally diffused, but presents an undulating character, the apex beat being slight, or scarcely recognisable; if it be recognisable, it will be observed to be below its normal position in the fifth interspace. The valvular sounds are without their wonted clearness, the first being weak, thin, and toneless, but of a somewhat high pitch. With such a series of symptoms it may be inferred, though there is dilatation, that this is associated with weakened parietes, due either to softening of the muscular struc-

ture, to atrophy, to fatty infiltration, or to fatty degeneration, but without hypertrophy. Should there, however, be in addition to the weakened shock and the increased amount of dulness, an impulse markedly irregular both in force and rhythm—being sometimes inappreciable and then presenting an undulatory character; whilst the pulse at the wrist, though partaking of these characteristics, is yet found not to be in unison with the heart's impulse; and should the valvular sounds also present similar inequalities, at one time being obtuse and nearly inaudible, at another occurring with more distinctness and with a flapping character, the first sound being always weak and toneless, the second weak and thin, but neither being accompanied by any murmur, we must infer softening with hypertrophy. If the dulness on percussion be obviously projected more towards the right side, and the lessened impulse, though not markedly irregular, be slow, while the valvular sounds are sharp and defined, the probability is that the heart is the subject of fatty deposit, but not necessarily dilated. Should the impulse be not only weak but extended, and, as it were, diffused horizontally, and having occasionally its force so far increased as to present, or rather to be accompanied by, a shock, and which shock, though thus perceptible, can scarcely be referred to any distinct point or localization; and in either case, whether there be shock or not, the impulse is both more gradual in development and slower in repetition than is natural, the valvular sounds being at the same time limited in area, dull, and somewhat prolonged, the first sound being disproportionately diminished in tone, the second weak, but on the occurrence of shock, flapping—we may, under such circumstances, infer dilatation with hypertrophy, but with sides weakened by fatty infiltration.

There are other important forms of heart disease which are characterized by a weak impulse; but as these are not associated with an increased area on percussion, and cannot, therefore, be referred to the same category of disordered action, we may, before proceeding to their consideration, pause to review those that have been enumerated in their more extended relations to

the animal economy. These forms comprise debility of muscular structure with dilatation, without and with hypertrophy, or with fatty infiltration or fatty degeneration, and will be found to include a tolerably well defined and compact group, presenting much the same general symptoms.

The general symptoms immediately connected with these several forms of heart disease are mainly (1) disturbance, occasional or permanent, in the respiratory functions; (2) debility on exertion; (3) occasional feelings of faintness, with (4) præcordial anxieties, flutterings, palpitation, and may be, pain.

The dyspnœa, or shortness of breathing, occasionally comes on spontaneously and without apparent cause, but is usually easily induced on bodily exertion, by shocks of the nervous system, and by mental exhaustion; at times this symptom may be so slight as to be scarcely appreciable; at others, passing into a laboured breathing, and even at times to a temporary suspension of respiratory action. In some cases, these phenomena of the respiration are gradually developed to an extreme condition, and then, when perhaps life appears almost extinct, as gradually recovered from.

A common and very characteristic symptom accompanying a heart exhibiting a deficiency of power in its impulse is the manifest debility on exertion. This, which may not be associated with some other affections of the heart, is ever present in the whole of the class now under consideration. There is a peculiar leg weariness and feebleness of power, especially referred to the knees.

The heart itself experiences flutterings rather than palpitations, and these flutterings are often much determined by the state of the stomach. They are not infrequently relieved by the evolving from the stomach a small amount of flatus. The relief from this is so instantaneous and effectual that the patient is apt to think that the flatulence is the sole cause of his difficulties. From the frequency with which this generation of wind in the stomach is associated with these low forms of heart disease, it is probably induced by them. Occasionally its presence induces discomfort, passing into pain.

For the most part, though the stomach feels sinking, as from

exhaustion and want of food, the appetite is feeble, and anything like heavy food or a full meal induces cardiac distress. The stomach requires frequent supplies of a light and easily digestible food, and not infrequently there is a desire for stimulants in small but repeated quantities.

The pulse, though presenting no uniform nor distinctly diagnostic character, yet more often than otherwise is feeble, soft, and small, often dicrotic and irregular in rhythm, especially after exertion. It is generally lower in frequency than is natural to the individual; and in some rare cases falls so low as to be less than half its normal amount. I have known it occasionally to fall to twenty-six beats in the minute. For the most part this condition of the pulse is associated with a shortened first sound—at times, indeed, to its entire suppression, so that the second sound only is to be heard. The surface of the skin has a tendency to coldness and pallor; the gradual absorption of the red tints of the face, and the assumption of a doughy whiteness, is very marked. The lips, too, partake of these changes.

In advanced cases, where the distress from feebleness in the systemic heart is added to by congestions of the liver and portal system, there occur hæmorrhages from piles, and occasionally large and alarming amounts of blood are voided by epistaxis. Still, experience shows that, under these circumstances, the patient rarely succumbs; these hæmorrhages cease voluntarily, and apparently afford relief. In some extreme cases, the attack is only brought to a conclusion by the occurrence of rigor, and the faintness and insensibility prevail for a time; life is almost invariably preserved. Œdema of the legs occurs, but only in advanced cases. During the whole course of an enfeebled heart there may occur feelings of giddiness, with a tendency to faintness; these feelings fill the patient with alarm, and he expresses himself as nervously conscious of their importance. When the portal system also becomes unduly loaded, the giddiness may pass into an attack of a much more serious character, amounting at times to pseudo-epileptic or even pseudo-apopletic fits. These attacks are often preceded by an "aura" referred to the stomach or bowels, and are probably associated

with some form of indigestion or generation of wind as before referred to.

Besides these symptoms, and which may be considered as proper to, and symptomatic of, that condition of the heart in which the impulse is abnormally deficient, there often occur affections of other organs, and which may also be assumed to be consequent to, and dependent upon, this condition. These are chiefly congestions of the liver and kidneys, but sometimes inducing from these latter the secretion of a pale limpid urine in large quantities, congestions also of the lungs, with occasional attacks of bronchitis, and eventually œdema of the lower extremity.

When the weakened impulse is accompanied by hypertrophy, associated with fatty deposits and infiltration, or with fatty metamorphosis, more especially the two latter, a distressing form of hypochondriasis is occasionally met with. If not depending on these conditions of the heart, it is intimately associated with them. The hypochondriasis is recurrent in its nature, characterized notably by want of sleep; and, if sleep be procured, not necessarily relieved by it. There is a craving for frequent food; great anxiety for relief during the paroxysms, which are prominently marked by fears of an instant dissolution. During the paroxysms, the unhappy patient is almost entirely unable to divert his thoughts from his own morbid feelings.

The physical condition of the heart whereby this series of symptoms is induced is primarily and essentially that of weakness in its muscular fibres. These are found to be flaccid, absorbed, or degenerated; and in some instances fatty matter is interposed. This latter, while inducing weakness of the fibrous structure by absorption and alteration by accumulation, impedes the action of the heart itself. The fatty matter is essentially a deposit, and whether deposited on the outside or within the heart, or infiltrated between its fibres, consists of fat contained in separate and distinct cells.

Debilities of the heart, such as have been now described, may be the process of years, or they may be the prompt result of recent inflammations, having their seat in the structure of the

heart itself or surrounding membrane. They are not infrequently associated with the gouty habit, and are often the immediate result of a gouty metastasis.

The depraved physical condition of the heart in these cases evidently accounts for the deficiency in the power of the impulse, so that, though there may, in some, be an increase of size, the heart has no longer the power to close, with vigour, on the blood it has to propel; the necessary dynamic force is wanting; and to this we must look for the *rationale* of the essential symptoms. It accounts for the impulse being so deficient in force that the external evidence of the systolic contraction is barely appreciable, whether by sight or touch. It also accounts for the fact that the valvular sounds are not so pronounced and defined as in a vigorous organ; as also for the irregularity and dicrotism in the pulse, and for those manifestations of the often-recurring sensations of general weakness and faintness consequent on exertion.

The general increase in the parietes of an enfeebled heart by dilatation or by fatty deposit, explains the diffusion of the impulse, though its force be diminished. Dilatation, moderate in amount, and without increase in the thickness of the parietes, accounts not only for the louder tone of the sounds then heard, but for their being sharp and clear. The column of blood which the several valves have to act upon is, in this case, rather increased than diminished, while the dilated walls communicate more readily to the ear the sounds thus induced. An increase in this simple form of dilatation accounts for the relative diminution of the first sound. The dynamic power of the enfeebled ventricle is unequal to closing with energy on its distending amount of blood—hence the auriculo-ventricular valves, in the exercise of their function of preventing regurgitation, do not promptly and forcibly close, and hence the normal amount of vibration is failed to be produced. The second sound is not interfered with to the same extent. On the occurrence of the diastole of the ventricle, the semilunar valves immediately and effectually close, and, as the column of blood is ample, the integrity of this sound is not very markedly interfered with. Should the ventricular

dilatation increase, the mitral valve becomes, to a certain extent, inoperative, and there is heard a faint and prolonged regurgitant murmur; and if this ventricular dilatation still further increase, the semilunar valves also become inoperative, and there is not only murmur instead of the second sound, but the rhythm of the column of blood is interfered with, so that an uncertain soft and undulating pulse is felt in the artery at the wrist.

The prognosis in each of the cases belonging to this category of disease is eminently unfavourable. For the most part, whether dependent on congenital weakness, or due to accidental sources of development, they insidiously progress and assume a chronic and incurable form. Unless the most judicious precautions be adopted they advance to the impairment of power, and eventually to the destruction of life.

It is, perhaps, doubtful whether the progress of disease in these cases is not inevitable, and all that we can hope to do is, by a careful prophylaxis, to prevent, not the inevitable advance, but its undue acceleration.

Of all the diseases of the heart, it is in this class that death pre-eminently takes place suddenly. With structural debility in the heart itself, there is associated the not infrequent tendency to sensations of a passing faintness. Any over-fatigue or undue mental shock or exhaustion, or indiscretion in diet, may not only induce these, but determine the occurrence of a perfect fainting fit, with hazard—more often consummated than not—of causing the immediate cessation of life itself.

The treatment is mainly prophylactic, may be slightly remedial, but can in no case be esteemed to be curative. The avoidance of all exhaustion—mental and bodily—of sudden physical exertions and mental shocks, of intemperance in diet, and of the depressing influences of cold, are the main means to be observed; at the same time the general health should be sustained by light nourishing food and the specific tendency to faintness by the occasional use of suitable stimulants and restoratives. As symptoms arise and require, the use of ferruginous tonics, sedatives and antispasmodics, may be adopted.

In addition to the above instances of debility of impulse in conjunction with an abnormal extension of dulness, there is yet another class of apparently similar cases to be considered. This class comprises those cases whereby an abnormal dulness is induced by physical complications external, but contiguous, to the heart—such as morbid deposits in the pericardium and pleura, the occurrence of tumours, etc. In these cases, though there be a diminished impulse associated with an abnormal dulness on percussion, it does not necessarily follow there is disease in the heart itself, or even that there is really a failure of impulse; it may be only masked. It is, therefore, necessary to ascertain if any of these sources of interference with the due appreciation of the abnormal impulse exist. In cases of pericardial effusion, though the heart's impulse be really not impaired, it may be so masked as in some cases to be scarcely appreciable, or even to be rendered completely and entirely imperceptible. This occurs more especially in the course of passive effusions rather than from effusions caused by active inflammation.

In these cases of pericardial effusion, the general indications of this diseased condition must be sought for. The cardiac region may be arched forward, and the intercostal spaces be obliterated by the bulging outwards of the integuments; and perhaps the characteristics of an œdema may be presented. The apex-beat, slight though it be, may be seen raised so as to range with a line even with the nipple. The hand laid over the surface discovers little or no movement; if there be any, it is weak and of varying force, but not undulatory. The undulatory movement is rather the accompaniment of dilatation of the cavaties of the heart itself, especially of the right ventricle, when occurring without effusion.

The dulness on percussion is very much more marked in these cases of effusion than in the cases previously referred to. It is also widened in area, and may even range above the right rib. It has, as Dr. Walshe points out, a tolerably uniform pyramidal shape; its base ranging with the sixth, rarely with the seventh rib, and extending its apex upwards, according to the extent of the fluid. At the apex the sounds are often nearly, if not

entirely, imperceptible. Should the first sound be heard, it will be weak and uncertain, with a muffled character, and anticipates the feeble indication of the impulse. At the base, the sounds, though they be faint, are audible and distinguishable: the first sound is here certainly louder than it is over the apex or the ventricle; the second sound is audible through the course of the arch of the aorta. Besides the above, there may also be the more specific indications of the existence of serous inflammation of the pericardium, by the presence of friction-sounds, and of the existence of fluid by the presence of ægophony at the edge of the dulness.

The impulse of the heart may also be obscured by an empyema, by pleuritic effusions, by deposits (solid and fluid) in the mediastinum, and by other contiguous tumours; but these are all causes which obviously present themselves for observation, and consideration, on account of their own specific lesions, rather than in relation to the accompanying condition of a diminished impulse.

We will now consider the existence of a diminished impulse under entirely different conditions. An impulse, feeble and very limited in extent, sometimes exists with an evidently very deficient area of percussion-dulness, both superficial and deep, and this evidently without any emphysema or other cause to interfere with the correctly ascertaining that the size of the heart is abnormally diminished. The cardiac sounds are everywhere at a minimum; but, if heard, may be a little sharper in tone than natural; the pulse is small and deficient in force. This state of things indicates a condition of muscular atrophy. If there be frequency and some palpitation in the heart's action, it is probably due to the atrophy being caused by, or associated with, a generally depraved condition of the blood, as in tubercular degenerations or carcinoma. In these cases the heart is not only diminished in size, but its fibres are degenerated, being pale, soft, and deficient in firmness. This may be simply caused by an anæmic state of the viscus, but it is more commonly due to a fatty degeneration of the fibres, and which is evidenced,

not by the deposit of separate fatty masses, but by the heart exhibiting both on its external and internal surfaces the appearance of buffy spots. Dr. Quain called attention to this subject in a paper published in the "Medico-Chirurgical Transactions" in 1850. In the Lumleian Lectures, 1872, he summarizes the distinctive character of this fatty metamorphosis by stating that "the tissue of the heart is paler than natural, light brown or buff in tint, the altered colour being most marked in spots and mottlings. The consistence is greatly diminished; sometimes it breaks down under pressure as would a lung consolidated by pneumonia." It is often greasy to the touch. It may affect part or the whole of the organ. In advanced cases the whole area is occupied by granules and fat globules, but not such fat as is found on the surface or between the fibres, since the globules are smaller, appear to have a mere albuminous envelope, and are extremely like the oil globules of milk. The diseased fibres appear friable and break up readily into small fragments. This change is found most frequently in the walls of the left ventricle, next in those of the right ventricle, then in the right auricle, and, least frequently, in the left auricle. It is generally more evident in the columnæ carneæ and inner layer of the muscular fibres than elsewhere. Dr. Quain's opinion is that the fatty matter is the result of a chemical and physical change in the composition of the muscular tissue itself, independently of those processes which we call vital. This fatty degeneration not only causes weakness, by its metamorphosis of fibre into fat, but may also, in some rare instances, cause diminution in bulk, though its general condition is, as previously stated, in association with hypertrophy. Atrophy of the heart then may be simple, as seen in phthisis and carcinoma, or in diseases where there is a general wasting of the muscular structure of the body; or it may be due to the specific wasting associated with pericardial adhesions—occurring not infrequently after the acute form of pericarditis; or it may be due to the specific metamorphosis of the fibres. These conditions of the heart, now weakened in power and diminished in bulk, offer important and fatal indications.

It is not easy in these several cases of anæmic or of simple muscular atrophy, or of fatty metamorphosis, to discriminate one from the other solely by the physical phenomena exhibited by the heart. We must seek to determine the precise form of affection rather from the general symptoms; but these, with their etiology, had better be referred to when considering the morbid states of the heart in the category of structural diseases next to be described, which comprises the morbid structural changes specified above, but unaccompanied by any diminution in the heart's bulk.

This category includes the very important range of cases, as regards diagnosis, in which the heart, though the impulse be feeble, is not enlarged, nor involved in any surrounding or contiguous dulness, in which the indications are that the heart, though weak, is in fact of the normal size. These cases may be due to structural, or only to purely dynamic, causes. Between these two classes of cases it will be well to discriminate, and then to find out in the latter where we may anticipate neither danger nor cause for alarm; *i.e.*, the existence of a normal condition, or of functional debility only. The character of the impulse, when the heart is feeble and small in size, has just been described. Should this character be well marked, though there be no indications of a want of proper bulk, and there be, in addition, an impulse always reduced in strength, and only very rarely exhibiting a visible *ictus*, and this rather having the appearance of an undulation than of a beat; while the sounds, the first being sharp and flapping, the second very thin, are generally very weak and toneless, and entirely free from any complication of murmur; with a pulse irregular in force or rhythm, but for the most part slow, we may surmise there is muscular weakness. Should the pulse be rather fast than otherwise, this weakness is then probably due to a state of anæmia. If, under these circumstances, the impulse of the heart, though generally feeble, be occasionally characterized by a forcible beat, it is very probable these conditions are associated with a concentric hypertrophy of weakened tissue. Should we, however, find these specific indications of muscular

or anæmic weakness greatly exaggerated, so that there is a variable impulse, always feeble, generally retarded, but with the character of a fluttering unsteadiness, indicative of great variability both in force and rhythm—at times even jerking and abrupt—and with the heart, as it were, largely projected forwards; the first sound flapping, short, weak, and toneless, and then obtuse and dull; the second weak and thin, no murmur; no jugular pulsation; the pulse at the same time weak and irregular, with loss of power, may be markedly slow, and little or no tendency to general or local arterial or venous congestion; if with all these the apex-beat, though difficult to define, be yet more distinct than in the cases of simple muscular or anæmic weakness, we may infer the presence of debility from fatty metamorphosis. Hence we see that in muscular weakness, there are certain indications; in anæmic weakness other indications; and in fatty metamorphosis others are superadded.

The general symptoms characterizing the two categories of disease above referred to—viz., debility of impulse, with diminished or with normal size—are those of physical weakness and easily-induced breathlessness. In muscular atrophy, the feeling of weakness is immediately experienced on the exercise of any undue exertion; and the patient, for the most part, shows a disinclination to submit himself to the test. In anæmia, there is a more general depression of vital force, and the breathlessness is more marked and more persistent than in simple muscular weakness. It is essentially a disease of young life, and associated with the nervous and hysterical temperaments, and is met with more commonly in females than in males.

By far the greater proportion, however, of the cases of weak impulse are due to a fatty degeneration or metamorphosis of the muscular fibres; and this, unlike that of anæmia, is rather a disease of males than of females, and in them occurring at a more advanced period of life—being rarely met with in those under forty years of age. Fatty metamorphosis is more a disease of the labouring than of the wealthy classes. This, probably, is

to be explained, as Dr. Quain suggests, by the causative conditions being both general and local. The former comprising impaired nutrition, starvation, extreme anæmia, phthisis, cancer, and acute adynamic diseases; the latter diseases of the arteries, especially the coronary, endocarditis and pericarditis. The particular form of fatty metamorphosis, now referred to—viz., that without hypertrophy or dilatation—is, however, more often met with in those whose occupations are not laborious. This probably is due to the heart, if predisposed to this form of degeneration, not being excited, by severe muscular exertion, to take on the conditions of hypertrophy or dilatation; but in whatever class it may occur there will be found associated with it a pale flabby skin, indicative of the constitutional tendency. When a state of fatty metamorphosis of the heart exists, the countenance is usually pale and sallow; sometimes the lips are livid, generally deficient in the natural colour; the tissues are soft, the manner is languid, the temper is apt to be dejected, and there is a sadness and melancholy foreboding not commonly met with in many other affections of the heart; muscular power is deficient and soon exhausted, and the effort to exertion is not very promptly aroused; there is evidently a depression of vital force; the appetite is feeble, digestion is weak and flatulent; there is not infrequently giddiness, headache, and syncope, and may be, coma. In advanced cases, there is marked uneasiness across the loins, and the feet and ankles show slight œdema; uneasiness of the heart, at times passing into pain, often supervenes, with occasional sensations of a palpitation, or rather flutter; and if this increase is often accompanied by a choking feeling, somewhat of the nature of a globus hystericus.

The respiration is irregular, often hurried, at others prolonged with not infrequent sighing. When the right ventricle is prominently the seat of weakness, the respiration may present peculiar characters, as described by Dr. Cheyne ("Dublin Hospital Reports," vol. ii. p. 217), in which a protracted period of apnœa is slowly recovered from to be followed by a few hurried respirations. The apnœa, as shown by Dr. Reid, is associated with an increased action of the heart, as evidenced

by acceleration of the pulse, followed by remissions during the hurry of the breathing. In the case described by Dr. Cheyne, the several phenomena occupied about a minute, and occurred in a person greatly diseased. It is probably a symptom solely due to very advanced disease. Dr. Stokes says (p. 324) he has never seen it except in cases of fatty degeneration. Assuming such to be the fact, it might be considered diagnostic of the existence of this form of disease. There are grounds, however, for considering it due to a neurosis of the vagus, and which neurosis may be determined by other diseases of the heart and great vessels emerging from it. Dr. Laycock has advanced this view, and gives cases confirmatory of it. An illustrative one in which this peculiar form of breathing was, in the final illness, developed to a remarkable degree had been under my careful observation for some twenty years. From early life this lady had been subject, on the slightest occasions, to distressing attacks of increased impulse of the heart. Naturally of a strong and even mind, during an attack the brain became disturbed, with heat and flushings of the face, so that the characteristics of her condition were those of a restless distress. The physical signs indicated weak and dilated walls of the right heart. The attacks of descending and ascending respiration frequently recurred during a period of six weeks, and invariably during sleep and sometimes during prolonged sleep; the respiration would very gradually subside into a perfect apnœa, and this often lasted for so long a period as to induce the conclusion she had breathed her last. Respiration was then gradually restored, the full breathing sometimes ending in a sigh or a yawn. She died at an advanced age.

In extreme cases the memory is often notably impaired; and mental efforts not only exhaust but induce irritability, and are not without risk to life. The tendency to feel faint becomes more marked, and is often accompanied with a distressing and alarming vertigo, and this vertigo sometimes heralds a more decided disturbance of the nervous system, so that something of the nature of a convulsion may take place. It does not present

the features of an epileptic convulsion, but rather of a violent spasmodic struggle for life.

It has been advanced by Mr. Canton that the appearance of the arcus senilis is concomitant with, and as such to be considered a symptom of, fatty metamorphosis of the heart. My own observation does not lead me to confirm this view. I have seen many cases of fatty degeneration where it has not occurred; while in many, especially amongst the aged, in which it has been strongly marked, there has obviously not been the least tendency to a fatty metamorphosis.

On comparing these general symptoms of an anæmic or fatty heart, accompanied with an enfeebled impulse, with those that characterize weakness of impulse, accompanied by evidences of dilatation, it will be seen that they are in many respects similar; still there are differences. In the anæmic and small fatty degenerated heart there is less evidence of distant local congestion; the lungs and the portal system are not so prone to be loaded, and hæmorrhages rarely or never occur; and œdema is only rarely, if at all, met with. The difficulties of breathing are also not so much the result of congestion as of the obvious nervous debility. There is also a more extreme state of general physical weakness; and this gives a distinctive quality to the occasional sensations of faintness and coma, and the less marked convulsive character of the attacks that occasionally supervene upon the occurrence of vertigo: and in the entire absence of valvular murmurs.

The proximate cause of the whole of the symptoms which characterize this class of heart disease must be found in deficiency of dynamic power, so that the heart is unable to effect other than a feebly expressed contraction on the blood which it has to propel ordinarily, and an utter incapacity to do anything beyond this without great general distress. If it be called on to do this excessive work its action immediately becomes hurried and irregular, and with the result of acquiring a still greater incapacity than belonged to it in its previously quiescent state. The remote cause is evidently alteration and

weakness of structure consequent upon an attenuated or degenerated condition of the muscular fibres.

The consideration of these anæmic, enfeebled, or degenerated forms of heart disease is not only interesting but important, from their association with pre-existent diseases, and also as influencing other and more serious lesions in the heart itself. Doubtless, each of these forms of disease may be fatal in itself; but, for the most part, they are to be considered, in the former case, as aiding in the general depreciation of the powers of life; and, in the latter, as the forerunners of those other more advanced conditions of disease, as dilatation, hypertrophy, and perhaps valvular inefficiency. Simple muscular debility is more apt to pass into excentric hypertrophy, while fatty metamorphosis rather determines to simple, if not to concentric, hypertrophy, the one tending to dilatation, the other to deposit. Both of these lesions are, for the most part, slow in their progress, and in the course of events, may often threaten the termination of life; still, under favourable circumstances, they are consistent with prolonged life. For the most part muscular debility, as it proceeds to excentric dilatation, leads to visceral congestions, which apparently kill, rather than the original heart disease; but in the case of fatty metamorphosis, death is, for the most part, determined by dynamic failure, or even by rupture of the heart itself.

It is obvious, from the nature of the symptoms, and the anatomical condition of the heart which produces them, that the prophylactic management of these cases mainly consists in quiet, in the avoidance of all excitement, bodily and mental, while the system generally is sought to be improved and invigorated by fresh air and a generous diet.

The medical treatment, as indicated by the evident feebleness of the general system, and of the heart in particular, finds employment in light preparations of iron, in antispasmodics, and stimulants; the occasional use of mineral acids with digitalis is often most beneficial; and, at times, sedatives,

in the forms of opium, hyoscyamus, hydrocyanic acid, and chloroform, are useful. The associated attacks of vertigo or faintness must be met by the stronger restoratives of æther, ammonia, or brandy.

The symptoms of a deficient impulse, without evidence of any increased size in the volume of the heart, occasionally develop themselves in an active form, and suddenly; sometimes, as it were, idiopathically or as symptomatic of an inflammatory condition of the substance of the heart, or by way of a metastasis, or during the course of other diseases. Illustrations of these several sources of a deficient impulse are, occasionally, to be observed in the course of certain forms of idiopathic febrile conditions of the heart itself, in pericarditis, in gouty metastasis, and as a concomitant of typhus, typhoid, and scarlet fevers.

The physical examination of the heart in these cases, reveals, perhaps, little more than the negative evidence of a deficient impulse, while the valvular sounds are often very indistinct, or are merged without murmur into one feeble sharp sound; or the first sound, being lessened or even obliterated in the left heart, is more audible in the right. The only distinctive peculiarity is that, during the frequently recurring paroxysms of distress of heart, which for the most part accompany these affections, both impulse and sounds are almost or entirely suspended, while there is an increase of the urgent symptoms; the pulse is small and irregular, the breathing hurried, and pain and anxiety at the præcordia increased. This is peculiarly the case in idiopathic inflammations, and in gouty or other metastases. In these the frequent recurrence of paroxysms of varying duration and intensity is peculiarly a characteristic. In fevers this is also appreciable, but not to so marked an extent, as the remissions are not so readily recognisable. In suppressed gout, inflammation of heart tissue is accompanied by great anxiety and fear of dissolution, with inability to use the least physical exertion. It notably incites to fatty degeneration.

The rhythm of the heart's action in these cases is generally,

but not always, accelerated. In some case of idiopathic inflammation, and more especially where there is a gouty metastasis, it is occasionally slower than natural. The pulse is invariably small and thin.

In the examination of these latter cases by the stethoscope an obvious uneasiness is often induced when pressure is made over the region of the heart. There are reasons, however, for concluding that pain is not experienced in the heart itself, but in the surrounding tissues, which, sympathizing in the disordered state of this organ, are thus prompted to protect it, and render immediately obvious the necessity for its being unmolested and kept quiet.

The general symptoms in these cases of asthenic inflammation, whether idiopathic or due to a gouty metastasis, are characterized by præcordial anxiety; an uneasiness rather than pain; the complexion assumes a sordid or livid aspect, with occasional flushings. The countenance has a varying expression of distress; there is a small, thin, and subdued pulse, with restlessness both of mind and body, more especially of the latter; excessive faintness is induced in any but the recumbent position, and yet a restlessness induces the patient to seek change from it. To the patient these symptoms excite sensations of anxious alarm; to the experienced observer they do not fail to indicate the reality of the danger, and that the result may too often be fatal, and that rapidly; occurring often, as it were, suddenly, or on some very slight exertion.

The physical condition, which is the immediate cause of these symptoms, is probably due to a sudden nervous depression in the vital powers of the heart, caused by the asthenic forms of inflammation, to which, whether idiopathically or by metastasis, it has become subject. Probably no very notable changes in its structure can be detected; but should the fatal termination be postponed, evidences of a softened structure are usually to be observed.

When these symptoms present themselves in typhus and typhoid fever, they usually occur early after fever has fully developed itself, becoming appreciable, about the sixth day, by

a sudden and notable prostration of power. The loss of impulse, on physical examination, is first to be detected at the apex (Stokes, p. 377) and to the left side. Sometimes the impulse becomes inappreciable everywhere; occasionally, but not always, the valvular sounds are also inaudible. In favourable cases, after the expiration of eight days from the time these symptoms have set in,—*i.e.*, the fourteenth of the fever—the heart shows indications of rallying, by a return of the impulse, and then of the valvular sounds. Recovery is always first indicated by a restoration of the impulse; it is the first to fail, and the first to be restored. The pulse, during this period, is always small, weak, and greatly accelerated.

In all these cases the tonicity of the heart's power is rapidly lessened, or even destroyed; and this is due to softening of its fibre; so much so, as in some cases, to disintegration of its structure. No alteration in volume takes place, but the left ventricle, which is the essential seat of the diseased condition, becomes livid in hue, soft in texture, and with so little power of resistance as to receive the impression of the finger pressed upon it as if œdematous; in parts all trace of muscular fibre is obliterated, and a dark homogeneous-like looking structure takes its place, resembling somewhat the cortical portion of the kidney.

The treatment of the above two classes of cases is not identical. In continued fever, when the powers of the heart are thus impaired, sustained stimulation may be useful, but certainly not always so; for, frequently, notwithstanding the prevailing reliance on stimulants in these cases, they are not only uncongenial to the patient, but evidently foment the disease by adding to an injurious irritability. Milk, light animal drinks, etc., are more often suitable. Still some use of stimulants is, for the most part, required. In the cases of idiopathic asthenic inflammation of the heart, and in metastases from gout and other diseases, stimulants are not well borne, in fact their exhibition, more often than otherwise, both induces repetition of the paroxysms and an increase in the severity of the symptoms that characterize them. Sedatives, alkalis, with light drinks, appear the more appropriate remedies.

The more manifest examples of a deficient systolic impulse of the heart in connexion with physical disease have been now reviewed. These being eliminated from the inquiry, it remains to consider when the existence of a deficient impulse is dependent solely on functional disorder, and whether as such it have hostile tendencies, or may be considered as entirely free from them, affording assurance of danger neither to life, nor threatening future injury to the heart itself; and, finally, it remains to consider when such deficient impulse is proper to the individual, and consistent with a sound and healthy condition, present and prospective.

Those in whom a feeble impulse occurs as a functional disorder are, irrespective of sex, usually of the leuco-phlegmatic temperament. This feeble impulse is met with, in both sexes, chiefly amongst those whose distinctive mental quality is that of being unimpassioned, and who are constitutionally prone to that form of indigestion characterized by cold and clammy extremities. The physical signs, on examination of the heart, are very negative. The impulse, though weak, may, however, be excited by exertion to more powerful action, and even to throbbing, without any sensations of distress, although it be, as is most probable, accompanied by an acceleration of the breathing. When thus excited to unusual action, it will be found that the sounds are more audible and somewhat altered in quality—the first sound suggestive, rather than being, of a sharp ringing tone, while the second is prolonged, and perhaps reduplicated; still, they are less sonorous and distinct in weak impulse from dilatation.

The general symptoms marking this disordered condition are a flatulent dyspepsia, anorexia, perhaps a depraved appetite, a foul breath, with tendency to constipation; indifference to exertion, both mental and bodily; a general languor, with lowness of spirits, passing at times into a distressing despondency; shortness of breath upon exertion; and, in extreme cases, a marked liability to œdematous swellings, not only of the feet and ankles, but of the face and the person generally. Where this state occurs in females of the hysterical constitution, there may be superadded a tendency to faint—

a casualty which is somewhat unusual in these cases. This disordered condition is not in itself dangerous, and is certainly very amenable to general and medical treatment; nevertheless, the neglect of its indications may lead to grave physical complications. In its simple form, it is to be regarded as a blood-disease rather than an affection of the heart.

Exercise, both of the mind and body, though at the time it may seem to increase some of the symptoms, by inducing exhaustion, is for the most part beneficial, and should always be enjoined. The dyspeptic tendencies must be sedulously counteracted by a well-regulated diet, with the occasional exhibition of such ferruginous tonics, and aloetic and other deobstruents, as the system will bear.

Where a feeble impulse appears to be the normal condition of the individual, it will generally be met with — perhaps more frequently in females than in males—in those of slight make, rather above than below the average height, having good but moderate appetites; and, though not robust, in the enjoyment of a moderately good state of health. They do not appear generally capable of great mental or physical efforts, and they rarely or never engage in violent or prolonged exertion—the quiet regular walk being esteemed to be exercise sufficient. If by any unaccustomed effort the heart's action be accelerated, it is unaccompanied by any very continued, exhausting, or painful disturbance of the breathing, and there is certainly no undue tendency to fainting. They usually present fair mental qualities, with remarkably easy even tempers, not readily roused to excitement; of good but quiet tastes, being æsthetically, rather than animally, disposed, finding their pleasures in the enjoyment of scenery and the cultivation of the fine arts—in books, music, or painting. They are often good instrumental musicians: they pass through life amiably and agreeably, without ambitions, and neither achieve nor even plan any great designs.

In reconsidering the preceding illustrations of a diminished and feeble impulse can we feel, without being too didactic, that approach has been made, by aid of the physical indications, to

the accurately and surely distinguishing that which depends on diseased and urgent conditions from that which has its origin in natural and harmless causes. In the former it has been shown there are not only the signs of the diseased conditions themselves, but for the most part the more the heart is thus impaired, whether this be from dilatation, from muscular weakness, from fatty infiltration, or from fatty metamorphosis, the more marked is the weakness of the impulse, while the sounds are, as the case may be, altered in character; they may be muffled almost to extinction, or they may be more sonorous and more distinct than is proper to the normal heart. In the latter, the heart has its normal position and dimensions, and though the impulse be feeble and the sounds partake of this character, they are natural in tone and quality.

We now pass on to the consideration of the opposite condition of the function of the heart—viz., where the heart's impulse is increased in force.

The distinctive symptoms of this form of affection is the occurrence of abnormal violence, very various in degree, in the beat of the heart; usually but not necessarily associated with increased frequency, and then exhibiting those phenomena which are comprehended under the term "palpitation." Generally this form of disturbance is obvious to the person himself, but not necessarily so. It may be a chronic disease, accompanied by few or even no very unpleasant indications to the patient, or it may assume a paroxysmal character. Whether chronic or paroxysmal, an increased impulse may present various modifications; it may be a mere occasional flutter, or it may amount to a rap or to a blow of uncertain frequency; it may be associated with feelings of weight and fulness, and anxiety about the præcordia, or even pain. In a paroxysmal attack there may be sensations of a choking, and the heart may seem to the sufferer to rise, as it were, into the throat; the respiration may also be more or less disturbed—now hurried, now suspended. Sometimes a train of more serious symptoms may be superadded, as vertigo, tinnitus aurium, impaired vision, with a feeling of dis-

tension of the eyeballs, clammy coldness of the extremities, fear of death, partial unconsciousness, with a feeling of faintness, or even actual syncope. A paroxysm such as this may be succeeded by a somewhat prolonged state of cerebral disturbance, as evidenced by heat of brow and vertex, with headache and an inaptitude to think or regulate the thoughts.

Such being the associated and distinctive symptoms of an increased impulse in the heart, it becomes a matter of interest to inquire to what this may be due—whether to dynamic or to statical causes—and then there will remain to be explained the origin and *rationale* of the correlative phenomena. An abnormally increased impulse, whether chronic or paroxysmal, whether slight or well marked, whether simple or associated with grave symptoms, looking to origin and also to consequences, may be of minor consideration or of the most serious import. It may be merely the effect of a passing emotion or of some temporary over-exertion, or it may be the characteristic symptoms of important structural changes, or even of some fatal spasmodic attack.

To accurately estimate the source of an increased impulse is always of the utmost importance; may be, in its achievement, the professional credit of the physician is at stake. Besides the necessity for a correct diagnosis in order to pursue the right course of treatment, there may be the anxious inquiry of the patient; and the answer to be rendered may be of the greatest importance to his future well-being and happiness. Again, increase of impulse is often presented in the case of those who offer themselves for examination when proposing their lives for assurance, and as also in the case of those who are candidates for the public service; their eligibility or otherwise is to be determined by the reliable interpretation of the "report" of the medical examiner.

To discriminate between an increased impulse caused by diseased physical conditions, from one solely of temporary or nervous origin, is therefore obviously of the first necessity. To do this we must first ascertain if there be any organic complications; and when these have been satisfactorily estimated and

eliminated, it is then further necessary, in order to arrive at a safe diagnosis, to discriminate between those abnormal impulses which may be indicative of acute inflammatory attacks, of coming structural changes, and of serious spasmodic complications; and those resulting from passing, and in no wise dangerous causes.

Occasionally the conclusion can be promptly arrived at that the abnormally increased impulse is solely due to passing nervous influences, and has neither present nor probably future organic complications; while at others this can only be done with difficulty, and after large investigations of the physical condition of the organ itself, with all concomitant circumstances; and then perhaps only after repeated examinations both of the heart and of these circumstances: even then it is often, indeed, a decision fraught with difficulty and doubt.

In the investigation of these cases it is our first and most imperative duty to separately consider and duly estimate the essential phenomena which mark this disorder in the heart's action. The first point to be carefully examined into is the condition of the visible *ictus ventriculi* in relation to amount, position, and character; and in order to do this the patient should be examined both in the recumbent and the erect positions. Normally, the amount of the *ictus* is thus not very remarkable—perhaps scarcely appreciable in the erect position, and not at all so in the recumbent. Any very pronounced appearance of the blow of the ventricle in both or in either of these positions must be considered as a departure from a healthy condition. The force of the impulse may be very variously increased, from only a very slightly exaggerated apex-beat to an obviously violent agitation of the whole heart; from an increase which is scarcely perceptible to a diffused and violently convulsive movement.

It may be granted that these departures from the normal impulse, be they great or small, are met with in organic diseases of the heart (rheumatic and other inflammations, hypertrophy, etc.), and in functional disorders (blood and nervous diseases); and also they may be apparently present, though not really

existing, from the contiguity of other diseased or misplaced structures.

Though there may not be a great deal to be learned from the increased condition of the impulse *per se*, yet a note is thereby sounded, exciting conjecture and showing the necessity for further observation. At any rate, it is necessary at once to eliminate from our inquiry all those cases in which there is really no alteration of force in the heart itself, but solely the appearance of it, from the accidental presence of the disturbing effects of contiguous morbid structures; and then, so far as is possible, to separate those, in which the impulse is essentially statical, from those in which it is dynamic only—that is to say, it must be sought to separate increased impulse caused by organic disease from that occurring in functional disorders. It may, however, be impossible to do this without considering the increased impulse in connexion with its position, its character, and the area over which it is diffused.

The natural position of the *ictus ventriculi* is a defined spot, small in extent, below and a little to the right of the left nipple in the interspace between the fifth and sixth ribs. Though the impulse be confined to this spot, it does not follow there may be no disease of the heart. On the other hand, we may assume, if there be permanent displacement from this position, that this displacement is not due to passing functional disorder, but either to organic disease in the heart itself; or to some alteration in the normal position of this organ by contiguous diseased structures, or may be to personal peculiarities.

The natural character of the *ictus* is that of a slight blow to the feel, occurring rhythmically. When visible, it presents the aspect of a slight and regularly recurring undulation; and whether to the feel, or to the sight, it occupies only a very small space. The normal action of the heart is even and continuous, coiling and uncoiling, thus alternately elongating and globing, and only in certain positions of the body is there any sensible approach of the heart to the anterior parietes, and when the *ictus ventriculi* is to be felt. But when the heart

becomes hypertrophied, or dilated, or agitated by convulsive and irregular action, as when under nervous or febrile impressions, besides the *ictus ventriculi*, there is to be felt a globular form, which is propelled forwards over a more extended area, and not confined to any one defined spot. The impulse may thus be greatly increased in force; yet if the evidence of it be confined to the site and limits of the normal impulse, it is not to be inferred there is, of a necessity, a diseased origin for this addition to its ordinary force.

The impulse, instead of being limited to its normal small spot, may be considerably enlarged in area, ascending even to the base of the heart. Over the whole of this space it may offer the character of a sharp blow, or of a diffused undulation, or of the heaving of a dull heavy weight For the most part, alterations in the normal character of the impulse, whether in force, position, or character, should excite attention. They are too often suggestive, if not confirmatory, of extensive disease in the heart itself; especially if associated with rhythmical irregularities, or a want of synchronousness with the pulse at the wrist. These several conditions of the heart's impulse, whether met with permanently, or only occurring paroxysmally, offer a wide field for observation, and, by themselves, separately or collectively, not infrequently supply ample grounds for diagnosis.

To appreciate more satisfactorily the value of an increased impulse, as a means of diagnosis, some of the more prominent instances of its occurrence may now be referred to, with the view of identifying it with those diseased or disordered states of the heart, of which it may be pathognomonic. In order to do this, it becomes necessary to separate those cases where an increased impulse is the characteristic sign of serious chronic or actively inflammatory diseases from those in which it is associated with nervous or dynamical disturbances only. It is necessary, however, before assuming there is an increased impulse, to ascertain there are no neighbouring organic consolidations, such as portions of indurated lung or liver, mediastinal tumours, pleuritic or pericardial adhesions or deposits; each of

these may give the character of an increased impulse, though it do not really exist. These sources of error need not here be dwelled upon—they belong to other investigations.

Supposing, however, that an increased impulse really do exist, and that, at the same time, the apex-beat is projected beyond the left nipple, it may be assumed that the area occupied by the heart is abnormally increased; and therefore that there is conclusive evidence of enlargement in this organ itself. If the impulse project not only to the left of the nipple, but also below the line of its normal level, whether it be only slightly increased or exhibit evidence of a maximum and most disturbing force, and in either case being for the most part slow, deliberate, and heaving, giving the impression of a massive weight feebly and slowly moved, and maintaining, in all these respects a tolerably uniform character, we may assume that the heart is not only enlarged, but that its walls are thickened. But if, after exhibiting this diffused heavy impulse, the heart fall back with a jog as it were, and the impulse itself is diffused over a large area, with a dulness on percussion, especially to the left of the sternum, we may further assume that the above diseased conditions are somewhat advanced. Extension rather than force appears to be the measure of the amount of diseased enlargement and thickening. There may be a considerable augmentation of impulse without enlargement; but an impulse diffused over a large area, and without sharpness in the blow, so that it rather gives the impression of a deep-seated, extended, and perhaps feeble beat, and wanting in a clear definition of its extent, is a sure indication of thickening and enlargement of considerable extent. It is said that when the impulse exhibits the above characters, as regards force, but is limited to an extent less in area than is natural, the presence of a simply concentric hypertrophy is indicated. This is, however, a very doubtful condition for the heart to exhibit; and if it really do occur, is only of so rare occurrence that it is the lot of but few practitioners to have met with, and determined its existence. If the impulse and dulness on percussion be projected over an area above as well as below the left nipple, and at the same time the

beat be dull and heavy, we may assume that there is an hypertrophied condition of the left ventricle; and, if this impulse and dulness are further traceable to the left of this vertical line, that the hypertrophy of this ventricle is far advanced, and involves a considerable amount of structural change; inasmuch as an hypertrophied condition of the walls of this cavity first induces vertical enlargement, and then, as it progresses, horizontal enlargement.

If the impulse be of the above character, and yet be not projected to the left of the nipple, while a dulness on percussion is, at the same time, traceable behind the lower part of the sternum and projects itself into the epigastrium, an enlargement of the right ventricle is indicated. Disease in this ventricle widens horizontally. This enlargement may be due solely to hypertrophy, more commonly, however, to hypertrophy with dilatation; but, in either case, it is necessary to bear in mind that, by elevating, and thus mechanically displacing, the left ventricle, an erroneous conclusion as to the existence of an hypertrophied condition of this ventricle may be inferred. In such case, the dulness traceable below and above the nipple does not necessarily indicate enlargement of the left ventricle. Nevertheless, so rarely is the former disassociated from the latter, that practically there is scarcely room for the doubt.

It has been stated that when hypertrophy of the heart is clearly indicated, there are no certain means of distinguishing which cavity is exclusively its seat; still, the existence of a basic dulness on the one hand, and on the other of a dulness projected to the right of, and below the, sternum should, as regards the condition of the ventricles, have its weight in our means of diagnosis. The presence and the condition of the liver in this diagnosis must not, however, be lost sight of as disturbing elements.

The indications of thickening and distension of the auricles are not so certain, but it will be borne in mind that the auricles are, generally speaking, only so diseased when ventricular disease is largely developed, or there exists extensive inefficiency in the mitral or tricuspid valves. If the dulness

on percussion and the impulse are traceable, as previously described, over a diffused area—whilst the force of the impulse is neither massive nor heaving, but abrupt, sharp, and rising as it were to the surface, presenting both to the hand, and to the eye, an undulatory vibration, and this diffused over nearly the whole area of percussion-dulness, now tolerably uniform in rhythm, now presenting much rhythmical disturbance, for the most part uniform in force, but on slight excitement statically increased; and thus presenting the phenomena of paroxysms of palpitation, perhaps of rare, perhaps of frequent recurrence—the presence of dilatation with hypertrophy may be inferred.

If the area of dulness be projected downwards as well as laterally, and the apex-beat be ill-defined and lost in a feeble weak impulse of an undulatory character, and which to the eye may appear strong and violent, and traceable over the whole area occupied by percussion-dulness, but offering to the pressure of the hand only a slight resistance; being, in fact, really a weak impulse, though, from impinging immediately on the surface, apparently a violent one,—it may, under these circumstances, be assumed that the heart is mainly enlarged by dilatation, and that its walls are only slightly, if at all, hypertrophied.

The distinctive differences between hypertrophy and dilatation may be thus stated. In hypertrophy the impulse is increased, heavy, diffused, and heaving; the sounds are dull, muffled, indistinct, and prolonged. In dilatation the impulse is diffused, prompt, and sharp, but feeble; giving the impression, not of solidity, but of weakness; the sounds are sharp and ringing. As these several indications relatively prevail, we may estimate the amount of hypertrophy and of dilatation.

Whether there be or be not percussion-dulness over an enlarged area, if the apex-beat be very distinct, and then diffuse itself into a superficial undulation, obvious both to the eye and to the hand, giving the impression of a mass moving immediately below the surface, and in some cases dimpling on the diastole, we may suspect, whether the heart be enlarged or not, that it is adherent to the pericardium, and perhaps, through it,

to the pleura also. If there be at the same time a bulging of the cartilages of the fourth and fifth ribs, this latter condition most probably co-exists. This præcordial fulness or bulging may also exist in hypertrophy with dilatation, though there be no pleural nor other adhesions; but it is never met with in cases of dilatation only.

The above illustrations of increased impulse comprise, in addition to those cases in which the impulse is rendered abnormally obvious by the existence of pericardial adhesions, some of the examples of hypertrophy, and of hypertrophy with dilatation, uncomplicated with other lesions. Doubtless these morbid conditions of the heart are very often associated with other affections of this organ, more especially with the various valvular diseases to which it is so liable. The co-existence of valvular disease with these affections is so frequently observed, that it has conduced to the opinion that the several conditions of hypertrophy, and of dilatation, are not only to be considered, in these cases, to be mainly due to the prior existence of the valvular disease, by reason of the forces exercised in resisting the obstructions and regurgitations of the blood thereby resulting, but that they compensate for, and alleviate the mischiefs of, the valvular diseases themselves. There can, however, be no doubt that the existence, both of hypertrophies and of dilatations, is not necessarily, nor even commonly, dependent on the pre-existence of valvular disease; inasmuch as these affections commence and progress to a fatal conclusion in a large number of instances, without any evidence of valvular disease; while, on the other hand, obstructive valvular disease is often developed, and becomes confirmed during the progress of hypertrophy and dilatation; moreover, extreme forms of valvular disease are occasionally seen without the co-existence of either of these conditions. My own observation leads me to infer they do not stand in the intimate relation of cause and effect, but are to be considered as separately due to the same, or to different, inflammatory or constitutional causes.

Considering the import and the very serious consequences of

a diseased state of the walls of the heart, I cannot bring myself to estimate the occurrence of hypertrophy or dilatation as beneficial in cases of valvular disease; but rather to regard valvular disease as less dangerous when separately occurring, than when the evidences of the presence of hypertrophy or of dilatation coexist,—that, in fact, as a general rule, these latter affections are more injurious, and more fatal in their tendencies, than are the several valvular diseases themselves. This subject will, however, be more appropriately discussed, when speaking especially of valvular diseases.

The symptoms of the incipient or first stage of simple or idiopathic hypertrophy are neither very distinctive, nor suggestive. Some indications of its existence may, however, be observed. Besides an occasional increase of impulse, perceptible to the patient, slight difficulties of breathing are, at times, experienced, rarely occurring spontaneously, but mainly on exertion, such as on ascending stairs or rising ground, or lifting weights. If exertion be energetic and prolonged there may be a passing feeling of giddiness, or a rush of blood to the head with headache, and there is often, after mental exertion, a tendency to drowsiness. A sensation of fulness and weight over the left mammary region, at times amounting to pain, is occasionally experienced. The pulse is regular, somewhat strong and full, and gives the impression of a thick cord; in some rare instances it has a thrill. The strength does not appear to be impaired; the countenance retains its colour, or, may be, assumes a more florid hue. There is not necessarily any tendency to dyspepsia; but there is usually a constipated habit, and if this be not counteracted by aperients, the straining to induce an evacuation confuses and embarrasses the head.

As the hypertrophy increases, the occasional violence of the impulse becomes more marked, and the difficulty of breathing more easily excited, and more urgent and persistent in its character. The hurry of breathing which was spoken of in connexion with a feeble impulse as, in itself, a symptom of very

grave import, is here not necessarily to be so considered; nor does it always appear to accumulate on the continuing of moderate exertion. In reference to this Dr. Hope (p. 215) says he has frequently "observed that an individual who pants on first setting out on a walk, is capable of sustaining great exertions without inconvenience when he gets warm, and the blood is freely determined to the surface." The pulse then loses much of its cordy character, and eventually is soft and even compressible, and somewhat uniformly accelerated. Should, however, the hypertrophy be in any way associated with the effects of a pericarditis, or its consecutive adhesions, the pulse is quickened from the commencement.

As the hypertrophied condition of the heart advances, increase of impulse, with deficiency of resonance, becomes markedly evident. The area of percussion dulness first extends laterally in the direction and region of the apex, and then upwards towards the base—a deficient basic resonance is chiefly due to this cause, and, if there be pericardial adhesions, is earlier and more clearly observed.

While investigating the impulse the observation of the sounds must not be neglected. In the early stages there may be little or no departure from the normal conditions; but as the hypertrophy becomes more developed, the sounds, if not characterized by specific and direct indications, certainly present some peculiarities. The first sound becomes lengthened in duration, and has a muffled character, and is sometimes, though the impulse is notably diffused and increased, scarcely to be traced over the ventricle or towards the apex. In these places it is comparatively less audible than at the base, giving to the ear rather the effect of motion than of sound. The second sound, though sufficiently audible, is changed in character; its tone being lowered, and its quality often harsh, metallic, vibrating, and prolonged, so that the post-systolic silence is shortened, or even obliterated; hence the sounds take up relatively a longer duration of time. There is, in fact, a marked disproportion between the impulse and the sounds—the former is increased, the latter are diminished; the opposite to what takes

place in dilatation when the impulse weakens and the sounds become louder and clearer.

The ordinary functions of the system, though the amount of the hypertrophy may be really very considerable, are not necessarily impaired, and the countenance, with the frame generally, may present the aspect of health. In the subjects of this disorder there is often a tendency to a fresh florid complexion, which gives the fallacious assurance of the health being perfect, or even robust; in this respect offering a marked difference to the pale, pasty look of the weak and unhealthy condition of those suffering from the anæmic or the fatty degenerated heart. The desire for physical exertion remains, and were it not for the increasing breathlessness, would not be difficult of accomplishment.

As the disease progresses complications ensue, and the special and general symptoms assume a more marked and urgent character. Epistaxis occasionally occurs. The florid countenance deepens in colour, and at times assumes a purple or a livid hue. Should the right ventricle become affected, as probably it will, there is a more marked tendency to hæmorrhages—more especially hæmoptysis. This is sometimes periodical in its recurrence, though more often induced by over-exertion; and when also grave cerebral disturbances may take place, as visual imperfections, or a vertigo passing into a temporary insensibility. The act of stooping invariably distresses, and not infrequently provokes the above difficulties. The digestive organs become also somewhat impaired, being liable to flatulencies and the generation of acids; and the liver is apt to be congested; conditions which aggravate the immediate symptoms of the enlarged heart. The occasional tendency to sleep of a lethargic character is often, more especially after meals, very marked under these circumstances. Hypertrophy, though frequently accompanied by œdema, is not necessarily so. The more advanced instances of the uncomplicated form of this disease may exist without the least indications of local serous deposits. The same may be said with regard to the association of hypertrophy with an albuminous state of the urine. It would appear that hyper-

trophy of the walls of the heart and the kidney degeneration, whether waxy or amyloid, may occur simultaneously; or may exist, the one without the other. Doubtless they often co-exist, but it remains to be proved that in all these cases, or even in the greater number of them, the one condition has uniformly preceded or been dependent on the other. Some cases that I have had the opportunity of carefully observing lead me to the conclusion that these affections may co-exist, not as cause and effect, but as having origin in the same general degeneration of the system. The morbid causes inducing the one may, or may not, cause the other; hence all descriptions of heart disease may occur without kidney degeneration, and as this latter, in all its forms, may occur without heart disease. The above observation does not apply to that kind of albuminous complication, which is occasionally met with in otherwise normal urine, occurring chiefly in cases of hypertrophy in the gouty subject. This form is not persistent, it comes and goes, and is apparently due only to temporary congestions, and not to diseased structure.

The symptoms caused by hypertrophies of the heart will be influenced and modified by the progress and extent of the disease, but doubtless they are all subject to very marked and serious exaggerations when the condition of dilatation is superadded to hypertrophy, and this complication is, by far, the more frequent condition that we have to deal with. In such cases the general symptoms and effects of dilatation present themselves in addition to those of hypertrophy. There is more breathlessness, a greater tendency to palpitation, and a more diffused impulse on the occasion of these paroxysms of palpitation. The deficient resonance on percussion is more extended, especially over the sternum, and at the apex, and it is projected lower down than is proper to the normal heart; it widens, becomes obtuse and squares. The sounds at the same time partake of the general characters of simple hypertrophy, with those of simple dilatation. They are notably increased, the first presenting the heavy, prolonged, heaving beat of hypertrophy, with the abrupt loud and flapping commencement of

dilatation, while the second sound is generally louder than is usual with hypertrophy alone. The sounds are also more diffused over both the anterior and posterior parietes of the chest, and during paroxysms of excitement of the heart may be heard over the whole chest, and that too with considerable clearness. They occupy also the larger relative amount of time which is common to hypertrophy alone. If the right ventricle be the subject of any considerable amount of dilatation through valvular inefficiency, the external jugular veins become the seat of very obvious undulations occurring synchronously with those of the arterial pulsation, presenting also a lesser though marked undulation during the diastole of the ventricle. This condition is often associated with disturbing throbbings and noises in the ears, and with a distressing sensation of choking resembling a globus hystericus. In consequence of the obstruction thus caused to the return of the blood to the heart from the subclavian veins, Dr. Dobell ("Affections of the Heart," p. 18) states there arises a symmetrical clubbing of the finger ends, but without incurvation of the nails, and, hence, that the existence of this condition is presumptive evidence of this form of heart disease.

When with or without hypertrophy, the right ventricle becomes the seat of dilatation, the symptoms above detailed become aggravated and added to, and there also ensue the pulmonary and other visceral complications which seriously embarrass the position of the patient. In the earlier stage of this condition general and local weaknesses show themselves, mental depression induces languor and a dislike to physical exertion, and physical exertion really fatigues and increases the depression. A more marked shortness of breath is also induced than occurs in simple hypertrophy. This shortness of breath, with tendency to sigh, occurs in spontaneous paroxysms, as does palpitation, but they are both excited and increased by preceding exertion; giddiness, fainting, and even worse cerebral symptoms more often occur than in simple hypertrophy, and are easily induced by slight mental worry, or on exertion. Pain in the region of the heart is not unusual, especially if the heart be, by any of these

or similar means, excited into unusual action. Sometimes this pain becomes a very urgent symptom, and gives great alarm to the patient. The pulse is small, weak, and irregular, and there is a tendency to capillary congestions.

As the dilatation increases, all the symptoms of labouring weakness likewise increase. The difficulty of breathing and the palpitation are more marked and more easily induced. Sleep is sometimes difficult, and not easily enjoyed in the recumbent position. The evidences of local congestions become evident. The circulation through the lungs is impeded, and the blood is not duly oxygenized; the complexion assumes a bilious, dusky hue, the lips become purple and glossy; there is, perhaps, pulmonary œdema, with spasmodic cough, the expectoration being serous and frothy, and not infrequently bloody. The liver and the kidneys partake of this capillary congestion; there are jaundice, piles, and local and general effusions; the face is puffy, œdema of the ankles takes place, then of the thighs and of the trunk, and perhaps ascites, with pleural and pericardial serous deposits. The kidneys in the larger number of cases refuse to do their office satisfactorily, and a small amount of high-coloured loaded urine is, with difficulty, passed. In some rarer cases they secrete, or rather pass off, as a constant habit, an enormous amount of pale fluid. Though this latter is weakening and very fatiguing to the patient, it is the better condition of the two, for it prevents the local congestions. During severe paroxysms of the advanced stages of dilated hypertrophy, the condition of the patient is pitiable and most difficult to alleviate. The restlessness is unceasing. To relieve the difficulty of breathing he sits up, dresses and undresses himself; fatigued, he lies down, then sits with the legs out of bed till they swell, become red and painful, and shine with tension. The face is congested, and the hands swollen to inefficiency. To relieve all this the horizontal position is tried, but for a few minutes only, for the breathing then becomes more embarrassed. Finally relief is sought, and best arrived at, in the sitting posture, and by supporting the head on the back of another chair; and then in a restless, drowsy, half-

conscious state, he moans and coughs, expectorating with difficulty a bloody mucus or serosity. He may, perhaps, experience anginal pains, and in a fatal syncope end his misery. Urgent as this state may be, it is sometimes protracted, comparatively speaking, for a long period; sometimes emerged from, but, then, only shortly to be renewed.

There is another symptom attending these forms of disease the mention of which must not be omitted. It is the occasional presence of murmurs of the heart. These murmurs may be due to functional disorders, as the passing effect of over-excitement, or of morbid states of the blood, or they may be the evidence of those valvular lesions with which these diseases are so largely associated, and their importance added to. The origin and bearing of these will, however, be better considered when speaking more exclusively of murmurs. Here we are speaking only of uncomplicated cases of hypertrophy without, and with, dilatation.

An hypertrophied heart presents, as its anatomical condition, a general increase of its fibres, both in size and number, this increased muscular tissue is also hardened, more dense in structure, and of a deeper red colour than natural. The carneæ columnæ may also be increased in thickness, and perhaps elongated. Still, in all this there may be no essential alteration in structure, no conversion or destruction of muscular fibre. It is still muscle, with a manifest fibrous texture.

The anatomical condition of dilatation with hypertrophy is, in the first stage, one of increase and induration, so that the cut surface of the walls of the ventricle are harder and thicker than natural; and then, secondly, of degeneration. The fibres lose their elasticity, become weakened and soft and flabby in structure, and, more probably than otherwise, pass into the fatty degenerative form of disease.

There is, however, another anatomical form of hypertrophy, the "connective tissue hypertrophy," for the description of

which we are indebted to Dr. Quain (Lumleian Lectures, 1872). "Under the microscope a heart affected with this disease presents not ~~only~~ the usually limited amount of intermuscular fibrillar tissue and connective tissue cells, but a decided hyperplasia of this in the shape of connective tissue in all stages of development from the globular to the spindle-shaped cell, and from this again to the bundle of fibrillæ. The muscular fibres are seen to be surrounded by this connective tissue, and more or less compressed by it. In many of the fibres granular and slightly fatty degeneration may be observed. In other parts of the heart the muscular fibres may be normal in appearance. The thickness of the walls is increased, as in simple hypertrophy, but their density and consistency is strikingly increased to a firm, tough, leathery character. When divided the cut edges do not collapse but remain stiffly prominent. In colour such a heart may vary from pale buff to deep purple, according to the amount of connective tisue and of blood present. But the change often occurs with less marked characters, and the hearts, thus affected, may not differ greatly from those hypertrophied by simple addition to the muscular fibres."

The diagnosis of this form of hypertrophy, which has its origin in a chronic interstitial inflammation or hyperplasia, Dr. Quain states, is to be found "in the strong heaving impulse with a dull and obscure first sound. The signs are those of increased strength, but the increased strength is spent, in great measure, in overcoming the restricting action of the fibrous tissue which surrounds the muscular fibres. Hence there is evidence of circulatory weakness in the system generally."

Hypertrophy, whether of muscle, of connective tissue or of fat, is mainly a disease of middle age, and the succeeding years of life, and a very large proportion of those who become subject to it, succumb eventually to disorders of the general system, and which are directly induced by it. From the evidence of post-mortem investigations it is estimated that, in advanced life, thirty-three per cent. are affected with heart disease; and that in the greater proportion of these hypertrophy is found to exist,

and to co-exist with dilatation; and that, in the remainder, dilatation alone is the morbid condition. It may be generally stated that hypertrophy is the disease of the left heart, and dilatation of the right; and that the former originates in middle life from inflammatory and active causes, and that the latter is the condition of more advanced years, and has its origin in the more passive causes of tissue degeneration and mechanical injuries. These morbid conditions may be somewhat accounted for by those laws of existence which provide that as age advances there should be a normal increase of the walls of the left ventricle, while the walls of the right ventricle, and of the auricles, remain as in early manhood, or, perchance, may be weakened.

In some rare instances hypertrophy is met with in early life, *i.e.*, before or about the age of puberty. It may then exist to an extent to bulge out the ribs, and thus form an undulating protuberance over the region of the heart. These cases are sometimes associated with a general fulness or fatty condition of the system, with a deep dusky red complexion. They are characterized by a dislike to muscular exertion and by occasional attacks of syncope. The left heart appears to be the chief seat of difficulty in these cases. Hypertrophy is also sometimes in early life associated with strumous weakness and an anæmic state of the blood. Pallor of surface with shortness and distress of breathing are here prominent symptoms. It is not generally speaking a disease of early manhood, unless violent muscular efforts or active inflammatory disorders as endocarditis, pericarditis, or acute rheumatism, excite it into action; or it may be the heritage of the disease which had originated in early years.

Sex offers some peculiarities, for hypertrophy more often occurs in males than in females—it has been stated in the proportion of two to one. It would appear that, in the female, the tendency is to dilatation. Probably this may be due, in the female, to a weaker muscular fibre, while the disproportionate liability of the male sex to hypertrophy finds its origin in the greater development of muscular energy induced by the more constant exercise of physical exertion, and, perhaps, also to the

being more generally submitted to the straining hurries and mental anxieties of life. It would appear there is a greater tendency to this form of heart disease in those who are deep-chested with a largely developed muscular system.

The relative bearings of hypertrophy and dilatation upon each other are worthy of consideration. It is more than probable that in the larger proportion of cases the first lesion or departure from health is a tendency to hypertrophy, and that dilatation is subsequent to, if not consequent upon, this. Still they are conditions of the heart which do, separately and independently, exist. They, however, generally are met with contemporaneously. It is not easy to trace hypertrophy as a consequence of dilatation; and whether the existing form of disease be hypertrophy, or dilatation, it is often very difficult, if not impossible, to decide from physical symptoms alone; as it is also often difficult to decide which of the ventricles is the chief seat of disease, or what its precise anatomical character may be. Here, however, experience comes to our aid, for though both hearts, or only one, may be affected, for the most part, in adult years, the left certainly is so. In advanced cases, the order of frequency may be thus summarized. In by far the larger number of cases hypertrophy, with dilatation, is found to exist; then hypertrophy without dilatation; then dilatation with atrophy; and then without atrophy; and, in some rare cases, atrophy without dilatation.

Of the parts of the heart, the left ventricle is by far the most frequent seat of wall disease—then the left auricle, and then the right ventricle—the right auricle is not often the subject of these forms of disease. The muscular hypertrophy does not determine to dilatation so much as the fatty degenerative, the fatty infiltration, or the connective tissue hypertrophies; and this latter is less prone to dilatation than are the two former.

The increased impulse, which is found to exist with an enlarged area of dulness on percussion, and which is the prominent evidence of the existence of hypertrophy, is doubtless due to the morbid accumulations, whether of muscle or of con-

nective tissue, in the walls of the heart. The remote causes of this condition are to be found essentially in vital, and not in mechanical or statical causes; on the contrary, the mechanical defects observable are not only the results of the changes produced by inflammatory processes, but are themselves the evidence of those pre-existing morbid conditions whereby an increased nutrition in the organ is effected.

These inflammatory processes are, for the most part, constitutional, and are chiefly observable where the system becomes depreciated by the rheumatic, gouty, and strumous tendencies; and those blood diseases associated with anæmia and uræmia; or excited by an inordinate supply of stimulating or highly nitrogenized food, alcoholic drinks, or by depressing mental shocks.

The dilatation which is associated with hypertrophy is probably consequent on the increased work which is done by the hypertrophied organ, and for which the strength of its fibres is not altogether adequate. It is also probable that these fibres themselves, in consequence of the disease which has caused the hypertrophy, are below the average strength of the healthy tissue, and thus readily yield to increased action. These remote and essential causes of hypertrophy and of dilatation may be induced by a large variety of exciting and proximate causes, some general, some mechanical, as various pectoral affections, or arterial and capillary diseases, or deformities of the walls of the chest; in fact, any of those causes which serve essentially to impede the circulation of the blood, whether through the lungs or through the general system. It is affirmed by some that hypertrophy, with dilatation of the right ventricle, is mainly due to an antecedent chronic disease of the lungs; but that this chronic disease of the lungs may be idiopathic, or may be the consequence of hypertrophy in the left heart.

These structural changes in the heart are generally excited into activity by excessive physical exertion, inducing a too continuously violent action of the heart; but perhaps the most fruitful source of these affections occurs under the severe exertion of the physical powers, combined with anxiety and

pre-occupation of the mind. Illustrations are offered in the violences of athletic sports, or by the excitement and muscular effort of hurrying to a railway train. It has been my lot to see many examples resulting from both of these causes. Valvular diseases are generally esteemed amongst the chief mechanical causes of hypertrophy. Doubtless these conditions are often found to co-exist, but it does not appear to me, as previously observed, that these latter depend on the former so frequently as has been assumed.

In the preceding sketch of hypertrophy, and hypertrophy with dilatation, it has been seen that the essential and characteristic symptoms are the increased impulse with an increased area of dulness on percussion. Besides these the sounds are generally somewhat muffled or dulled; the first is notably prolonged, as also the second, but less markedly so, and this has often a harsh metallic tone; there are also occasional attacks of hurried breathing; and in the event of extreme dilatation of the right ventricle double jugular undulation.

The *rationale* of these various phenomena may be thus summarily noted.

The increased impulse is essentially due, primarily, to the excited condition of the heart, induced by inflammatory and other causes; and, secondarily, to the superadded force resulting from the anatomical condition of increased size; and this latter accounts for the increased area of dulness on percussion; while both this and the abnormally increased impulse are rendered more obvious by the mass of the heart being projected towards the anterior pectoral parietes—especially when pleuritic and pericardial adhesions are portions of the diseased conditions.

The cause of the dulness and prolongation of the sounds has a correlative explanation; for this modification of their tone and duration is due to the denser medium through which they are transmitted, being thus ill-adapted, by reason of texture and thickness, for the conveyance of those sounds which are caused by the arrest of the moving column of blood on

the closure of the valves; while the muffled metallic character of the second sound is due to the force and suddenness with which the aortic valves are closed, and to their being nearer to the surface of the chest, especially when the hypertrophy is complicated with dilatation.

The shortness of breathing is caused, perhaps, partly by a want of consonance between the action of an excited and an enfeebled heart and the rhythmical action of the lungs; but, mainly, by congestion of the cellular tissue of the lungs, through engorgement and in more extreme cases by effusion and œdema. Hence the lung, in cases of hypertrophy, often becomes the seat of sudden and serious lesions, lesions which are sometimes the first evidence offered that a diseased heart exists.

The undulations in the jugular veins, observable in extensive dilatations of the right ventricle, have their explanation in the auriculo-ventricular insufficiency, and are obviously double; one undulation being synchronous with the systole of the ventricle, and when the blood is forced back into the vein; and the other, when the blood is drawn in again by its diastole. The disorders that arise, as occasional consequences of these forms of heart disease, are very important. Though they be neither essential nor diagnostic of their existence, they are more frequently than otherwise associated with them. Amongst these are valvular insufficiencies and diseases of the arteries; congestions of the lungs and liver, and of the head; and, if the disease progress, the tonic power of the capillaries is surmounted and effusions take place; hence the frequent dropsies noticeable in these cases. Puffiness and œdema of the face or of the legs are often the first examples observable of these conditions, and then perhaps the pericardium and the pectoral and abdominal cavities become the seat of embarrassing effusions. Capillary congestion also induces hæmorrhages, as epistaxis and hæmoptoë, or may be visceral fluxes of an urgent character. If these be checked or restrained, congestive inflammations show themselves, more especially of the lungs and of the membranes of the heart. In some cases the cerebrum becomes the seat of disturbance, producing, may be, convulsions; more generally

stupor, or, where the arterial system is exaggerated, as it often is, apoplexy. But amid the complications which embarrass an hypertrophied heart, the co-existence of an anæmic state of the blood is the most distressing—so long as the blood retains its normal character there may be some relief, some remissions of distress—but when an anæmic condition is superadded, there is then little to be done, and little relief to be anticipated. Where hypertrophy exists in the young subject, or is complicated with an albuminuria, this complication is not infrequent—an albuminous condition of the urine is unfortunately a frequent concomitant; and, when it does co-exist, is a complication of the gravest import, imparting to the case an unusual irritability, with tendency to inflammations in the larger organs and the serous membranes; and the serous effusions, of which they are in such case, so frequently the seat, assume a character of urgency they might not otherwise possess. It is a vexed question whether the albuminuria be the cause of the hypertrophy, or the hypertrophy and heart disease the cause of the albuminuria by inducing congestion of the kidney and those inflammatory conditions known as Bright's disease. My own impression, as stated previously from much careful observation, is that they are not necessarily dependent on each other—but are the consequential effects—simultaneous or otherwise—of the same constitutional disorder.

The prognosis in cases of hypertrophy, with and without dilatation, must be greatly determined by the amount and character of the disease as indicated by its symptoms; the constitutional tendencies; the complication with disorder of other organs; and also by the period of life.

In early life hypertrophy, even to protuberance of the thorax, may exist, and be recovered from. It has even been assumed by some that this is a normal condition which naturally subsides on the approach of puberty. There can be no doubt that such a condition is occasionally met with, and that, unlike hypertrophy in advanced years, if there be no superadded dilatation, may, by good management, subside into a healthy condition. On the

other hand, there are grounds for concluding that such an hypertrophied condition may remain for years without developing active symptoms, and then in middle or after life spring up into active disease. The developed disease can thus be traced in after life, especially when complicated with dilatation, to some such preceding, though distant, cause. The prospects of recovery must be considered too remote to be entertained as a substantial reality. Nevertheless, if the hypertrophy be without complications, and the conditions of existence be suitable, with care, life may be prolonged, and even without experiencing other distressing symptoms than those attending an easily excited shortness of breath; but if undue exertions, bodily or mental, are pursued, and indiscretions in diet indulged in, life is perilled, and its conditions become painful to be borne. Hence the regimen to be observed is to be temperate in all things, to avoid a full and luxurious diet, and the exhaustion of mind and body by unsuitable exertions. The latter precautions should be urgently enjoined, for there appear to be, in the early stages, an energy of mind and a vigour of body which incline to exertion and social indulgence.

It is, perhaps, hazardous for any man after the age of fifty-five years, to engage in violent or unaccustomed exertions, or to do anything which may unduly hurry the heart's action, or induce shortness of breathing. Serious pectoral diseases, more especially of the heart, are often thus originated. This may in great measure depend on the tendency in the muscular structure of the left ventricle to increase disproportionately to the other parts of the heart, so that between the ages of fifty and sixty this increase of size becomes an established condition. Morbid hypertrophy is thus an exaggeration of the normal condition, but an exaggeration of very serious import. A precaution especially to be observed is to avoid stooping, or using any exertion in the bent position. The doing this embarrasses the heart, and loads the head, and may lead to grave cerebral complications as well as to severe pectoral distress. On the whole, hypertrophy with its several consequences, local and general, must be classed as amongst the most fatal of heart diseases.

The treatment in these cases, whether of hypertrophy, or of hypertrophy with dilatation, cannot in adult and advanced life, be considered curative, but it is alleviative and repressive. The regimen must, in all respects, be moderate, and the diet restricted to below the ordinary requirements, both as regards solids and stimulating drinks. The local application of sedatives is certainly comforting, and may be beneficial, more especially in the form of the belladonna plaister. In the early and simple form of hypertrophy, following the indications of Nature, which so often relieves congestion in these cases by local hæmorrhages, I have sometimes resorted, even in these later days, to the abstraction of small quantities of blood, chiefly by leeches; but cupping offers a safe and convenient mode of doing this. Should the dyspnœa be painful and prolonged, the relief by these small emissions of blood is more urgently called for; but such means should always be assisted by the exhibition of a few grains of blue pill, colchicum, and digitalis. This mode of treatment is, however, admissible only in the early stages, and must in no case be pursued to an extent to depress the vital powers, or to threaten the system with an anæmic condition of the blood. When the coexistence of dilatation is well marked light bitters and steel medicines are useful. A combination of the acid tincture of steel with digitalis has found much favour in recent years. It certainly is useful, and proves generally grateful and supporting to the patient. The employment of suitable sedatives is not only occasionally indicated, but is often followed by great relief. The chief of these are opium, aconite, belladonna, æther, chloroform, hydrocyanic acid, and hyoscyamus. During their exhibition the free action of the bowels, without purging, and of the kidneys, should be maintained, and if there be any tendency to visceral or capillary congestions, the aperients should be combined with small doses of mercury, but mercury must be avoided if there be the least evidence of anæmia or of albuminuria.

In the preceding pages it has been seen that an increased impulse in the heart's action, if associated with an hypertrophied

condition of this organ, or with dilatations or adhesions, is diagnostic of urgent and, eventually, of probably fatal disease.

We now proceed to consider the indications of this symptom where the evidences of these grave complications do not exist. Doubtless many cases of an increased impulse presented to us, are, where there may be no evidences of the above conditions, as in the first stages of myocarditis, endocarditis, and pericarditis in fevers, or it may be accompanied by intrinsic symptoms of other affections of the heart as murmurs, pain, etc. But these, presenting features, marked and proper to themselves, belong to a category of cases it is not here proposed to enter upon. The cases now to be considered are those in which an increased impulse is the distinctive and perhaps exclusive feature of the heart's disturbance, having its origin not in structural, but in functional causes.

In these cases the impulse is of varying intensity—it may be but slight, or it may be excessive; but whether slight or excessive it is usually perceptible, perhaps anxiously so, to the patient. On examination it is found to be abrupt in its stroke, presenting the characteristics of a sudden bound, and communicating the impression of a fremitus or agitation; sometimes suggesting the blow of a strong heart, or the flapping of a weak one.

This bounding of the impulse is usually confined to the apex, which beats in its natural position; but if the attack be in any way violent, the impulse may be diffused towards the base, and perhaps to the right of the sternum, and thus occupies a more extended space than is usual in a healthy and unexcited heart. Though apparently forcible, it is not really so; it does not raise the hand with the material heave of an hypertrophied heart. If there be an anæmic condition, the palpitation and fremitus may be felt at the base and over the aortic area. The right ventricle is sometimes loaded with blood, and gives the impression of increased size, but this, being distension and not dilatation, subsides.

The sounds are loud and clear, and louder and clearer than is

natural to a heart not in excited impulse. In these respects they are sometimes very pronounced and greatly exaggerated, being often audible, both in the mammary and the epigastric regions; indicating that the whole heart partakes of the undue commotion. The first sound is invariably abrupt; the second generally not altered, but may be reduplicated, and in some rare instances this may be the case with the first sound also; both sounds sometimes possess a metallic ringing tone, which in some cases almost assumes the character of a murmur. Careful observation will show this not to be systolic, as are nervous murmurs.

Such are the general characteristics of the exaggerations in the functionally excited impulse and sounds; but whether these exaggerations be more or less, they will be found to act in unison with each other. The impulse and sounds increase together and diminish together. Sometimes there may really be an evanescent systolic murmur.

On percussion there is an absence of abnormal dulness. The præcordial resonance, save where it is normally dull, is clear and well defined.

The pulse is sharp, jerking—not full, and differs in all respects from the full, strong, hard pulse of inflammations, and is obviously not equal in force to the apparent violence of the heart's action; in some instances, especially where congestion of the right ventricle exists, the pulse is evidently contracted and diminished in force; nor does it always beat in unison with the systole of the heart. It is often rhythmically disturbed. The breathing, though varied by occasional sighs, is not hurried; more usually it is lessened in frequency.

The attacks are essentially paroxysmal, the intervals exhibiting the heart free from all diseased action—both impulse and sounds being normal—nevertheless the paroxysms may be very protracted; and with only slight intermissions may continue for days or for months. These Dr. Latham (p. 250, vol. ii.) has styled cases of "mock hypertrophy." It must, however, be borne in mind that, in organic diseases, the normal rhythm and force of the heart occasionally reassert themselves; but

then there remain the other characteristics of the pre-existing organic affection.

An increased impulse presenting the above distinctive characters may, for the most part, be assumed to be functional or sympathetic in its origin, and not dependent on any organic disease in the heart itself. It is evidently an error or variation in dynamic power, and not due to a permanent development of organic force; quick and abrupt, it is an exaggeration, sometimes highly marked, of the natural nervous susceptibilities of the heart. There is obviously the absence of many grave indications of organic disease, especially of those of hypertrophy and of dilatation, which are more particularly the organic diseases of the heart inducing the presence of a violent impulse, and from which it is so often necessary to make a true diagnosis. The functional impulse is different in its phenomena, as it is in its origin, from the impulse occurring in these organic diseases. In hypertrophy it is gradual and heaving; in dilatation it is undulating. Nevertheless, the conclusion that an increased impulse is due only to functional disorder, must not be too absolutely arrived at, for we have to deal with this symptom in subacute inflammations of the heart and in incipient stages of hypertrophy and of dilatation, and before the physical signs of these conditions are developed.

The distinctive and specific symptoms of a functional increase of impulse, whether this be intrinsical to the heart itself or sympathetically caused by other diseases, may, moreover, offer many varieties for observation, and be also often found accompanied by other symptoms, which, though thus associated with the cardiac disorder, are not to be considered as distinctive or indicative of heart affection. It may be useful to refer to some of these.

The impulse, though still retaining its character of a want of real force, may be diffused over the sternal region, and, apparently, be very violent—so much so indeed, that it has been said to have agitated the whole frame of the patient, and thus even to shake the bed on which he lies. In the instances I

have seen it has appeared to me that the agitation of body was not caused by the motion of the heart, but by the accompanying nervous agitation; still the visual and felt effects of the impulse were undoubtedly diffused and very considerable.

This nervous increase of impulse, even when only slight, is usually more appreciated by the patient, more painful, and more a source of anxiety than is that attending organic disease, especially in its earlier stages. At times this anxiety amounts to a fear of immediately impending death. It is therefore necessary fully to appreciate that the presence and amount of these symptoms are determined by local nervous sensibility, rather than by diseased structure. Bearing also in mind that normally the heart's impulse is inaudible, and that without structural disease it is never accompanied by friction sounds, the sounds exhibited by the impulse of a nervous heart are marked and characteristic, such as the sharp flap or blow; or the sibilant murmur,—the "wheish," "wheish"—audible both to patient and attendant.

Other nervous sensations often accompany the paroxysm, as great anxiety of the præcordia, a feeling of choking, at times amounting to a true globus hystericus, flushing of the face, singing in the ears, pain in the head, disturbance and dimness of sight, incapacity of using the intellectual faculties, even to insensibility, with no corresponding evidence of congestion of the brain—for the eye will invariably be found susceptible to the irritation of light. The voluntary muscles may refuse to act, so that the gait is tottering, or the patient reclines or grasps adjacent objects to steady himself—yet at the time there is neither paralysis nor vertigo. In three cases I am, at present, familiar with, temporary loss of power in both legs accompanies a severe attack. Sometimes there may be a tendency to syncope and to clammy perspiration, with cold shivering. Pricking pains are at times complained of, but these are usually more permanent than the paroxysms of excited impulse, and rather belong to the morbid state of health that induces them.

The respiration, though not generally, may be much em-

barrassed, irregular, and oppressed during the paroxysm. It is rarely accelerated, but may present the phenomenon of a short inspiration with a prolonged expiration. If there be no associated pectoral disease there is none simulated by the attack, unless it be oppression of the heart itself. But this, as well as the attack, often appears to be relieved, and even to pass off by a few sighs or deep inspirations, or by short coughs.

If the paroxysms have been induced by a congested or loaded state of the right heart from using undue exertion, the breathing may be accelerated and accompanied by dyspnœa, and even apnœa, with a short dry paroxysmal cough. The dyspnœa does not, however, appear to be a measure, or dependent on the frequency, of the impulse. As a general rule dyspnœa is absent in these cases, but present in organic disease.

The sounds, always exaggerated, at times become very much so, and, then, usually with a sharp metallic ring; occasionally a kind of remitting humming sound is superadded, and may be heard by the patient; but this is never constant. Sometimes the sounds are heard over a great extent of surface; but this extent is no measure of their intensity, for they may not be loud but clear only. Extent and intensity are neither identical, nor have they the same sources of origin. The first sound, abrupt and short, is occasionally heard at the mitral apex to be both loud and clear, and may sometimes, though rarely, have the metallic ring. Occasionally there is a pericardial rub accompanying the mitral apex shock, simulating the friction sound of a pericarditis, but there is never a true friction sound. The second sound, more frequently than the first sound, presents the systolic basic metallic ring. Sometimes it becomes lower pitched and less clicking than in an ordinary paroxysm of palpitation; and may even, as also the first sound, so lose sharpness and abruptness, as to assume the character of a soft murmur; but this is usually in cases of spanæmia, or anæmia, and will be more particularly referred to when speaking of these affections.

The paroxysms may exist for a few minutes only, or last for days, or may even be protracted into months; they may be

solitary, or recur only at long intervals, or they may be so frequent as to present the aspect of being continuous.

In the intervals the impulse may drop to its natural force and frequency, and the sounds be unaccompanied by any exaggerations; nevertheless, though the attack may have subsided, there may yet be some slight irritability of the heart's action perceptible.

Exercise, which aggravates increased impulse from organic disease, and induces a hurry of breathing, relieves functional palpitation. Paroxysmal palpitation, in the former, more frequently comes on during and after exercise; in the latter, when quiet; and the paroxysms are usually more marked in the recumbent position and in early night.

Should the nervous increase of impulse be associated with a congestive state of the system, the pulse loses its jerking character and becomes feeble; but if the condition of the system be that of plethora it has a force and fulness not usually observable in ordinary cases; or it may be quick, small, and intermitting. The head symptoms, too, become more marked, especially as regards local pain, tinnitus aurium, and flushings of face.

When the palpitation is associated with scurvy or chlorosis, or a generally anæmic state, the attacks present less the character of being paroxysmal—they are on the contrary somewhat persistent; there is also more pectoral complication, even to dyspnœa. The headache in some of these cases is so intense and bewildering that the mind becomes alarmed with vague apprehensions of danger expressed amid an evidently distressing state of restlessness. Local puffiness and œdema show themselves with œdema of the face and extremities; the pectoral integuments over the region of the heart often present this condition. In some extreme cases of chlorosis a protrusion of the eyeballs, and an enlargement of the thymous gland is met with. It is probable that the morbid conditions of the system which induce these are the cause of the palpitation also. But they will be referred to subsequently. This group of symptoms indicates a morbid condition of the system with a weak, irri-

table, and excited heart; nevertheless, it is not one which can be positively stated to be organically changed or diseased. Paroxysmal palpitation, however, often occurs with a diseased condition of the heart, and when this condition is so little developed as to be most difficult of detection. This is chiefly in incipient cases of hypertrophy with dilatation, and is often met with, in old people, when the strength begins to fail. These cases are generally attended by great distress, both of mind and body. The paroxysms are recurrent, marked by dyspnœa, and often by a sonorous breathing; and these both alarm and distress the patient. Pains are felt over the whole chest, and especially in the epigastrium, then the head symptoms become urgent, a tinnitus aurium alarms, and the mind is agitated by a feeling of impending death. This so agitates the sufferer that he often presents the inconsistencies of praying for release by death, and yet urging every means to be tried to save life. Instances of these exaggerated forms of palpitation are sometimes induced by slight chronic inflammation of the endocardium, or even of the valves, but there is then generally a considerable amount of local pain.

In the absence of any certain and well-defined acoustic sign, it may be very difficult to distinguish and separate these several instances of an increased impulse having origin in, and associated with, diseased structure, from those of a purely functional nature. Still, as a general rule, if the impulse and sounds during an attack increase together, we may assume there is no incipient hypertrophy; as we may, if they simultaneously diminish, there is neither atrophy nor dilatation; and, perhaps, the absence of fixed pain may determine the increased impulse not to be due to local inflammations. The history of the case, the physical condition of the patient, and other surrounding circumstances will also aid our judgment.

It is probable that the proximate cause of this functionally increased impulse or palpitation, not being due to organic disease of the heart itself, owes its origin to some over-stimulation of the excitability of its muscular structure by functional

errors of the intrinsical cardiac ganglia of Remak, and of those nerves which, proceeding from the ganglia of the great sympathetic, supply the heart. At any rate it is difficult to refer its origin to other proximate sources. Remotely, the nervous error may be due to many causes—it may be excited by certain idiopathic conditions of the heart, and may thus be inherent and not due to extrinsically exciting causes. Under these circumstances the increased impulse probably indicates that the heart is on the threshold of disease; or it may be due to a deterioration in the quality of the blood itself, as in scurvy or chlorosis, when the amount of serum is disproportionately large; or in spanæmia, when the blood globules are diseased; or in plethora, when the fibrine predominates; or it may be sympathetically excited, and thus indirectly due to other existing morbid conditions of the system. Amongst the chief of these, separate from inflammations, must be classed gouty and rheumatic irritations; hepatic, stomach, and alvine disorders; debility from all sources, especially in the advanced weakness of fevers; after exhausting discharges, large depletions, hæmorrhages, together with the several varieties of mental emotions and the exhaustions from protracted mental and physical exertions.

These nervous palpitations are met with at all ages, but largely occur in early life; more especially in the years approaching puberty, and for a few years afterwards. The palpitations of after life, though sometimes nervous, are more usually of an organic origin; still in middle age and in advanced life we occasionally meet with very severe and distressing instances of their occurrence. Both sexes are liable to these attacks, but perhaps the male more frequently than the female. In the latter the hysterical constitution disposes to them, especially if there be any uterine irregularities. In the male they are met with in the irritable temperament, and where nervous depression is apt to pass into hypochondriasis.

The careful observation of a paroxysm of this form of nervous affection of the heart shows it to possess some distinctive

features. That while there is no abnormal dulness, nor displacement of the apex beat, no lung embarrassments nor incipient inflammations, the agitation of the heart is not only excessive, but is in its character sudden, and apparently increased in strength even to violence, having often rather the features of spasm than the calmness of rhythmic order. The sounds, too, are *pari passu*, increased in sharpness and intensity, and diffused over a larger area. Nevertheless, this is only an apparent increase of power, and hence the pulse does not partake of the simulated force of the heart. Observation also shows that, when it occurs in plethoric or chlorotic subjects, they alike suffer from consequential disturbance of the functions of the brain, while the latter in addition suffer from pectoral embarrassments and dropsical effusions. Assuming that the cause of this spasmodic impulse is remotely due to error or excitement of the nerves supplying, and the ganglia belonging to, the heart; and as these chiefly and mainly originate in and belong to the vagus and sympathetic system, we must look, for the exciting causes, to those circumstances which, perchance, may embarrass the due and healthy action of those nerves; such as the existence of a heart weak in itself or the false stimulation of the blood, as when this is in excess in plethora, or diseased in chlorosis; or undue exertion; or engorgement, from obstruction; or the being too stimulating in itself, or, extrinsically, by nervous emotions and influences, as dyspepsia, hysteria, etc.

In the spasms thus excited we must find the *rationale* of the specific, and occasional, heart symptoms exhibited. Hence the abruptness of the heart's action, its excited apex beat, its basic agitation diffused over the aortic region, and there having the character of a fremitus; hence, too, the greater personal anxiety and alarm experienced than in hypertrophy. In hypertrophy, though there is increase of impulse, there is also increase both of size and power, so that the palpitations, though violent and prolonged, yet are really effected by adequate muscular effort. In prolonged functional or mock hypertrophy these latter conditions are deficient. The spasm which modifies the motor power does not give the measured action of real

power, but substitutes in its place its weak and irregular action.

When the spasmodic impulse is influenced by over-exertion the heart palpitates in order to relieve itself of the venous blood that overloads the right heart and induces a remora in the capillary system; hence, too, the impulse is fluttering by reason of the ventricular contraction being impaired by the blood which overloads it. This is more especially the case when depressing passions are the exciting cause. The impulse is in fact proportioned only to the real force exercised, and not to the seeming energy and activity, fallaciously, exhibited. Hence, too, the valves and the contiguous tissues, being vibrating media, by reason of the very abrupt and forcible stoppage of the current of blood, whereby its momentum is suddenly arrested, not only yield the characteristic basic thrill, but convey the sounds to the surface loud and clear.

The clanging, or metallic ringing, sound which is, so often, characteristic of the excited valvular action, is differently accounted for by those who have described it. It has been referred to the impinging of the ventricle against the chest (Joy); to valvular tension (Hope); to the violence of the semi-lunar valvular contractions in a vibrating medium (Wood); etc. I would rather regard it as a modification of the second sound, caused, as in the preceding case, by increased vibrations in the current of the blood, when associated with an aortic thrill. The vibrations are thus prolonged so that they acquire this character of a clangor.

The remitting humming sound occasionally heard in anæmic cases may also be due to a similar cause, having its seat in the auriculo-ventricular valves, or the muscles and tendons belonging to them; but the consideration of this sound rather belongs to the subject of murmurs.

The reduplication of the second sound, and sometimes of the first, occasionally to be observed, is probably due to a want of consent between the two ventricles—the spasm which rules the action of the heart inducing in them some slight error of synchronous contraction—so that the valves, whether mitral or

auriculo-ventricular, do not close simultaneously. That spasmodic irregularity is the immediate cause of this phenomenon is confirmed by its being rarely observable in cases of organic disease.

In addition to these symptoms proper to the heart itself, there are, occasionally, some extrinsically to it.

The respiration has been shown, generally speaking, not to be disturbed; and this, which is a material aid in forming a diagnosis, is due to the current of blood not being interfered with by any degeneration in the structure of the heart. Sometimes, however, the attack is accompanied by sighs, or a dry cough; chiefly in those cases in which the agitated impulse is excited or accompanied by a loaded right heart, caused by over-exertion, or when a remora of venous blood takes place in the lungs by reason of exhausting or depressing passions. This sighing and coughing are obviously efforts of Nature to relieve these conditions. It has been attempted to explain the cause of the remora by stating that the respiration influences the capacity of the chest, and that the amount of blood in the lungs has reference to this capacity; also that the act of inspiration induces a flow of venous blood through the right heart into the lungs, and that expiration forces it onwards to supply the left heart with arterialized blood. Hence, if the inspiration be weak or relatively depressed, the right heart becomes loaded by the blood being kept back, and to relieve this there is sighing or coughing. Proof, however, is required that the relative amount of blood is influenced by the rhythmic acts of respiration. Rapid breathing, natural or artificial, does not necessarily increase the heart's action. The converse is perhaps the real explanation; and that it is the heart which regulates the supply of blood to the lungs; so that when there is this over-action, by reason of increased impulse, too much blood—and this is necessarily venous—is thrown into the lungs, and thus the venous blood here becomes disproportionately large, and there then arises cough to repair the error: or during a fit of palpitation with depressed feelings, the nervous energy of the lungs, being inactive, fails in inducing a due amount of respiration; hence the blood sent to the lungs is

not duly oxygenized, and sighing, or perhaps yawning, are the suggested efforts to relieve it. In either case the right heart becomes as a consequence loaded or distended, and Nature supplies the remedy.

Shortness of breath, and even apnœa, are occasionally seen to be associated with an excited impulse; but this is usually in cases of chlorosis, and probably the morbid condition of the blood causes these disturbances of the breathing by requiring a larger amount of oxygen than can be afforded by the ordinary rhythmical respiration. The occasional headache and other cerebral disorders are also probably due to this condition of the blood. The œdema not infrequent in these cases is to be attributed partly to this cause and partly to the congestion caused in some of the larger organs by being overloaded with a venous blood.

The pulse is ordinarily sharp and jerking, yet without the character of force, from the systole being the result of spasm rather than of sustained dynamic energy; but this character may be modified if the heart be congested or in a state of plethora. That it is not always in unison with the systole is due to the occasional irregularity in the systolic action of the two hearts; and here it must be borne in mind, that it is the systole, of the right ventricle, that prominently presents itself for observation and to the feel, while the pulse-beat indicates the conclusion of that of the left ventricle.

The freedom of the heart from all agitation and other indications of disease, before and after an attack, is due to its being a healthy organ; but, during an attack, liable to the morbid influences of spasm and congestive loading.

From the history of functional palpitation it may be assumed, whether the paroxysms be short or prolonged, occurring rarely or frequently recurring, that the termination will for the most part be a restoration to health. Nevertheless, cases may occur in which the functional disorder may originate and pass into organic disease. Thus severe and repeated attacks characterized by a loading of the right heart may induce a tendency to per-

manent dilatation. This more often happens when the attacks are associated with diseased conditions of the liver; and it is in these cases chiefly that local dropsies take place. Frequently recurring attacks, by shocking and exhausting the nervous system, and by inducing congestion of the brain, may induce deterioration of brain tissue and permanent impairment of its functions.

When associated with scurvy and anæmia, the prognosis is not so favourable. Still in the young and middle aged there is good chance of ultimate recovery. As these diseases are subdued the attacks of palpitation subside; when occurring in the aged, or in those having a constitutional tendency to hypochondriasis, or when associated with organic diseases, or excited by inflammations of the endocardium, a less favourable prognosis must be given.

In those prone to this affection many precautions should be observed. The young and the plethoric must avoid extremes of diet and exercise, the food should be moderate in quantity, and unstimulating in quality, exercise should be unfatiguing, and chiefly taken in the open air. Hot and ill-ventilated rooms and the postponement of sleep by late hours, and the midnight dance, should be especially avoided. The moral discipline must be sought in a healthy exercise of the brain and the keeping down, by a well-ordered intellectual culture, the tendency to vain imaginings and emotional passions. The constitutionally nervous and irritable in mind must sedulously avoid exciting situations, as well as exhaustion by overwork. The sluggish and hypochondriacal must resist the temptations to inaction, seeking to overcome these tendencies by exercise, by cold bathing, more especially by means of the shower-bath; and, if the bowels be confined, by the judicious use of aperients.

The treatment must have reference to the paroxysm; and to its exciting causes. The objects to be sought being the mitigation of the violence of the impulse and to cut short the attack; and then to prevent its recurrence.

To follow out the first indication it should be ascertained, where possible, if the attack be essentially due to irritability in the heart itself; or if it have its origin in some coexisting excentrical cause. For the most part the attack is relieved by antispasmodics—as ammonia, camphor, æther, assafœtida, musk, valerian, sumbul, etc., but where irritability of the heart itself is the cause of the attack, it is generally best met by sedatives—as opium, hyoscyamus, hydrocyanic acid, and (it has been proposed) digitalis. If the attack be due to stomach derangements, as from the presence of the gouty acids, an alkali may be useful. In extreme cases, and where head symptoms supervene, the extraction of a small quantity of blood by leeches or venæsection may be useful. The mental treatment is of the greatest moment. A confident and cheering prognosis conduces to recovery and prevents the nervousness which exhausts and tends to prolong the disorder.

The paroxysm being allayed, it is then well to examine carefully into the state of health and find out where may be the sources of this error of increased impulse. The dyspepsia must be relieved; the liver is to be set right; the uterine functions are to be restored to regularity; loaded bowels relieved; plethora subdued; spanæmia and chlorosis strengthened into health; exhaustion compensated for, and debility converted into strength; and the exhausted and over-worked must seek renovation in travel and cheerful recreation.

CHAPTER VIII.

ERRORS IN THE RHYTHMICAL ACTION OF THE HEART.

THE action of the heart, besides the various errors of impulse just described, is subject to perversions or errors of rhythm. These latter may exist independently of the former, or they may be associated with, or superadded to, them. Impulse, normal or otherwise, depends on the single muscular contraction that produces it. Rhythm depends on the order and regularity of a succession of these muscular contractions. By this succession the heart presents for observation two rhythmical features. One of motion, whereby the blood is propelled, and one of sound produced, on the disturbance or arrest of this motion, by the sudden closure of the valves. This latter depending upon, and caused by, the former. The motor power and the order of rhythmical contractions are mutually independent of each other, and are not due to the same causes. Force may be varied while rhythmical order remains unaltered, and irregularity of rhythm may occur with unaltered motor power.

Impulse, as just observed, depends on the contractile energy of the ventricle. The cause of the rhythmical succession of these impulses is more obscure, and perhaps, in the present day, not to be satisfactorily explained. The subject is one of deep interest; it is, therefore, not surprising that various theories have been advanced to account for it. Amongst the more noteworthy of these are those that attribute it to the agency of the cerebellum and the eighth pair of nerves (Willis); to irritability of muscular fibre, independently of the nerves (Haller); the lot or consequence of accumulated force (Arnott); the necessity or

law of rhythmic nutrition (Paget); and partly to a healthy nutrition, partly to the inherent vitality or power of moving residing in muscles, and partly to the influence exerted on the cardiac movements by the ganglia and nerves situated on the surface and in the substance of the heart, and other parts of the body (Pettigrew). We may conclude that these theories, considering who their authors are, have been not only cautiously advanced, but ably supported by ingenious arguments. Nevertheless they do not succeed in satisfactorily explaining the mode whereby these rhythmical phenomena of the heart are effected. We must content ourselves with the knowledge that the movements of the heart commence in the fleshy elements which constitute its muscular fibres, and that these movements depend on nerve agency, and not upon the immediate agency of the stimulus of the blood. Rhythmical movement of the heart will continue, though the stimulus of the blood be withdrawn; while it ceases, though it have this stimulus, if the nerve power be destroyed. The fact that nerve power cannot continue to exist without the stimulus and nutrition of the blood, does not impugn the conclusions to be deduced from the above positions. Though we may admit all this, the *rationale* of rhythmical movements is thereby in no way explained, and we are constrained to fall back upon the comprehensive conclusion, that it has its origin in vital, though unexplained, causes.

To the physician, there remain for study and consideration the several disturbances, whether simple or complex, to which the rhythmical order of the heart's motion is liable. By the patient they may, or may not, be recognised; generally they are so recognised, and are then often the source of the greatest anxiety. By the physician their presence and bearing are appreciated by the touch or by the ear; presenting to each of these senses very different phenomena, both as regards effect and origin. By the touch we can only appreciate the successive rhythmical errors in the frequency and the force of the systolic movements, and in the duration of the pauses between them. Thus there may be inequality in the strength of successive beats, or there may be an occasional impulse more or less

powerful than those, preceding or, succeeding it; or the impulse may be diminished or increased in energy, and may be doubled or even tripled; or the recurrence of the impulse may be so irregular as to constitute intermittence; or there may be that kind of uncertain vibratory or vermicular motion, to which the term "fremitus" or "thrill" has been given. To the ear is revealed irregularity in the recurrence and duration of the sounds, from the slightest appreciable pause to the most rapid and confused trembling, with very manifest alterations in their tone and pitch; as also those other sounds which are superadded to, or take the place of, the sounds, and to which a great variety of names have been given, but which are here comprised under the term of "murmurs." These murmurs of the heart may be independent of the sounds, or may be a diseased quality of them, so that the sounds are overlaid by, or pass into, the murmur. The normal rhythm of the sounds is thus interfered with, but the sounds themselves, as previously observed (p. 29), if not thus absorbed, always exist; a deficiency of valvular sound, and of murmur, never coexists, one or the other there must be.

We therefore see that each rhythmical error of the impulse indicates error in the succession of the ventricular systole, while rhythmical error of sound indicates error in the valves, or in the valvular openings; or of the volume or condition of the column of blood passing through the heart.

In the course of observing these rhythmical irregularities, it will be found they present, in their several relations, great varieties; but the particular and minute enumeration of these is rather satisfying to curiosity than instructive. It will be only necessary to refer to those amongst them which are sufficiently typical and distinctive, so as to enable us either by themselves, or in conjunction with other concomitant circumstances, to indicate the condition of the heart, whether diseased or otherwise. In doing this it will be necessary to bear in mind the vital laws that regulate the recurrence of the muscular contractions and expansions of the heart; and which are assumed to be immediately due to the agencies of the vagus nerves, and

the nerves and ganglia proper to the heart, proceeding from the ganglia of the great sympathetic system of nerves, excited and nourished by the blood it circulates; as also the hydraulic laws which order and regulate the sounds and murmurs.

In considering how far error of rhythm, whether of impulse or sound (including murmur), is indicative of but slight ailment, or of serious disease, it is necessary to study it under its more specific manifestations, paying every attention to special peculiarities. Investigation must, therefore, be directed not only to its phenomena, but to the causes material and functional which induce it. Rhythmical irregularity may be of no moment, but there is required some positive and more negative evidence to prove that it is not so. Though we know it may be due only to nervous and functional causes, we also know that it may be due to diseased conditions of the walls of the heart or of its lining membranes, to valvular lesions, etc.; it is therefore necessary to ascertain that it is not complicated with these latter sources of evil before affirming it to be due only to the former.

It will be useful to consider the errors of rhythm under different conditions—(1) solely of impulse, and appreciable by the touch and sight alone; (2) of impulse and of sounds, but unaccompanied by murmurs; (3) of impulse and sounds accompanied by murmurs; and (4) the association of each or all of these with ascertainable structural heart disease.

In any consideration of the above, it may be premised that if rhythmical errors, whether of impulse or of sound, be found to be associated with murmur, there is, presumably, an indication of evil, and that such indication can only be satisfactorily set aside by ascertaining that the associated murmur is not of diseased, but only of functional origin. If, however, the existence of structural disease, whether of valves or of walls, be clearly evidenced, then a disturbance of rhythm, however slight, must be considered as indicative of serious disease. Premising this, we may then consider the value, in diagnosis, of errors of rhythm in those cases in which they are not thus obviously

associated with structural disease, or with murmurs, or an altered condition of the sounds. Rhythmical errors under these circumstances are met with under many forms. But whatever may be the forms in which they occur, they are referable solely to perverted frequency, and to perverted force, in the action of the heart. They may be slight and scarcely to be appreciated, or they may be very marked; now easily produced and as promptly subsiding; now a life-long ailment.

The question arises, are there in these uncomplicated and simple forms of rhythmical error any specific characters in the phenomena they exhibit which will enable us to infer the precise lesion or functional disturbance on which their existence depends; and, hence, whether we may consider them as a means of diagnosis?

In a previous page (p. 74) it was shown that the rhythmical errors of force were mainly appreciable, and usually estimated, by the pulse, and some few of the indications thus revealed were briefly referred to. In pursuing the subject, it is necessary to consider what the conditions may be which excite and determine the action of the heart to these errors. Rhythmical errors of force may be functional, or they may be dependent on statical causes; hence they may be determined by those agencies proper or external to the heart which may effect alterations in its force; and the same may be said of the errors in frequency. We have thus a wide range of causes to investigate, including sympathetic nervous disorders; disorders of the ganglia and nerves of the heart; disordered conditions of the blood; hypertrophied, or weak and dilated ventricles; general and specific inflammations (rheumatic, gouty, and uræmic); abnormal and congenital diseases of the heart itself, and of its membranes, etc.

It may be that, in the present day, without reference to sound (or murmur) or to other evidence of obvious structural disease, these rhythmical disturbances, however marked they may be, do not justify a very definite or positive conclusion as to their immediate origin. Surmise is alone excited. We may suspect the existence of a degenerated muscular structure;

of valvular disease; of a deficiency of nervous power; of inflammations, as pericarditis, endocarditis, myocarditis, etc., bearing always in mind that, for the most part, rhythmical irregularity indicates irritability and weakness, rather than increased strength. Increased strength, as in hypertrophy, does not generally determine to rhythmical irregularity.

A sudden pulse shock after a prolonged pause may be due to an impaired nervous energy; but, if the pulse shock be also strong, and instead of being sudden has the character of slowness, we may suspect the impaired nervous energy is due to some injury to the great nervous centres of the spine or head. If the impulse be weak, slow, and yet sharp, the muscular structure of the heart is probably so impaired as not to be able to receive its duly required nerve power, though this latter be really not impaired. The more rapid beat, sharp yet weak, with a diminished pause, indicates the weakness of fever, or the want of a due nourishment, as in spanæmia, etc. An intermitting pulse during, or after, fever, or other diseases, indicates weakness either in the muscle of the heart or in its nerve power, and therefore is of evil omen. Nevertheless, though we may surmise evil, we are not justified in coming to a positive conclusion that it really exists.

Cases occasionally present themselves in which rhythmical irregularity appears to be the normal condition. Some are marked by an extension of the pause (slowness); and some by its apparent extinction, so that there exists a rapidity of beat which defies all analysis; the impression conveyed being one of unresolvable confusion. Both these classes of cases present the remarkable feature of losing much of their distinctive irregularity when under the influence of febrile attacks. The slow pulse becomes quicker and more steady; the rapid one less frequent and more distinct. These cases will be, subsequently, more particularly referred to.

In order to estimate the value of rhythmical irregularity as a means of diagnosis, we must not be satisfied solely with

the evidence afforded by touch, but must seek for aid in the signs revealed to the ear; and these will mainly be those produced by irregular valvular contractions, or by the superaddition of murmurs. The indications to be derived from these latter will be more particularly considered in the succeeding chapter; here the indications afforded by rhythmical irregularity in the sounds will alone be referred to. It must be borne in mind that the valvular sounds generated in two hearts are presented to us, and that these may not be in unison either in force or time. The first and second sounds may be relatively increased, or diminished, or reduplicated, as the blood, irregularly impelled by either ventricle, impinges on the walls; or is urged, or checked in its course, through the valvular openings. As each valvular sound has its normal special peculiarities, it is therefore necessary, before we can satisfactorily appreciate the existence or the nature of error in either of them, to carefully consider these, as well as their modifications by distance, or by the medium through which they are transmitted; and we must also well consider the alterations to which they are capable of being submitted. They may be intensified and heard over a larger area than natural, or they may be less loud and heard over a more limited space; they may be prolonged or shortened in duration; they may be moved from their natural position; their pitch may be elevated into thinness, or into a flapping sound, or toned down to a soft dulness; or the rhythm of all the sounds may be relatively interfered with by a reduplication of one, or both, of the sounds; or the pauses between the sounds may be so lessened as to obliterate all appreciation of rhythmical regularity, or so lengthened, especially the post-diastolic, as to give a distinctive character to the pulse.

Supposing the pulse, though regular as to its pauses, be sluggish and diffused in its movements, suggesting to the touch that the ventricular contractions are somewhat prolonged and retarded, and inducing to the suspicion that the walls of the ventricle are unduly thickened, we then examine the sounds; and find that the first, though dull in pitch, is intensified in

tone, and prolonged, with an increased area of transmission, so that it becomes lengthened in duration, even to running into the second sound, while towards the apex it is dull, muffled, and toneless, or even inaudible; and the second sound is heard short, loud, and well toned, perhaps accentuated. We may suspect, if not assume, the presence, in different degrees, of hypertrophy, and the probabilities are, that the other conditions of an hypertrophy will confirm the diagnosis. But other rhythmical disturbances may coexist. The first sound, besides the above characteristics, may be reduplicated and weakened, showing that the ventricular contractions are not in unison; due probably to those of the left ventricle being prolonged by the enfeebling and induration, which have taken place, in its walls; and should the position of the first sound on the right side be lowered, as well as this reduplicated sound on the left side, there is probably some hypertrophy of the right ventricle as well as of the left. At any rate, the suspicion thus aroused should be carefully investigated.

When, with the above evidence of a prolonging of the left ventricular contraction, the first sound is found to be loud, less in duration, and transmitted over a large area, slightly raised in position, and conducted to the left of the nipple, while the second sound is yet louder, sharper, and more toned—in fact, if both sounds are intensified, with a reduplication of the first, and with alteration in its position, we may suspect there is a commencing hypertrophy, with dilatation. The explanation being that the ventricular parietes are indurated, and at the same time undergoing the process of dilatation, so that the power of contraction is not only enfeebled, but prolonged; and this prolongation is greater, and therefore more marked, in the thicker walls of the left ventricle (p. 111).

When the pulse is weak, soft, and irregular, but not pausing in the beat, we suspect weak walls, with dilatation. If we then find that the first sound is shortened, rapid, and unequal, has a feeble sharpness, or is accentuated at the commencement, is extended in area, but weak over the whole of this area, and more especially at the apex; while the apex beat, on excitement,

is knocking, rough, or murmur-like, as in incipient inflammation, but which character disappears on rest; and that the second sound is clear, sharp, flapping, and has a raised pitch, but still is somewhat continuous with the first sound, so as to make it somewhat difficult to separate and define them; and that the confusion thus induced may, under excitement, pass at the base into a well-marked fremitus; and if, with this morbid condition of the sounds, the periods of silence are unequal, it may, with tolerable certainty, be inferred that the walls of the ventricle are weak and attenuated, and passing into a state of dilatation. The inequality in the periods of silence may exist independently of the rhythmical revolution of the action of the heart; and this is due to some of the ventricular contractions being shorter and weaker than those immediately preceding and succeeding them.

Should the pulse be so feeble as to be indistinctly appreciable, and communicating only a feeble fluttering movement, we infer with certainty a weak condition of the walls of the ventricle; but if we have superadded the special valvular sound previously described (p. 95), so that the disturbed rhythm is characterized, not only by deficient force, but by want of tone, we infer fatty degeneration. If, with all this feebleness and rhythmic confusion, the second sound be heard at the base dull and slightly clanging, there is probably some deposit and rigidity of the semilunar valves, and of the contiguous parts of the aorta, and the presence of the smallest possible amount of thrill makes this tolerably certain.

If with quickness and a general feebleness of pulse, the first sound be disproportionately weak, the presence of established inflammation of the heart or its membranes may be suspected, and probably on investigation, other evidence of its presence will be discovered. In confirmed and critical cases of this nature, as in the soft and weakened hearts of typhus and typhoid fevers, the first sound may become so weak as to disappear. In these cases it is of special interest to carefully observe the relative amount of tone in the first and second sounds. While

the second sound remains, the gradually progressive feebleness, or extinction of the first, marks the progress of an inflammatory weakening and degeneration. The contraction of the ventricle is thus shown to be the result of the feeble effort of a heart rendered powerless by disease. Should the pulse, besides the above characters of quickness and feebleness, pass into an irregular excitement, we may suspect incipient specific inflammation; and this may be inferred more certainly to be the case if the valvular sounds are intensified, and with the first sound having a ringing character on the systole; or if there be a suddenly recurring irregularity in the sounds, so that at intervals they pass into tumultuous action. Should there be a prolongation of the first sound, with reduplication of the second sound, the inflammation is probably of a nature to embarrass, if not to weaken the ventricular structure, as is found to be the case in endocarditis and myocarditis.

With the above characters of pulse, should the sounds be weak and muffled, but otherwise unaltered, the impending inflammation is probably that of a rheumatic pericarditis. Though the ventricles are unaffected, they are by sympathy excited to quickness, but not to force; the tendency in this first stage of the disease is one of alarm and caution to the heart, so that it seeks ease in rest and quietness.

The preceding illustrations of rhythmical disturbance, both of impulse and of valvular sound, are due to error in the contractile agency of the ventricle; and the urgency of the condition must be measured by the estimated physical cause for these rhythmical disturbances. Bearing always in mind, that, as the heart is loaded with blood, resonance and loudness are, proportionately, modified, these disturbances may, to a certain extent, be constituted as tests of the condition of the muscular structure of the heart. Experience teaches us that the more obvious and important sources of rhythmical disorder of the impulse and of the sounds are to be found as the results of the inflammatory diseases of the heart and its investing membranes—endocarditis, myocarditis, and pericarditis—and these form a group of diseases

so intimately linked together that, in practice, it is difficult to disentangle them. For the most part they generally, in some degree, coexist. Myocarditis is so almost invariably associated with, if not dependent upon, pericarditis or endocarditis, that to treat of it as a separate disease would be an inquiry, not of practical interest, but only of curious investigation. It may be that simple and uncomplicated cases of myocarditis occur, but they are most rare. It is with pericarditis and endocarditis we have prominently, and indeed most frequently, to deal. Though these are essentially separate diseases and their symptoms sufficiently distinctive, they are so commonly associated with each other, and so commonly the forerunners, if not the cause, of the associated myocarditis, that they may all be well considered together. The general symptoms are, in the main, the same, but the special symptoms sufficiently mark the differences of each.

The general symptoms are more or less of a hot, dry, febrile condition, probably ushered in by a rigor, with præcordial uneasiness, generally passing early into pain. The pulse is at the commencement slow, sometimes pausing, then becomes accelerated, irregular, and intermitting; may, for a time, be hard and bounding, but, as inflammation extends, becomes weak, small, rapid, and irregular. The breathing is difficult, short, sometimes catching, and accompanied by a short dry cough, There is a general restlessness, with an incapability of lying excepting on the back; the expression of the countenance is anxious and distressed.

With the progress of disease, the symptoms are aggravated; the general distress is increased; the breathing is short and suffocative; and there is a tendency to syncope. The face is flushed, puffed, and haggard; and perspirations evidence the general distress and the severity of the heart pain. The pulse is quick, irregular, and small; while the impulse of the heart is tumultuous, diffused, and undulating, and there is pain on pressure over the region of the ventricle. The brain becomes disturbed, and a restless delirium often sets in.

In endocarditis the pain may be developed less early than in

pericarditis, while the impulse may be more violent and felt over an increased area, and accompanied by a basic fremitus. The common physical signs are the diffused, rapid, and abrupt impulse with an increased intensity of the valvular sounds. The distinctive characters are, in pericarditis, friction sounds and, perhaps, an increased area of dulness from effusion into the sac of the pericardium; in endocarditis, murmurs with increased percussion dulness from consequent enlargement of the ventricular walls. In severe attacks of both these forms of inflammation, cerebro-spinal complications not unfrequently occur. In the attack, convulsions of an epileptic character may take place, and, subsequently, chorea. The proximate causes of these disorders are inflammation of the pericardium, with lymph deposits and effusions of serum; inflammation of the endocardium, with valvular degenerations; and of the muscular structure of the heart itself. The remote causes are the injurious effects of cold and moisture when the body, by overheating and exhaustion, is rendered susceptible of their injurious influences, and, especially, if there be the coexistence of a rheumatism in any of its forms, but more especially that of the acute form. So frequently are these inflammations of the heart and its membranes dependent on this latter cause, that they have been divided (Ormerod) into two classes—the rheumatic and the non-rheumatic. These latter comprise the advanced stages of albuminous diseases of the kidneys, inflammatory pectoral affections, as pleurisy and pneumonia, eruptive diseases, more especially scarlet fever and degenerations of the blood, as scurvy, ichorrhæmia, and pyæmia.

The predisposing causes of these affections are the gouty and rheumatic habit, especially when these occur in full and plethoric subjects, or are developed in the early and adult periods of life. It is generally affirmed that the youthful male is more prone to them than the female. I have more often met with them in the female, especially at that period of life immediately succeeding the establishment of the catamenia. For the most part, these cases have proved, from an early association with a chlorotic, or anæmic, condition of the blood, severe and very intractable. Examples often present themselves in the persons of domestic

servants; a class of persons well, and perhaps redundantly fed, but exposed to great alternations of temperature, to draughts, cold stone floors, and often sleeping in confined, ill-ventilated chambers.

The termination of these cases is, more often than might have been expected, in partial or complete recovery; but too often in chronic disease. Thus pericarditis may result in pericardial effusions and adhesions, and endocarditis in permanent mischief to any one or to all the valves; while both, more often than otherwise, set up disease in the muscular structure of the heart, and hence many of the degenerations that, in after life, are here met with. The tendency and the immediate results of pericarditis are to muscular atrophy; of endocarditis to hypertrophy and dilatation. So frequent, insidious, and gradually progressive, are these several effects that, in apparently perfect recoveries from lengthened and confirmed cases of each of these inflammations, a most cautious prognosis should be given.

It is probable that the first stage of these disorders is one of simple inflammation of the serous membranes, pericardial or endocardial, and that hence the muscle of the heart, without being affected, takes alarm, and the local anxiety, with slowness of pulse, ensues; that, in the second stage, the spread of inflammation to the muscular structure, and to the nerves and ganglia connected therewith, induces pain and rapidity, with irregularity of rhythmical motion; while, in the third stage, there are the evidences of exhaustion of power and degeneration of tissue.

The treatment must be prompted by these indications. For the incipient stage, inflammation is to be counteracted by the emission of blood and mercurial purges; the extension of the disease to the muscular and nervous tissues, by opium and alkalies, and more especially the bromides and iodides of potass; the third, or stage of exhaustion, by slight stimulants, restoratives, and tonics; and if there be the complication of a chlorosis the ferruginous tonics are especially useful. When occurring in advanced stages of Bright's disease, the bromides, with light tonics, appear to be occasionally useful, but it is a complication accompanied

by very great general and local distress. This distress is not always to be relieved.

Separately from, and independently of, all inflammatory affection, error of rhythm, both of impulse and of sound, may be induced by accidental and purely statical causes, such as the rupture of the tendinous cords, the rupture of a valve, accumulations of coagula in the ventricles, more especially in the right one, etc.; but these instances of disease are generally also associated with a murmur, and if their existence is to be diagnosed, it is rather from this morbid sign than from the rhythmical errors. They not unfrequently occur as curiosities of medical experience, and it must not be lost sight of that they may present themselves for observation.

Errors of rhythm may be observed, occasionally, under circumstances which lead to the conclusion they are mainly due to some diseased condition of the valves. In one class of these cases, the indications are a doubling of the pulse-shock, followed by a pause of ordinary, or somewhat prolonged duration. Both the pulse-shocks are feeble, the former of the two being obviously the weaker. The pulse under the circumstances may be said to be dicrotous. On examining the heart, there is also to be observed a double impulse, indicative of there being, for one pause, two ventricular contractions, rapidly and almost continuously following each other. The pause is ordinarily lengthened. The first sound, generally indistinct, sometimes passes into a murmur, and is always extended, even to being prolonged into the second sound. It may be stated in another way: the first sound is not only disordered in character, but is divided and lengthened by imperfectly completed systolic contractions, while the pause or diastole is not materially disarranged. Sometimes the pulse-shocks are deficient when the doubled impulses of the ventricle, though weak, are appreciable, thus presenting the characteristics of a false intermittence.

This doubled impulse appears to be due to the ventricle being inadequately supplied with blood, and in order to com-

pensate for this, after the first systole an imperfect diastole ensues, and is followed by a repetition of the systolic contraction. On investigation it is generally found that this inadequate blood supply does not depend on any muscular weakness of the ventricle, but is rather due to valvular disease, or to a feeble nervous energy. In the former case there probably exists some advanced constriction of the mitral valve, whereby there arises an obstruction to the free flow of blood into the ventricle. This constriction, if the diastole be active, induces murmur, but as it is usually weak and lagging, the sounds emitted are not well marked, either as valve sounds or as murmur. This form of rhythmical irregularity is, however, not necessarily due to this cause. It may be the result of a morbid and irritably feeble nervous energy, so that, succeeding a sluggish diastole, the first imperfect systole takes place, followed by another to compensate for the deficiency of the former one. But in this latter form of disorder the valve sound, though prolonged, is recognisable as a valve sound, and there is no accidental occurrence of murmur.

A redoubled pulse beat is also, occasionally, to be observed when there is regurgitant disease of the aortic valves. This is due to the ventricle reacting on the blood returned to it, from a dilated aorta, on the first commencement of the diastole, and before the perfect, and more efficient, diastole is effected.

The rhythmical character of the second sound is also sometimes altered. It is, at times, found to be intensified, and lengthened by the valves and contiguous tissues being thickened, or becoming flaccid, and inelastic, from antecedent inflammation. In other cases, the second sound is shortened, and this has been said to be due to a weak, paper-like character of the sigmoid valve. It is more probably due to a feeble, but rapid, contraction of the ventricle, in an irritable nervous heart.

Rhythmical error, having for its chief characteristic alterations in the valvular sounds, is conspicuously observable in another class of diseases—the nervous or functional blood diseases. These diseases may exist idiopathically or in conjunction with other diseases of the heart. When so conjoined, rhythmical

irregularity is always of serious import. Here we shall speak only of those cases which are independent of any such complication. Under these circumstances, if the sounds generally are intensified, the pitch of the first sound being heightened, sometimes even to the extent of being a short sharp knock, while the second sound, save in intensity, is not materially altered, we infer there is a nervous heart. In extreme cases, there may be so much ventricular irregularity as to induce, in place of the normal rhythmical sounds, an ill-defined "fremitus," and so much force as to induce a metallic ringing, with a rubbing murmur on the systole.

Thus the impulse of the heart, which in health is rarely appreciable, and its friction never, respectively become so to the touch and to the ear. These abnormal conditions may exist to such an extent as to entirely obscure the first sound.

The fremitus in these cases is neither perfect nor well defined, as it is when occurring in organic disease; at least, it is not found to be so in the heart itself. It may, however, be a well-defined fremitus, under the circumstances of functional disorder, in the aorta and in the course of the great vessels, and, in fact, often is so.

Occasionally a reduplication of the second sound is observable. Generally both the reduplicated sounds are more clear, defined, and uniform than when the reduplication occurs in connexion with organic disease. This may be accounted for by the fact that, in addition to the excited agitation of a nervous, but physically perfect, heart, there exists a spasmodic want of consonance in the action of the two ventricles, so that their systole is not effected in unison.

As a general rule, these cases of rhythmical irregularity are not subject to any complication with a globus hystericus, so frequent in other nervous affections.

Irregularity and rapidity of pulse appears to be in some rare cases the normal condition, instead of only a passing paroxysm. Three notable cases of this kind have passed under my observation. In each the habitual rapidity of the rhythmical irregularity was such as to defy any very satisfactory or perfect

analysis of the sounds—motion and sound were involved in a restless confusion. When under the influence of a febrile attack, the heart's action, in each of these cases, presented the curious phenomena of losing this habitual irregularity and assuming a tolerable regularity of systolic impulse, though weak and without energy, with a well-defined diastolic intermission. Sir Thomas Watson has described a similar case. Two of the cases, occurring in males, attained advanced ages; the other, a female, still survives, approaching a mature period of life. The physical examination indicated in each the presence of a small heart with feeble and most obviously irritable action. The general constitutional tendency was to repose; there was no great breadth or energy of mental power, with a very evident dislike to active bodily exertion: nevertheless they were not altogether incapable of undergoing a tolerable amount of it. This nervous irritability of the heart appeared to be coincident with special nervous irritability, marked, in one case, by occasional epileptic attacks; in another, by anomalous cerebral attacks which, in advanced age, developed into congestive and convulsive disease of the brain; in the third, the case that still survives, an anxious and nervous irritability is the chief characteristic. On my first seeing this lady, now some fifteen years ago, I found her pacing her garden in a fever of excitement, and, at a rate, which one could have scarcely thought possible with the soundest heart. She had not long before been formally told that she had a fatal heart disease. The suggestion that this might not be the case reassured her, and she soon resumed her ordinary composure.

Looking to the general history of these cases of nervous irritability of the heart, and comparing it with its occurrence under other circumstances, may we not assume that, if the heart be small, this condition of irritability and irregularity is not in itself indicative of fatal tendency, but that if it be associated with large or, in any wise, diseased hearts, the tendency is to a fatal termination.

Should the pulse be intermitting, with a marked deficiency of

power, and the valvular sounds be, at the same time, disproportionately intensified, the first sound having a clicking quality and the second shortened, the irregularity is concluded to be due to a thinning of the blood or a deficiency in its quantity, as in anæmia, chlorosis, scurvy, or exhaustion after hæmorrhages ; for the less the heart is loaded with blood, especially if it be thin in quality, the louder the sounds and the sharper their resonance. In hæmorrhagic exhaustion, aortic thrill is superadded ; in anæmia and chlorosis, thrill of the pulmonary artery. If the pulse be fluttering, irregular, and without force, with a false palpitation over the heart, and the pause is so prolonged as to pass into syncope, there is that nervous condition of the heart which, in the female, is associated with hysteria ; in the male with a low excitable nervous condition.

It may be assumed that intermittence is an undue prolongation of the pause, and that this prolongation is due to an inability in the heart to, rhythmically, resume its systolic action; are we, then, justified in asserting, supposing this prolongation of the pause on the one hand, and the absence of contracting power in the ventricle on the other, constitute syncope, that therefore intermittence, as evidenced in the pulse, is allied to, if it be not in quality, syncope ? The subject will be referred to in a subsequent page.

It must always be borne in mind that the above symptoms of rhythmical irregularity in the heart, being due to a morbid nervous excitement, may be greatly interfered with, even overlaid and masked, by the urgency and magnitude of the diseases that cause it, by the nature of the antecedent bodily condition, and by temperament, either separately or conjointly. The special description of these sources of difficulty, however, more particularly belongs to the history of each of these diseases.

The preceding illustrations of error of rhythm, whether with, or without, valvular disorder, indicate the existence of a disordered action in the heart, which may, or may not, be grave in its nature. The conclusions as to this must be mainly arrived at from the indications, which may be afforded by the ascertainable

conditions of the walls, and the valves, of the heart itself. Though, in all cases, the remote cause of rhythmical irregularity is probably a morbid condition of nerve power, due, may be, to a disordered condition of the circulating fluid, or of the structure of the heart; yet excepting in very rare cases, the proximate physical cause must be sought in the disturbances that ensue in the systole and diastole of the ventricles. Hence if rhythmical irregularity, however slight, or whatever may be its nature, be met with in conjunction with evidences of structural disease of the heart, it must be deemed to be of serious import. But it has been shown to exist independently of these, and under circumstances of less moment. It may be due to modified nervous influences, centric or reflex; due to direct mischief to the ganglia and the nerves that supply the heart; to mental and bodily exhaustion; to shocks and excitements, pleasurable and painful; to sudden changes of temperature; it may be caused by, and supervene on, many diseases, as gout, rheumatism, indigestion, fever, and inflammations; to the specific influence on the vagus of various poisons, and amongst these in some persons must be esteemed various articles of diet, more especially tea. This grateful, and most useful, article of diet to the many is, in its effects, singularly baneful to the very few. I have known it in the case of one person, who is generally robust, certainly has no heart disease, but is somewhat gouty, and, if indiscreet in diet, dyspeptic, produce immediately ill effects; a single cup of tea induces rhythmical irregularity, so that the otherwise ordinarily quiet and even pulse becomes small and hurried, and, if the use of the tea be persevered in, the distress of heart and the sleepless anxiety become urgent. A few doses of sal volatile, or of one of the fixed alkalis soon, in this case, restore the heart to its normal condition.

The predisposing causes of these rhythmical irregularities are, in young people the debility, on overgrowth; in older persons, the nervous temperament, and the gouty and dyspeptic stomach.

These attacks are, at times, very persistent; and, especially so, when coexistent with, and dependent upon, a morbid condition of the heart; in other cases they may, on the sub-

siding of the causes that induced them, rapidly and entirely pass away, and apparently leave no traces of their having existed. As a general rule they cause no subsequent disorder; or, if any follow, it is rather to be traced to the predisposing, and immediately exciting causes of the rhythmical irregularities.

In these cases we are justified in saying that danger is indicated when there coexists ascertained disease in the heart itself. The danger belonging to, and being measured by, the character and amount of this disease. Where no such ascertained disease exists, the rhythmical irregularity is not conclusive of the special physical condition of the heart, but it may be such as to conduce to a well-grounded suspicion of its existence; and may also aid towards forming a correct diagnosis. In other cases it indicates little else than dyspeptic irregularities, or anæmic states of the blood; or is the accidental symptom of inflammatory and constitutional diseases, etc., etc. In all these cases the prognosis must be derived from the urgency of the exciting disease rather than from the rhythmical irregularity. But whatever may be its immediate source the prophylaxis is to generally strengthen the nervous condition of the system, and to observe a regimen that may not result in indigestions, or the development of gout, or in any way conduce to exhaustion of mind or body. The treatment is, after ascertaining the source of the rhythmical irregularity, whether this be physical, or functional, dyspeptic, anæmic, or hysterical, duly to minister to these several affections, so as at once to counteract, and remove, the causes that induce it.

It was mentioned incidentally, in a preceding page, that in the course of these rhythmical irregularities the pause might be so lengthened as to pass into syncope. Seeing that the termination of life, in a large number of persons having heart disease, is immediately produced by this cause, it is, if not of importance, at any rate of interest, to consider the phenomena that characterize an attack of syncope, and to determine, if possible, its nature and origin. Syncope, or fainting, it is said, may occur instantaneously; generally speaking, it has a premonitory stage, usually of a few

seconds, or of a few minutes' duration, but generally sufficiently prolonged to give warning of the coming attack. This is marked by feelings of languor and præcordial anxiety, disturbance of the respiratory functions, giddiness, nausea, flatulence, partial sweats, obscurity of vision, singing in the ears, confusion of mental faculties, a marked sensation of "sinking" at the epigastrium, hurried and feeble throbbing of the heart, with an alarm, amounting, at times, to a conviction of approaching death.

When the attack is confirmed, and syncope takes place, sensibility and mental power are arrested; the pupils of the eyes are dilated; respiration is materially impeded; the muscles, voluntary and involuntary, save that of the heart, are powerless; the arteries are pulseless; the heart has no sensible impulse; and the valvular sounds are feeble, toneless, and hurried; but they exist, and both are to be traced, though at the apex it is not easy to recognise the presence of the second sound.

If we carefully consider the nature of the above symptoms, we see they may be referred to two categories; those affecting the nervous system, and those affecting the heart. What, then, is syncope? Is it a nervous or brain affection (leipothymia); or is it a heart affection? Seeing how coincident the various symptoms are, it may be difficult to unravel this problem. The nature, sequence, and the complications of the phenomena may assist us.

Doubtless syncope cannot be said to be complete without a cessation, almost to annihilation, of the heart's action. Feeble though the contractions of the heart may be, still it contracts. The functions of the respiratory system and of the brain are equally, if not more completely, suspended; and the same may be said of the nervous system generally. All the functions of the body, voluntary and involuntary, save that of the heart, which depend on the exercise of a healthy nervous energy, are effectually arrested. It would, therefore, appear that in a completely accomplished attack of syncope, the heart is the only organ that retains power—diminished and imperfect though this be; as long as this faint echo of power exists, life is preserved; and only ceases, if the attack be so protracted, or so severe, as to

effectually annihilate this power. It is said the rhythmical action of the heart cannot be arrested for a longer period than a minute. Recovery is then impossible, and death ensues.

Now, what are the conditions, and the exciting causes of syncope? Is it necessary that there should be a feeble, or a diseased, heart for its development? Certainly not, for we find that it constantly occurs in those in whom there is not the slightest ground for assuming the existence of disease or feebleness, or even tendency to disorder, of the heart. On the other hand, the remarkable fact presents itself, that every variety of diseased heart may exist, and run its course to a fatal termination, without the slightest manifestation of syncope. Doubtless syncope occurs more frequently in those who have diseased hearts, than in those not so affected; and, in the history of heart disease, the consideration of this disorder is an important element, as the termination of life is often due to its occurrence; but the larger number of those afflicted with heart disease do not die from this cause. If we carefully analyze the symptoms of a syncope, we find the greater proportion of them intimately allied to many of those, essentially, characteristic of disorder of the nervous system, and that these may occur, independently of any complication, with a fainting at the heart. The after effects, too, of an attack of syncope are more often those of the nervous system; even to relaxation of the sphincters, convulsions, or partial paralysis. Moreover, the symptoms of syncope developed in the heart are allied to, if not identical with, many of the manifestations of disorder which take place in this organ, many of which have just been described as illustrations of the nervous rhythmical disorders of the heart, and which are notably caused by disorder in the nervous system; such as those ordinarily induced by indigestion, fright, mental emotions, and similar causes.

On considering all these circumstances, may we not assume that syncope has its primary origin in, and is essentially a disorder of, the nervous system, and that one of its main and characteristic seats is in the heart; that if the heart be naturally feeble, or if it be diseased, it is not only more liable to the attacks, but the attacks themselves are correspondingly more developed, and

more important, as regards the safety of the individual? Under these circumstances, if the nervous energy be too long withheld, the heart is less able to exercise a power of restoration, and to re-establish its adequate rhythmical phenomena. The same may be said of an attack of syncope when there is confirmed disease of the brain. Though there be confirmed disease, whether in the brain or in the heart, it will be understood that syncope often occurs without inducing a fatal termination.

I will here refer to three typical cases that have recently presented themselves to my observation. A lady, who had been recently the subject of a slight attack of bronchitis whilst standing with her back to a hot fire, suddenly exclaimed with an expression of alarm, "I am falling," and threw herself into a chair. She immediately lost consciousness. The respiration was partially suspended, but the heart's action was scarcely disturbed. After two or three minutes, she so far recovered as to complain of pain in the head. She neither referred to, nor remembered to have experienced, any alarm of the heart. The subsequent disturbance was cerebral.

A gentleman of advanced years, of uniformly good health, quiet and most regular in his habits, was alarmed whilst taking his usual walking exercise by a sudden and frequently recurring giddiness. He assured me that, for a moment, he evidently lost consciousness, and, had he not previously made a broad base with his legs, he should, as he anticipated, have fallen to the ground. He, at the same time, experienced a fluttering or palpitation of the heart. From the general history of this case, I concluded these attacks were due to some acid indigestion, and a few days' medical treatment confirmed this, as he was entirely relieved from any recurrence of the attacks. The general symptoms did not suggest the presence of any disease of the heart, but the indications of the pulse lead to this conclusion; and, on examination, this proved to be the case. The apex beat was to the left of the nipple, and there were also the other indications of dilatation and weakness.

A lady of middle age, who for some years has been, presumably, the subject of hypertrophy with fatty degeneration of the

heart, and, more recently, of a confirmed albuminuria, presents the following symptoms:—The pulse is small but regular, the breathing unembarrassed, and there are, apparently, neither cerebral, pulmonary, nor stomach complications. She is very cumbrous from œdematous swellings of the abdomen and legs, so that for some time she had not taken rest in the horizontal position. Whilst in this state it was sought to relieve her by active purgatives, but in the midst of doing this, a distressing state of failure in the power of the heart's action supervened, occurring in paroxysms, lasting, may be more or less, for an hour. Though speaking of faintness, and showing, as regards the pulse and the respiration, a considerable amount of debility with disturbance of rhythmical action, she neither exhibited nor experienced any cerebral distress.

If we regard carefully the symptoms of the preceding cases, we see that the first is evidently one of well-marked syncope without any organic complication, the attack being due solely to morbid nervous influences. The second is an illustration of vertigo—probably a transient form of syncope—occurring in one, who, as it so accidentally happened, had a structurally diseased heart, and which was thus more obviously affected than it otherwise might have been. The third case is probably not one of syncope, but of a true physical failure in the power of the heart caused by nervous exhaustion. The heart itself being in the last stage of structural disease and inefficiency.

But syncope may, and often does, take place in confirmed cases of heart disease, more especially of the nature of hypertrophy, and of dilatation with fatty degeneration. I have seen, in such cases, hæmorrhages of very considerable amount, concluded by the occurrence of a perfect, though not fatal, attack of syncope; the pulse being weak and the pause prolonged with the most entire suspension of mental and nervous power. Such an attack has lasted for many minutes, and then, perhaps, after a few long-drawn sighs, and a struggle towards, but not to the recovery of, consciousness, the state of syncope has recurred. The return of conciousness appeared always to be the indication of the termination of the attack. Cases of an analogous

kind to those now enumerated, but with varying symptoms, constantly occur. There may be cerebral incompetency without entire failure of the heart; and there may be failure of the heart without cerebral incompetency.

If we carefully consider these several classes of cases, we find that, in those which are marked by the general and specific symptoms of a syncope, the pulse-beat is not more feeble, nor the diastolic pause more prolonged, than often occurs in similar, as also in other, diseased conditions of the heart, without inducing syncope; and that in other cases syncope may occur without the existence of a prolonged diastolic pause. But no complete attack of syncope can take place without the entire suspension of consciousness and voluntary power. Supposing the above to be a true representation of the phenomena, we may conclude that failure in the power of the heart is not the specific cause but only a feature of syncope. That, in fact, syncope is really a *deliquium animi*, a leipothymia, and not, primarily, an affection of the heart.

The heart affection, in an attack of syncope, is probably due to some morbid condition of the nervous system. There may be interference with the vagus supplying the heart; but, more probably, the seat of the affection is in the sympathetic ganglionic system, and the nerves and ganglia thence proceeding would then communicate to the heart, as to other organs, the widely-spread manifestations of the syncope thus originated.

The practical bearing of the question is that, irrespective of the nature of the disease which exists in the heart, the assurance may be entertained, where there is no general liability to syncope, that the subject of such disease will not thus have his life suddenly cut short; and, where there is this tendency, means general and special should be taken to counteract it.

CHAPTER IX.

THE PATHOLOGICAL INDICATIONS OF ENDOCARDIAL MURMURS.

THE pathological indications, to be derived from the errors taking place in the normal phenomena of the heart, have been hitherto considered irrespective of the occurrence of murmurs. In a preceding chapter (IV.) these have been described, together with their origin, in relation to the several valves, and the circulating fluid. Here it is proposed to consider the disordered conditions with which they are associated, in order to discriminate, so far as may be, the extent to which valvular disease, presumably, is the measure of danger to health and life. To do this it is necessary to consider the circumstances causing, and caused by, them; or in any way influencing, or associated with, their existence.

These murmurs may be caused by lesions to which the valves are subject, mainly the results of inflammatory and degenerative processes and statical injuries, and of congenital imperfections; by the want of a proper adjustment of the valves they have to close; as, also, by the nature of the fluid that passes through these apertures. The valves may be thickened—have deposited upon them false membrane, lymph, earthy, bony, and atheromatous matters. They may be stiffened, wrinkled, and uneven; may be adherent to neighbouring parts and to each other; may be the subject of ulcers and erosions; and of violences and injuries causing disruption of their normal continuity. Hence they acquire conditions which neither admit an even, or full, flow of blood through the apertures to which they are attached, nor

adequately close them against its return. The valves, though healthy themselves, may, however, prove to be inefficient from disease in neighbouring parts. The most ordinarily occurring of these are dilatations of the walls of the ventricles by which the apertures are so enlarged that the valves become insufficient to close them, as was their wont; or the murmurs may be caused by the blood itself; this being so altered whether in quantity or quality as in passing through normal apertures to originate sonorous vibrations. These several valvular lesions, and sources of murmur, may occur separately, or in association with each other. When considering the phenomena incidental to a case of murmur, whether simple or complicated, it must be borne in mind that, beside the physical valvular condition which may originate a murmur, and which condition may be fixed and unalterable, the question of "force," consequent on heart power, must be taken into account. Force, more or less, is necessary for the manifestation of a murmur. It may be that a murmur, which under a given amount of force is always to be recognised, may, on a diminution of that force, be materially lessened, or absolutely undeveloped. This evanescent character of murmurs is conspicuously observable in cases of very advanced valvular disease. It will, therefore, be understood that extent, and intensity, of a murmur are no sure measures of the amount and nature of disease.

The appreciation of this modification of a murmur is not only, under such circumstances, of importance, but it has a further practical bearing; for it is sometimes necessary, where there arises the suspicion of a murmur, to test it by inducing some unwonted amount of action in the heart.

Though murmurs, persistent for years, may thus entirely disappear, it is but rarely found that the normal sounds are re-established. They are superseded by muffled and obscurely marked sounds, recognisable neither as murmurs nor rhythmical valvular sounds, but only as roughened, or "to-and-fro," vibrations. Nevertheless in some cases murmurs, which, for a time, have taken the place of normal valvular sounds, may pass away, and the valvular sounds be again heard. In the former case, debility

and loss of power, are, generally, the source of the disappearance of murmur; in the latter the subsidence of valvular insufficiency, or the restoration from a morbid condition of the blood. Hence, not only the condition of the valves, but the condition of the walls of the heart, or the motor power of the blood, and the health of the system, as influencing the condition of the blood, become elements of serious consideration.

Before referring to the value of the murmurs occurring in the several valves, we are, from experience, fully justified in asserting, as a general proposition, that the existence of any one of them, if there be also present other recognisable disease of the heart, whether active or chronic, is to be considered of serious import; and experience also justifies us in stating that neither of them, without such complications, is to be classed as symptomatic of necessarily fatal disease; for, apparently, very marked congenital or subsequently acquired murmurs may exist for the natural duration of life, and, in some instances, even without ostensibly inconveniencing the subject of them.

Nevertheless murmurs have their significance, and, for the most part, this is to originate a suspicion of the existence of valvular injury; and that this injury is indicative of danger, present or prospective. But can we from the murmur itself precisely indicate the nature and the amount of the valvular injury, or can we predicate its consequences? We can do something, but we cannot do all. We can recognise systolic from regurgitant disorder, and that is information of the greatest significance; but beyond this we can do but little.

A feeble sound may be associated with the worst of valvular injuries, while one that is loud and harsh may be caused by injury comparatively unimportant. This seeming divergence, of cause and effect, is so marked that the axiom has been propounded "the louder the murmur, the less the actual amount of mischief inflicted." For the true indications of the state of the valve and its vital influences, we must look to other, and more general conditions than those, solely, of the murmur, which emanates from it.

The pathological anatomist details the valvular lesions; the physician determines their source and origin. The chief of these, besides congenital errors, may be ranged under the several heads of endocarditis, more especially of the rheumatic character; gout, and the gouty diathesis; and violences.

The tendency of endocarditis to induce valvular diseases is so conspicuous, that the history of their existence could not be considered without reference to this source of origin. They are not only the immediate consequences of an acute attack of this disease, but are developed in the course of its chronic and subacute form.

The progress of endocarditis in its chronic form is often very insidious, for pain is not a necessary element of its existence. There may be general febrile and other serious symptoms, but little directly to indicate that the heart is the special seat of mischief. The specific effects of inflammation of the endocardium, and of the valves, are thus often fully developed before suspicion has been aroused. They occur at all ages, but more notably so after the middle period of life.

The general symptoms are a slight febrile state; great prostration of strength; a quick and irregular pulse; suddenly occurring palpitations; rhythmical irregularity and intermissions, with præcordial anxiety, or even oppression. But the weakness and general symptoms are more marked than is the local distress. Though there may be no distinct evidence of valvular disease, there are indications of disturbance. The first sound is soft, obscure, and blowing; the second diminished in sharpness. As the disease progresses, various difficulties may ensue, and amongst these are, notably, structural changes in the valves, and in the walls of the ventricles, and then the evidences of valvular lesions, and of hypertrophy, manifest themselves.

In more severe forms of chronic inflammation of the endocardium the heat, thirst, restlessness, and accelerated and irregular pulse, are, sometimes, more marked. The attack assumes a paroxysmal character, occurring so regularly, as to simulate a case of ague. In this form the murmur, still soft, is difficult to

define from the rapidity and rhythmical irregularities of the heart's action. It is probable that, in each of these forms, there is also some degree of myocarditis, as there undoubtedly is when the endocarditis is of an acute character.

Experience also shows that valvular disease sometimes follows symptoms of even a less marked, slower, and more insidious form of endocarditis. With slight occasional attacks of dyspnœa, and some passing feeling of oppression of the præcordia, there occur an abiding tendency to despondency, restlessness, and want of sleep. These latter symptoms subside after a time, but rarely before the valvular disease is confirmed. This form of chronic endocarditis is mainly due to the gouty diathesis, and affects the aortic valves. The preceding forms belong rather to the class of rheumatic inflammations, and chiefly affect the mitral valve, and to a less extent the aortic. A case illustrating the chronic gouty form presented itself to me under peculiar circumstances. I examined a gentleman aged 63, for an assurance office. Of a gouty habit, he was, as regards the heart, free from all suspicion of disease. Three years afterwards, he having, in the meantime, fallen into an anxious, restless, and an hypochondriacal state, but from all of which he had entirely recovered, I re-examined him, and found, to his surprise, a well-marked systolic aortic murmur; for he had experienced no pain in the region of the heart. He maintained his wonted energy for six years, and then infirmity accumulated rapidly upon him. At the age of 74 he succumbed to all the inconveniences of confirmed heart disease.

The results of these inflammatory diseases of the endocardium, whether acute or chronic, are, as regards the valves, the lesions so commonly observed. Those of rheumatic origin, for the most part, commence in lymph deposits, those of gouty origin in atheromatous degenerations. The deposits, which are at first soft and of an uniform round shape, in due time, flatten, become warty, or cartilaginous in density, and then the seat of bony deposit. The valves otherwise are thickened, sometimes glued down, or portions glued together. False membranes or bands may be formed, and the endocardium generally is

thickened, indurated, roughened and loses its normal polish. The valves both aortic and mitral, are thus structurally and functionally impaired; the aortic, more often than the mitral, in the chronic and gouty forms of disease; the mitral valve in the acute and rheumatic forms, but neither valve is exempt in either case.

The injuries, which the valves experience from violences, are, mainly, examples of simple rupture of the fine muscular structures attached to the mitral and tricuspid valves, or of one, or more, of the segments of the aortic valves. They are but rare sources of mischief, and rather belong to the curiosities of medical experience.

Valvular diseases, however induced, are sufficiently bad in themselves, but, for the most part, they do not stand alone. They are found intimately associated with many of the diseases occurring in the important organs of the body. The more prominent of these are diseases of the lungs, with nervous disturbance of breathing, congestions in the liver and kidney, œdema, and dropsy; but the most serious are the structural changes in the muscle of the heart itself—more especially hypertrophy and dilatation. It is commonly stated that hypertrophy and dilatation are the mechanical results of valvular obstruction, and also that the operation of the hypertrophy, thus induced, is of the nature of a serviceable compensation for the, otherwise, injurious consequences of the obstructive disease. I have ventured to suggest, where they are associated together, that the mechanical origin of the hypertrophy is not, in all cases, evident; and I would add that the theory of the hypertrophy being a serviceable and beneficial compensation, against the evils of obstruction, is not tenable. Doubtless valvular disease, and hypertrophy, are commonly coexisting conditions; but, for the most part, the exciting causes of the former are also sources of the latter; and examples are not infrequently presented for observation in which the valvular obstruction is considerable, and presumably of long standing, and yet there exists but little or no hypertrophy. As regards

prognosis, the question is an important one; and, at any rate, is worth examining from another point of view than that of the recognised one. The issues raised are, whether the hypertrophy associated with valvular lesion is due to statical causes depending on this lesion, or is it of independent and vital origin?

Cases have occurred within my own observation in which there has been the extremest amount of aortic valvular disease, with little or no ventricular enlargement; and similar cases are recorded in nearly every work that details the pathological anatomy of heart disease. Dr. Peacock details two cases that may be thus classified; and Dr. Tilt, who, while saying that enormous enlargement is observed in cases in which the contraction or insufficiency is small, states, on the other hand, that in some instances in which the obstruction must have been extremely great, the size of the heart has been found slightly or not at all increased; and he refers, especially, to a case in which the aortic orifice was so much contracted as hardly to admit of the passage of a small probe. Dr. Chambers, in his "Decennium Pathologicum," states that, in the cases he analyzed of chronic disease of the valves, including mitral and aortic combined, aortic alone and mitral alone, the walls were healthy in 30 per cent. Disease of the aortic valves alone is stated to be about 25 per cent.

From the history of some of these cases there is reason to infer they were of congenital origin, and thus a life-long existence of the extremest obstruction had failed to induce hypertrophy, or, in some of them, even the suspicion of the existence of heart disease.

Let us consider the converse of these conditions; the presence of well-marked and extensive hypertrophy, with only the slightest amount of valvular lesion, and such as, it may reasonably be assumed, to produce neither obstruction nor regurgitation. Post-mortem examinations very constantly exhibit these conditions; a few slight vegetations on the faces or edges of the valves, with largely thickened walls. Then we have the further fact that mitral obstruction, without the least trace of any aortic valve

disease, and, therefore, supplying none of the elements, according to the mechanical theory, for increased energy in the ventricle, is almost as constantly associated with an hypertrophied condition of the walls, as is aortic valve disease. Some of the most remarkable cases of hypertrophy have occurred under these circumstances.

Cases also occur in which the right ventricle is hypertrophied as well as the left, though the aortic valves are alone diseased, and though at the same time there are no indications of any coexisting pulmonary congestion; and then there are the numerous cases in which hypertrophy exists, though there be no valvular disease. The phenomena, which are generally attributed to valvular disease, are notably observed in these cases.

If we look to the origin of valvular disease, we find, in the larger proportion of instances, it is to be referred to causes which also produce hypertrophy, but more especially to endocarditis in some one or other of its forms. This promptly induces myocarditis, and, consequently, structural disease in the walls, which is generally fully established before the valvular disease; so that instead of the hypertrophy being the result of the valvular disease, it not only coexists with, but precedes, it.

It may perhaps be said when rupture of the aortic valves takes place, through violence, that it is subsequently found there is a condition of ventricular hypertrophy; but it does not follow that, in these cases, there may not have been, on the one hand antecedent wall disease, or, on the other, that it might not be set up by the irritation thus caused to the heart generally, and to the endocardium more particularly. At any rate, looking to the amount of obstruction and regurgitation induced under these circumstances, and also to the amount of hypertrophy, there rarely exists sufficient physical cause, or time for its development, to permit the conclusion that the hypertrophy has been the result of merely mechanical operations.

Seeing that the aortic valves may be obstructive, without inducing hypertrophy, and that hypertrophy is generally a con-

dition antecedent, and therefore independent of valvular disease, one may reasonably infer that the hypertrophy is not the statical or mechanical result of the valvular disease, but that it has an independent, though probably coexisting vital origin. It is doubtless probable, if disease be set up in the ventricle, that the effects of subsequent or contemporaneous valvular disease may serve to irritate and increase the ventricular disease; but judging from the cases, congenital and otherwise, in which no hypertrophy has been developed, it is also probable that valvular disease is not adequate, in an otherwise healthy heart, to set up disease, or to overcome the strength and elasticity of its muscular fibres. If we take the various elements of heart disease in their ever changing proportions of valvular obstruction, of hypertrophy, and of the existence of either, or both of these, in their relation to the duration of time, it is difficult to arrive at any law of cause and effect. We see everywhere a coincident, and mutual, origin and dependence of disease; and that congestion and inflammation in one portion of an organ affect other portions of it or other organs. Hence the disease in one portion of the heart may induce disease in another portion; and though there may be a consenting dependence and mutual influences in all the larger organs, the lungs, the liver, the kidneys, the brain, it yet by no means follows that these diseases are all to be referred to statical causes, to the exclusion of those which are vital and independent in their origin.

It is generally understood that dilatation is preceded by hypertrophy, and this is probably the case. The inflammatory processes which primarily induce the hypertrophy, sooner or later subvert the muscular structure of the heart, and then dilatation, partial or general, sets in. It may fairly be assumed that, in such event, the ponding back of the blood by valvular obstruction has its direct influence.

In respect to the second position, that in the accident of valvular obstruction, the enlargement of the ventricle is conservative and compensative, a long and careful clinical study of the

cases in which these conditions coexist, has assured me that the opposite is the result; that valvular disease is comparatively harmless so long as the walls are not diseased, but if they be diseased and show activity in this direction, then danger threatens; and that where they both coexist, whether in an active or chronic form, the difficulties that arise are rather those proper to hypertrophy and to dilatation, and such as are found to prevail without valvular disease. That in fact there are, superadded to the valvular difficulties, the injurious consequences of inflammatory and vital morbid processes in the more essential portions of the heart's structure.

The general statement is that the increased power of the ventricle compensates for the smallness of the aperture and for the obstruction thence resulting. Is this position tenable? Is it a physical truth that the smaller the aperture the greater the force required to project fluid through it. The very opposite appears to me to be the hydraulic law here applicable, the larger the aperture the greater the power required to project fluid through it. A disproportionate amount of force applied to the projection of a fluid through tubes may strain and rupture these tubes, provided they be elastic or destructible as are the valvular openings, while the fluid itself, being neither elastic nor capable of compression, affords no compensation to the force applied. If this be true, any unwonted force, such as is assumed to be the case in hypertrophy, applied to a diseased and obstructive valve, supposing also that more blood was forced through it than was proper to its restricted aperture, would result in the mischievous consequences of undue dilatation or rupture. If increase of force were the compensation for smallness of aperture, any means whereby this could be effected would be beneficial. Now we know, as a clinical fact, that increase of force in the case of valvular disease is especially injurious, for in cases where there may, or may not, exist evidence of hypertrophy or of dilatation, any means which augments the contractile power of the heart, as unusual exertion, emotional feelings, etc., immediately induces marked inconvenience, and perhaps urgent breathlessness and pain in the region of the heart. Accelera-

tion of rhythmical action, without increase of force, if not conservative might, theoretically, be deemed compensative; but experience shows that, if the valves be diseased, both increase of force and acceleration of rhythmical action are injurious; while safety remains in quietude both of mind and body.

Hitherto the murmurs referred to have depended on valvular changes, but there are also the murmurs which have their origin in a deficient amount, or a disordered condition, of the blood. These inorganic murmurs have characters that soon realize their true origin to the practised ear. They are systolic, basic, and chiefly heard in the præcordial region, with conduction in course of the great vessels. There is no apex murmur, but, at the apex, synchronously with the murmur, the first sound is clearly defined, and has a metallic ringing sound. The tone of these murmurs is musical, cooing, soft and of low pitch, and with these characters they are traceable for some distance in the contiguous large vessels. They are remittent, and not intermittent, as is the murmur transmitted through the jugular veins when there is insufficiency of the tricuspid valve. As a rule they are always accompanied by palpitation, which is constant, while the murmurs are not so; they may occur suddenly, and as suddenly subside, or they may persist for days and weeks. The palpitation generally has quickness, is spasmodic, and does not convince one of force, it has activity but not power, but when excessive, the murmur is increased in tone, and sometimes, indeed, is only to be traced when the heart is excited or agitated.

The immediate causes of these inorganic murmurs are (1) deficiency in the general amount of blood; and (2) alterations in the proportions and quality of the constituents of the blood. The former constitutes anæmia; the latter comprises spanæmia, chlorosis, and the allied degenerative blood diseases.

The anæmic murmur has its seat in the aortic valves, and is readily traceable through the larger emergent arteries; but it is not a murmur of frequent occurrence. It is remarkable the

large amount of blood, and the frequency of the discharges, which may be drained from the system, provided there be no diseased condition of the blood itself, without inducing the presence of a murmur. Under these circumstances, though there be no murmur, the first sound is generally flapping in character, and the second sound ringing in tone.

The general symptoms of anæmia besides those of the heart are extreme debility marked by hurried breathing, anxiety of the præcordia, and palpitation on the slightest exertion, even to syncope of an alarming character. The pulse is small, quick, feeble, and jerking; and I have met with aortic thrill, though it is said this only occurs when the aortic valves are diseased. The countenance is pale, thin, and transparent; the lips and mucous surfaces are, as it were, bloodless; there is no œdema; the bowels are confined; the urine scanty; the appetite feeble; drinks are agreeable. With great nervous sensibility there is languor, and the tendency is to inaction; mental exertion fatigues; there is headache, and at times, throbbing of the temples, and the noise in the ears is of the greatest intensity—a lady once described it to me as that of a steam hammer;—the sight is feeble and disturbed by "muscæ volitantes." It is chiefly due, besides hæmorrhages, active and passive, to deficient nourishment, and to scurvy. These cases do not bear any purging or depletive measures. The cure is in rest of mind and body, good food, bark, wine, light ferruginous remedies, opium, fresh air and cold sponging, and the special avoidance of all purgative or depletive remedies.

The murmurs in chlorosis and spanæmia, and when they occur in ichorrhœa and leucocythæmia, have their seat for the most part in the pulmonary valves, and are not traceable in the course of the larger arteries. They are generally associated with the venous hum to be heard in the jugular veins. The general symptoms are debility more or less marked with an abiding hurry of breathing and palpitation, greatly increased on exertion. Though syncope occasionally occurs, there is not the marked tendency to it there is in anæmia, nor is its danger so

urgent; it partakes more of the character of a hysterical fainting; there is anxiety of the præcordia, with pain, dull, heavy, and persistent, below the left nipple; the pulse is small, weak, and quick, throbbing or bounding, often intermitting; the countenance is pale, puffed, doughy, and wax-like, and presents that character which has given to this disease the name of the "green sickness;" blushing is easily excited, giving brilliancy of expression to the eyes, the lips are bloodless, the tongue pale, coated, and enlarged, and generally exhibits the impressions of the teeth; there is a general tendency to puffiness of the integuments, and often to œdema; the bowels are confined and flatulent; and the peristaltic movements often accompanied by a borborigmus; the urine, large in quantity, is pale, free, and of low specific gravity; the appetite is capricious, sometimes excessive, more often feeble and depraved, desiring unusual aliments, and rejecting those ordinarily used; the breath is loaded, and there are evidences of congestion of the liver; piles are not infrequent; exertion both of mind and body is irksome; the sensibilities are blunted, and there is a tendency to hysteria, with singing in the ears, headache, and vertigo.

It is in the course of the protracted and chronic form of these affections the peculiar swellings of the thyroid gland, and the protrusion of the eyeballs (exophthalmos) occur. In this district the complication of swelling of the thyroid gland is often met with, and occasionally cases of exophthalmos. Within the last four years two cases of this latter have been under my own charge, and one recently, in the person of a young woman, under the care of Dr. Lewis Shapter. The symptoms were severe and aggravated, and she succumbed to the exhausting effects of the disorder. During life there was an abiding systolic murmur, with undulating pulsation diffused over the pulmonary area; the pulse was small and quick, and the respiration hurried and easily excited. The thyroid swelling materially interfered with the taking a due amount of nourishment. The main feature as regards the heart exhibited on the post-mortem examination was dilatation of the right ventricle, but with no disease of the

pulmonary valves, nor obvious insufficiency of the tricuspid. It may therefore be assumed the murmur heard was a blood murmur and not organic.

The causes which predispose to chlorosis and spanæmia are the strumous diathesis, the hysterical, hypochondriacal, gouty, and dyspeptic constitutions; and their development is excited by bad diet, overwork, the sleeping in ill-ventilated rooms, rheumatic fever, late hours, exhausting pleasures, indiscretions of youth, and those social positions which induce a morbid state of the moral feelings, sorrow, vain longings, and, in females, the general causes that induce amenorrhœa, and other irregularities in the uterine functions.

The treatment consists in strengthening the system, more especially by the aid of steel medicines and the warm gums, unloading the liver and bowels with aperients, fresh air, skin frictions, the cold bath, and in rousing the torpid system to healthy exertion both of mind and body.

When these morbid states of the blood coexist with organic disease, they aggravate, and render it, more painful and urgent, and more difficult of alleviation. They also complicate the diagnosis of its murmurs. This difficulty frequently presents itself after an attack of acute rheumatism, when the systolic spanæmic murmur masks a systolic mitral, and, may be, even an organic aortic, murmur; and the same may occur with other chronic valvular murmurs. Whenever there is a blood murmur some caution must be exercised in pronouncing the non-existence of a chronic murmur. After an inorganic murmur subsides there sometimes remains for observation an unsuspected pre-existing organic murmur.

Besides the inorganic murmurs just described, endocardial murmurs are met with, which apparently depend on functional disorder in the actions of the heart itself. These are chiefly exhibited under the influence of morbid excitements, specific or general. The murmurs heard occasionally in chorea and epilepsy, and after violent exercise, are of this nature.

The question arises what is the immediate origin of these endocardial inorganic murmurs. Is it due to the mechanical consequences of the alterations in the quantity or density of the blood; or is it due to irregularity in some of the rhythmical actions of the heart from false stimulation? When murmurs are produced by over-exertion, or in chorea, epilepsy, etc., we reasonably assume them to be due to faulty muscular or tendinous operations. The immediate cause of blood murmurs is not so easily stated. In anæmia there is a lessened amount of fluid, and that which remains may be altered in quality; in chlorosis and spanæmia the blood is lowered in density, has relatively a less proportion of its red constituents, and these are altered in quality, being smaller in size. Doubtless the less dense the fluid passing through tubes, the more high-pitched and distinct are the vibrations of sound caused by it; oil or treacle forced through a French-horn or the tube of an organ would fail to produce the clear vibrations which the air does. This law of the production of variety in the tone and character of sound by fluids of different densities may, to a certain extent, be applicable to the generation of these blood murmurs. It must, however, be borne in mind that the blood being lessened in quantity, or altered in quality, morbidly excites the heart to irregular action, and that this irregular action is violent and spasmodic in character, and is, without intermission, abiding in duration, while the murmurs are usually, not persistent but, remittent. The immediate origin of these inorganic murmurs is a subject of much interest, and well worthy of accurate investigation.

The organic murmurs emitted by the several valves may be now briefly considered; and, in doing so, it will be convenient to refer, first in order, to those of the mitral valve. They are either systolic, regurgitant, or both, and argue greater or less disease of the valve or its inaccurate adjustment from disease of the ventricle. Obstructive disease in the valve may exist with, or independently of, regurgitant incompetency. Theoretically a diastolic, or, as it is now generally styled, a pre-systolic, murmur

should, thereby, be heard. Though the obstructive disease may, by reason of hardened deposits and contractions, so narrow the aperture that the flow is materially lessened in amount, yet no recognisable pre-systolic murmur may be present. This is generally to be accounted for by the feebleness of the ventricular diastole, so that the force by which the blood is drawn through the valve is not adequate to produce a murmur. But should this obstructive or flow mitral murmur really exist, it must be pre-systolic. Nevertheless, it may neither be distinct nor recognisable, as there almost always coexists a regurgitant murmur, and the pre-systolic runs into and is, as it were, absorbed by the more sonorous regurgitant murmur. The pre-systolic is better appreciated by its occupying the time of the normal pause, or rest. The pause is diminished, the murmur is prolonged, and is soft and musical in its commencing tone, becoming sharper and harsher towards its close. Should there be no regurgitant murmur there may be a doubtful first sound, but this would only be appreciable on the right side of the sternum, and is probably not due to the closure of the mitral valve, but to that of the tricuspid. But, be this as it may, the pre-systolic is very likely to be confounded with a regurgitant mitral murmur, and into which, practically, in almost all cases, it merges.

The presence of an obstruction in the valve necessarily limits, so far as it exists, the amount of blood supplied to the heart; and this deficiency, by withholding from the heart its natural stimulus, generally induces rhythmical irregularity, and, at the same time, distension of the auricle and pulmonary complications, in consequence of the blood being ponded back in the auricle, and upon the lungs. Whether an obstructive condition of the valve may, or may not, produce a murmur, it will most assuredly induce the general symptoms and inconveniences which belong to the condition of insufficiency or regurgitation usually indicated by the systolic or regurgitant murmur, but probably not to so grave, or so persistent, extent. For although the blood is in both cases ponded back in its flow, or forced back upon the lungs, so that there ensues an equal

amount of congestion, and of deficiency of blood flowing onwards from the heart, and, therefore, an inadequate systemic supply takes place, yet obstructive mitral disease is not necessarily associated with disease of the left ventricle; as is so very commonly the case when mitral insufficiency exists. Moreover the ill effects of mitral regurgitation are likely to be more rapidly and energetically developed, than are those of obstructive disease, by reason of the systolic being greater than the diastolic force.

Though the mitral regurgitant murmur may be caused by a diseased condition of the valve, which both permits regurgitation and induces obstruction to the flow, it may also be due to causes which are entirely independent of any such conditions. There may be a regurgitant murmur though the mitral valve be not in any way diseased. It may, in fact, be due to any of those causes which induce deficiency of closing power; and conspicuous examples of this want of adjustment occur, when, though the valves are healthy, there is extensive dilatation of the aperture or disease of the walls, especially towards the apex, of the ventricle, or rigidness of the tendinous cords.

The existence of these several sources of valvular incompetency constitutes disease, and experience shows that in some cases it may arise from permanent and incurable physical conditions, whilst in others it may be due to temporary ailment only. As the disorders induced by permanent disease of the mitral valve are generally serious in their nature, and painful and fatal in their tendencies, it is useful to discriminate between those that are transient and those only susceptible of alleviation.

The general symptoms and the consequences of an obstructive mitral flow and of regurgitation are, in the main, very similar, and may, as far as any practical inferences are concerned, be considered together. Bearing also in mind that the predominant murmur generally heard, though the physical conditions of obstruction may exist, is almost invariably systolic and

regurgitant, and presents the character of being usually soft in tone, low in pitch, and prolonged; and, also, that it takes the place of the first sound. Being systolic it must not be confounded with the aortic flow murmur. As regards the left ventricle there are only two systolic murmurs—the mitral regurgitant and the aortic flow. This latter, as will be shown, may be due to functional, as well as to statical, causes, while the mitral murmur is only due to statical; morbid conditions of the blood not appearing to be adequate to produce it. Hence the murmur of anæmia, or the murmur met with occasionally in the hearts of feeble children, or in cases of amenorrhœa in females, are never instances of a mitral murmur. Whatever may be the precise condition of the valve, the existence of a permanent murmur, especially if it be associated with local fremitus, indicates insufficiency of closing power, and this results more or less in the blood being forced back into the auricle, and which thus becomes unduly distended, and, after a time, permanently dilated, and perhaps hypertrophied, as may be also the ventricle, while, at the same time, the supply to the aorta, and to the system generally, is, by so much, deficient. In such case an aortic thrill may sometimes be felt; but this must not be confounded with the thrill which is occasionally associated with the murmur attendant on constrictive semilunar disease. By way of compensation for this deficiency of blood to the system quickness in the heart's action is induced, and is generally, but not always, accompanied by rhythmical irregularity, with a weak, small, unequal, and perhaps dicrotic pulse; the appetite is capricious, and there is generally a disinclination to take animal food; the mouth is dry and clammy, with persistent thirst, and there is a relish for sub-acid drinks; the surface is dry, and sometimes exhales the sour smell of rheumatism; the mental expression is markedly one of anxious pain, and even of anguish, characterized not so much by irritability as by a subdued yielding to disease, with a shrinking within oneself; great disinclination to exertion or movement of any kind.

The symptoms, special and general, of hypertrophy, and

eventually of dilatation, being usually concomitants of an acute attack of mitral valve disease, must also be taken into account.

The secondary symptoms have great significance. The lungs become loaded with blood, then permanently congested, and in extreme cases, hepatized; the breathing is oppressed, the respiration sibilant; there is a short, frequent cough, with a pituitous frothy expectoration, at times tinged with blood; or there may be occasional hæmorrhages. As the disease advances the right heart and cavæ become loaded and distended, and venous congestion is the result. The other main organs then participate in the difficulties; the liver is gorged with a black bile, and shows indications of being enlarged, and anasarca, and, may be, effusion into the cavities, supervene, the function of the kidneys being, at the same time, arrested; the brain is also affected; there are occasional giddiness, headache, and lethargies, so that its functions are impaired. This state is generally very persistent, and months are often passed in a listless, semi-conscious state; the countenance is puffed and turgid; the cheeks suffused with dark blood, and the lips and the extremities become livid from the universal prevalence of capillary obstruction; the digestion is depraved, and there is an abiding nausea and flatulency.

These symptoms sometimes supervene rapidly on the first attack, or they may, in variously modified forms, continue for years, and during which notable relief to the more urgent of them, by judicious management, is often obtained. This amelioration rarely continues long, and a recurrence of the disease in its more formidable aspects takes place. With a feeling that death may occur at any moment life is thus often prolonged; sometimes in a state of oppressive suffering, or half stupor. For the most part it is a form of disease that is terminated, not so much from any sudden failure of the heart, as from the secondary effects upon the large organs, or from dropsical effusions, especially into the parenchyma of the lungs.

Such is the not unusual history of a case of disease of the

mitral valve. Insufficiency of closing power alone may induce all the symptoms detailed, but generally it is associated with other lesions of the heart; the mitral valve itself may not only be insufficient, but may be thickened, inelastic, contracted, and torn; may be the subject of adventitious deposits, and thus both obstruct flow while permitting regurgitation. The auricle and the ventricle may be hypertrophied or dilated, or, as they usually are, both; they may otherwise be degenerated in structure, etc. Such are the more formidable aspects of this form of heart disease. Unfortunately it is not an unfrequent one; only a little less so than that of aortic valve disease. On the other hand it may exist for years, indeed, through a long life with an amount of regurgitation so slight, as not seriously to incommode the health or well-being of the individual. This is probably due partly to the surrounding tissues not being implicated in the original disease, and partly to the resistance offered by the auricle, aided by the pericardium, to the shock of the increased volume of blood being so effectual that the regurgitation only temporarily distends it, and without causing pulmonary congestion.

It may also exist for years, and then subside as a murmur. As an illustration of this the following case not only offers an example, but shows the propriety of not being too dogmatic in the prognosis to be made. T. L., æt. 53, a broad-chested vigorous man, by occupation a cowkeeper. Seventeen years ago had a severe attack of rheumatic fever, which left a remarkably loud and pervading mitral murmur. He presented no other indications of disease, and was enabled to pursue his occupation which necessitated his being at work in the fields by the dawn of day. For twelve years he was occasionally under observation, and the murmur was always loud and persistent. Five years ago having contracted a bronchitis the murmur ceased, and there was a roughened and somewhat obscure systolic first sound. He is now for a third time under treatment, having general rheumatism. The impulse is increased; at the apex there is an obscure muffled first sound; beneath the sternum and to the right of it both sounds are clear; no sound is

to be heard at the point of the left scapula; the respiration at times is slightly hurried. It is probable that for the first twelve years the condition was rather that of deflection in the current of the blood, than of amount regurgitated; that after the bronchitis, and by the shaking of the cough, the cause of this was lessened or removed, and left a roughened valve only; and that, now, structural change in the parietes is commencing.

The pre-systolic murmur, though generally associated with the regurgitant, may, as just observed, occur independently of it, and therefore indicate to a certain extent, a less amount of present and future danger; and it may also occur without an amount of obstruction adequate to induce a ponding back of the blood, as when slight lymph deposits on the edges of the valve take place; and it may also be induced by temporary rigidities due to rheumatic and other inflammatory disorders. These are not infrequent sources of a pre-systolic murmur, and deserve very careful consideration. Their diagnosis must be sought mainly in the entire absence of pulmonary difficulties, and partially, in there being no evidence of engorgement of the left auricle. Assuming these conditions, and also that there is no irregularity in the impulse of the heart's action, and no evidence of wall disease, a pre-systolic, or flow, murmur through the mitral valve, whatever suspicions it may originate, does not absolutely imply vital unsoundness.

The presence of a regurgitant mitral murmur must not always be too readily assumed. It has just been observed that this murmur very often confuses a due appreciation of the pre-systolic, but the converse also obtains. The pre-systolic, and that, too, from only slight causes, is stated to be by some recent observers, so sonorous, predominant, and prolonged, as to conduce to the conclusion there is regurgitant disease where really none exists.

Though there be a regurgitant mitral murmur, if it be not persistent, or if there be no evidence of weak or dilated heart, or of pulmonary complications, the actual presence of diseased structure is not to be inferred. Murmur may be due to many

temporary causes, from faulty adjustment of the valve to the auriculo-ventricular opening in consequence of strain from over-exertion; from disturbance in the muscular appendages of the valve, so that spasmodic action is induced, as in epilepsy, chorea, or after excessive masturbation; or from ventricular dilatation induced by a loaded and a congested organ. It becomes important where these sources of a mitral murmur obtain, to recognise them, so as to modify the unfavourable prognosis one might otherwise be disposed to make. At the same time, though they be clearly recognised, we must not too summarily pronounce them to be indicative of no future danger, for they may lay foundation for serious valvular and wall mischief. This is especially the case if there be, as not unfrequently happens, a coexisting spanæmic condition of the blood; and when, perchance, there may be, thus resulting, a murmur in the pulmonary orifice to add to the difficulties of the just appreciation of the state of the mitral valve.

Besides the congenital and other causes which induce valvular disease in general, mitral valvular disease has its special exciting causes in rheumatic inflammations, in gout, and in chronic blood diseases, and then in deficient valvular adjustment due to dilatations of the ventricle and to congestive pulmonary diseases. The former of these special sources of disease is met with, for the most part, in early and middle life, the latter more usually in advanced years. The prophylaxis is, in early life, to especially avoid conditions that induce rheumatism, or foster the development of the gouty cachexia. In the advanced period of life to avoid all over-exertion that may strain the heart, or the neglect of those pulmonary and blood diseases that may originate valvular insufficiency.

Where the diseased condition of the valve is ascertained, all sources of undue excitement in the heart must be avoided; while over-action in the heart itself is to be tranquillized. The aim is to prevent regurgitation, and congestion of the lungs. If local congestions take place, specific means to lessen and remove these must be adopted, together with general means to

relieve the associated errors of functional disturbance. Congestion of the lungs is relieved by expectorants, sedatives, and demulcents; of the liver, by aperients; and the kidneys, by diuretics: æther, and the nitrous æther are effective general medicines.

The want of appetite, so frequent in advanced stages of the disease, is often relieved by antacids, with light bitters, and the citrate of iron, or other light forms of chalybeate salts. Acids, which are useful in the early stages, cannot now be well borne. Stimulants are injurious: they induce heat and restlessness.

Long observation has shown that the aortic valves are subject to lesions depending on (1) congenital, (2) inflammatory, (3) degenerative, and (4) accidental causes, and experience has shown that the lesions thus established may induce murmurs both of flow and of regurgitation; also that the murmurs are, in some cases, associated with diseased conditions of the valve indicative of fatal tendencies, while in others, the conditions are consistent with prolonged life, and without sensible inconvenience; and experience has also shown they are the seat of murmurs, though there be no kind of valvular disease. The flow aortic murmur is essentially systolic, and may, therefore, easily be confounded, as previously pointed out, with the mitral regurgitant murmur. When it occurs uncomplicated with other valvular sounds, the first sound is audible at the apex, but the second sound is ordinarily difficult to trace, or only towards the sternum, and this may probably be, not the aortic, but that of the pulmonary semilunar valves. The murmur itself often offers a wide range of sound for observation; it may be only a slight purring tremor; it may have a soft musical pitch; it may be blowing, rasping, sawing, or grating. It is generally sharper and more harsh in tone than the mitral regurgitant murmur.

The former class of murmurs may probably be materially determined, as regards the character of these sounds, by the nature of the valvular lesion, for the valve may be only

roughened by slight lymph deposits, or it may be thickened by dense cartilaginous or hard, bony degeneration; or its aperture may be contracted to very small dimensions. Though these several conditions may produce a relative loudness of murmur, it is not to be assumed intensity of sound is a sure measure of mischief. There may be the loudest and the harshest of murmurs and the smallest possible amount of mischief. Let the valvular lesions be what they may, provided they induce no great obstruction to the flow of blood, and there also coexist no complications with other structural disease of the heart, this systolic murmur may be both intense and persistent for years without any apparent detriment to health. But when in addition to the systolic murmur there are evidences of an obstruction to the free flow of blood, as by an obvious deficiency in the systemic supply, with rhythmical irregularity in the heart's action, and with aortic thrill, a serious condition, both as regards the heart itself, and the system generally, is to be inferred.

In the early stage of this form of valvular disease there may not be much to attract attention; there may be only slight irregularity of pulse, with a halting breathlessness on exertion, and perhaps a passing indication of an uneasiness, or slight pain, in the direction of the base of the heart. But as disease advances restlessness and irritability set in; the skin becomes pallid, and is somewhat puffed. The countenance has an anxious expression very different from the puffed and placid expression in the cases of fatty degeneration or mitral disease. There is a general sense of exhaustion and weakness; the breathing, on exertion, is short and panting, not from congestion, but from innervation; and sudden, sharp pain may then also occur, and occasionally with a sensation, if the exertion be continued, of faintness, or rather, as if all physical power would cease. Unlike the complications of a mitral murmur, the pulmonary congestions are not prominent symptoms: there may, perhaps, be occasionally a slight, nervous cough, but this is not accompanied by expectoration, nor are there the venous congestions, and, even in extreme cases, there may be neither dropsy nor œdema.

Should pulmonary or venous congestions occur, they are probably to be attributed to the development of complications with other diseased states of the heart, more especially hypertrophy and dilatation of the ventricle, with mitral valve disturbance, and, indirectly, with congestion of the right heart. But the more important complication is that of insufficiency, which, in advanced stages of constrictive aortic disease, may be said, invariably, to take place, so that, besides the constrictive, there is regurgitant disease. The valves in one or more of their segments may become so thickened, or so shrivelled, or so otherwise involved in a diseased condition of structure, that their function of effectually closing the aortic aperture is impaired.

Though this insufficiency may occur as the sequel of the original disease causing the primary state of constriction, it may also exist independently of this latter. Regurgitation may, therefore, exist without obstruction to the flow. However it may arise, the symptoms will be the same; the diastolic murmur thus induced will take the place of, and annihilate, the second sound. It is softer, not so rough, and more prolonged, than the systolic flow murmur; it is musical and cooing, and may be in these characters even loud; it is heard mainly towards the centre of the sternum, and, when it is associated with a constrictive murmur, presents a "to-and-fro" sawing character; the constrictive murmur being harsh, the regurgitant soft. It sometimes communicates a thrill to the left of the sternum. The pulse presents its peculiar characters (p. 75) now hard and full, but suddenly falls, it rises against the finger rapidly, and rapidly sinks. The visible arteries present a marked prominence, are contorted, and wriggle as it were; the superficial small arteries do not look full or congested. In some rare instances there is developed a sensitiveness to these arterial movements. A lady whom I saw some twenty years since, and who had aortic valvular disease, felt, as she expressed it, the beating of every artery in her body, and a slight humming sound was clearly emitted by many of them. She lived for years a quiet and uncomplaining life, and was not apparently greatly inconvenienced by the disease.

As the symptoms of regurgitant aortic disease are developed the muscular structure of the heart undergoes structural change, and hypertrophy and dilatation of the ventricle take place. The association of these conditions presents a hopeless and inevitably fatal tendency. The supply of arterial blood is too little for the requirements of the system, and the blood being ponded back, the lungs become oppressed, and the right heart, with all its evil consequences, distended. The local and general symptoms then culminate in the greatest distress.

The prognosis in disease of the aortic valves, whether of obstruction or of regurgitation, or of both, must, in the main, be esteemed to be calamitous. The causes that usually induce the valvular disease, also commonly induce hypertrophy, more especially in those of an advanced age, and of a gouty constitution. So soon as hypertrophy and dilatation become active conditions, so soon do the more serious symptoms show themselves. In old age, beside the tendency to cartilaginous changes, and calcareous and bony deposits, the semilunar valves sometimes become relatively too small for the aortic aperture. This aperture being then abnormally distended, both murmur and palpitations are induced; symptoms of great significance after fifty or sixty years of age.

It is said that a regurgitant is worse than a flow murmur; certainly, if associated with palpitation, more or less marked, shortness of breath on exertion and præcordial uneasiness, we must consider it a fatal form of disease, for there is then also evidently dilatation. These cases of aortic insufficiency sometimes end suddenly.

Should, however, the muscle of the heart retain its integrity, and the valvular obstruction be inadequate to retard the blood upon the lungs, or the valvular insufficiency to deprive the system of its requisite supply, aortic disease may be deemed the least dangerous of the valvular organic diseases. As a general rule, it may be stated that, *per se*, the systolic murmur is less dangerous than the regurgitant; and that if the systolic murmur be unassociated with other heart disease, with palpitations,

or pulmonary disorder, while the apex beat is normal, and the first sound distinctly heard, coincidently with the murmur, below the left nipple, and the murmur itself only slightly heard there, but distinctly audible below the angle of the left scapula, the indications are, that the valvular disease is not of a serious nature, nor portending hostile influence to health. We must also bear in mind that the systolic aortic murmur may be a blood or functional murmur only.

Seeing the tendency to aortic disease in advanced life, and that the gouty constitution determines to the changes that the valves then undergo, everything that tends to foster gouty disease should be avoided; and, also, as the valves, by reason of the structural thickenings, and rigidities, thus induced, are susceptible of rupture and impairment, all violent exertion. When valvular disease is established, quietness of mind and of body should be enjoined, and only a moderate use of stimulants, with light and nutritious food permitted. Rash and futile attempts at cure must not be attempted. If we cannot repair these chronic diseases, we must avoid, in any wise, lowering or injuring the constitution, lest we increase that we would subdue.

Murmurs occurring in the right heart are very difficult, and, at times, impossible of recognition; not only from their nearness of origin, and similarity in sound, to those of the left heart, but from these latter being louder, more preponderating and generally coexisting, with the former. Hence, though we really may be listening to murmurs of the right heart, they may be confounded with, or attributed to, murmurs of the left heart; the more especially, as experience has taught us, that, comparatively speaking, they are of rare occurrence. Disease in the pulmonic semilunar valves so seldom occurs as barely to make the investigation of a murmur, there occurring, a matter of any great practical interest. It is not so, however, with murmurs originating in the tricuspid valve. The flow tricuspid murmur is, from the feebleness of the ventricular diastole, so low in tone, and so masked and overpowered by the loudness of the invari-

ably coexisting venous hum, that it is impossible to distinguish and separate it; and, perhaps, the conditions attending its existence are rarely of any great moment to the well-being of the animal economy. The valvular insufficiency, which causes the systolic regurgitant tricuspid murmur is, however, of more importance, and it is of the greatest consequence not only to recognise it, but, being aware of its existence, to anxiously watch its effects upon the liver, the kidneys, and the capillary system. Though from similarity of sound we may not be able to distinguish the regurgitant tricuspid valve murmur from a systolic mitral murmur, there are grounds for valid suspicion of its existence when the murmur is audible at the second intercostal space, and is not conducted into the carotids, and is not to be heard at the apex of the left scapula.

The origin of this systolic murmur is rarely due to disease in the structure of the valve itself, but usually to insufficiency, induced by dilatation in the ventricle. In practice, this is what we have to deal with; and, in the history of heart disease, in its advanced stages, and in the later periods of life, its occurrence is by no means infrequent, and the effects of it are marked and calamitous.

The symptoms are nearly identical with those of right ventricular dilatation. Besides the murmur, the area of dulness is increased, the impulse is communicated to the right of the sternum, and below it, and is intermittent in force and rhythm; there is a marked turgescence of the jugular veins, with undulating and intermitting pulsation, and with venous hum. The countenance is livid to darkness, showing the amount of venous congestion that prevails; the cerebral symptoms also indicate engorgement of the vessels of the brain, a wandering restlessness, with thoughts of imaginary errors alternating with a semi-comatose state, sometimes headache. The appetite is capricious, and stimulants flush, increase restlessness, and are generally injurious. The pectoral symptoms are marked by dyspnœa and a short, dry cough. There is a tendency to congestion of the kidney, with deficient amount of urine, and of the liver with œdema; the general condition is one of great weakness and

prostration of strength. As these symptoms accumulate, coma supervenes, and slowly life is terminated.

Such are the main symptoms of this affection in its more aggravated form, and, sooner or later, they are generally developed; for, when dilatation of the ventricle is so advanced as to induce permanent valvular insufficiency of the tricuspid valve, venous congestion oppresses the system. It is, however, generally, though a fatal, a lingering disease, and the urgency of its symptoms may be gradually and separately developed.

The immediate cause of this valvular insufficiency is due rarely to structural changes in the valves themselves, but to dilatation, and a loaded state, of the ventricle by the ponding back of the blood from the lungs. This may be, primarily, due to emphysema, bronchitis, or pneumonia, and remotely to disease of the left ventricle, and more especially to obstruction and insufficiency of the mitral valve. It has been affirmed that mitral insufficiency will directly induce the congestion and dilatation of the right ventricle, but this is very doubtful. The intermediate pectoral congestions are generally the exciting cause.

To a certain extent, tricuspid regurgitation into the auricle and cavæ is a salutary provision, the heart and the lungs being partially relieved thereby; and though the blood is thus ponded back upon the system, so that the liver, the kidneys, and the cellular membrane are liable to become loaded, yet the disease may persist without effecting these complications.

A case has recently passed under my careful observation which well epitomized this form of heart disease. A lady, æt. 72, after feeling some little shortness of breathing, consequent on an attack of bronchitis, very rapidly indicated the presence, to the fullest extent, of tricuspid valvular disease; shortness of breath, palpitation, soft-blowing murmur projected to the right of the sternum, and not traceable in the great arteries, or at the point of the left scapula; venous hum; lividity of countenance; congested liver; urine scanty and loaded, with a large amount of œdema. By steadily evacuating the bowels, and aiding the liver and the kidneys in their functions, the congestive disorders in great measure subsided, and the murmur was only to be occa-

sionally heard, and then only when congestions re-established themselves. The palpitation was always present, œdema entirely disappeared; but with lividity of countenance and skin, occasional bilious congestions, and cerebral inconsistences, life was prolonged for eighteen months. The *post-mortem* examination exhibited pulmonary congestion, with dilated right ventricle, and insufficiency of the tricuspid valves. There was no other presumable source of murmur than this insufficiency.

The prognosis in these cases, however we may relieve and ward off effects, must be bad; there is little or no hope of recovery; still much may be done to postpone difficulties, and much to alleviate them. Seeing that pulmonary disease is an immediate cause of the ponding back of the blood upon the right ventricle, everything must be done to prevent and alleviate this; and the treatment must serve to unload the other congested organs, mainly by the judicious use of general and specific purgatives with diuretics; and such remedies as may strengthen the heart without loading the system: a combination of the nitro-muriatic acid with æther and digitaline, is most useful in these cases.

CHAPTER X.

PAIN OF THE HEART.

Pain in the region of the heart is always important in the estimation of the patient, and, generally so, of the physician. It presents itself for observation in every phase; from the slightest uneasiness to the acutest agony; from the mere momentary sensation to the distress indicative of immediate danger, and culminating in the fatal form of disease so graphically described by Heberden. It is necessary to consider if these various examples of pain, including angina pectoris, are to be considered as identical in their seat and origin, or as presenting distinct, and to be determined, differences. If identical, then intensity and complications become of main importance. If not identical, it becomes necessary to discriminate between them, by establishing what may be their differences; and then to appreciate the gravity attendant upon each, as also the complications that may influence this gravity.

On carefully reviewing the symptoms of these several forms of pain of the heart, we find that angina pectoris presents features that do not belong to the others, and that they may therefore be classed in two groups, the one comprising the general forms of pain, the other that of angina pectoris. The distinctive character of the one being pain; of the other, spasm.

Amid the general forms of pain in the region of the heart, examples of varying intensity are often exhibited in association with the inflammatory diseases of this organ and its membranes,

as pericarditis and endocarditis; and with the chronic degenerative structural diseases to which it is so liable, as fatty degeneration, and aortic valve disease. It may be mentioned here that of all the chronic diseases of the heart, pain is more particularly the accompaniment of that of the aortic valve. When this valve is diseased, pain is promptly induced on the least over-exertion, and peremptorily forbids its continuance. The presence of this ingress of pain is not devoid of an indication of special danger. Sudden death being, occasionally, one of its accidents.

It must, however, be borne in mind that these several inflammatory diseases and structural degenerations not only may, and do, frequently exist without any symptoms of pain, and that pain is often associated with functional disorder only. Hence it may be inferred that structural diseases, though they may be a remote cause of pain, are not its immediate cause. This conclusion is further borne out by the fact that there may be the presence of pain in its severest forms without fatal consequences, or any indication of abiding disease; while death may attend it, though no obvious lesion, nor pre-existing abiding disorder, are to be detected.

Be this as it may, if pain be associated with recognisable inflammatory, or chronic, diseases of the heart, it is, for the most part, to be regarded as an urgent symptom, and one adding to the danger or urgency of the disease with which it is associated. Assuming this to be a recognised position, we may pass on to the consideration of pain where no evidence of physical disease exists, and endeavour to estimate in such cases its true nature and origin, and its value in arriving at a correct prognosis.

Pain, uncomplicated with obvious organic disease, is met with mainly in those of a nervous temperament, in the gouty and dyspeptic, and in the course of certain of the blood diseases, and as an accompaniment of special female disorders.

In anæmia, and when the system has been suddenly drained of its blood, pain occasionally is met with as an urgent symptom, being intense in character and sudden in its ingress and recurrence. The point to which attention is directed is obviously

the apex of the heart; and the pain there felt appears to be associated with its projection forwards. It is a more frequent accompaniment of these cases than is the systolic murmur or the thrill which, when they occur, have their seat in the aortic area. In spanæmia and chlorosis there often occurs, besides the hurried breathing and palpitation, a dull heavy pain, of a somewhat persistent character. This is felt immediately under the left breast, and is always sensibly increased on exertion. A systolic murmur is a more frequent accompaniment of these cases than of anæmia, and has its seat usually in the valvular opening of the pulmonary artery, and is heard above, and more to the right of, the seat of pain.

Pain, referred to the intercostal space below the left breast or seventh rib, and very circumscribed in its area, is not infrequently met with, both in the robust and the emaciated; it occurs especially in the hysterical and the hypochondriacal, and is often sought to be relieved by long-drawn sighs; it occurs in intermittent paroxysms, or the pain may remit or persist for a lengthened time. Save the anxiety it may cause, it does not affect the health, or embarrass the other organs; and generally a well-regulated amount of exercise relieves, certainly does not aggravate, it.

A severer form of pain is also met with; for the most part confined to the region of the heart generally, but often extending through it to the præcordium, and to the left shoulder, to the neck, and to the stomach; sometimes, though rarely, extending to the arms. At times this pain is very acute, and assumes an urgent intensity. For the most part it presents more of the remittent than intermittent character; the pain recurring in frequent paroxysms, and lasting may be for hours, or for days. It occurs more often when at rest than on exertion. Indeed, it is often relieved by a healthy amount of exercise.

Severe and urgent as this form of pain appears to be, the sounds of the heart are in noways simultaneously affected; there may, however, be rhythmical disturbance, chiefly in frequency, with occasional intermittence. The respiration is rarely disturbed;

certainly not materially hurried, even on exertion. But, if affected, it presents the characters, not of an asthma, but of the holding the breath in consequence of the intensity of the pain. When a prolonged and severe paroxysm of this form of pain has subsided, it generally leaves a dull aching weight in the region of the heart, and which may last for days. It appears to be essentially of dyspeptic origin; is met with in the gouty and the high feeder; is induced by mental anxiety, moral emotions, indiscreet diet,—more especially tea and thin wines. Two cases of this form of affection have presented peculiarities of some similarity. The heart appeared to assume a writhing or twisting motion, so that, for relief, the left arm would be turned over the breast, and the body twisted over with it, the breath being forcibly held. Both cases occurred in females, and the pain apparently had its exciting cause in some excentrical irritation in connexion with the uterine system; one developed ovarian disease, and the other invariably feels a preceding aura from the left iliac region.

These several forms of pain have analogies with each other; their differences mainly being in seat, in intensity, and in their complications with other disorders. Their seat is usually fixed and defined; they are irregularly intermittent, or remittent, and do not partake of the nature of cramp or spasmodic constriction, and apparently they have little or no influence on the heart's action, or on that of the respiratory organs. For the most part, though their manifestation may be severe, and therefore alarming to the patient, they do not, simply and unassociated, indicate the presence of disease of a fatal tendency. They are to be esteemed as capable of alleviation, and generally of cure. If there be danger, it is chiefly due to complications with organic disease of the heart, or other contiguous organs.

These pains appear to have their seat, primarily, in the pneumogastric or cardiac nerves; and, secondarily, in the nerves of the brachial plexus and cerebro-spinal nerves supplying the front of the thorax. In their symptoms, they partake of the nature, and obey the laws, indications, and phenomena of ordi-

nary nerve-pains, such as tic-doloreux, or those of a sciatica, or lumbago.

The prognosis, and the treatment of these affections require that their origin, seat, intensity, persistency, and complications should be well considered; each being measure of disorder, and guide to the means for alleviation.

The pains originating in disordered conditions of the blood usually find relief; the anæmic in the restoration of the strength of the system by generous diet and tonics; the spanæmic in altering the condition of the blood by the usual treatment adopted in a chlorosis, as the exhibition of warm aloetic purgatives, aromatic gums, and such ferruginous medicines as the system will bear. Urgent cases of spanæmic pain, even when associated with a basic murmur, generally yield to these.

The intercostal pain is somewhat persistent, and difficult of alleviation. To successfully treat it, the attendant conditions must be carefully considered. If occurring in the weak and emaciated, tonics and good food are indicated; if in the plethoric and hypochondriacal—and these conditions often coexist—a strictly regulated diet, with exercise, must be enjoined, and so likewise when occurring in the hysterical and dyspeptic. In the hysterical, and especially in the hypochondriacal, this pain is most persistent and difficult of cure, and sometimes resists all the means devised for its alleviation.

The severe paroxysmal or recurrent pain, and which appears to have its seat mainly in the branches of the vagus nerve, is essentially of dyspeptic origin, and requires, with well-regulated diet and exercise, very careful medical management. The indications for the most part are to correct an acid or gouty diathesis. Alkalies, and the alkaline mineral waters, light bitter infusions, and warm alterative aperients, are often most useful in these cases. It is in this form of heart-pain that tea is so often an eminently injurious article of diet: the smallest quantity sometimes inducing an attack.

In very urgent and persistent cases the application of leeches is useful to the region of the heart, or even the unloading the

general system by the lancet. I have seen cases in which the pain has subsided as the blood was being withdrawn. The dull pain, which succeeds an attack, is usually relieved by warm stimulants and gentle exercise in the open air.

We pass on to the consideration of that marked and severer pain experienced in the formidable and very fatal disease termed "angina pectoris."

The prominent symptoms of a first attack, are a painful constriction in the præcordial region, extending beneath and towards the left of the sternum, and a difficulty of breathing so urgent as to induce a feeling of immediately impending suffocation; the internal conviction is that death is then to ensue. The constriction is so acutely painful as to have been described in the strongest epithets; the stomach is oppressed and flatulent; the countenance is pale, and the expression one of the greatest anxiety, or of horror. The attack occurs suddenly, usually on exertion, which it peremptorily arrests; it may continue for a few minutes and then, with a few deep sighs, entirely subside, leaving only a sensation of numbness over the region of the heart, and the neighbouring pectoral region. The mental faculties are not affected, and there is a perfect appreciation and an accurate recollection of the events that have occurred. The recurrence of an attack is uncertain. Days or months may elapse before a second takes place; usually, however, the time is not very distant before a second, and a succession of attacks, ensue. As the disease progresses, the attacks are of longer duration, with more marked and urgent symptoms. The constriction is more intense, and the pain is projected down the left arm, in the course of the nerve of Wrisberg, over the chest, and towards the neck, and may even extend to the right arm and to the back. Death sometimes ensues on the first attack; but, more often, a rapid succession of attacks is the precursor of the fatal one.

If uncomplicated with any very obvious disease of the heart, experience has shown that it chiefly occurs in the well-fed, and those of the nervous, rather than in those of the spare and lymphatic temperaments; that it is a disease of adult age, and

that the male is more liable to it than the female. The following case, however, illustrates its occurrence in a female, æt. 22. In apparently good health, she expectorated a small quantity of blood; ten days afterwards, whilst walking, she experienced a sufficiently well marked attack of angina; a week afterwards, a second; three days elapsed when a third, more severe and more urgent, occurred. The only occasion on which I saw her was the following day, dressed, in the accustomed sitting room, and as if she were not in any way the subject of fatal disorder. A careful examination of the heart and the lungs yielded no trace of disease. In three days she experienced another attack, and on the following, an attack, in which she died.

Experience has shown that angina occurs in association with several of the diseases of the heart, as induration of the coronary arteries, diseases of the aortic valves, and of the aorta, fatty deposit and fatty degeneration; in fact, with almost every lesion to which the heart is liable; but then it occurs without the presence of these lesions. It can, therefore, be scarcely said to have its anatomical seat in any, or either, of them, the more especially as these several lesions pass through all their phases to the fatal conclusion without the occurrence of angina. I have notes of several cases in which the heart was slightly, or extensively, diseased. As I write this, a case, under my charge, has just succumbed to an attack; a male, æt. 38, florid complexion, well nourished, with antecedent regurgitant disease of the aortic valves, of some months' duration, and, latterly, with slight pulmonary congestion. Three weeks before his death, he experienced the first attack. The exhibition of æther, opium, and ammonia was grateful to him. A week afterwards the second attack occurred. He awoke suddenly, hastened out of bed, seized the back of a chair, and leaned over it, gazed about wildly, stamped, moaned, the perspiration streamed from him, the heart's action was violent and irregular, the murmurs indistinct, the pulse bounding, breathing oppressed, pain across the chest intense, and the stomach painful and distended with flatulence. This attack lasted about half an hour, and was apparently relieved by the subcutaneous injection of digitaline. Three nights afterwards

he had a third attack, and, subsequently, six others. These latter attacks lasted generally about an hour. In one of them he seemed only semi-conscious, and wandered about the ward. The later attacks were relieved by the subcutaneous injection of half a grain of the muriate of morphia. This relieved the spasm, and induced some three or four hours' sleep. So intense was the pectoral pain, and the fear of impending death, that he dreaded sleep lest spasm and death should then overtake him. The last and fatal attack had only commenced; in some two minutes he was dead.

In a case which occurred two years ago, the heart itself was not diseased, but there was a large aneurismal tumour in the mediastinum, with disease of the emergent aorta. Besides this, I have notes of only two cases where no disease in the heart could be detected. In one of these, I witnessed the whole course of a paroxysm. A gentleman, æt. 51, of robust frame, well fed, capable of, and taking, a large amount of exercise, both on foot and on horseback, had, at distant periods, experienced three or four attacks of gout. These had been tolerably severe in character, but not of long duration, and confined to the feet. Whilst walking up a steep hill, on a cold, windy evening in October, he was arrested by an attack of angina. He walked home, slept the early part of the night, but was awakened towards morning by another attack, and he experienced four others in the succeeding forty-eight hours. When I saw him at the expiration of this time, he was in bed, calm, looking pale, made no complaint of pain, the pulse was small, skin soft, respiration natural, urine pale and frequent. He dreaded the attacks, and was anxious to sit out of bed to meet them. Whilst examining the region of the heart, an attack came on. His impulse was to get out of bed, and he sat at the side of it; the breathing was partially arrested with, at distant intervals, deep sighing respiration; the abdomen was distended, and the diaphragm appeared inactive. The mental anguish was obviously very great; he broke into profuse perspiration; there was no expressed desire for cold air; on the contrary, warmth appeared at the time, and generally, grateful to him; the heart's action was subdued, weak, and irregular; the pulse small. The attack

lasted some ten minutes, and passed off with a slow, prolonged breathing, and a general sense of weight about the chest; the stomach and bowels were incommoded by flatulence, and its discharge gave great relief. He referred to the pain of the heart, and over the sternal region, and towards the neck, as most severe. Æther, ammonia, and stimulants generally, afforded no relief during the attack, and were, as he expressed it, injurious to him, both then and afterwards. During the intermissions sedatives, opium, hyoscyamus and hydrocyanic acid, if not useful, did not offend him, and he had sleep under their influence. The attacks, however, increased in frequency, duration, and intensity, and, thirty-six hours afterwards, he died.

The theories of the cause of this disease have been numerous. Its origin has been attributed to spasm, cramp, neuralgia, epilepsy, specific disintegration of the heart's structure, ossification of the coronary arteries, to a heart loaded with blood, to enlarged liver, to gout, and, latterly, to paralysis. If the cases of angina, in which there exists evidence of structural disease in the heart, be excluded from consideration, it is, for the most part, found that, during the intermissions, there are presented for observation no very obvious nor essential morbid conditions; and that, during the paroxysm, the phenomena have relations with those exhibited by muscle when in a state of spasm.

The pectoral constriction, which assumes so many forms, and is of such varying intensity, and, from its effects upon the respiratory organs, may perhaps suggest the fear of impending death, is not only always present, but is so prominent a symptom, as to stamp the attack with the vague suspicion that it may be of the nature of an asthma. It is, however, to be distinguished from asthma, not only by the character of the associated pain, but by the occasional deep and effective inspiration that is voluntarily made; and by its being unaccompanied by a wheezing, or any other bronchial symptoms; by no craving for fresh air; and by the remission being free from a mucous expectoration.

The sudden occurrence of the attack; the pain commencing at the lower portion of the sternum, and spreading over the

chest and in the course of the thoracic and brachial nerves; the fixed condition of the diaphragm and of the muscles of the chest, and which is only overcome by a determined voluntary effort, the distended stomach, the acid flatulent dyspepsia, the intensity of the spasm, the mental distress, the anticipation of immediate dissolution, all point to the fact that the muscular spasm is due to some direct and immediate nervous influence.

Considering the very marked and distinctive symptoms which are thus seen to characterize the attack of angina, it might be assumed that it has its seat in some exclusive and specific morbid anatomical condition. Though it must be acknowledged, in the present state of medical knowledge, that this has not been ascertained, yet there has been observed, as a general anatomical fact, that the muscular fibres of the heart present, when examined shortly after death, the character of relaxation and flaccidity, and not of contractility; the rigor mortis does not appear to have been established. Assuming this to argue a pre-existing condition of paralysis, there is then, to account for it, the preceding affection of the nerves. It may also be that the spasm and pain experienced in the heart are analogous to, if not identical with, what is observed to occur in other muscles when wasted and paralyzed by a deficiency of the due amount of a healthy nervous stimulus; or it may be, by reason of the disorder of the nervous filaments pervading the muscular fibres, the tonicity or vital elasticity of the muscular structure of the heart may be impaired; so that, failing to propel the necessary column of blood, the heart becomes over-distended; and distension alone will induce pain.

If we look to the seat and to the concomitants of the pectoral constriction and of the pain, there are grounds for concluding that both the vagus and the great sympathetic nerves are materially involved; the former in the diffused pectoral spasm, and the latter in the difficulties experienced by the heart. To go a step further; considering that, during an attack, the rhythmical action and the power of the heart are not always

and certainly not necessarily disturbed, it might almost be inferred that the spasm of the diaphragm, which always exists, and of the pectoral muscles, where the chief seat of pain is, that the vagus and the phrenic nerves are the primary seat of the disease, and that their intimate communication with the great sympathetic and the cardiac nerves induces the ultimate failure and paralysis of the heart, through the direct agency of these latter. The two systems of nerves are, however, so intermingled that it is difficult to determine their special relations to the spasm; but it is evident, so long as the voluntary muscles are sufficiently unaffected as to be capable of overcoming the spasm, set up in themselves and in the involuntary muscles, life is preserved.

Pain is so conspicuous a feature in an attack of angina pectoris that it always forms part of its definition. But is pain a necessary element of the disease? though it generally exists, does it always? may there not be fatal spasms without pain? I have seen cases which have excited the suspicion that this may occur. There have been sudden pectoral spasms with all the expressed anxiety and fear of an angina; the sighing respiration, the profuse perspiration, and yet little or no complaint of any painful sensation; and such cases have died in an attack. A case I am now anxiously watching presents features of this kind. A lady of somewhat stout habit, æt. 62, and having, as I conclude, fatty deposit of the heart, and, may be, some slight amount of emphysema of the lung, experiences the sudden accession of a diaphragmatic and pectoral spasm; the countenance becomes suffused, the expression anxious, the respiration retarded, then deep and sighing, the pulse is small and contracted, and there is the immediate breaking out of a profuse and universal perspiration; she distinctly says she feels no pain of any kind. The attacks last from five to ten minutes; they are not of frequent occurrence, but, when they do occur, are apparently excited by only slight causes. Save the chronic disease of the heart, the intermediate state is not unsatisfactory. The appetite is good, she takes moderate

exercise, is of a cheerful temperament, and certainly neither nervous nor hysterical.

These attacks appear to be so nearly allied to those of a true angina pectoris that I would venture so to class them, designating them as cases of painless or dumb angina. Together with angina they may be referred to the class of somewhat analogous cases of nervous spasm and morbid muscular contraction; examples of which may be found in laryngismus stridulus, or pertussis.

Death by drowning appears in some instances to be due to cramp or spasm of the heart, and perhaps paralysis of the cardiac nerves, and may therefore, under such circumstances, possibly have alliance with angina. I allude to those cases in which the swimmer sinks suddenly, and without struggle, to the bottom; or, may be, after uttering a single piercing cry, as if taken with some sudden pain. In these cases the body has been recovered beneath the precise spot whence it had, in life, disappeared; and there evidently had been no effort of a drowning struggle.

The treatment of angina obviously divides itself into that proper during the attack, and that during the intermission.

During the spasm, experience shows that the aim must be to calm, and not to stimulate, and that for this purpose opium, æther, and chloroform are appropriate agents; the hypodermic administration of the salts of morphia is often attended by marked success.

The use of the nitrite of amyl has recently been advocated. Dr. Madden, in a personal and very interesting memoir ("Practitioner," December, 1872) of his own case and experiences, details the effects of this agent. The attacks, which had generally lasted some twenty minutes, and often recurred after an intermission of only three hours, and having resisted the means previously applied, were signally relieved by it. The first five drops inhaled, "strangled," in two

minutes, the attack, and under its use they gradually became less intense and then less frequent, till comparatively speaking he was relieved from them. Dr. Madden says, "I cannot profess to give a full scientific description of the phenomena presented by the nitrite of amyl in action. The presence of intense pain is not favourable to the exercise of calm, philosophic analysis; and I can only tell what I *felt*. The first effect was often bronchial irritation, causing cough, then quickened circulation, then a sense of great fulness in the temples, and burning of the ears; then a violent commotion in the chest, tumultuous action of the heart, and quick respiration. The angina pain then died out first in the chest, next in the left upper arm, and last of all in the wrist, where it was usually extremely severe. In speaking of my first experiment with the amyl, I said the spasm was, as it were, strangled; this word accurately expresses the sensation. I felt as if a new power was suddenly called into play, which seized hold of, and by a violent effort crushed out the force previously in action. It was not by any means, in itself, a pleasant process; but I delighted in it, for I knew the end would be relief. When the pain had ceased there was generally for some time a strong involuntary tendency to suspension of breathing, each prolonged pause being followed by a very deep inspiration. There was not at any time the slightest confusion of thought or disturbance of vision, but occasionally slight and transient headache.

"As regards physical signs, the rasping sound was soon modified; but a loud blowing systolic murmur, heard at the base of the heart along the aorta and in the subclavians, especially the right, continued throughout the illness.

"I have omitted to mention one curious feeling which I commonly had. The front of the chest seemed to be bulged out in a convex prominence, which suddenly terminated at the lower end of the sternum in a sharp and deep depression towards the spine. This was a purely subjective phenomenon. There was no contraction of the diaphragm, and no retraction of the abdominal walls. But though the hand laid upon the

parts convinced my mind of their normal condition, it in no way modified the sensation.

"All these things appear to me to indicate the nervous system as the chief field of action of the amyl. In *slight* commencing attacks merely smelling the cotton wool on which a previous dose had been poured was sufficient to relieve the pain. It acted like a gentle anæsthetic, without any quickening of the circulation. But in a *severe* attack the full action of the drug with its concomitant vascular commotion, was quite essential. The pain never began to yield until the heart was violently affected. I soon learned to know when I had taken enough, and probably thus avoided unpleasant after consequences."

The treatment during the intermissions must be guided by the physical condition and constitutional tendencies of the patient. If of a robust habit, with a gouty diathesis, alkalis, with warm alterative aperients, having special reference to the tendency to flatulent distension of the stomach and bowels, are useful. If the system be naturally weak, or reduced by disease, a generous diet, with ferruginous tonics, will be required. Stimulants, as a rule, are not serviceable. But in all cases violent and protracted exertion, and anything that may arouse emotional excitement, must be specially avoided.

CHAPTER XI.

DISEASES OF THE LUNGS IN CONNEXION WITH DISEASES OF THE HEART.

In the course of the preceding pages casual mention has been made of the intimate association of diseases of the heart with those in other organs. The more prominent of these are diseases of the lungs, the liver, the kidney, and the brain; but by far the most so are inflammations and congestions of the lungs, and, as the result of these, pulmonary hæmorrhages, effusions, deposits, consolidations, and structural degenerations. Hence, concurrently with disease of the heart, we often have to minister to the occurrence of pneumonia, emphysema, œdema, bronchitis, and marked disturbances in the acts of respiration. In doing this it is necessary to consider to what extent the disease of the heart may have been caused by that of the lung; or whether the disease of the lung is a consequence of the disease of the heart; or whether they are coincident with, or depending upon the same exciting causes; and above all we must consider if either, in consequence of this connexion, presents peculiar and specific conditions, separating it from those it exhibits ordinarily and independently.

If we regard with attention the structure of the lung in its relations to the heart and the circulation of the blood, and also the intimate connexion of the nerves and ganglia that are distributed to these organs, we cannot fail to appreciate how necessary the integrity of either one must be to the well-being of the other.

The first prominent feature that presents itself is that the

branches of the pulmonary artery supply to the air-cells a fine plexus of vessels. Though these branches traverse the bronchial tubes, they are in no wise distributed to them, but pass on directly to the air-cells. In these the blood is aërated, and then is returned to the heart by the pulmonary veins. When we consider the delicacy of the structures concerned, and the importance to the heart that their functions should not be impaired, we can well understand that any morbid condition of the blood conveyed by the branches of the pulmonary artery to this plexus; any obstruction to its free flow through the plexus; or any degeneration of the tissues concerned, so that the aëration of the blood is impeded; each and all, constitute circumstances fraught with evil consequences: the lungs are injured and the heart is disturbed.

Now we see that these results do take place, and that the air-cells become subject to inflammations, degenerations, distensions, and œdema. Each of these diseased conditions, being also of independent origin, is capable of injuriously influencing the heart; and each may be induced by antecedent morbid action in the heart. If we consider the disorders of the air-cells independently, and having no reference to, nor dependence upon, any other diseased action in the lungs, perhaps the heart is more influenced by them, than they are by the heart; hence pneumonia, emphysema, or œdema of the air-cells, unless there be an antecedent bronchitis, are rarely the immediate consequences of any diseased condition of the heart, but they are very inducive of it. If, however, there be a co-existing bronchitis, then diseases of the heart materially embarrass the functions of the air-cells.

Pneumonia, by involving in its inflammatory processes the plexus of vessels proceeding from the pulmonary artery, prevents the necessary aëration of the blood, and directly and indirectly impedes its free circulation: hence congestion, not only of the lung, but of the right ventricle and auricle, and larger veins, takes place; and, if the pneumonia be widely prevalent, the left

heart is moreover deprived of its due amount of blood, and the blood, supplied to it, is impure.

Besides the general symptoms of fever, and dry crepitation, the respiration is hurried and the heart's action increased in frequency, and these symptoms may sometimes suddenly assume an urgent and alarming character. There may be delirium, and often is, if the attack be acute, and complicated with rheumatism. The mischief set up in the heart is primarily the result of inflammatory processes, and secondarily of obstruction, and often causes both ventricular and valvular degenerations. Pneumonia is rarely induced by antecedent disease of the heart, unless the bronchial tubes are previously also implicated, and the blood is alike ponded back upon them and upon the air-cells, and then its form is usually chronic, barely inflammatory, rarely acute. Be this as it may, the local symptoms are not very different from those of an acute attack. There is a dry crepitation induced by the presence of a tenacious sero-mucus, and there is a tendency to local congestions, hepatization, and œdema. The symptoms that may occur are often urgent in the extreme, and unless timely relieved may rapidly pass to a fatal termination.

Pneumonia, by the structural changes it induces in the walls of the air-cells, renders them liable to degenerations and distensions; and hence reduces them to an emphysematous condition; the consequences of which are peculiarly injurious to the heart.

Emphysema presents itself mainly in the form termed lobular and the results are that the air-cells become subject to distension, perforation, and atrophy. Hence serious mischiefs occur, the lightest of which is that the capillaries, which compose the plexus, being wider apart, are relatively diminished in number and therefore present a less amount of blood for aëration. From the changed structural condition of the cells, and from their abnormal distension, the blood, in its passage through the plexus, is not only impeded, but, from the feeble respiration by which a less amount of air is inspired, it is inadequately aërated. Though this state may exist only to a small degree, its symptoms

will manifest themselves, and its consequences eventually, if not immediately, be felt. As the disease progresses these difficulties accumulate. The plexus of vessels occupies really, as well as relatively, a less area; the capillaries that compose it, from being ruptured, or absorbed, are diminished in number and eventually become entirely obliterated: subsequently the supplying branches from the pulmonary artery partake of this destruction, and, as far as these vessels are concerned, the circulation of the blood ceases. As these changes take place, an anastomosis is established between the terminal branches of the pulmonary and bronchial arteries, and with the pulmonary veins; so that, through its means, the flow of blood from the right ventricle to the left auricle is effected. The immediate result of this destruction of the pulmonary plexus is that the previously diseased membrane, which forms the air-cell, fades in colour, becomes anæmic and atrophied, loses its elastic softness, and, may be, is perforated. The general symptoms are a feebleness of respiration, the inspiration short, the expiration prolonged; the nostrils are distended, and obviously expand on inspiration; the corners of the mouth are depressed, and the lower lip falls, is full, and pendulous. Asthmatic symptoms develop themselves paroxysmally, but the breathing is not painful, only oppressed and suffocative. There is a short and feeble cough, with the difficult expectoration of a small amount of viscid phlegm. The voice is feeble, and speech not capable of being prolonged. The gait is stooping, the movements slow, and indicative of torpor; the countenance is dull, puffy, and loaded; the complexion dusky, the result of venous congestion, and the general appearance is anæmic, wasted, and cachectic; the pulse is small; the temperature of the surface is decreased, for there is not only less blood circulated, but this is less oxygenized. The chest is arched; there is greater resonance generally, and especially over the præcordial region, and this, instead of being diminished during expiration, is increased. The impulse of the heart is not perceptible in its normal position, and the sounds are faint and obscure from the emphysematous lung overlapping it. Below the

ensiform cartilage the impulse is very marked, and the sounds are distinct.

Whether emphysema be proximately due to fatty degeneration (Williams), or to fibrous degeneration (Jenner), it is for the most part immediately induced by, or associated with, bronchitis; though Louis affirms it may occur spontaneously and independently. The association of emphysema and bronchitis aggravates both, and renders each doubly dangerous. Thus suffocative spasm of a fatal tendency is induced by the copiousness of the sero-mucous secretion (bronchorrhœa) of the bronchitis and the difficulty of expectorating it, caused by the emphysema. Urgent though these attacks may be, they are not due to any acute form of disease. When the lungs are emphysematous they are rarely the subject of pneumonic inflammations, or even of structural congestions, and this is probably due to a low state of vitality, as seen in the anæmic and cachectic state of the membrane of the cells.

Is emphysema the cause of, or is it caused by, the associated disease of the heart? There can be little doubt that, in these associated cases, if the emphysema has originated in a pneumonia, or some other degenerative source, the disease of the heart will be found to be subsequent to the emphysema; and that, probably, the disease of the heart has been induced by that of the lung. As a general observation, disease of the heart does not set up emphysema; for every variety of the former may exist without the latter; while general observation shows that emphysema rarely occurs without the association of the two affections. But if disease of the heart do not originate emphysema, it may cause bronchitis, and emphysema is commonly associated with bronchitis, and, may be, is not unusually caused by it. So far, therefore, disease of the heart may be said, indirectly, to be the cause of emphysema.

The consequences of an emphysema upon the heart are that the walls become hypertrophied and dilated, and eventually the valves take on disease. These morbid changes take place in both hearts, and are attributed, commonly, to the obstruction

thus caused in the pulmonary circulation. In the right heart there is, doubtless, a ponding back of the blood, and congestion and distension may have their influences; but that the same phenomena, hypertrophy, dilatation, and valvular disease, in the left heart should be equally due to this obstruction, which can induce there neither congestion nor distension, is scarcely possible. Moreover, in other morbid conditions of the lungs where there may really be a greater amount of obstruction, as in phthisis, pneumonic consolidations, etc., these structural lesions of the heart do not necessarily occur. They are more probably due to vital and not to mechanical causes; to the impure blood and to the general disease induced thereby.

It has just been observed that emphysema may be indirectly the consequence of disease of the heart. This is ordinarily the result of valvular obstruction of the left heart, and it will always be found that bronchitis has been the intermediate agent, and that, without the pre-existence of this, emphysema would not be met with. Whenever this does occur, the distress which an emphysema causes to the right heart is very greatly increased. Under any circumstances the consequential results are congestions of the venous system, of the liver and kidneys, and a general anasarca. Coma is also sometimes superinduced. Very rarely hæmoptysis, and, if it occur, only in small quantity; and then perhaps it is due, not to congestion, but to the rupture of the degenerated walls of an air-cell.

When congestive diseases of the heart are established, the lung is liable, especially if emphysematous, to become the seat of œdema. This local form of congestion adds, not only to the embarrassments of the emphysema, but is often the precursor of the fatal termination. The air-cells become swollen and loaded with a tenacious, sticky, and sometimes sanguineous, sero-mucus. The deposit takes place especially in the lower and posterior lobes. There is a crepitation, almost, if not entirely, undistinguishable from the crepitation of a pneumonia; the respiration is rapid and oppressed; there is a short, frequent cough, with scanty expectoration of a frothy, watery fluid. This pulmonary

œdema is usually associated with a general anasarca, and, perhaps, with albuminuria. It may be a chronic affection, may come on slowly, or may be developed so rapidly as to threaten immediate dissolution. This latter is especially the case when an extreme condition of valvular obstruction in the left heart is established. Œdema is so frequently the result of mitral disease, that its urgency may almost be taken as the measure of the amount of valvular obstruction. The mechanical agency of the heart in causing pulmonary œdema may, however, be said never to be called into power without there being also present some of the conditions of a bronchitis. Under urgent circumstances of this kind the poor sufferer lies on the right side, so as to let the heart be as free, and as little oppressed in its action as possible; but the passive and mechanical origin of the œdema is not to be overcome. With a pale and anæmic aspect, with an occasional bright flush of the cheek, the last few hours are passed in a resigned, unrepining, and perhaps half-conscious state.

Pulmonary disorders which have difficulty of breathing (dyspnœa, orthopnœa) for a marked and permanent symptom, may be regarded as amongst the sources whence originate hypertrophies, dilatations, and, perhaps, valvular lesions of the heart. Illustrations of this position may be observed in emphysema, chronic pneumonia, cirrhosis, and in chronic bronchitis; and it may also be observed after the long persistence in violent exertion. It is probable that the operation of these affections on the heart is in some respects simply mechanical. The phenomena that take place in bronchitis will serve to illustrate the sequence of events.

Bronchitis, unless frequently recurring, or chronic in form, does not generally materially interfere with the functions of the heart; for, although the bronchial tubes may, to a certain extent, be obstructed, yet, as they are nourished by the bronchial arteries, and, so far, are independent of the pulmonary system, obstruction in them does not throw back the blood upon the right heart, so as to cause congestion there.

But if the bronchitis is of frequent recurrence, or assumes the chronic form, it may be accompanied by asthmatic complications; and both the bronchitis and the asthma may so congest the air-cells, that the capillary flow of blood through them is impeded. The immediate results of this are a distension of the right ventricle, and insufficiency of the tricuspid valve, with auricular and jugular turgescence. These latter are provisions of safety, and tend to relieve the lung from the ill consequences of congestion. They only continue so long as the bronchial spasm obtains. If the bronchial disorders are persistent, and structural disease of the parietes of the heart develop itself, the distension of the ventricles becomes permanent, and then constitutes dilatation. But still, all must not be set down to the mechanical forces of the congestion, for there is, generally, a pre-existing condition of hypertrophy; and hypertrophy may be considered as the vital effect of inflammation—be this acute or chronic. Dilatation of the ventricles, especially of the right, is always of serious import, and one that will go on from bad to worse, not only as regards the heart, but other organs. It is a fruitful source of heart disease in old people, and of much more frequent occurrence than is generally suspected. We have, however, more often to deal with bronchitis as the result of a pre-existing heart disease. Disease of the left heart, more especially, if associated with obstructive valvular disease, leads to many congestive lung affections, but the chief of these is bronchitis, in all its forms; and with its complications of hæmorrhages, pulmonary apoplexy, œdema, etc.

So long as the blood supplied to the bronchial tubes by the bronchial arteries is returned to the left heart by the pulmonary veins, and even so long as the blood, when obstructed in the pulmonary veins, finds primary relief by regurgitation into the bronchial arteries from the readiness of their communications with each other (Waters, Guillot), the valvular disease may not cause bronchitis; but when the blood is forced back, as it is in extreme cases of mitral valve disease, into the pulmonary plexus of the air-cells, congestion and disease of the bronchial tubes are then induced. Hence, also, the right side of the heart

becomes loaded. But, as before observed, the pulmonary plexus is rarely affected, directly and primarily, by obstructive disease of the left heart; only secondarily, and not until pulmonary venous congestion has been established in the bronchial tubes.

Bronchitis, when caused by valvular obstruction, is always a persistent disease. In its slighter and non-inflammatory form it yields evidence of engorgement, causing the secretion or exudation of a sero-mucous or pituitous fluid. The expectoration of this is difficult, and the cough, necessary to expel it, often distressing. Occasionally the secretion is very considerable, and passes into a bronchorrhœa; the seat of this is generally in the smaller tubes of the lower lobes.

If the mitral obstruction, or even regurgitation, be confirmed, congestion ensues, and then the bronchial disease becomes inflammatory in its aspects; chronic thickening and obstruction of the minute bronchial tubes is very liable to set in, the expectoration becomes muco-purulent, with occasional hæmoptysis and, eventually, œdema takes place in the air-cells and in the areolar tissue of the bronchial tubes. The œdema is, however, of mechanical, and not of inflammatory origin. The physical signs are a crepitation not unlike that of a pneumonia mingled with mucous râles. Though the resonance on percussion may not be generally impaired, there is, here and there, a local dulness, caused by lobular congestion. These often set in suddenly, and if the exudation be sanguineous, the tendency is to pulmonary apoplexy. The hæmoptysis is, in general, small in quantity. In some cases it is of frequent occurrence. This, as well as the muco-purulent expectoration, though often regarded with anxiety by the patient, are beneficial, and a little observation convinces that they relieve the more urgent symptoms both of the heart, and of a difficult expectoration. They also indicate the treatment to be pursued in case of the occurrence of a congestive apoplexy of the lung setting in. It has been thought by some that the hæmoptysis is due to the congestion which takes place in the right heart, but the force of the right heart, in urging on the blood, is too feeble for this. It is doubtless due, as in other cases, to the mitral obstruction.

Considering the amount of degenerative disease that takes place in phthisis pulmonalis, and the consequently recurring irritability of the heart's action, it might be supposed that this organ would take on some notable form of disease; and, seeing also that the obstruction to the pulmonary circulation is very marked, it might likewise be concluded, assuming the correctness of the mechanical theory of the production of hypertrophy and of dilatation, that these conditions would, more especially, be developed. Experience, however, shows that the changes in the heart are only those common to other chronic diseases; wasting of its substance, as wasting elsewhere takes place; and dilatation, when fatty degeneration in the heart itself exists, as it occasionally does. The exceptions to these only prove the rule.

The anatomical facts observed are that the tubercles, whatever may be their origin, do not acquire nourishment from the pulmonary system, but that they eventually become "connected with the bronchial arteries, or with those supplying the thoracic parietes" (Louis); that the adventitious vascular system thus developed occupies largely the lobes, to the exclusion of the pulmonary artery (Guillot); and that the cavities also become supplied by tufts, and by a vascular web connected with vessels proceeding from the aorta; we therefore see that in the course of the evolution of a phthisis pulmonalis the circulation is transformed from the pulmonary to the aortic system, and that the lungs, as the disease progresses, acquire increased capacity for arterial blood, and lose it for venous.

We have in all this three remarkable circumstances: (1) that by the obliteration, more or less, of the plexus of the pulmonary artery, the venous blood is not aërated, but passes on directly to the left heart by the pulmonary veins; (2) that blood proceeding from the aorta is aërated in the lungs; and (3) that this blood returns to the heart not only by the bronchial and azygous veins, but very mainly by the pulmonary veins. The facility with which this transformation of the circulation may be effected, is evident when it is seen that by injecting the pulmonary veins the whole vascular system may be filled;

the branches of the pulmonary veins, the capillaries of the air-sacs, the pulmonary arteries, together with the vessels of the bronchial tubes, blood-vessels, lymphatics, and areolar tissue (Waters); in fact, whatever may be the supplying source, Nature provides that the blood shall be returned to the left heart for systemic circulation.

The occasional lividity or cyanosis of the lips, and mucous surfaces in phthisis, and in other diseases, as emphysema, pneumonia, and in asthma, from atony and spasm of the air-cells, is probably due to this transformation of the circulation, so far as, in each case, the extent of the disease influences it, from the pulmonary to the aortic system. The blue blood is thus primarily sent, through the pulmonary artery to the bronchial artery, and then on by the pulmonary veins to the left heart, and is only secondarily aërated through the aortic circulation. Hence the systemic blood is more or less cyanosed.

The prominent fact as regards the heart is that it is, comparatively speaking, inadequately supplied with perfectly arterialized blood. It is probably due to this that the vital processes of inflammation are not set up in the otherwise irritable and excited organ; and that hence the vice of hypertrophied muscular tissue is not added to the other difficulties of a pulmonary consumption. The wasting and the occasional dilatation are the result of the ordinary laws of muscular decay.

An arterial murmur, not permanent in character, and separate from any emitted by the heart, is occasionally met with in the course of an auscultation of the lungs. A not unusual seat of this murmur is towards the acromial end of the left clavicular region; it is also heard in the right clavicular region, and may be in other portions of the lungs. This murmur is always soft, but is sometimes associated with a kind of click, which is apparently due to some pulmonary complication, occurring with the act of a deep expiration. It has been considered by some observers that this murmur is to be esteemed as an indication of a tuberculated lung.

But it is certainly, in very many cases, neither dependent upon, nor even associated with, a tubercular deposit; and I have known it to occur where there was neither symptom, nor suspicion, of pulmonary disease, but only a well-marked hysterical condition. I recorded the history of such a case in 1838. This lady is still living, and free from all pectoral disease. The symptom has been a marked one on several occasions—occasions generally under circumstances of excitement.

It is probably due to abnormal pressure or distension of an artery, either by reason of the presence of tubercular deposit, or some enlarged or morbid condition of the lung. In these cases I had thought its seat to be in the common carotid artery, where this emerges from the aorta; others have regarded its seat to be in the subclavian artery. As the murmur is not conducted into the carotid, or the branches of the subclavian, arteries, its seat is probably not in either of these, but in the bronchial arteries.

A case of some interest has recently presented itself in which a murmur of this kind is audible over the whole pulmonary area:

H. G., æt. 30, a miller, transferred to my charge from the surgeon under whose care he had been for an ulcered leg, presented (February 27, 1874) the conditions of a tall, broad-chested, well-nourished man, large, fleshy, and muscular, countenance heavy in expression, complexion dusky, and suffuses readily on exertion; frequent short cough, with copious expectoration of a clear viscid phlegm, and which he had experienced for some seven or eight months; chest slightly more arched than natural, uniformly resonant, no appreciable dulness in any particular part; increased resonance over the region of the heart. The general indications were those of an universal, but slightly developed, lobar emphysema. The respiration was slow and feeble, but easily hurried on exertion; expiration more prolonged than the inspiration; there were occasional bronchial râles, especially if a forced or deep inspiration were made. To these symptoms there was super-

added over the whole area of the lungs a soft murmur, synchronous with the systole of the heart. The heart's action was somewhat feeble, the impulse neither increased in force nor extended in area; the apex beat normal, and not perceptible in the recumbent position. The valvular sounds distinct and free from murmur. In the emergent aorta and in the course of the carotids there was a soft murmur; no murmur in the abdominal aorta. The pulse small and feeble, more marked in the right arm than in the left; there was no area of dulness, or impulse, or local exaggeration of murmur to lead to suspicion of an aneurismal dilatation. (14th April.) Cough much relieved, the murmur less intense, and on the anterior portion of the right lung almost inappreciable, but may be made evident on exertion, and is then generally increased over the whole pulmonary area; the face also flushes, and even passes into lividity if exertion be continued.

In the above there are indications of the presence of an emphysema and of bronchitis; no indications of disease in the heart itself, and no sure evidence of any aneurismal or arterial disease. To what, then, is the pervading arterial murmur in the lung due? Is it a conducted murmur from the aorta, or has it an independent seat in the plexus supplied by the pulmonary artery to the air-cells; or in the minute divisions of the bronchial artery?

Whether conducted or of independent origin, we may infer its seat is not in the plexus of the pulmonary artery, as its associations are with the murmur in the aorta and carotid arteries. These associations do not exclude the consideration that its seat may be in the minute divisions of the bronchial artery. We must bear in mind that the bronchial artery separates into twigs which accompany the minute subdivisions of the bronchial tubes, so that each terminal bronchial tube is accompanied by two or three very flexuous arterial twigs, and these anastomose freely. Whether conducted or self-originating, this is probably the seat of the murmur. Considering the improbability of any such uniform conduction of a murmur having its seat in the aorta, and considering, also,

there are evidences of congestive bronchitis and of emphysema, it is not impossible the murmur may have its independent origin in some abnormal condition of the terminal bronchial tubes, with dilatation of the air-cells, producing pressure on the minute branches of the bronchial artery. If this be the case, we may suppose the murmur, audible in the aorta and in the carotids, is due to convection from that induced in the bronchial arteries.

The correct appreciation of the seat and origin of this murmur is involved in some difficulty. Further observation may enable this to be cleared away. In the meantime the fact remains, that the whole pulmonary area may be the seat of an arterial murmur. The explanation of the symptoms exhibited by this patient appears to be that, incidental to his occupation as a miller, bronchitis with emphysema became established in the minute and terminal structure of the bronchial tubes, that these pulmonary affections have disturbed the equable flow of blood through the bronchial arteries, and are now in the course of injuriously operating on the heart and large vessels.

The respiration is often disturbed in affections of the heart, sometimes prominently and painfully so, though there may be little or no disease of the lung; or only such as would not, independently of the heart, induce the character of breathing experienced. Besides the painful breathing associated with the inflammatory diseases of the heart, there are respiratory errors which are interesting, not only as examples of disordered action, but from their significance as indicators of the condition of the heart. These may be ranged under the two heads of (1) hurried breathing, and (2) apnœal breathing; difficulty and straightness may be the qualities of both varieties.

Hurried breathing may be excited on exertion; may occur spontaneously and paroxysmally, or it may be a persistent feature. It occurs on exertion, more especially when the condition of the heart is that of a simple hypertrophy, but when in addition, or even without the hypertrophy, there is fatty

deposit, or ventricular dilatation, the hurry of breathing, besides being excited by exertion, occurs spontaneously, and in the recumbent position may be increased even to intolerance; it is especially indicative of structural feebleness and dilatation. It is markedly associated with straightness in angina pectoris. In the case of confirmed valvular diseases, the hurry of breathing is sometimes persistent; more usually so in mitral than in aortic valve disease. In mitral it is often the first symptom which attracts attention. In aortic valve disease there is usually some antecedent local pain with palpitation. It is an urgent symptom in these affections. All these forms of cardiac breathing may be much increased, if there be also present any congestive diseases of the lung, as emphysema, bronchitis, asthma; and it is necessary, when these are present, not to overlook disease of the heart as a cause possibly co-existing.

The apnœal forms of breathing are the sighing, the "ascending and descending" (p. 96), and the cessation on sleep. Dr. Laycock, who has detailed some interesting cases of these two latter forms of breathing, attributes them to a neurosis of the vagus ("Dublin Journal of Medical Science," July, 1873). The sighing breathing is often associated with fatty degeneration of the heart, and with that condition of the nervous system which disposes to hypochondriasis. It may be concluded that the whole of these errors of breathing are due, directly or indirectly, to functional disturbance of the ganglionic or vagus nerves.

CHAPTER XII.

SUMMARY AND CONCLUSION.

On reviewing the history of a large number of cases of disorder of the heart, the attention is arrested by the different features presented for observation, more especially in respect of time and intensity. Some examples are rapid in their course; some the process of years; some appear to be stationary, and some to pass away and resolve themselves into the normal state; some are painful, and immediately demonstrate themselves; some are covert and inappreciable.

It is important to recognise the existence of these varying conditions, not only as a portion of the history of the diseases themselves, but in order to arrive at a correct prognosis and the true indications of treatment.

The ancient popular impression, and indeed the professional one also, that all diseases of the heart were necessarily and early fatal, and generally suddenly so, has long been discarded; and, in its place, the consolatory truths have been established, that some may be cured, some may be tolerated, some consistent with a long life passed free from pectoral embarrassments, and even in happy ignorance of being the subject of disease; perhaps the larger proportion of the most prevailing forms may permit, not only the attainment of the ordinary term of life, but, when life ceases, may be so completely in abeyance as not to be the direct cause of death.

The consciousness to the individual of the presence of heart disease is no measurer nor criterion of its urgency. It may be but a slight attack with no threatening consequences, and yet its symptoms may be marked by great severity; or its tendencies

may be fatal, and yet the symptoms demonstrated scarcely appreciable. It is even said that the apparent urgency is sometimes in the inverse ratio of the gravity of its development.

Disease of the heart has nevertheless its anxieties, its pains and distresses, its fatal tendencies, and its sudden conclusions of life. It has been seen that it is mainly to be studied under the forms of inflammation, valvular imperfection, and structural degeneration.

For the most part the inflammatory diseases of the heart, if early and efficiently administered to, are not fatal; but they are apt to be the origin of evil consequences, and, specially, to be the cause of structural mischiefs, which progress unfavourably to the well-being of the individual, and materially serve to influence the duration of life. These mischiefs are hypertrophies, valvular imperfections, and degenerative lesions. Each of these may, however, have an independent origin, and be found to be established without any demonstrable reference to an antecedent inflammation; or they may be associated with, and dependent on, each other.

The progress of hypertrophy is generally marked by the increase of impulse; valvular disease, for the most part, proclaims itself; degenerative diseases are often insidious, slow, and covert—"go on till discovered, and then go on." These several diseases are capable, may be, of alleviation, or of being arrested in their course, but they are incapable of being cured. As they are, so must they remain; unless they proceed to a worse state of structural degeneration.

Valvular imperfection is not often a fatal disease, perhaps only very rarely so, without the concurrence of the secondary effects which they themselves not uncommonly produce, or of some co-existing wall disease. But when either of these are co-established with the valvular disease, the results are usually calamitous. It is difficult, perhaps impossible, to arrive at a correct appreciation of the relative fatality thus induced by valvular disease, whether it be considered as a whole or in detail. Though there may be no want of the copious enumeration of fatal cases, together with accurate and minute histories of the structural lesions attending

them, yet these furnish no sufficient data for a comparison, inasmuch as there does not, and probably never can, exist an accurate and sufficient record of the number of cases which are not fatal. In other words, the amount neither of valvular disease, nor of its varieties, has ever been ascertained, and without this no true inferences can be deduced. The morbid anatomist may carefully detail the kind and the amount of diseased structure he discovers, and, so far, his statistical deductions are accurate and reliable, but the physician cannot tell what may be that other number, in which similar disease exists; and who do not die in consequence, or may pass on in the crowd as not having it; neither in the cases that he does investigate can he, in the larger number, trace when or by what means the disease commenced. A recent observer, taking for his data the revelations afforded by post-mortem investigations, states, if I understand him rightly, that the most fatal of the valvular diseases is the aortic regurgitant; then, in succession, the mitral regurgitant, the mitral obstructive, and the aortic obstructive. If we look to the evidence afforded by clinical observation, one would rather regard the diseases in the valves of the right heart, when they are established, as the most fatal, and then those of the mitral valve. The disorders consequent on the aortic valves are those of failure and deficiency; those of the auriculo-ventricular valves of congestion—and certainly the contemplation of the consequences of congestion lead to the conclusion the causes, which induce this, are the most fatal. In whatever valve the disease exists, it may, however, be assumed, that if it be gradual in its origin and only slowly progressive in its course, life may be long sustained: but that if its origin be violent, its course will be rapid, and lead to an early fatal termination. Age too is an element; the mitral is the disease of early life, the aortic of middle and later years.

The degenerative diseases are those of hypertrophy, dilatation, fatty deposit, fatty degeneration, and connective tissue degeneration. These may each occur in connexion with, or independently of, valvular disease; when so connected they are earlier developed, and more rapid in their course. Unconnected

with valvular diseases, they may be slow in progress, and, perhaps, not tend to shorten life. The most fatal of the tissue degenerations is that of dilatation, and this is probably due to its being an advanced stage of the others. The next is the fatty degeneration, which, by metamorphosis of the muscular fibre into fat, impairs the action of the heart. The muscular hypertrophy, the fatty deposit, and the connective tissue degeneration, are consistent with life so long as they are not sufficient to embarrass its propulsive power. But sudden death takes place in these if overtaxed. In estimating the probable danger of these forms of disease, constitutional predisposition, age, co-existing conditions of health, as gout, cachexia, chlorosis, etc., may be duly considered, and their influences weighed.

On reviewing the whole question, it may be stated that sudden death, taking the full range and amount of heart disease into account, is an event of only rare occurrence; that its occurrence in the case of uncomplicated valvular disease is especially rare; and that when it does occur, in cases where valvular disease exists with other lesions of the heart, it may be equally attributed to them as to the valvular disease; that it usually takes place in those cases where there is dilatation and degeneration, more especially of the nature of a fatty metamorphosis, to such extent as to impair the dynamic efficiency of the heart; that death usually happens through the secondary effects of congestion and of anæmic debility; that in the larger number of instances the disease is slow of development; and that life may be prolonged, notwithstanding, to its natural term, and without eventually succumbing apparently either to its direct or indirect influences.

Whatever may be the nature of the disease, its contemplation teaches us much worthy of consideration. One great lesson is that, though only slight in itself, it may be the forerunner of consequences which are urgent and fraught with danger; and that, therefore, in the management of heart disease, the abstaining from all which may over-exert, excite, or over-stimulate the already disordered organ, should be strenuously inculcated; and another

great lesson is that, above all things, the physician must bear in mind, as he can neither restore nor repair the physical mischief that exists, he must, while seeking to succour and to alleviate the attendant distress, and to arrest the progress of mischief, avoid to do that which may, in any way, weaken the heart or impoverish the general condition of the health.

When the existence of disease has been carefully and accurately ascertained, the question arises as to the policy of communicating to the patient and to his friends the precise condition of things. To friends there can be neither doubt nor objection that the communication should be candidly made, so that they may be fully aware both of the prospective dangers and favourable aspects of the case. Unless there be some pressing condition, strongly and markedly indicating the probability of an early, or, may be, sudden dissolution, it is, nevertheless, always advisable so to guard and explain the opinion that the friends be not looking forward, in abiding alarm and anxiety, to the occurrence of such an event. The greater probability of this not being the result, possible though it may be, should be strongly and clearly inculcated upon them. It is distressing both to friends and to patient that an anxiety of this kind should be entertained. To the patient, my own conviction is, that it is the proper course to state to him distinctly the nature of his disease, and fully to explain to him his position in reference to it. My own experience has been that this course, so far from injuriously alarming, has been attended by beneficial results. He is better satisfied to know his real position, and he reconciles himself to the necessities it entails upon him. Unless the immediate state of things contraindicate it, we are more than justified in giving the assurance that the tendencies of heart disease are rarely to a suddenly fatal termination, and that its presence, with due care, is consistent with a prolonged and not painful existence. It is our pleasant duty fully to explain this.

Physicians are occasionally called upon to advise as to the

probabilities of life when disease of the heart is a condition. Assurance offices look to their medical advisers, not only for accurate information, but for sound and guiding opinions. Are such lives insurable, and upon what terms? It may at once be stated that all active and inflammatory forms of heart disease, and all existing blood affections, are uninsurable at any appreciable rate. Then we have to deal with chronic disease, and it has just been stated that there are forms of disease in which it is justifiable to state to the patient the possibility that life may be prolonged, and so that the ordinary term of life may even be attained. Diseases capable of activity and an early fatal result may remain latent for years. Would an assurance office be justified in such case in granting an assurance? I think not. No rates appear adequate to cover the risk of even seemingly trivial diseases, and amongst such must not be included any indications of hypertrophy, dilatation, or degeneration of the parietes, or any errors of rhythm, or valvular irregularities in association with them. None of these can be even considered as trivial diseases, and they certainly are not insurable.

True, many affected with disease of the heart do live for years, and may even attain to advanced ages; but can these be reduced to a rule? Can they be placed in well-defined classes, so as to illustrate a law of life assurance? If not assurance would be a speculation and not, which it really is, an art founded on the science of probabilities. The principle that must guide the practice of assurance offices is the assurance of selected lives from such classes as they may admit to assurance. The bearings of this argument may be illustrated by the two classes of "Rupture" and "Drunkards." The increase on the ordinary rates in "rupture" is commonly 10 per cent. The numbers of those dying in consequence of it are ascertained to be in such proportion that this rate is proved to be adequate, and, therefore, a safe law of assurance. With regard to "drunkards," though some live to good ages, the tenure of life in this class is so uncertain that no law can be established, and they are practicably uninsurable. They, in fact, belong

to a class that has no certain relation nor reference to the life law of the population in general. The same may be said with regard to diseases of the heart. An assurance office must not be guided by the fact that some survive to good ages, but, before involving in such business, must ascertain the numbers that survive at each period of life from amongst those that have heart disease; and then having discriminated and separated into classes each separate form of disease, it would be requisite solely to insure favourable and selected cases.

The only form of diseased action which I can contemplate as being entertained by an assurance office is where a valvular murmur is associated with no degenerative wall disease, and with neither regurgitant nor obstructive valvular disease, and this can only be inferred by the entire absence of all pulmonary and systemic congestions and irregularities. Perhaps the only insurable valvular murmur is that which indicates slight but not obstructive deposits on the valves, and these it would probably not be safe to insure, without an increase of some seven, or ten years, to the rates of the ordinary period of life of the person proposed for assurance.

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