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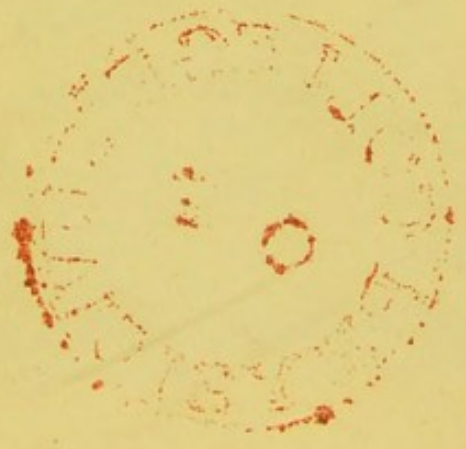

DISEASES OF THE HEART,

THEIR

PATHOLOGY, DIAGNOSIS,

AND

TREATMENT.

BY

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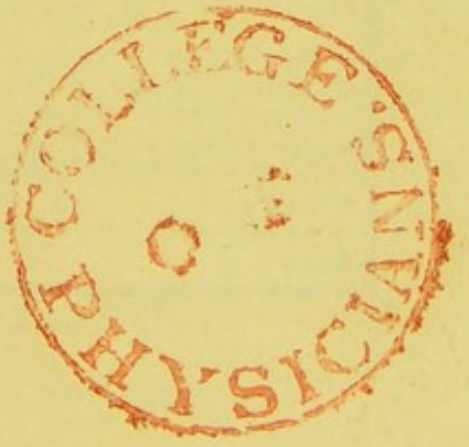

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PREFACE.

In the pages which follow, I do not pretend to offer the reader a full and didactic relation of all that may be said concerning Diseases of the Heart; but, on the contrary, have rather wished to exclude therefrom all matters which did not seem to have a direct and immediate reference to their practical consideration, or which might overshadow and obscure their main and most essential features.

I have been particularly anxious to place in their true positions two divisions of the subject—namely, the Pathology and the Treatment of acute cardiac diseases; and have endeavoured to state, in clear terms, the knowledge which we possess of the one, and what reasonable degree of faith

experience warrants us in placing in the efficacy of the remedies which we employ in the other.

Pathology, I have reminded the reader, is something more than mere pathological anatomy—something beyond the study of structural changes. It comprises, besides this, all that we know of disease through derangements of functions observed during life, and through the effects of remedies. There is, indeed, a physiology of disease as well as of health—a pathological physiology. Pathology enables us to observe certain effects produced by disease during life and after death. And it does more than this; it enables us, by reason, to follow the disease some short step beyond these, its visible manifestations. There are many diseases, indeed, of whose pathology we know nothing, excepting what we learn of it through disturbances of function manifested during life: of these, pathological anatomy renders us no account whatever. What, for instance, do we know of the pathology of tetanus or of hydrophobia, beyond those striking disorders of functions which characterize them during life? Reason,

nevertheless, gives us the full assurance that such disorders are the resultants of anterior changes in the system, though anatomy cannot find them out. And thus, pathology has also to do with facts where neither the sight nor the touch can take cognizance, and which can be reached only by a process of reasoning.

A clear comprehension of these undeniable facts, brings us to a right estimate of the meaning of pathological changes of structure, and gives us a key to their proper consideration as objects of our therapeutics. Inflammation of a joint may be the result of local injury; and it may be the result—the local manifestation—of a constitutional disease. The inflammatory processes in both may be much alike; but how unlike are the ideas which the inflammations excite in the mind of the physician!

Such is, in a few words, the view which I take of the pathology, and such the manner in which I have wished to apply it to the interpretation of diseases of the heart: remembering that pathology and the right cure of disease, must necessarily go

hand in hand, or, in other words, that a knowledge of its pathology is essentially necessary to a right understanding of the treatment of disease.

With regard to the treatment of acute cardiac diseases, I would observe, that I have not attempted to avoid a discussion of the difficulties which beset the subject. There is no class of acute diseases — and it is useless to attempt to conceal the fact — in which the difficulties of treatment are more strongly illustrated. And need we be surprised if doubts and difficulties rest upon their treatment, when we recollect how incomplete is our knowledge of their pathology, and that only a portion of its entire history is yet revealed to us? We are, it is true, as men of action in the face of disease, compelled to act, and must therefore select some particular line of procedure; but in doing this, it is our duty to keep this fact fairly in view,—namely, that their true *ratio medendi* is yet to be discovered—is a problem yet to be worked out. The proof of this assertion is, I believe, to be found in the past and the present history of the treatment of

these diseases; to which I would refer him who doubts its truth. The statement may, perhaps, appear strange to the ears of some of my readers; and as the fact (as I deem it) involved therein is one of extreme interest to the profession, I shall take the liberty of here stating briefly the grounds which I feel justify me in making it.

Let us, then, consider what these acute cardiac diseases are, and what the remedies we have to oppose to them. Here is pericarditis, for instance, —in its acute form, a typical representation of the thing, whatever it be in its essence, denominated inflammation. Here is a malady which may destroy life at once; and if it kill not, will yet leave behind it, we must ever dread, a disorganization of one of the tripods of life—the great central and ever-active organ of the circulation; a disorganization which will, in its turn, give occasion to other secondary disorders, which sad experience tells us have, for the most part, but one issue, and that a fatal one.

What now are the antidotes which medical science, resting upon the accumulated experience

of the past and the present, proposes for such a bane? A skilful diagnosis has enabled us to trace most anxiously, from day to day, and with surprising accuracy, the progress of the malady; and we can even sometimes anticipate its attacks. Scarcely sooner can pathology demonstrate the existence of the inflammation to the eye, than does our diagnosis reveal it to the ear. If ever disease demanded the physician's earnest care, this surely does. Its progress is rapid; it may kill at once; and it may leave permanent disorganization of structure. What then has medicine to oppose to this destructive malady? Some few years gone by, the practice was, to meet the violence of the inflammation by the extremest antiphlogistic measures; the lancet was plied with a most unsparing hand, and with the most unhesitating faith in the propriety of its use. But where are the believers in or imitators of such a practice now? This heroic and *certain* method, as it was called, of arresting the destructive agent, —of *extinguishing* the disease,—has been convicted of error, and condemned by later authority as

"uncertain and very dangerous." Now-a-days, the inexperienced are rather warned by their masters against the use of the lancet, than told of the benefits of blood-letting. We find expressions of regret at the disappointed expectations of former hopes and faith in the remedy; warnings of the dangers which it may lead to; caution advised in its use; and no estimate offered of its actual service.

And again, does modern observation still ech the praises which were once so warmly bestowed on mercury as an arrester of cardiac inflammations? "After blood-letting, rapid induction of the mercurial action is of the greatest consequence", wrote an authority, in a most unhesitating style, some fifteen years ago. And now, when we turn to the last best authorities of the day, who have given the world the benefit of their long and great experience, and seek for guidance, what do we find? We find one of the most observant and practical physicians amongst us, admitting, that the firm faith which he himself once reposed in the efficacy of this remedy, had been undermined by

the truth-telling effects of further experience: and then again, if we look to the latest-published work of authority for consolation, we find that the portions of it which treat of the remedies to be applied to these acute diseases, are just the most unsatisfactory portions of it.

And all the other methods of combating acute cardiac inflammation, which have been from time to time well recommended, and are now in practice amongst us,—what shall we say of them? Let me put the question in another form; and, appealing to the experience of individuals, ask, What is the treatment they adopt in arresting the inflammation? In the answer to this question will be found the fact,—which any one who has had occasions of observing the practice of different physicians must well know,—that there is no general accordance in opinion or in action as to the course pursued by them. To all of us it has happened, that the hopes and the confidence of to-day, derived from some happy issue which the disease has had in our hands, under one method of treatment, have been belied by the reverses of

the morrow. Is it not a fact, moreover, that the greater the experience the physician gains, the more cautious and quiet—the more expectant, the term is—he becomes in his treatment? What other cause is it, but repeated failures in treatment, that has driven some physicians to the pass of leaving the disease and the system to struggle for the mastery between them,—physicians, we must remember, whose honour, and reputation, and scientific knowledge, are above all doubt. They have done so, they tell us; and we are bound to respect their opinions, as the conclusions forced upon them by long and well-gathered experience.

A firm conviction in the efficacy of any particular treatment, founded on the results of our own personal experience, though natural enough, often exercises an influence most baneful, and grievously obstructive to the progress of the medical art. There is, innate and constantly alive within us, an ever-hasty desire of generalizing upon, and drawing conclusions from, the results of our observation. However isolated a fact, however barren it may yet intrinsically be, the eager mind still strives to

poses some favoured plan of treatment that has found great favour in our eyes.

What would I infer from this? That the physician is to remain an idle spectator; that he is to sit quietly looking on, while the disease is running riot and committing its worst ravages? Assuredly not;—the man of action must act, even when struggling through the mist of errors: but this I would infer, that he must act reasonably, that is, with a full acknowledgment and appreciation of the difficulties which bestrew every step he takes; cautiously, and with the sentiment that each act is an experiment, and that the experiment may be unhappy as well as happy in its results, and that it may be for evil as well as for good.

I have dwelt upon this not over-agreeable subject, because I feel that it is one which must ere long claim more earnestly than it has yet done the attention of the profession, not only as it relates to the class of diseases here particularly put in question, but as it affects most other acute diseases. Every day's new experience in pathology teaches us, that in the treatment of disease, we have much

to unlearn as well as much to learn. And how can we expect to progress in a right direction, while error still clings to and clogs our footsteps? The very discordant opinions which equally honest and equally skilled observers maintain,—observers not living in separate ages, or in different countries, or in separate cities, but exercising their art upon the same disease, under the same roof, in the same public hospitals,—must have a meaning. Is it not one which is, oftener than we care to confess, responded to by our consciences at the bedside of the patient? From the follies and wickednesses of others we may learn much; and does not homœopathy teach us this, that the human body has suffered from a want of discrimination in the use of remedies on the part of its legitimate professors? To what other cause can be attributed the successes of that remarkable imposture?

How are we to reconcile but in one way the unhesitating opinions which men hold to-day, and the equally unhesitating, but exactly opposite opinions respecting treatment of disease which they hold to-morrow? We may say, diseases change

their type, and many persons would seem to take the supposition for granted as an undeniable fact; but what proof have we of it? When we come to calmly investigate the grounds upon which such a conviction on their part rests, we find what a "baseless fabric of a vision" they form; the conviction becomes a mere vague idea, resting on some floating notions of the mind. How, for example, must we proceed, if we would prove the dissimilarity between pneumonia of to-day and the pneumonia of twenty years ago? evidently by the comparison of the history of the disease now with its history then. Well, I fancy he must be a bold man who would attempt to work it out by the aid of facts. Differences in type at different epochs! why how many differences in type does not the same disease present at the same epoch! What are all the different modifications in symptoms which disease presents to us, except changes in its type, and changes, indeed, chargeable to the influence of age, sex, climate, and the thousand ever varying accidents which beset the life of man? Are we to believe that the human body has so

changed its conditions, or that diseases have so totally altered their nature, as therein to find an explanation of the fact of the wholesale blood-lettings, which characterized the treatment of disease in the last generation, and the almost entire abstinence from blood-letting, which characterizes the treatment of the present race of practitioners?

Is it unfair or untrue to say that, in the opinions which we form as to the effects of remedies on disease, we very frequently permit ourselves to be guided to most illogical conclusions? and that in forming our conclusions on too narrow a basis, we thus often ascribe to therapeutical agents, as regulators and controllers of disease, a power and efficacy which the reason of the case in no way justifies? If this be untrue, then I ask him who denies its truth, to explain satisfactorily how it is that there is such a want of uniformity, nay, such an actual diversity, exhibited in the treatment of the same disease by practitioners in different countries, or different epochs in the same country, or at the same time and in the same place, by physicians practising upon indivi-

duals subject to precisely the same external influences.

I surely need not linger to give proofs of this diversity of opinions. If the induction of mercurialization be, as we are taught here in England and in France, the true and orthodox treatment of primary syphilis,—how comes it that such an employment of mercury is positively anathematized on the other side of the Tweed?* Here the remedy is a sheet-anchor, there it is proscribed as a positive poison; symptoms which one man regards as the consequences of syphilis, are by the other proclaimed to be the results of mercurial poisoning! Now, is anyone to believe that syphilis is one thing in Edinburgh, and another thing in London and Paris; that what is a cure for it in one place becomes a poison when used in another?

Facts like these the experience of every one will supply him with in abundance,—indeed, but too abundantly; and to what conclusions do they

* I am of course supposing, that the opinion of those who speak with authority—*ex cathêdrâ*—expresses the opinion of the country.

inevitably lead? manifestly to this, and I do not shrink from stating it, namely, that more faith is placed by us in the power and efficacy of therapeutical agents as regulating and controlling acute diseases than reason warrants; and that the exact amount and degree of influence exerted over such diseases by remedies are, in very many cases, not readily demonstrable.

It is difficult to write on this subject without the fear of being misconceived. I trust, however, that no one will suppose, from anything here said, that I am silly enough to be sceptical as to the uses, advantages, and power over diseases, which therapeutical agents possess; or that I am desirous of running my head against the solid rampart of real facts, which medicine has stored up concerning the treatment of disease, and of which she possesses a goodly store. What I do fear is, that we are very apt to attribute to the operation of our remedies, effects which are due to other agencies; and that inordinate attention to purely therapeutical treatment of disease may distract our attention from the proper management of it, by

a due consideration of diet, habits, mode of life, etc., etc.

But, as I have already said, as practitioners of medicine we must act; and the particular treatment recommended in the following pages, as applicable to the cure of acute cardiac diseases, is that which is recommended to us by the experience of wise and experienced practitioners, and therefore that which I adopt without hesitation, under the restriction of precautions here prescribed. That its actual influence over the disease has yet to be demonstrated, may be said without injury or blame to our art. The problem is one whose solution is manifestly most difficult. The remedy employed may be alike; but reflecting for a moment on the thousand ever-varying influences, subtile and inappreciable to our senses, to which the body of man is subjected; and remembering how dissimilar in some one or other particular (of which we can take no cognizance) of its organization is the human frame, we see at once how it is that every case must be specially affected, after its own fashion, by that remedy; affected in a way dis-

similar from every other case. The conditions of the problem, so to speak, differ in every case, and therefore so must the results.*

But some main general conclusions, it might be suggested, have resulted from the inquiry. Though individual facts offer shades of difference, they may possess some common features of agreement. This we cannot deny; and it is here that we apply for help to the large experience of those practitioners in whose clear-seeing observation we have full confidence.

But I must not further pursue this subject here. Perhaps I have said more than enough to impress upon the reader the great difficulties which, in my opinion, surround the treatment of acute cardiac diseases, and the caution which it behoves the practitioner to use in his dealing with their cure. I trust I may not be accused of presumption in speaking thus freely on this topic.

* It must be distinctly understood, that I am speaking solely of acute inflammations of the heart. As regards its chronic diseases, or rather, those disorders which result from its disorganization—it is admitted, on all hands, that medicine gives relief in a manner which is most manifest and unequivocal.

What I have ventured to express, is the result of impressions forced on my mind by neither a partial nor a limited observation; and perhaps, as still further justification, I may be permitted to add, that the class of diseases here particularly referred to, is one which has long been with me, in an especial manner, the subject of consideration.

W. O. MARKHAM.

Clarges Street.
February 1856.

DISEASES OF THE HEART.

INTRODUCTORY CHAPTER.

GENERAL REMARKS ON THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CARDIAC DISEASES.

It has been my desire in the following pages to place before the practitioner and the student a summary of the positive knowledge, which medical science has acquired up to the present time concerning diseases of the heart; and I venture to believe, notwithstanding the many excellent works on these subjects which have been of late years produced for the benefit of the profession, that there is yet room for another, written in the manner which I have here desired to follow out.

I have been anxious to keep the following objects continually in view: to eschew, as far as possible, all theoretical considerations, except in so far as they tell, in a clear and consequent form, upon the practical bearings of the subject treated of; or, in other words, to accept nothing which is not manifestly capable of being usefully applied, directly or indirectly, to the elucidation of the diseases of the

heart: and carefully to select all the available materials with which experience has up to the present time furnished us, in order to place them to the hand of the practitioner ready for his use. I have especially desired, also, to make them all converge, and be subservient, to one particular end,—the end and last object of all medical knowledge,—the treatment of disease.

The history of diseases of the heart strikingly illustrates the advantages which are to be derived from the carefully following out a correct method of observation. No class of diseases, in fact, more clearly demonstrates the progress which the medical art has made during the past and present generations. Our knowledge of every part of their history, of their pathology, their diagnosis, and their treatment, has been greatly widened; the gross errors which obscured the subject removed; and what we do know is placed on the sure and firm basis of facts resulting from direct observation.

The study of pathological anatomy, that is, the study of the diseased conditions of the organs and parts of the body, was necessarily the first stepping-stone which conducted us to a right understanding of their diagnosis and their treatment. It enabled us, by appeals to material facts, to correct in great part the erroneous views previously held concerning the nature of diseases,—views which mere fancy had ofttimes engendered; it gave us sure ground

whereon to establish a path of observation, by following out which we may reasonably hope in the end to arrive at their true comprehension. From pathology arose the physical diagnosis of internal diseases, a method of investigation by which we have been enabled to attain an unhoped-for degree of certainty in our discrimination of those diseases. The conclusions, indeed, which we derive from the facts obtained by the aid of physical diagnosis, are altogether founded upon our knowledge of their pathology. And thus through the study of their pathology and physical diagnosis, it has necessarily resulted, that the special objects of our treatment in cardiac diseases have become distinctly placed before us.

The study of pathology, thus rightly pursued and rightly understood, necessarily forced the physician to forsake the regions of imagination, wherein he had been too fondly wont to seek his ideas of the nature and treatment of diseases, and to search for them in the positive abnormal changes of the parts and organs of the body: it taught him, in fact, to connect alteration in function with alteration of organization. From the moment this method of analysing disease was adopted in practice, medicine assumed something of a scientific position; practice took its stand on facts, on the results of actual observation.

The progress which medical knowledge, thus

pursued, has made is really surprising ; much more so than, perhaps, at a mere outside view of the matter, we may be apt to imagine. To learn the full value of our gain, it is necessary to look back into the past history of medicine, and compare the knowledge then possessed by its practitioners, before the date when pathology and diagnosis, as here described, were made parts of its study.

We have, indeed, at length obtained something of a sure footing in our knowledge of the nature and treatment of disease. We can measure the amount and the value of that knowledge nicely ; we can see clearly enough how far pathology leads us, how far therefore it becomes a guide to us in practice ; we know at a glance where our knowledge is based on the demonstrated, and where it is derived simply from hypothesis ; and if we are still compelled in practice to proceed and act without the guide of positive demonstration, we have at least the satisfaction of knowing that all we do is founded on the best and most reasonable knowledge, which our means enable us to lay hold upon, that is, is founded on the reason of the thing.

Perhaps it may not be uninstructive here shortly to glance at the actual position of our knowledge of diseases of the heart—of their nature, diagnosis, and treatment—thus reduced to its true proportions by modern pathology and diagnosis. How far, we may ask, does pathological anatomy lead us

upwards to an insight into their real nature? What amount of information, capable of being put to practical use, does physical diagnosis afford us, concerning the pathological conditions of internal organs? What power does treatment exercise over these pathological conditions? What are its effects in opposing, counteracting, or removing the primary diseases and their secondary consequences?

No one fact of greater importance, for it bears immediately on the practical treatment of disease, has resulted from modern pathological investigations than this, viz.: that local changes of structure are, in the very great majority of instances where they become the proper subjects of medical treatment, connected with some general disorder of the whole system; the structural alteration, unlike the consequences resulting from traumatic injury, not being essentially *the disease,* but only one, and as it were the partial, expression of some widely extended influence; some poisoning of the blood; some deranged state of the nervous centres; some faultiness in the nutritive powers; some depraved condition of the absorbing or secreting forces. As our views of pathology have been widened by more extended observation, local changes, local, so called idiopathic, inflammations, have become less and less recognized by us as, *per se, the diseases.* We have been taught to look beyond these local manifestations of disease; and in doing so find reasons

enough to believe, that, in the very great majority of instances at least, what we have been accustomed to consider as simply the products of local inflammatory action, are in reality but the partial consequences of a wide spread influence; having relations to, and connections with, other disorders of structures, situated perhaps at distant parts of the body, and to which, apparently, they bear no immediate analogy in their characters.

Applying these observations to the diseases of the heart, we may ask in what instances, except in cases of traumatic injuries, can the acute diseases of this organ—its inflammations—be regarded as purely idiopathic? When do we recognize the signs which tell of the existence of endocardial or pericardial inflammations, and not find also present some connecting links, which refer them to a general condition of the body, which shew that they are but parts of a general disordered condition? The cases in which there can be a pretence of saying that the endocarditis or the pericarditis has arisen independent of such connexion are indeed so rare, that they may be well ignored as obscure or ill observed, and as quite unfit to serve as data for argument. How important does this fact—even though it be not an universal one—become in daily practice! The physician, thus regarding the matter, of necessity draws the important conclusions from it, viz., that in the treatment of these diseases he

has two distinct facts to deal with,—the force and extent of the local inflammation, and the nature and character of the particular disordered state of the body with which it is associated. By these two facts, not by one to the exclusion of the other, his treatment must be directed, and they must be the guides and the measure of his remedies. Endocarditis, for example, connected with rheumatic fever, and endocarditis connected with uræmic disease, may yield to physical diagnosis precisely similar indications of their presence ; but how utterly different in the two cases do the particular inflammations become when we search out and find their *general* connexion ! how different the prognosis, how different the treatment, how dissimilar the entire character of the diseases !

And it is equally the same with pericardial inflammation. The local signs, which alone tell with unerring certainty of the existence of the inflammation, may be alike, but how vastly different do co-existing circumstances render its significations. In the case where it is, for instance, associated with rheumatic fever, the local inflammation becomes of every importance ; it is that to which the physician in an especial manner directs his remedies ; it so predominates in the train of circumstances with which it is connected, as for the moment to render these other circumstances of the malady subordinate to it ; the general, specific character of the

disease is lost, as it were, in the importance of the local inflammation. Then, on the other hand, consider the pericarditis as arising during the course of uræmia, of pyæmia, or of phlebitis, and we find how unimportant, and of how secondary a value it is, in comparison with the other and more general particulars of these diseases.

And then again, as regards the different degenerations of textures which affect certain parts of the heart, and give occasion to disorders of its functions, do we not find these more or less connected with disturbances of the nutritive or other functions in other parts of the body? Atheromatous and calcareous matters deposited in the inner membrane of the heart or in the coats of its great vessels, tell of a general disordered tendency of the arterial system throughout the body. Fatty degeneration of its muscular tissue is no isolated complaint. Hence, should diagnosis give us just grounds for believing that we have to deal with any of these particular conditions, we know whereto our remedies are to be directed; not surely to these the mere local accidents, but against that general cause which has produced them, if haply we may stay its action. And we have also to deal with the consequences of the disease, and with the secondary disorders which result from the local organic mischief. The valves, we will suppose, for instance, are rendered defective through the degeneration of

their structures—are ossified; we cannot supply their defects, or change the morbid structure, which has rendered them defective, but we can assist the different organs in their attempts to struggle against the results of those defects. The muscular tissue is degenerated; we cannot restore it to its original state of integrity, but we can, by careful hygienic rules, supporting the general health and aiding the powers of the system, hope to stop the progress of the degeneration, and, at all events, ward off for a time the dangers which are to be anticipated from such a condition of the heart.

Modern pathology, again, has taught us the true meaning of hypertrophy of the heart. The time has been when the whole efforts of the physician were exerted in the attempt to reduce this enlargement of the organ; but now we understand, that so far from being the special object against which to direct our remedies, it is an actual provision of nature, brought into action in order to supply an unnatural need. The circulation of blood is interfered with, either in the heart from defect of its valves, or in the great vessels from structural changes, or in the peripheral extremities of the vessels from obstructions situated there, etc.; and to overcome these obstacles, and to supply these defects, it is requisite that the heart's power should be increased: it is consequently increased in size, and so increased in force.

Pathological anatomy, aided by the modern microscope and modern chemistry, has thus given us clear and succinct ideas of cardiac diseases. It has brought us to a certain point in their history, but there it leaves us; it shows us structural changes, abnormal conditions, and diseased products; but why these structural changes, why these abnormal conditions, why these diseased products, it tells us not, or tells us very indistinctly. Here the microscope and chemistry fail us. That anterior fact which provokes these changes eludes us. What that effective cause is which, in connection with rheumatic fever, so often induces endocardial and pericardial inflammations; and why, in rheumatic fever, again, the heart so often escapes unscathed, we know not. Why in one disease the pericarditis is so prominent, and why in another so subordinate a fact; why it should be at one time the especial object of treatment, and why at another quite a secondary object,—all this has yet to be explained.

The *physical diagnosis of cardiac diseases*—that is, the knowledge of the diseased conditions of the heart, derived from alterations in the natural character of the heart's sounds, and from the presence of sounds (coincident with the heart's movements) where none exist in health—necessarily premises a knowledge of the pathological states of the heart. Their pathology is involved in their diagnosis; an abnormal sound has no meaning,

when its idea is separated from the pathological condition which produced it. The one, indeed, to a great extent illustrates the other, inasmuch as defect of organization of the heart is very frequently associated with complete change in, or partial modification of, the natural characters of its sounds. Pathology describes, after death, the physical changes which disease has produced, and diagnosis enables us, with more or less of certainty, to ascertain their existence during life.

It would be quite out of place here to linger over the immense advantages which medicine has gained through the discovery of the physical diagnosis of internal diseases. The study thereof is now an essential part of the physician's education. The great accuracy with which it often guides us to the seat of the disease, and the insight it gives us of its nature, have rendered it a most attractive study. Here we find that acute observation will lead us to something approaching the demonstrative, which is enough of itself to make the study grateful to the practitioner of medicine. The charm of the study, however, appears occasionally to lead its cultivators into a fault which respected authorities have warned us to beware of: it has been thought that the study of these physical signs may be too minutely prosecuted; that relying thereon, observers have pretended to a much greater accuracy in their diagnosis than the nature of the subject admitted

of; and that, by lingering over these signs, they are apt at last, in the application of their treatment and in forming their diagnosis, not to give sufficient consideration to the general symptoms, and unfairly to subordinate them to the local signs.

That the objection is in part good, no one can deny; and I refer to it here, as a warning well worthy the student's attention. By overrating the value of the physical signs, and too fondly trusting our powers of physical diagnosis, we are apt to forget, or too lightly to pass over, the still more valuable facts which the general symptoms present to us. We say, still more valuable, for we believe it is undoubtedly true, that in the great majority of cardiac diseases, and in the secondary disorders to which they give rise, the general symptoms regulate and guide, and are the measures of our treatment, rather than the physical signs: these point to the seat of the disease; those direct the treatment. We have endeavoured in the following pages to point out the real value which, as practical men, we should attach to the physical signs and the general symptoms, giving to each its proper share of attention; and to show how, upon a consideration, not of one alone, but of both aids, we should found our diagnosis, and how fallacious the one must be to us if accepted to the exclusion of the other.

But putting aside all fine discriminations in phy-

sical diagnosis of cardiac diseases, which, after all, are of very minor importance to the practitioner in directing his treatment of them, I will here just state in outline the broad and important facts which it reveals to us, and which, it may be added, would either be very indistinctly perceived or totally concealed from us, were we deprived of its aid. It is, indeed, most satisfactory to be assured, that great skill in the practice of physical diagnosis is not requisite, to arrive at such an amount of knowledge as shall suffice to indicate surely to the observer—at least, in the very large majority of cardiac diseases—that the heart is affected. That degree of acute observation which pretends so nicely to discriminate, during life, all the minute differences of diseased structure, the exact positions and relations of them, which, after death, when they become the direct object of vision, pathology points out to us, is certainly not necessary to guide the hand of the practitioner.

The following are the broad facts which he requires to be master of; and to know them—that is, to be able to arrive at the power of discriminating them—demands no more than an ordinary degree of attention, such as any one capable of the practice of medicine is surely able to apply thereto.

Pericarditis and endocarditis, inflammation of the inner and of the outer membranes of the heart, have their clear and distinctive physical signs.

There is no mistaking, say what we will upon the subject, an ordinary every-day friction sound for an endocardial murmur. Cases may and do occur, in which it is difficult, and even impossible, to tell the difference between them, that is to determine whether the murmur heard be endocardial or exocardial; but such cases are quite exceptional. Now it is very evident, that without the aid of the physical signs, however well marked the general symptoms might be, however high the accompanying fever might run, we should be unable with certainty to localize the inflammation. In like manner, through the aid of physical diagnosis, the injuries and defects of the heart's valvular apparatus become known to us; hypertrophy and dilatation of the heart, and pericardial effusions.

Here, indeed, are summed up, in a few words, all the leading features of heart diseases.—All of them are made known to us by physical diagnosis; and without its aid could only be indistinctly guessed at.

The advances made in the pathology and diagnosis of heart diseases, have necessarily been followed by improved methods of treating them. The objects towards which our healing efforts are to be directed, have been thereby distinctly placed before us: diagnosis leads us to the seat of the malady, and pathology then tells us of the structural changes there induced by it. But though pathology tells us much, it does not give us the whole

history of the nature of the malady: it points out to us certain anatomical or chemical changes of parts of the body; but what works these changes, and how they are worked, science has yet to explain.

Hence, then, in agreement with what pathology teaches us, the physician has, in the treatment of cardiac inflammations and degenerations, two separate and clearly marked indications before him, requiring his attention; viz.—first, the local inflammation, and the anatomical changes of parts; and secondly, the cause—the specific agent—which produces those changes.

Both these facts evidently demand due and separate attention, and must never be lost sight of; their consideration at once gives to our treatment a clearness and distinctness in its direction. It has to combat a local inflammation and its distinctive tendencies, and it has to neutralize a specific agency producing such inflammation; the inflammation and its pathological consequences are clearly known to us,—the general specific agent is beyond our knowledge. We have to deal, then, with inflammation, but inflammation produced by a specific cause; and herein naturally lies the great difficulty which besets this story of the treatment of cardiac diseases. In the treatment of the local disorders of the inflammation, we have ordinary principles of medicine to guide us; but in the treatment of the

cause thereof we have no guide whatever, beyond those doubtful hints which successful treatment seems to furnish to us.

And here it is that we reap the benefits of a sound pathology. There is, at all events, no obscurity in the *terms* of our treatment: we know exactly what we have to deal with; in how much the conditions of the case are analogous to those which demand the practice of ordinary principles, and therefore clear; and in how far they are hypothetical, and therefore the practice applied purely experimental. Any one who will fairly weigh these considerations can, we should imagine, readily understand why it is that so much doubt, and difficulty, and obscurity, and such variety of authoritative opinions, surround this question of the treatment of acute cardiac diseases.

There is another cause besides this, also, which adds to the difficulty, and it is this,—that unfortunately the physical diagnosis, which demonstrates the fact of the cardiac derangement, does not help us *until the mischief is done.* We cannot catch the inflammation in its stages of congestion, as we can the inflammation, for instance, of the eye; the physical signs which determine the fact are the results of inflammation that has reached beyond its first period. The very first sign of it heard may be the tale of a permanent injury,—of a ruptured or disorganized valve for instance. It is true that we

may occasionally suspect the existence of the local disease, and the suspicion may even warrant our acting as though the fact had been already fully verified by the diagnosis, but still it is only suspicion; and, as a rule, it may fairly be said that our remedies are not directed especially to the heart affection, until such times as the physical signs have declared that the organ is affected.

I may here shortly exemplify what has been said above, by referring to the history of acute rheumatism. No one can doubt the specific nature of the disease: it is general in its effects; the inflamed joints are assuredly not the essence of the disease; neither is the cardiac inflammation which so often accompanies it. These pathological marks are its secondary results, its consequences, its visible manifestations. Now I think the testimony of all physicians will bear me out in saying, that, as a rule, the main treatment of rheumatism is general, like the cause producing it; its object being to neutralize, or destroy, or eliminate, that specific agent which induces the specific action; but occasionally it happens, that the local inflammation runs high, beyond the ordinary specific degree of it,—so high, indeed, as even to disorganize a joint: then of course it demands local treatment. Now it does so almost invariably in the case of the cardiac inflammations which accompany rheumatism; and here it is, in particular, that the local inflammation by

its character and position becomes of such importance; it entirely overshadows the general specific character of the disease, so that the attention of the physician is almost wholly absorbed in the attempt at subduing the local inflammation.

Thus, in cardiac inflammations we have two facts to deal with: we have the specific action, and the local inflammatory action thereby excited. Now the ordinary inflammatory action we may hope to meet by ordinary antiphlogistic treatment, modified by attendant circumstances; but the specific action producing it, the cause which throws the malady on the heart, we know not how to deal with, for we know not what it is, nor do we even know of its existence, until the ordinary signs of inflammation indicate the fact; nor has experiment in treatment yet enabled us to neutralize its action. Diagnosis is late: it tells us that the mischief is done, not of its doing. And herein, if we mistake not, lies the dark side,—for a dark side there is of our knowledge of the treatment of these diseases. I have hereafter endeavoured to show what is the treatment, under all the doubts and difficulties which rest upon it, most reasonably adapted thereto. The general treatment may be said, upon the whole, to be that of acute rheumatism,—a conclusion to which the history of its pathology would naturally lead us. But where it differs from that of acute rheumatism is in this chiefly, that in acute rheu-

matism the local inflammation of the joints rarely is of that severity as to require any particular local treatment, whilst in cardiac diseases it is generally of such a character as to demand the local and general treatment of ordinary inflammation.

But whatever obscurity may involve the treatment of acute cardiac diseases, none happily attaches itself to that of their chronic forms, or rather of the secondary consequences which result from the disorganization of the heart's structures produced by the acute inflammation, or from degenerations of its tissues. Here pathology points out pretty clearly the work which is to be done, *i.e.* the exact nature of the disease,—of the mischief which is to be remedied; and thereby it indicates the distinct objects which require the application of our remedies. There are, indeed, no class of disorders in which the skilful physician can afford greater temporary relief to the distresses of his patient. This he does by ministering to the functional derangements; by relieving the different organs and parts from the mechanical pressure exerted upon them through the impeded circulation; by sustaining the weakness of the heart, and regulating its action.

I have so often spoken of a "specific agent" as being the cause of the visible signs of disease, that it may be well, perhaps, before I conclude this chapter, to explain what idea I attach to the term.

Such like expressions are clearly open to much criticism; but the imperfection of our knowledge obliges us to make use of them. When I speak of a specific agent, then, it must be understood that I am speaking of a thing of whose essential nature we are totally ignorant. We are sure that some antecedent act or cause must exist, to give occasion to the visible and tangible signs of disorder, just as we know that some agency must give rise to the phenomena of gravity or magnetism, though of the nature of that agency we have not the faintest conception. What I mean thereby is simply the cause or causes which excite the local signs of disease. I do not wish to create in the mind of the reader the notion that some actual tangible body is there present; it does not follow because in certain cases we find effects produced in the body when certain matters—urea, bile, uric acid for instance—are accumulated in the blood, that therefore in all cases where general signs of disease present themselves, a tangible agent is there present in the blood, and produces them. The fact is, that in diseases properly termed specific, the cause producing the symptoms has hitherto eluded all our researches. We know no more of the real nature of typhus, than we did before animal chemistry was a science, that is, no more in a positive sense. We have no kind of certain knowledge whatever on the subject; its point of departure, whether in the solids or the

fluids, is still just the battle of to-day, as it was the battle of days gone by. Humourism, indeed, which a few years since was proudly triumphant, seems once again tending to succumb to solidism.

Giving this wide signification to the term "specific agents," I may also add, that it would be taking a very narrow view of the subject, to suppose that this cause is necessarily in all cases of a simple nature. Why may not the cause, as we call it, be in fact made up of a series of antecedents and sequences? the last sequence being the change visible to our sight. If we thus, in scarlatina, were haply to find some subtle and anomalous body always present in the blood, still even then we should have to ask, But why came it there? was it engendered by some fermentation? was it formed by disordered nervous influence? Was that which caused the fermentation absorbed from without? did it primarily act on the nervous centres? These and similar hypotheses naturally suggest themselves to us, and they are fairly questions worthy of consideration, in so far as they point out the numerous difficulties which involve the subject of the origin of diseases. Unfortunately, however, their discussion yields us no positive knowledge on the matter. Chemistry and the microscope have done their best, in the hands of the best men; but they have really as yet told us nothing concerning the original cause of such diseases. What they

have done, nevertheless, is an immense service to science. They have cleared away great masses of errors; they have laid solid foundations for future researches, and have pointed out the methods which we must pursue, in following out our researches into the nature of diseases; and they have also, in numerous instances, brought us nearer to the original cause of the disease, by taking us one step higher in the history of the diseased process, than our unaided powers enabled us to attain.

CHAPTER I.

PATHOLOGY OF PERICARDITIS.

Anatomical description.—Peculiarities of the Inflammation.—Its special characters.—Effects of the Pericarditis on the different Organs of the Body.—Effects on the Heart itself.—Pericardial adhesions.—Relation of Pericarditis to Endocarditis, and to Fatty Degeneration of the Heart.—Effects of Pericarditis on organs and parts around and external to the Pericardium: on the Pleuræ and Lungs: on the Œsophagus.—White Patches (Spots) on the Heart.

PERICARDITIS, anatomically described, consists in an inflammation of the whole or a part of the serous membrane which covers the outer surface of the heart, and the roots of the aorta and pulmonary artery. The appearances presented by the inflammation are much the same as those which are observed during the progress of inflammation of other serous membranes of the body. We have the stages of congestion, of exudation, of growth of pseudo-membrane, of absorption, of adhesion, and of organization or of degeneration of the products exuded. As the description of these things is the subject matter of general pathology, we shall not linger to give any minute account of them here.

The anatomical changes characteristic of the first stage of pericarditis are the following:

The superficial vessels of the serous membrane become injected with red blood, forming tuft-like arborescent patches, having little ecchymoses sometimes scattered between them. The surface of the membrane loses its naturally smooth, shining and semi-transparent appearance; inflammatory exudations of various kinds are poured out into the cavity of the sac, and pseudo-membranous growths form upon it. The surface of the false membrane is uneven, flocculent, and sometimes honeycombed; in colour and thickness it varies much. The character of the inflammatory exudation also differs considerably in different cases. After a time, the membranous growth takes a more consistent form; new vessels shoot out—in all probability, from the parent vessels of the serous membrane—and spread into it; it thus becomes organized, and is finally converted into a kind of areolar tissue. The inflammatory exudation is at the same time gradually absorbed; and at length, by the cohesion and union of the opposed surfaces of the false membrane, and its complete organization, the two layers of the pericardium become united, and thus a vascular anastomosis is established between them. The union so formed between the pericardial surfaces is complete or partial, and the adherent medium loose or intimate.

But instead of adhesions being formed, and of the production of organized membranes, the exudation sometimes takes on a purulent or sanious character; or it may be metamorphosed into cheesy, tuberculous, calcareous, and fatty matters. The formative impulse given to the exudation depends upon the constitution, the condition of health, and the idiosyncrasy of the individual, and upon the character of the disease, *i.e.*, upon its constitutional relations and its cause.

Hence we may say, that the first and immediate consequences of pericarditis are, the pouring out of inflammatory exudations into the serous sac—of serous, albuminous, fibrinous, or purulent exudations, tinged or not with blood corpuscles or with the dissolved colouring matter of the blood; and the formation of a false membranous growth on the serous surfaces. Secondarily follow absorption of the serum, and more or less complete adhesion of the pericardial surfaces, through the medium of the false membrane upon them; or the exudation takes on a purulent nature, becomes of a "low character," and then the organization of membranous growth is interrupted; or it is converted into fatty, tubercular, or other matters; or again, the serum may increase rapidly and in such abundance, as indeed, by its pressure upon the heart, rapidly to destroy life.*

* Rokitansky, in the lately published edition of his *General*

Thus far, the description of this inflammation is the description of the inflammation of serous membranes in general; but there are certain facts peculiar, or rather incidental, to pericarditis, to which we will now briefly refer.

It is necessary to recollect, that the immediate and secondary effects of the inflammation are exercised upon an organ whose uninterrupted action is essential to existence, and whose temporary derangements even may endanger life. One same general cause may excite pleurisy and may excite pericarditis; but in the eye of the physician, the two inflammations are of vastly different importance; and they are so, simply because of the different anatom-

Pathology, speaking of the inflammation of serous membranes, tells us that the *proper inflammatory exudation* and the *membranous growth* are to be carefully distinguished the one from the other. The exudation of inflammation is in all cases alike, differing only in the amount of elementary forms which it contains; these elementary forms—cells, nuclei, and granules—are incapable of further development. The *pseudo-membranous* production is not, as has been hitherto taught, a plastic, organizable portion of the exudation, but is an actual growth of the areolar substratum, in which are formed round, oval, spindle-shaped cells, which gradually coalesce, and are fused down into a hyaline, or striped, fibrous, membranous layer, giving to the inflamed membrane its well-known dull, flocculent appearance. From this layer arises a secondary layer of cell-growth; and so arise other layers, the materials being brought to them by the formation of a new vascular system: the vascular system springs out of that of the original structure, and gradually spreads out into the new-formed membranous layers. Inflammation is not a necessary ingredient of such new formations; they occasionally take place without any visible signs of its concurrence; it is rather an exciting cause.

ical relations of the pleura and of the pericardium: that is, of the difference in the nature of the organs which they invest. Such an amount of effusion as in the one case scarcely suffices to impede the respiration, is enough to exert a deadly influence on the circulation in the other. The inflammation of the pericardium, moreover, by its contiguity, affects the muscular tissue of the heart; an organ whose integrity and constancy in action cannot fail for a moment, without periling life. These are reasons —and sufficient reasons—why the slightest pericardial friction sounds excite such deep anxiety; and there are others.

It is most important that the student, while studying the pathology of pericarditis, should not confine his ideas of its nature to the local manifestations of it. Pericarditis, except in the rare cases, where it is the result of traumatic injury, or is excited by contiguous disease, is not an idiopathic inflammation; it is one of the consequences of some cause operating generally in the body—of blood disease, it may be; and this is the guise under which it chiefly comes under our observation as physicians.

Thus considered, the local disease, the pericarditis *per se*, will be found to vary infinitely in the importance which is to be attributed to it. It may be a subordinate item in the general disease, or it may be the most important and the most prominent

item ; it may be strictly sthenic, and it may be purely asthenic. Connected with rheumatic fever, it becomes, for the moment, the sole object towards which the physician directs his curative art ; and again, on the other hand, in some low fever, in phlebitis for example, so little marked may it be in its effects, so insignificant in the train of co-existing symptoms, as sometimes even entirely to elude observation, and to demand, should it be discovered, no local or special consideration.

The anatomical changes which represent the disease to us, be it remembered, are not the disease ; what may be more truly called so, viz., the exciting cause of the local disorders, exists prior to them ; the changes are but the consequences of the cause which is working in the system. And these causes again, though pathology cannot clearly trace them out, are various, and they attack the vigorous and they attack the weak ; and certain of them operate in particular at one period of life, and some at another ; and they fall upon other organs and parts of the body besides the pericardium. The same cause which produces the rheumatic pericarditis is that which, in all probability, produces the rheumatic joints, and which produces the endocarditis, and the pneumonia, and the pleurisy : and so again, that cause which in disease of the kidney excites pericarditis, is the same which, for instance, gives occasion to inflammation of other serous membranes

and parts of the body. Hence, then, in almost every case which comes under our observation, pericarditis cannot be rightly treated of simply as a local inflammation; it is one of the resultants, or is the single resultant, of some general disease of the body. To look upon pericarditis, in such case, as the disease, is surely much the same as if we were, in particular instances, to designate typhus as a pneumonia or a cerebritis, whenever the lungs, in the one case, or the brain, in the other, happen to be the organs on which the disease has, in an especial manner, exerted its influence. In pericarditis certainly, as in the pneumonia and cerebritis of typhus, the local inflammation may be so violent as to demand especial treatment, but here likewise must we remember that the disease is general, and that therefore, in the general character of the disease, and in the general bodily condition of the patient, we must still seek our guides to direct us in the management of the local disease.

I trust I have said enough to show the exceeding value of a right and true understanding of the pathological history of pericarditis; and though out of place here, I cannot help remarking, that it is, perhaps, from want of a due appreciation of this subject, that have arisen those strangely discordant methods of treating pericarditis, which have, at various times, been recommended by different observers.

Having given above an account of the local products of the inflammation, I will now proceed to trace the effects and consequences which ensue to other parts, as results of the pericarditis.

The intimate anatomical relations existing between the heart and its investing membrane might, *à priori*, lead us to the conclusion, that inflammation of the latter could scarcely fail to produce some influence upon the former; and experience fully justifies the conclusion.

In the first period of the pericarditis, the heart's action is peculiarly excited, as shown by the violence, rapidity, and irregularity of its pulsations. This excitement may be in part attributed to the general disorder; but it depends in a much greater degree upon the contiguous pericardial inflammation, which acts as a cause of local irritation to the muscular tissue of the organ. After a time, however, when exudations into the pericardial sac and adhesions of its surfaces have taken place, the action of the heart becomes enfeebled and impeded; and it appears to be so in proportion to the degree and extent of these exudations and adhesions. There are, indeed, three ways in which the action of the organ appears to be interfered with, viz., by the mechanical pressure of the fluid exerted upon it; by the pericardial adhesions; and by a partial paralysis of its muscular tissue, resulting from the influence of the contiguous inflammation, whereby

its nutrition and innervation are injured. The muscles of the heart, in all probability, are affected after the same fashion as the intercostal muscles are in advanced cases of empyema. "In both cases," says Dr. Stokes, "we have the common condition of inflammation of a tissue in close connexion with the muscle itself; in both we observe, first, the evidences of excitement, and then those of depressed vitality." In illustration of the same fact may also be mentioned, the paralysis of the muscular coat of the intestines in peritonitis, evidenced by their tympanitic condition.

Exudations of solid or fluid matters into the pericardium must necessarily interfere with the heart's action. They act injuriously upon it in several different ways. They exercise a baneful influence on the nutrition of the muscular structure of the heart. At the onset of the pericarditis, the structure is immoderately gorged with blood; but after it has been for some time subjected to the effects of the effusion, it becomes of a brownish red colour, is softer than natural, and has a macerated appearance. This condition has been supposed to be the consequence of inflammation; but it more probably results from the action of the fluid effusion interfering with the heart's nutrition; from whence a defective power of the muscles naturally ensues.

When the fluid effusion is of large amount, it

acts, moreover, mechanically as an impediment to the free movements of the heart. The pressure on the surface of the organ, when the pericardium is much distended, must be considerable; and with great deference to such an authority, I cannot but think that Dr. Stokes has much undervalued its influence. The fluid pressure necessarily acts equally upon all parts of the organ, and of course, therefore, with infinitely greater effect upon the auricles than upon the ventricles; their feebler muscular structure rendering them particularly obnoxious to its influence.* The large arterial trunks must also resent the pressure. To this pressure, thus exerted, may fairly be attributed, in part at least, the impeded respiration and circulation which often arise so rapidly in the progress of this disease. The auricles are compressed, and the return of the blood, both

* I have lately seen a case of pericardial effusion, in which the heart looked as if it had been squeezed up by the hand: the auricles were both of them thickly coated with plastic growths, crumpled down, and scarcely to be recognized, as the heart lay at the back of the sac; the aorta, too, looked precisely as though it had been compressed at one point. There were twenty ounces of fluid in the sac in this case. We may ask here, incidentally, if it is probable or possible that an aorta could be thus compressed without giving rise to a murmur? If so, then aortic murmurs may arise during pericarditis, independently of endocarditis; and thus murmurs, having the ordinary characters of endocardial murmurs, which have been heard in cases of pericarditis during life (and which, in consequence of no trace of endocarditis being found after death, have been considered as pericardial), might, after all, have been in reality endocardial murmurs, produced after the fashion here suggested.

from the systemic and pulmonary circulation, thus partly arrested in its current towards the heart. Solid fibrinous exudations, spread upon the pericardial surfaces, likewise interfere with the heart's action, especially when collected in large masses about the base of the heart. Not unfrequently we find the fibrinous matter closely coating the arterial trunks, and so thickly covering and embracing the auricles and the base of the heart, as to give that part of it the appearance of one uniform mass. Is it possible that, in such case, there is no mechanical pressure exerted on the parts referred to, and so as greatly to impede the circulation? The inflammatory action, when it affects that part of the pericardium which covers the roots of the great vessels, may likewise partially injure the elasticity, and otherwise interfere with the nutrition of their coats, and so become a cause of their subsequent dilatation, and of other abnormal changes taking place within them.

Pericardial Adhesions.—The opinions of authors differ considerably respecting the particular effects produced on the heart, through adhesions of its pericardial surfaces. Dr. Hope's experience led him to conclude, that complete adhesion of the pericardium is always followed by hypertrophy and dilatation of the heart; but further observation has not confirmed his opinion.* Dr. Barlow, indeed,

* Dr. Hope mentions that bellows murmur is one of the

has even stated, that the very opposite condition to hypertrophy—viz., atrophy of the heart—is the most frequent consequence of such adhesions. Skoda and Kolletschka deny that hypertrophy of the ventricles is a common consequence of pericardial adhesions: they generally found dilatation present, and but rarely associated with hypertrophy. Dr. Stokes says, that obliteration of the pericardium does not necessarily induce any manifest change in the condition of the heart. This is an opinion to which I cannot subscribe: it is scarcely possible to believe that the free action of the heart is not interfered with, through adhesions of its investing membrane, and that such adhesions do not, in a greater or less degree, act injuriously on its muscular structure.

The discrepancies of authors on this subject may perhaps be, in part, explained by the following suggestions: That their observations were made at different—earlier or later—periods of the existence of the adhesions; that not sufficient attention was paid to the nature of the adhesions—whether they were loose, or so intimate as to prevent all motion

ordinary signs of pericardial adhesions. Now, wherever such a murmur is present, the valves are, as a general rule, defective, but from defect of the valves hypertrophy usually results; wherefore it seems not unfair to suggest, that the hypertrophy, which Dr. Hope placed to the account of the pericardial adhesions, might have been rather the consequence of co-existing valvular disease.

of the heart within the pericardium; whether they were between surfaces of the membrane not naturally opposite to each other;* or whether there co-existed exo-pericardial adhesions,—adhesions to the surrounding surfaces of the lungs, adhesions to the diaphragm, and to the soft and solid parts of the framework of the chest; all of which would manifestly produce greater interference with the heart's action than the union of the pericardial surfaces alone.† Moreover, we know that inflammation of the pericardium may leave the muscular tissue in very different conditions, as regards its integrity; perhaps unchanged, perhaps atrophied, perhaps softened and degenerated; and such altered conditions of its structure will necessarily modify the consequences, whatever they be, which result from pericardial adhesions. And then, again, with pericarditis is frequently associated endocarditis; and with endocarditis, diseases of the valves; and with dis-

* We may readily understand how such kind of adhesions form when the heart is displaced from its natural position by serous effusions, and thus how the ordinary relations of the pericardial surfaces are lost; we know that partial adhesions will take place, even though a considerable amount of serum is present; and in such case, after the serum is absorbed, and the parts tend to regain their former relative positions, the adhesions necessarily, we must suppose, produce an unnatural drag upon the heart's movements.

† Dr. T. K. Chambers shows, in his *Decennium Pathologicum*, that, in a large number of cases, death occurs at a much earlier age when the adhesions are universal, than when they are only partial. "The consequences of partial adhesion are much less rapidly fatal than of universal adhesion."

eases of the valves, hypertrophy and dilatation of the heart. A consideration of these various accidental circumstances, all or any of which may complicate the subject of pericardial adhesions, will explain, perhaps, to some extent, the discrepant opinions of authors respecting the consequences which result from such adhesions. Where the muscular structure of the heart is unaffected, and the other parts of the organ (with the exception of the adhesions of its pericardial surfaces) are healthy, we may fairly, as a general rule, admit the opinion of Dr. Hope as correct, viz., that hypertrophy of the heart is the result of pericardial adhesions.

Relation of Pericarditis to Endocarditis.—The exact relation in which pericarditis stands to endocarditis, whether as cause or effect, or as a concurrent inflammation, has not yet been clearly determined. It is well known, that inflammation of the endocardium does very frequently arise during the progress of pericarditis, that it may precede it, and that both inflammations may arise simultaneously. But it appears to be chiefly in cases of rheumatic pericarditis, that we observe the association of endocarditis with pericarditis; and it may be added, that endocarditis is rarely met with in cases of non-rheumatic pericarditis. Dr. Ormerod found the physical signs, which may be considered as denoting the effects of present or past endocarditis, in nine-tenths of his cases of rheumatic pericarditis. It

must not be forgotten, however, in thus calculating the relation of endocarditis to pericarditis, that endocarditis frequently arises independently of pericarditis, and that therefore the signs—the bruits—by which its presence is manifested to us, may, in cases of acute pericarditis, result from old valvular diseases; they may also be simulated by functional murmurs.

Inasmuch as pericarditis, in those cases in which it is most frequently found associated with endocarditis, is justly considered as the result of some disturbing cause working generally in the body, we may very fairly attribute the endocarditis to a similar cause, and thus look upon it, in such cases, simply as one of the local manifestations of the presence of the general disorder of the body. We can in no way regard the endocarditis as spreading by contiguity from the pericarditis.

Relation to Fatty Degeneration of the Heart.—Pericarditis is one of the numerous causes to which fatty degeneration of the heart has been ascribed; but here, as in the instance of many other supposed causes of fatty degeneration, we must be slow in admitting the relation of the affections, for we know that fatty degeneration is very common where no sign of past or present pericarditis exists; that it may co-exist with every abnormal condition of the heart; and that it may be the sole morbid condition present. Still, pericarditis may indirectly be a

cause of fatty degeneration, in so far as it interferes with the proper nutrition of the muscular structure of the heart; and in a like manner it may be the cause of atrophy of the organ.

Effects of the Pericarditis on Parts External to the Pericardium.—Inflammation of the pericardium sometimes, though I believe rarely, extends by contiguity to the parts external to and around the pericardium. In such cases, the usual products of inflammation are exuded into and upon those parts, and adhesions are formed between the outer surface of the pericardial sac and the portions of the lung in contact with it, and also between the pericardium and the thoracic walls. The consequence of such adhesions is, that, in the first case, the lung can no longer, as in health, glide freely over the pericardium during the respiratory movements; and that, in the second, the heart itself (when its pericardial surfaces are likewise adherent) becomes directly attached to the thoracic walls and to the lungs. The exact effects produced on the heart's actions by these attachments has not yet been shown, but reasoning *à priori*, we may be sure that such adhesions must, in a greater or less degree, be injurious to the free play of the organ.

Effects on the Pleuræ and Lungs.—More or less well-marked signs of pleurisy and pneumonia generally attest the co-existence of these diseases in cases of pericarditis, especially of rheumatic peri-

carditis. They appear to arise in two ways: first, through irritation exciting by contiguity inflammation in parts which are already predisposed thereto; thus we generally find the pleurisy and pneumonia most developed in those parts of the lungs which are contiguous to the pericardium, and particularly in the left side of the thorax: and, secondly, the pleurisy and pneumonia seem to arise independently of any local relationship, and as consequences of the same general diseased action which caused the pericarditis. In these cases it is certain, at all events, that the pleuræ and the lungs are readily obnoxious to inflammatory attacks.*

* Considerable obscurity still hangs over this most interesting and important part of the history of the pathology of pericarditis, viz., its true relation to the pulmonary affections which so generally accompany it. We have spoken of the spread of the pericarditic inflammation by contiguity of textures, but there are great objections to this explanation, notwithstanding the fact that the left lung is more early and more frequently affected than the right lung, in pericarditis; for if it were a true one, how comes it that we rarely ever find that pleuritic inflammation, however contiguous to the pericardium and however intense, excites pericarditis? and then again, we often find the pulmonary affections, the pleurisy and pneumonia, out of all proportion, in intensity, to the pericarditis; and besides this, in fatal cases of pericarditis the pulmonary tissue itself, not merely the pleura, is found deeply involved in inflammatory changes, changes which can hardly be attributed to any spreading by contiguity. And not unfrequently, also, it happens, that the pulmonary and pleural affections declare themselves before any physical signs exist to show that the pericardium is damaged; and they may also declare themselves in parts of the lungs, quite distant and apart from the neighbourhood of the pericardium.

When the effusion into the pericardium is considerable, the free action of the lungs is hindered by the pressure thereby exerted upon them; the left lung in particular is thus affected; it is often found pressed upwards and backwards, and very considerably reduced in size, especially in its lower lobe. The pericardial effusion, consequently, acts doubly as an impediment to the respiration: first, indirectly, by its pressure, within the pericardium, upon the heart (particularly on its auricles) and the great vessels, whereby the pulmonary circulation is interrupted; and then, directly, by its mechanically compressing the lungs and the bronchi. The effusion, when abundant, occasionally interferes with deglutition by its pressure on the œsophagus, a symptom which would, perhaps, be more frequently observed, if it were more frequently sought for. The pericardial inflammation, should it spread to the diaphragm, may likewise interfere with the respiration by restricting the movements of the muscle.

We shall not stop to consider here the many other effects which frequently arise in different organs and parts of the body as consequences of pericarditis. They result chiefly, in a secondary manner, from the impediments thereby offered to the circulation, and are much the same in character as the effects which are produced by other diseases of the heart, and will therefore come under our no-

tice when speaking of those diseases. They consist chiefly in congestions of the lungs, the abdominal organs, and the brain; in effusions into the parenchymata of those organs, into the cellular tissue generally, and into the serous cavities of the body, etc. The very serious and important affections which occasionally manifest themselves in disturbances of the cerebral functions, in cases of acute pericarditis, will also be considered hereafter; pathology has hitherto failed to throw any light upon their exact nature.

White Patches on the Heart.—The milk-white patches, or spots, so frequently met with on the pericardial surface of the heart, particularly on its anterior and right side, and along the course of the coronary vessels, have generally been considered (chiefly in consequence of the researches of Mr. Paget) as the results of inflammation. Later investigations have thrown doubt upon the correctness of this opinion. There is reason, indeed, to believe that in many instances these fibrinous patches are of non-inflammatory origin: "We incline to the belief," says Dr. H. Jones, "that, in the majority of cases, these white patches are the result either of the simple exudation of unhealthy plasma, or of such plasma effused from vessels in a moderate state of congestion." Similar kinds of exudations are supposed to produce the white patches and thickenings of the peritoneum, so often found on the surfaces of the

liver and spleen, and to be connected with particular conditions of the blood. This appears, also, to have been the opinion of Skoda and Kolletschka, expressed by them many years ago in a paper on pericarditis (*Oësterr. Jahrbücher*, vol. xxviii): "These white spots," they say, "the products of the inflammatory deposit, must be distinguished from those which are the results of partial thickenings and opacities of the subserous tissue; these are not produced by inflammation, but by repeated exudations. The two kinds may be readily distinguished: the inflammatory kind being found *upon*, the other *beneath*, the serous membrane."

Mr. Paget's main reason for supposing them to be of inflammatory origin is, that when they are present, pericardial adhesions almost invariably coexist about the roots of the great vessels of the heart; but to this it may be replied, that such adhesions are of a peculiar character, and that their inflammatory origin is not certain; and particularly that they are not of the nature of those adhesions which we ordinarily meet with as the results of pericarditis. Dr. T. K. Chambers, in his *Decen. Patholog.*, shows us that only three times in one hundred and sixty cases were these white patches found associated with pericardial adhesions, and that they do not seem to have been conjoined with the *ordinary* cases of adhesion; a fact which cer-

tainly tells against their being generally of inflammatory origin.

The opinion that these patches have a double mode of origin is probably correct, and explains the opposite views which authors take of their nature. It is confirmed by M. Bizot. Those of inflammatory origin are deposited upon the serous membrane, and are removable; the non-inflammatory arise from exudations into the serous or sub-serous tissue, constituting an hypertrophy of the membrane.

The important inferences which Dr. Latham has drawn from the existence of these patches cannot be sustained, if the above views are correct.* The opinion of Dr. Baillie as to their importance, seems to be more in accordance with the actual state of our knowledge on the subject. "*This spot,*" says Dr. Baillie (vol. ii, p. 17), "consists of an adventitious membrane, formed on a part of the pericardium which covers the heart, and may be easily dissected off, so as to leave the pericardium entire. It is an appearance, I believe, of no consequence whatever, and is so very common that it can hardly be considered as disease."

* "All those white spots upon the surface of the heart .... have at length been demonstrably shown by Mr. Paget to be the effects of inflammation," etc. (Dr. Latham *On the Heart*, i, 345.)

CHAPTER II.

CAUSES OF PERICARDITIS.

Local Causes.—General Causes.—Rheumatic Pericarditis.—Non-rheumatic Pericarditis.—Relation of Pericarditis to Rheumatic Fever.

INFLAMMATION of the pericardium may, in almost all instances, be traced either to local injury, or to some general morbid condition of the system. In no case can we regard it as idiopathic. As the consequence of local irritation, the inflammation may result from traumatic injuries, from blows and wounds; it may be excited by the presence of tubercle or cancer within the serous membrane, or by their spreading into it from without; abscesses opening into the sac from the parts around, from the liver, the mediastina, etc.; ulceration of the œsophagus, spreading through into the pericardium, and permitting the contents of the stomach and œsophagus to pass into its cavity; neighbouring pleurisy, peritonitis, and pneumonia;—all these, and similar local sources of irritation, may excite inflammation in the membrane.

The occurrence of tubercular disease in the heart

is very rare, and only provoked, as Dr. Chambers says, by the strongest diathesis. Malignant disease is somewhat less rare. When these diseases do arise in and affect the pericardium, they necessarily, sooner or later, provoke pericarditis, and are therefore occasionally local causes of pericarditis.

Pericarditis excited by these local causes is, comparatively, of rare occurrence; it is better known to us as the local manifestation of some general affection of the body, or as the product of some morbid condition of the blood. Daily observation, indeed, forces this conclusion upon us. Thus we find, in a large majority of the cases of pericarditis which come under our notice, that the disease presents itself in association with rheumatic fever or with Bright's disease of the kidney; and in these diseases we have proof enough of the existence of a morbid agent in the system at large.

Modern pathology teaches us, that the ideas which we possess concerning the essential nature and causes of pericarditis are very imperfect; and that what we are frequently pleased to designate a cause of the disease, is in reality merely a co-existing manifestation of it, and in many instances arises from the self-same cause. Acute rheumatism cannot be called the cause of pericarditis; both the affections are the resultants of some anterior morbid state of the body, and, as it would seem, of the same morbid state; and what may be fairly said of

their association and connexion is this: that the morbid element more frequently displays its presence in the joints than it does in the heart. To represent one as a cause of the other, is something the same as if, in a case of typhus, we were to speak of ulceration of the ilium as the consequence of a pneumonia, which haply may have pre-existed in the course of that disease. A right understanding of the causes and nature of pericarditis is of every importance to a right understanding of the treatment of the disease.

Dr. T. K. Chambers, in his *Decennium Pathologicum*, enables us to see what were probably the direct (so called) causes (or relations to other diseases) of pericarditis, in one hundred and thirty-five cases, in which recent signs of inflammation were found after death.

	Cases.
Rheumatic fever	18
Do., with diseased heart and kidneys	1
Uræmia from diseased kidneys, either alone, or with diseased heart .	36
Diseased heart and dropsy . .	18
Pyæmia	18
Pneumonia	10
Pleurisy	5
Vomicæ in the lungs . . .	8
Erysipelas	4
Typhus	3
Other causes	13

A glance at this table will at once show us, as Dr. Chambers remarks, how decidedly the constitutional predominate over the local (so called) causes of pericarditis. In fact, the number of what are now called causes will doubtless yet be diminished, as our knowledge of the pathology of the disease advances. Pneumonia and pleurisy, for instance, are here given as causes; but how far they are really such, must, in all cases, be very doubtful. The mere co-existence of the pleurisy with the pericarditis, or the circumstance of diagnosis having determined that the pleurisy preceded pericarditis, are no proofs that the pleurisy is the cause of the pericarditis. *This* we do know, for we have constant demonstrations of the fact,—that inflammation of the pleural membrane, even where it immediately surrounds the pericardium, may exist, and yet leave the pericardium perfectly intact; and we also know that the active agent, whatever it be, which determines the pericarditis, also tends to render the lungs and pleura readily obnoxious to the inflammation: so that it must be ever difficult for us, in the present state of our knowledge, to decide whether the pleurisy is the cause of the pericarditis, or whether both are but local signs of some general disorder of the constitution. The mere precedence in time, is no proof of one disease being the cause of the other.

Dr. Ormerod, for the better comprehension of

these so called causes of pericarditis, has reduced them all under two heads, viz.—

1. Rheumatic pericarditis.
2. Non-rheumatic pericarditis.

A consideration of the different characters of these two classes of pericarditis, points out to us the practical utility of such a division. A study of their clinical history presents us with marked points of distinction, which are well worthy our best attention: by its aid we obtain a clue to the character and the treatment of the disease in each particular instance, and also learn how its nature varies, and how varied and opposite its proper treatment must consequently be, under different circumstances. We will therefore proceed to point out the different characters which belong to the rheumatic and to the non-rheumatic classes of pericarditis.

Rheumatic Pericarditis occurs at a comparatively early period of life; the average age in sixty-one of Dr. Ormerod's cases being twenty-one. The subjects of the disease are, for the most part, healthy in constitution; and as regards sex, women appear as often as—perhaps somewhat more often than—men to be affected by it. The symptoms of the rheumatic pericarditis are prominent and well marked; the disease, except in rare instances, is associated with, and, in fact, preceded by, affections of the joints of a peculiar character, and such as

rarely fail to excite the attention of the observer. The local inflammation, moreover, frequently reacts with violence on the general system; and in such case, when death ensues, it is to the immediate consequences of the pericarditis,—to the action of the inflammation on the heart itself,—that the fatal result is ascribable. But though the symptoms are severe, the disease, comparatively speaking, does not often destroy life at once; that is, by its direct and immediate effects. Death, when it occurs in acute pericarditis, is chiefly to be ascribed to the abundance of the exudation, whereby the heart is overwhelmed.*

Non-rheumatic Pericarditis, on the other hand, occurs at a later period of life; forty-two being the average age of persons affected by it, in twenty-four cases reported by Dr. Ormerod. The subjects of the disease were, with a single exception, all of the male sex. The inflammation does not so much attack the healthy, as those whose constitutions are either originally weak, or have been debilitated and depraved through indulgence in ill-habits of living. It is far more serious than rheumatic peri-

* It has not yet been satisfactorily determined, whether the heart is, or not, more frequently involved in the disease, in proportion to the severity of the rheumatic fever, and of the arthritic affections. Authorities have given opposite opinions on the subject. Judging from my own experience, I should be inclined to answer the question in the affirmative; and more particularly in so far as the female sex is concerned.

carditis in its consequences. Dr. Ormerod informs us, as the results of his experience, that 91·6 per cent. of the non-rheumatic cases were fatal, whilst of the rheumatic sort 18 per cent. only were fatal. The local symptoms, again, of non-rheumatic pericarditis are at times very obscure; they are often, indeed, so obscure, as not to direct the observer, by any local signs, to the seat of the disease; and thus it not unfrequently happens, that the existence of the pericarditis is first discovered after death. In correspondence with this fact, it may be observed, that the local inflammation does not appear to exercise, by reaction (as happens in the case of rheumatic pericarditis), any particular influence over the patient's general condition, so as to appear, in any direct way, a cause of death. Moreover, the pathological changes found after death (compared with those resulting from the rheumatic pericarditis) are less extensive; a circumstance which perhaps explains, in some degree, the absence of local symptoms in association with the disease during life.

It is not, therefore, by the violence or the effects of the local inflammation that life is destroyed, in cases of non-rheumatic pericarditis; death rather results from the action of that particular disease which has, in fact, preceded, and has probably given rise to, the pericarditis. Thus, for example, when pericarditis appears in the course of kidney disease

—the most frequent exciting cause of non-rheumatic pericarditis—it generally appears at an advanced period of the renal disorder; but kidney disease (of the character here inferred) is, as we well know, for other reasons, fatal in its consequences, and entirely independent of its action on the heart. The serous membranes, under the influence of uræmic disease, readily take on an inflammatory action; and, of all the membranes, the pleural and the pericardial appear to be the most frequently subjected to it.

Thus, in non-rheumatic pericarditis, the local inflammation plays a subordinate part; its share as a disturber of the system is but small; it is little more than a local sign (unimportant in its immediate effects) of a serious general disorder. In rheumatic pericarditis, on the other hand, the local inflammation must be considered as the all-important fact: its consequences fall at once, and with violence, upon the heart; the inflammation is sthenic, and its products may be considerable in amount, so as to act directly on the heart, both impeding mechanically, and otherwise seriously affecting its actions; the general disease of the constitution is subordinated in importance to the local inflammation, which it has engendered.

As we have already said, pericarditis is chiefly known to us, in practice, in connexion with rheumatism and with Bright's disease of the kidney;

the non-rheumatic class must, therefore, be in great part constituted of cases in which the latter disease is present. It is impossible to do more than speak approximatively, in a statistical point of view, regarding the relation in which rheumatism and Bright's disease stand to pericarditis; but we may estimate roughly, that pericarditis is about equally frequent in relation to the two diseases.* In Dr. Chambers' table, rheumatic fever figures as a less frequent cause of pericarditis than uræmia; and other causes, indeed, seem as active as the rheumatic fever in producing the disease. Dr. Ormerod's account appears to tell a very different tale; for therein we find, that of eighty-five cases of pericarditis, sixty-one were associated with acute rheumatism, and that twenty-four were of non-rheumatic origin. The apparent discrepancy, however, is readily reconciled, by the consideration that Dr. Ormerod's cases were some of them observed during life, and others after death; whilst Dr. Chambers' statistics are the results solely of post-mortem records; and, as we have already said, acute rheumatic pericarditis is rarely fatal,—non-rheumatic

* Dr. Taylor's conclusions were, that acute rheumatism, and Bright's disease *in its advanced stages*, have an equal tendency to produce pericarditis and endocarditis; but that acute rheumatism has a greater tendency to produce those inflammations than has Bright's disease when in its earlier stages; and that Bright's disease *in its advanced form* is, much more frequently than acute rheumatism, complicated with inflammation of the pleura, lungs, peritoneum, and parts within the head.

pericarditis, on the other hand, being in most cases connected with uræmic disease, almost invariably is so.

Relation of Pericarditis to Rheumatic Fever.—In what proportion of cases of rheumatic fever we may expect that pericarditis will arise, is not very certain. M. Bouillaud and Dr. Hope have doubtless made the proportion too great: these observers found pericarditis present in about one-half of their cases of acute rheumatism. Later observers come to a much nearer agreement. Dr. Taylor makes the number about one case of pericarditis in nine of rheumatic fever; Dr. Budd, about one in eight; Dr. Latham, about one in eight. We must, however, recollect that, in all probability, cases of subacute rheumatism were included in these; so that no definite conclusions can be drawn, even from these statements. Moreover, statistics on this head, to be of any value, must take separate cognizance of the age and sex of the individuals attacked by the pericarditis. The young—particularly young females—appear infinitely more liable to the inflammation, in connexion with acute rheumatism, than do adult males. If one might judge roughly, from a tolerably large number of observations, one would say that, as a rule, the young female attacked by acute rheumatism rarely escapes coincident pericardial inflammation; whereas, on the other hand, acute rheumatism in the adult male is, in by far

the majority of cases, not accompanied with pericarditis.

It is scarcely necessary to refer, in particular, to any other of what have been called causes of pericarditis. When pericarditis appears in connexion with typhus, with any of the exanthemata, with erysipelas, with pyæmia, and such like diseases, it is very manifestly an accident, associated with some particular form of those diseases, and an accident entirely subordinate to the general disorder; its coincidence with these diseases, moreover, is, comparatively speaking, not common.

CHAPTER III.

PHYSICAL SIGNS OF PERICARDITIS.

Impulse and Sounds of the Heart.—Friction Sound.—Conditions necessary for its production.—Distinction between Endocardial Murmurs and Pericardial Friction Sounds.—Percussion Sounds, how modified by Pericarditis.

In describing the symptoms and signs of pericarditis, I shall give them as they occur in typical forms of the inflammation, as, for instance, in association with acute rheumatism. At the same time, it is necessary to remember, that the disease comes before us under very various guises; now with all its features prominently marked, and then again latent and insidious, both in its origin and in its progress. Here the disease may present the signs of most acute inflammation, very distressing local symptoms directing the physician to the seat of the mischief; and there again so little do the symptoms bear with them the mark of any local inflammation, that the disease may entirely escape notice, if not wisely sought for.

In its typical form, as we have said, with all its characteristic marks, it is best known to us in

association with rheumatic fever—a fever which, for the most part, attacks the youthful and the vigorous, and is of a strictly inflammatory character. In its latent and disguised form, it is the associate of atonic diseases, those which fall on the cachectic, the feeble and the aged body; this form is known to us chiefly in connexion with Bright's disease of the kidney.

The attention of the student must be carefully directed to the importance of keeping constantly in view these distinctions. Indeed, it may be truly said, that these two classes of pericardial inflammation, as they are called, resemble each other in no particular, except in that of having a similar locality,—their causes, their nature, their general symptoms, their treatment, their history, are all essentially different.

Of the Heart's Sounds, Impulse, etc.—At the onset of the inflammation, the heart's sounds are generally louder, and its impulse stronger and more widely spread, than natural. The force and rapidity of its action are, at first, increased by the local irritation; but as the inflammation advances, and when effusion gathers in the pericardium, the movements of the organ become feeble; its impulse is irregular, weak, trembling, and even imperceptible; and its sounds are weak, altered in character, or superseded by morbid sounds. When much effusion has taken place into the pericardium, and the patient lies re-

cumbent, the heart naturally gravitates to the back of the pericardium ; in consequence of which, a considerable quantity of fluid is interposed between it and the thoracic walls. Under such circumstances, the natural sounds and the impulse of the heart become weak, and sometimes almost imperceptible. The heart's movements are impeded by the mechanical action of the effusion. And not alone by such mechanical interference. The contractility of the muscular structure itself becomes, at length, affected by the near contact of the inflammatory process which is going on in the serous membrane ; its nutrition and its vitality thereby become impaired ; the result of which is, that it is at last partially paralysed, and after the same fashion, we may suppose, as are the intercostal muscles in pleurisy, and the intestinal muscular tissue in peritonitis. When the heart is thus paralysed, it is well to recollect, that a small amount of exudation may have as equally enfeebling an effect upon it, as a much larger amount would have under ordinary conditions, so long as its muscular force remained intact.* From this fact, also, we may draw the conclusion, that the nature of the heart's normal

* We do not here take into consideration the general condition of the system, the tonic or atonic nature of the inflammation, and the many constitutional influences which in particular cases, *per se*, powerfully affect the nature of the sounds and impulse of the heart ; but these are facts of every importance clinically to the physician.

sounds, heard in pericarditis, enable us to judge approximatively only of the quantity of pericardial effusion.

Moreover it should be remembered, that a heart already hypertrophied, may become the seat of pericardial inflammation; and in such case, we may expect the sounds and impulse to remain loud and distinct, even when a considerable amount of effusion exists in the sac.

The physical signs characteristic of pericarditis are: double or single friction sound, and increase of dulness on percussion, heard over the præcordial region; and occasionally a tactile fremitus perceptible to the hand at the same part.

Friction Sound.—A double or single friction sound heard over the heart, synchronous with its movements, and confined to the præcordial region, is the surest diagnostic sign we possess of the presence of pericarditis; it is, indeed, the first positive sign which enables us to determine with certainty the local character of the inflammation. Fever, præcordial pain or tenderness, and all the other general symptoms of pericarditis may exist, yet until this friction sound—however gently rubbing—be heard, we cannot with certainty affirm that the pericardium is the part attacked. The friction sound appears at a very early stage of the inflammation; it may remain audible through the whole period of the existence of the inflammation, or it

may be heard at the commencement, rapidly pass away, and appear once again when absorption of the fluid part of the exudation has taken place. The friction sound usually accompanies both the systole and the diastole of the heart; occasionally it is heard during the systole only; in some very rare cases, only during the diastole. The smallest amount of lymph exuded on the surface of the pericardial membrane, will, I believe, give occasion to the friction sound during the heart's movements, provided no fluid come between them;* it is not impossible, indeed, that mere dryness of the pericardial surfaces may be sufficient for its production.

The loudness and character of the friction sound depend partly upon the amount of lymph deposited on the serous surfaces, and upon the roughness of the parts where the surfaces come in contact; but still they bear no exact or constant relation to the amount of lymph deposited, or to the violence of the inflammation, for they are modified by several other accidental circumstances. Thus, the intensity of the sound depends not only upon the roughness

* This fact is drawn in part from analogy. I once distinctly heard pleuritic friction sounds in a lad a few hours before his death. The very thin layer of lymph—however readily seen when looked for—which occasioned the sounds, would, I believe, have passed unnoticed, except for the fact of the friction having been heard during life. Let any one close his ear with the hollow of one hand, and rub over the back of it with a finger of the other, and he will find that the gentlest friction he can practise is plainly appreciable by the ear.

of the surface of the fibrinous deposit, but also upon the area of roughened surfaces which rub upon each other, upon the force of the heart's action, and upon the amount of fluid effusion in the sac. These accidents necessarily modify the loudness and clearness of the sound; but still, with rare exceptions, its essential character is ever that of a rubbing sound. Whatever particular well-known sound fancy may please to liken it to, it is still a friction sound, bearing but one import to the physician's ear. Its varieties are only degrees of the same sound, embracing the gentlest rubbing, and reaching up to a rough creaking and even scratching sound.

Conditions necessary for the Production of the Friction Sound.—We may gather, from the above, what the conditions are which are requisite for the production of the friction sound: they are, the deposition of solid or semi-solid matters on the surface of the membrane; the contact of its opposing surfaces, and a certain degree of force in the action of the heart. Serous, hæmorrhagic, or purulent effusions, will not of themselves produce a friction sound; nor will the sound arise when the heart's action is very feeble, nor whenever such an amount of effusion is present as suffices to prevent the surfaces coming in contact, and so rubbing on each other—a condition, indeed, which very rarely obtains.

What amount of effusion is able, in any given case, to prevent the friction sound arising, cannot be determined: the size of the heart itself; the amount of solid deposit on the serous surfaces; the weak or strong action of the organ; the situation of the plastic deposit;—these, and other circumstances, necessarily influence the result, and cannot be decided, with any degree of certainty, in such cases. This much, however, appears certain, that (to use the words of Dr. Walshe) "no conceivable amount of fluid will of necessity totally annul friction sound." When the quantity of serous fluid is large, the friction sound, if it still exist, will be heard chiefly about the base of the heart and the large vessels; and here the sound often lingers, when it is no longer audible over other parts of the præcordial region. The reason of this seems to be, that the opposing surfaces of the membrane are less liable to be separated by the fluid around the roots of the great vessels and at the base of the heart, than at other parts of the organ.

Sometimes, although very rarely, friction sound does not avail us as a sign; for it may be absent altogether, or it may have been present and passed away before the patient came under our observation. Generally speaking, it does not last long; though to this there are frequent exceptions. It terminates either in the death of the patient, or in adhesion of the pericardial surface, or in resolution

of the inflammation; perhaps sometimes in the formation of "white spots."* There appears no reason, if the inflammation be not considerable, why the membrane should not regain its original integrity; but it is doubtful if it ever does so, when the friction sound has been once fairly established.

Difference between Endocardial Murmurs and Pericardial Friction Sounds.—It is in certain cases difficult, if not impossible, to distinguish the pericardial friction sound from an endocardial murmur: a great authority in stethoscopy asserts, indeed, that there is no kind of endocardial murmur (with the exception of the whistling murmur) which may not resemble a pericardial friction sound. We certainly not unfrequently meet with cases, in which the most practised ear will hardly venture to draw the difference, or decide the nature of the murmur, from the mere character of its sounds. In such cases, we must endeavour to enlighten our diagnosis by other signs: we must judge of the nature of the

* As a *proof* of this, a very interesting case is related in the *Medical Gazette*, vol. xlvii. p. 843, by Dr. Morehead. In this case, a rubbing creaking murmur was heard over a spot one inch and a half in diameter, internal to the nipple, the patient having at the same time other symptoms of pericarditis. He recovered under treatment, and died nine months afterwards of cholera. There were no pericardial adhesions, but over the centre of the anterior wall of the right ventricle was an opaque patch, about half an inch long and a quarter of an inch broad, which by moderate traction was readily removed from the surface of the pericardium in the form of a thin layer of areolar tissue.

sound by the period of its occurrence, and by the presence or absence of signs of endocardial disease. The friction sound does not coincide exactly with the heart's sounds: it commences rather *before* the systolic sound begins; and, as a rule, it is not audible in the direction of the current of the blood, in the vessels and through the heart. The friction sounds, moreover, do not replace the heart's natural sounds, except when they are so loud as to overpower them; their duration is generally short; they change their seat; they vanish and reappear; they are generally confined to the præcordial region, and they vary in force and situation from day to day. Pressure of the stethoscope, Dr. Sibson has shown, increases the intensity of the pericardial friction sound, in certain cases, but does not affect an endocardial murmur: to this later remark, however, Dr. Walshe does not altogether give assent.

But these differential signs must be received with caution, and for the following reasons: an endocardial murmur may be, and very frequently is, present in pericarditis, and may thus, consequently, complicate the pericardial friction sounds;* the heart's action may be so rapid, that the relation in time of the pericardial murmur to the heart's sounds cannot be determined; a pericardial friction sound, again, may be heard beyond the præcordial region: I have,

* Dr. Watson says, according to his experience, that endocarditis is invariably complicated with pericarditis.

indeed, heard a friction sound precisely resembling, as far as the ear could judge, a rough aortic systolic bruit, conveyed up to the top of the sternum, in a case where, after death, the aorta and its valves were found perfectly sound, but the base of the heart and the vessels covered with much roughened lymph.

The other signs, also, proposed for the recognition of the pericardial friction sound, are modified by accidental circumstances, by the force of the heart, the quantity of fluid effusion, etc., etc. Even pressure by the stethoscope does not appear to be an infallible sign. Dr. Walshe states that he has heard a mitral murmur increased in force by it. It must also be remembered, that the effects of the pressure can hardly be made to bear upon the pericardial surfaces, when the walls of the thorax are stiff and unyielding; neither can the pressure be exerted upon the affected parts, except when it can be made to bear directly against the roughened surfaces of the pericardium, and therefore not in cases where the friction sounds proceed from the sides and back parts of the heart.

A blowing murmur—endocardial—has been occasionally observed along the aorta and over the left ventricle, in cases of pericarditis in which, after death, no valvular lesion existed. The cause of such murmurs may be attributed—either to pressure upon the aorta, produced by the exudation of

lymph upon it, together with loss of its elasticity, caused by the inflammatory process; or, to some irregular action in the heart's muscular movements, involving those of its columnæ carneæ, whereby the function of the auriculo-ventricular valves is rendered temporarily incomplete; or again, they may possibly, I have thought, be ascribed to the pericardial adhesions, these being of such a character as to prevent the columnæ carneæ from freely contracting, so that the mitral orifice is left partially unclosed during the heart's systole.

A pericardial friction sound may also be confounded with an exo-pericardial friction sound, arising from the rubbing of the outer surface of the pericardium against some opposing surface of the pulmonary pleura, when this is covered with plastic exudation. I believe there is no sign, except that regarding their locality, by which these murmurs may be distinguished the one from the other.

Sounds—certain bronchial râles, for example—resembling friction murmurs, often arise within the lungs; these, and likewise pleuritic friction sounds, are readily distinguished from pericardial friction sounds by the circumstance of their cessation during the arrestment of the respiratory movements.

Pericarditis: Percussion Signs.—Effusion into the pericardium increases the ordinary degree of præcordial percussion dulness, and in proportion to its amount; when the effusion is very considerable,

the percussion may be extensively dull, reaching over the greater portion of the anterior surface of the left side of the thorax, and beyond the right border of the sternum; resistance also is, in such case, felt by the finger on percussion. The dulness arising from pericardial effusion is more marked than that which arises from hypertrophy and dilatation of the heart. Practised from day to day, percussion often enables us to follow the increase or diminution of the fluid in the pericardium; it does not, however, give us any accurate information as to the actual amount of effusion which has taken place. There are, indeed, several sources of error, which may mislead us in judging of the amount of effusion by percussion; and these it is well to note. A small amount of pericardial effusion, for instance, may be associated with hypertrophy of the heart, with malignant disease, with enlargement of the left lobe of the liver, with aneurism of the aorta, with condensation of the lung, and with pleuritic effusion; and consequently the præcordial dulness may, in such cases, be increased out of proportion to the amount of pericardial effusion. A careful analysis of the signs and symptoms present, however, will generally enable us to come to a tolerably correct diagnosis as to the nature of the co-existing diseases, and thereby prevent our falling into the errors referred to.

On the other hand, in emphysema of the lungs,

when these organs overlap the heart to an unnatural extent, and in atrophy of the heart, the amount of effusion present may appear, on percussion, less than it really is. An inflated stomach, also, modifies much the percussion sound.

As a general rule, we find in health that about two and a-half to three inches of the heart's surface is uncovered by the lungs; that surface may be rudely likened to a triangle: a line drawn perpendicularly down the centre of the sternum defines the position of the anterior edge of the right lung, and constitutes the right side of the triangle; its left side is an oblique line drawn from the fourth left sterno-costal articulation, downwards through the cartilages of the fifth and sixth ribs; these two lines meet above, directly beneath the level of the fourth rib; the base joins the sides above the cartilages of the sixth rib.

Between this surface of the heart and the thoracic walls, only fat and intercellular tissue naturally intervene; hence when dulness on percussion exists beyond the limits here indicated, we may suspect an abnormal condition of the parts beneath. The dulness in itself of course simply indicates that solid or fluid matters, containing no air, exist beneath the thoracic walls in those parts, where a certain amount of air ought naturally to be present; the diagnostic value of the dulness must be decided by other signs.

The fluid effusion collects, in the first instance, about the base of the heart and the roots of the great vessels; and it is in their direction,—that is, in the long diameter of the heart,—that the abnormal dulness is first observed; but as the quantity of effusion increases, the dulness reaches across to the right, or beyond the right border, of the sternum, upwards towards the clavicle, and towards the left lateral region,—that is, in the direction of the heart's breadth; the heart being heavier sinks downwards and backwards in the pericardium, when the patient lies recumbent.

Solid exudation rarely exists by itself in such quantity as to increase the dull percussion; when it does, the symptoms attending it are much less severe, and the pulse remains good and regular; as a rule, the greater the fluid effusion, the greater is the danger which threatens life.

We may gather from the above, that increased præcordial dulness is, in itself, no certain sign of pericardial effusion; but judged of by the light of other signs, it becomes so. It differs from pleuritic effusion in this, that it commences at the anterior, whilst pleuritic effusion commences at the back and sides of the thorax. In hypertrophy of the heart, the pericardial dulness is not so well marked, nor is the resistance so complete as in pericardial effusion: and besides this, hypertrophy has its special signs to distinguish it; in effusion the dulness in-

creases most markedly in an upward direction, in hypertrophy downward and towards the left side. In fact, there are very few instances in which the careful observer can be deceived in his diagnosis, especially if he has watched the progress of the pericardial inflammation from its commencement.

Enlargement of the liver, particularly of its right lobe, occasionally pushes the heart upwards and to the left; encroaching on the præcordial region, and increasing much the præcordial percussion dulness. Such an enlargement of the liver would hardly fail of being recognized.

It is not necessary to refer particularly to the other abnormal conditions which may produce increased præcordial percussion dulness; their co-existing symptoms will generally enable us to distinguish between them and the pericardial effusion.

Together with the friction sound and other signs of pericardial effusion, a *fremitus* is sometimes felt over the præcordial region when the hand is laid thereon; it is doubtless occasioned by the same cause as that which gives rise to the friction sound, but is not often met with. It has been observed in cases in which effusion of lymph has taken place into the pericardium, and the pericardium has thereby been united to the thoracic walls.

CHAPTER IV.

PERICARDITIS.—GENERAL SYMPTOMS.

Pulse.—Respiration.—Pain.—Cerebral Symptoms.—Dysphagia.

THE general symptoms* attending acute pericarditis are those of inflammatory fever, of the rheumatic type; and with these are usually present, at the same time, local pain and oppression about the region of the heart,—pointing to that organ as the part upon which the inflammation has fallen.

The *heart's beat,* at the commencement of the attack, is stronger, fuller, more widely spread, and more frequent than natural; but it soon loses its regularity, some of its beats being perceptibly stronger than others; and with the progress of the

* We must premise, that the symptoms here given are those of a typical case of rheumatic pericarditis; practically, the *symptoms* of pericarditis are very varying, and sometimes so obscure as to throw little light on the diagnosis of the disease; but happily the *physical signs* rarely fail to serve us; they remain constant, and are readily seized by the attentive observer. From which we may conclude, how important it is ever carefully to watch for these signs in all those diseases in which experience has taught us that pericarditis may possibly arise.

disease, and as effusion into the pericardium takes place and the vital power of the heart is weakened, the impulse diminishes in force, becoming weak, trembling, and at length even imperceptible.

The *pulse* at the wrist varies much in its characters; for the most part it corresponds to the nature of the heart's impulse: at first hard, frequent and strong, it becomes weak, fluttering and irregular in the latter stage of the disease, and when the pericardial effusion is large. It retains its regularity more when the exudation is solid. Sometimes it remains of a natural strength, even when the impulse of the heart is hardly perceptible; or it may be very weak when the heart's impulse is violent, in which case we may expect the co-existence of hypertrophy of the left side of the heart, with valvular disease. A weak, fluttering, irregular and intermittent pulse is ever an anxious sign, for it indicates an advanced condition of the disease, and an enfeebled state of the vital powers. In no disease is the pulse more subject to sudden changes than in this; the least movement or mental excitement raising its frequency in a most remarkable degree. When neither pleurisy nor pneumonia co-exist, the pulse is often much more frequent than natural, in proportion to the number of the respiratory movements.

The Respiration.—Several causes tend, directly and indirectly, to quicken the respiratory move-

ments and impede the respiration in pericarditis; and these causes act conjointly or separately. The due expansion of the thorax is prevented by the pain; the free return of the blood from the lungs is impeded by the pericardial effusion, which restrains the action of the heart and compresses the auricles, as already explained. The motion of the diaphragm also is frequently affected in consequence of the pain which it occasions. Pleurisy and pneumonia are almost constantly associates of acute pericarditis; and when present, they also necessarily interfere with, and increase the disturbance of, and embarrass the respiration; and more or less so, according to their degree and extent. And again, where the pericardial effusion is considerable, it mechanically presses upon the lungs, particularly the left lung.

By all these different circumstances the respiration is impeded. The respiratory movement of the upper part of the chest is increased, and that of the epigastrium arrested; the expansion of the ribs over the heart and left side of the chest is also partially arrested, particularly when the costal pleura participates in the inflammation; the difficulty of breathing is much increased by the slightest motion or mental agitation; sometimes, even when the exudation is small, the patient breathes much quicker than natural, and perhaps without being aware of his doing so.

In young subjects, and in those in whom the elasticity of the ribs and their cartilages permit of it, a bulging of the præcordial region is sometimes observed, where the pericardial effusion is considerable.

The distension of the jugular veins indicates an obstruction of the circulation; and their degree of distension is a good test of the amount of the impediment. When the veins do not collapse, but remain constantly distended during inspiration and expiration, we may be sure that the circulation is seriously obstructed.

Hence the effects on the respiration, resulting from the pericarditis, depend upon several circumstances:—upon the amount of existing pericarditic and pleuritic effusions; upon the degree of pain; upon the condition of the muscular structure of the heart; upon the inflammation and paralysis of the diaphragm; upon the presence or absence of pneumonia and pleurisy; and upon the extent and degree of these.

When the inflammation runs high, and the lungs and pleura become implicated in it, the symptoms are especially severe: the face expresses extreme anxiety and suffering; the patient fears the slightest motion, and even speaking a few words will render him almost breathless; the features are livid and the lips blue; the respiration is painful and rapid, and the ordinary signs of asphyxia gradually appear.

Difficult respiration is an important symptom, when it comes on suddenly. The fluttering, faltering heart, and the respiratory anguish, as Dr. Latham remarks, almost always occur late in the course of the disease.

Pain, when present, is generally most severe at the onset of the inflammation; but of all the symptoms of pericarditis, it is the most inconstant, and therefore the least trustworthy. It is felt chiefly about the præcordia and at the scrobiculus cordis, and is increased on deeply breathing, and by pressure—particularly by pressure at the epigastrium: it is increased also by motion. Sometimes it reaches to the shoulder and down the left arm. When the pain is felt extensively over the thorax, we may surely infer that pleurisy accompanies the pericarditis. The tenderness on pressure, often felt at the epigastrium, may accidentally be the result, it is well to remember, of diaphragmatic pleurisy or of gastritis, as well as of pericardial inflammation. The stomach, indeed, often sympathizes with the inflamed pericardium, and then vomiting, etc., occur. The integuments at the præcordia are sometimes œdematous.

The tongue is dry and red, or moist and coated with white epithelium; the thirst is great, and there is much general fever; the distress of the patient is often extreme, when the symptoms are severe. At first he is very restless, and if rheumatic

pains hinder him not, frequently varies his position in bed; but with the progress of the disease, movement is restricted, and then he lies for the most part on his back. Often he requires his head and shoulders to be raised, feeling that he breathes more freely when he is high in bed. If one lung is much compressed by the pericarditic or pleuritic effusion, he tends to lie towards that side where the effusion is. Whatever position he assumes, is painful and irksome to him, and yet some impending dread forbids him to change it. In the worst stages of the disease, cold sweats come over him, lividity of face, rapid respiration, feelings of suffocation, and then, at length, coma closes the struggle for life.

Cerebral Symptoms.—In pericarditis, symptoms indicating disturbance of the nervous centres are always of very serious import: the pathology of these affections has yet to be written, for their nature is not indicated by any local changes hitherto discovered, to which they can be ascribed. When they appear, they are generally the precursors of a fatal termination of the disease; in young persons they often present themselves under the form of choreal disease. Sudden movements of the body, twitchings of the limbs, throwing about of the head, great restlessness, delirium, distortion of the features, startings in the sleep, tetanic spasms, are the usual manifestations of these disturbances of

the nervous centres. "Wild delirium, epileptic or tetanic convulsions, chorea, coma, fatuity, are the greatest and the rarest of these symptoms; and mutterings, reveries, transitions from torpor to excitement, subsultus, are the least and most frequent, but they are all akin one to an other. The least may mount up to the greatest, and the greatest run down to the least." (Latham.) In pericarditis associated with Bright's disease of the kidney, the head symptoms may, perhaps, be attributed to the uræmic condition of the blood; but their cause in rheumatic pericarditis is not clear; whether it is a poisonous agent in the blood acting directly on the brain and spinal marrow, or whether it is excitement, arising in a secondary or reflex manner from the local disease, we know not. This much we may say, that the old doctrine of metastasis gives no explanation of these cerebral symptoms.

Difficulty of swallowing is occasionally observed when the pericarditic effusion is considerable; it depends in all probability upon the pressure of the fluid in the pericardium exerted on the œsophagus, whereby the passage of the food into the stomach is mechanically impeded; the difficulty is increased when the patient lies recumbent. The same pressure has been thought, also, in rare cases, to have acted upon the trachea, and so to have interfered with the respiration.

The condition of the skin depends, in great part,

upon the nature of the pericarditis, whether rheumatic or not: when the disease is of rheumatic origin, the state of the skin, as well as many of the general symptoms, are those peculiar to acute rheumatism.

In some rare cases of rheumatic pericarditis, the inflammation of the pericardium precedes the affections of the joints; sometimes it comes on when the arthritic affections have already ceased to exist; but much the most frequently the pericardial inflammation supervenes during their continuance. It has not yet been clearly ascertained, whether the severity of the articular, bears any relation to that of the pericardial inflammation. "Sometimes we find the one affection is violent, and the other absent or slight; and then again both the rheumatism and the pericarditis are violent, and their symptoms increase and decrease in force together. The fact is, that pericarditis may be equally looked for, whether the acute rheumatism be severe or mild,—whether its seat be fixed or shifting, and at the beginning as well as during the progress of the disease." (Latham.) Such seems the experience of physicians in London; but it does not correspond with that of Dr. Stokes in Dublin, who says: "The liability to all forms of carditis in rheumatism is in proportion to the severity and obstinacy of the fever." The question is one which is manifestly very difficult of solution.

I have frequently had occasion to corroborate a fact stated by Dr. Watson,—a law he calls it,—namely, that the younger the patient is who suffers acute rheumatism, the more likely will he be to have rheumatic carditis.

CHAPTER V.

PERICARDITIS.—DIAGNOSIS.

General Diagnosis.—Differential Diagnosis.—Prognosis.

THE local and general symptoms of pericarditis, as we have seen, are often very obscure; and none of those above described point with unerring certainty to the pericardium as the seat of inflammation. We must therefore be mainly guided in our diagnosis of the disease by the physical signs, viz., the friction sounds, and the increased dulness of percussion over the præcordial region. Guided by these, and aided by the general and local symptoms, a correct diagnosis will rarely fail us.* These physical signs, therefore, are of every importance to the student; but he must distinctly understand, that there are some possible sources of error, into

* Corvisart, to whom the friction sign was unknown, speaks of the "scrupulous attention and subtile sagacity" requisite for the diagnosis of pericarditis. The idea which he held of the fatality of this disease probably arose from the circumstance, that the slighter cases of pericarditis escaped his observation altogether; that he, in fact, recognized those only in which the local and general symptoms were severe and fatal.

which he may fall in judging of them. These we have already spoken of, but will now more particularly refer to again. We are told to found our diagnosis of pericarditis on the acoustic character of the friction sounds, to which the inflammation gives rise; on their concentration or diffusion; on their double or single nature; on their amount of extension over the thorax; on their transmission or not along the vessels; on the presence of tactile signs; on their constancy or variability in seat, and on the effect of treatment in modifying the sounds.

But there is not one of these particulars to which exception may not be taken: thus, as we have already seen, the pericardial sound may resemble an endocardial murmur; it may be single; it may be diffused; it may be transmitted along the vessels—at all events, to a certain extent, along the aorta. The tactile signs also are rare, and when present may be the consequence of pleurisy; moreover, endocardial murmurs, arising in consequence of co-existing endocardial inflammation, are very commonly present in pericarditis, and so complicate the pericardial friction sound; and lastly, the friction sound may be exo-pericardial or pleuritic.

Dulness of percussion over the præcordial region has also other indications besides that of pericardial effusion: it may depend upon hypertrophy of the heart; upon aneurism; upon malignant or other

tumours; upon œdematous or inflammatory consolidation of the lungs; upon simple serous effusion into the pericardium; upon a large development of fat in the anterior mediastinum, and upon pleuritic effusion.

These are possible sources of error which may mislead our diagnosis, and which therefore it is well to be aware of and guard against. But after all, in practice, there is happily little chance of the careful observer being misled into a wrong diagnosis by any of them; for to each of these accidents special characters are attached, which serve to distinguish them when not representatives of pericarditis. In the great majority of cases of pericarditis, therefore, which come under our notice, we may conclude that the physical signs at once and readily declare the nature of the malady; and when, as occasionally in some rare instance may happen, they permit a doubt on the subject, their comparison with the general symptoms, added to a due consideration of the whole history of the case, will rarely fail to remove the difficulty.

Prognosis.—The immediate results of an attack of pericarditis,—in so far, at least, as the disease imperils life,—we may anticipate will be favourable. Unfortunately, however, it happens, that in the greater number of cases, the pericarditic attack leaves behind it, in the heart, more or less clearly marked traces of permanent injury; and we may

add, that in all probability it predisposes the organ to a repetition of the inflammatory attack. I have already described, in speaking of the pathology of pericarditis, the secondary disturbances, which result to the heart and other organs of the body, as consequences of the pericarditis; and to the account there given of them I must here refer the reader. The most important consequences resulting from the inflammation, are those which ensue through injury of the heart's valves, caused by the endocarditis which so very frequently accompanies the pericarditis. The evils supposed to result from pericardial adhesions have already been referred to.

It is impossible to give a favourable prognosis of the future health of a person who has been once attacked by rheumatic pericarditis. He must ever be a subject of watchful care. To him, if to any one, that motto of Corvisart is truly appropriate—*Adhærit lateri lethalis arundo.* He may have recovered from the immediate effects of the inflammation; but in all probability he has done so with a damaged heart; he is conscious in himself, that he is not the man he was before the attack; his breathing is shorter and quicker; he has an occasional cough; he is not able to undergo the exertion which he was once equal to; occasional palpitations also trouble him, especially if he walk fast or run; in short, he bears about him, in a more or less marked form, the symptoms of heart disease. Rare,

indeed, are the cases in which the heart, once attacked, escapes permanent injury; rarer still those, in which it remains ever after free from a repetition of the attack. No time appears to grant immunity therefrom: years may pass away, and seemingly all traces of the pericarditis with them; but still, some slight mischief remains—too slight, it may be, to warn the patient of its presence, and yet enough to tell a tale to the ear of the physician, of permanent injury done to the heart; and then, at length, some cause exciting, inflammation once again falls upon the injured organ, and with increased violence in its effects. Or gradually and almost imperceptibly, and in a chronic way, the injury which has so long lain quiescent, and has even left its victim unconscious of its presence, gathers strength: the imperfect valve, for instance, by a slow process of disease affecting its structure, becomes more imperfect, until, at length, the imperfection manifests itself by symptoms to the patient, as well as by signs to the physician; and then begins the struggle for life, which, continuously maintained, or interrupted by some short peaceful intervals, ends but in death.

In such cases, the prognosis we give as to the possible prolongation of existence, depends very much upon the condition of life of the patient, and upon his powers of restraint over himself. The poor—those who live by their daily manual labour

—have, as a rule, but small hopes of prolonged life before them; they are still exposed to the causes, which were perhaps the original sources of their malady, and their very exertions to gain the means of living increase the severity of the symptoms which are destroying them. There are no cases, met with in hospital practice, whose progress is more sad to watch than these. Removed from the immediate provoking causes of their sufferings, and subjected to proper hygienic rules, the improved change in the bodily condition of such patients is often rapid, and indeed surprising: the dropsical symptoms, the difficult breathing, the præcordial pain, the palpitations, etc., quietly disappear, and the patients after a time leave the hospital, with the belief that they are cured of their disease. Then once more they return, as they must, to their labours, and once more these labours, and the other attendant circumstances of their condition, provoke the rapid recurrence of the evils, which they vainly hoped they had for ever left behind them, when they quitted the hospital. In such cases, prognosis has little favourable to hope for.

But the prognosis of the disease, as it occurs in those of better condition in life, and who are able to bestow their whole attention on care of their bodily condition, is often very hopeful for prolonged existence. In no class of chronic maladies can it be said, that medicine acts more efficiently, in the

relief of symptoms, than in this class, or that it has more power, in warding off the consequences of organic disease, when that disease has not reached beyond a certain stage of progress. Hence, therefore, to such-like subjects of cardiac disease, who are able and willing to submit to treatment, medicine can often hold out hopes of a lengthening of their days.

Upon a general consideration of the subject, we may say of the termination of *rheumatic pericarditis*, that the disease is rarely fatal in its first attack; but that the attack seldom leaves the heart perfectly restored to its previously healthy condition. In most instances, it leaves the organ more or less damaged; or, if not damaged, it at least renders the heart readily obnoxious to recurring attacks of the inflammation, and thus, earlier or later, induces fatal structural changes of some or other of its parts.

Non-rheumatic pericarditis, on the other hand, is generally fatal; or, more correctly speaking, it is associated with a fatal malady, and occurs at a fatal period of that malady's progress. It is not so much the pericarditis, in such case, which causes death, as the general disease which provokes the pericarditis. The pericarditis is but one of the symptoms of a fatal disorder,—for, instance, of uræmic disease; and it is not by any means necessarily a prominent symptom, as it generally is in cases of rheumatic pericarditis.

CHAPTER VI.

PERICARDITIS.—TREATMENT.

General Remarks.—Bleeding.—Mercury.—Opium.—General and Local Treatment.—Paracentesis of the Pericardium.—Pericardial Adhesions.

In considering the treatment of pericarditis, it is necessary to call to mind a fact already referred to, namely, that pericarditis (excepting in the case where it is the consequence of traumatic injury) is not an idiopathic inflammation; that is, not idiopathic in the sense in which we use the term, as applied to ordinary inflammation of the lungs.*

* I say advisedly, "as usually applied to inflammation of the lungs"; for I do not wish to be here understood, as considering that so-called idiopathic pneumonia is really and truly invariably such. I have already referred to the fact, that pathology is daily diminishing the number of those idiopathic local diseases, and referring them to some more general and exciting cause, present in the body. There are many reasons which justify the belief that "idiopathic pneumonia" may possibly be merely the local signs of a general disorder. We must remember, therefore, that "idiopathic" means merely, and no more than, this, viz., that, so far as we at present see, the inflammation is a local disorder, provoked by local causes, and having no relation to any particular general disorder of the system; and admitting, as we must, our very imperfect know-

Pericarditis is manifested either secondarily, as the result of some other disease present elsewhere in the body,—of purulent absorption, for instance; or as one of the local expressions of some general disorder of the system,—of its fluids or its solids,—as in the case of rheumatic fever; or the inflammation is the immediate consequence of traumatic injury.

It is of every importance to keep these distinctions in the origin of the disease clearly in view: unless they are properly recognized by us, we shall never arrive at a true and rational method of treating pericarditis. Excluding from consideration those cases of pericarditis which are the result of traumatic injuries, we find that we have to deal with disorders, in which diathesis plays a part; and therefore to attempt to treat them as simple local inflammations, and without reference to their particular diathetic character, is to treat them erroneously. Equally manifest is it, that no general directions of treatment can be prescribed,

ledge concerning the exciting causes of diseases in general, and marking, as before said, the more enlarged views which modern pathology has opened to us concerning their nature, we are at least inclined to be cautious in localizing *the disease* in the existing and perhaps accidental derangements of parts. Even while I write, an example of what I am stating comes before me. Dr. Addison, in a most interesting memoir on diseases of the supra-renal capsules, tells us that he once spoke of a peculiar, and what he termed, an "idiopathic anæmia"; this anæmia he now associates with disease of the supra-renal capsules. He formerly called it "idiopathic", simply because there was no apparent cause exciting it.

which shall be adapted to any given case: each demands a particular consideration—a consideration founded, in the first place, upon the cause or relation of the disease to other diseases; and secondly, upon the age, constitution, and condition in life of the patient. Such are the data upon which a rational treatment must be founded.

It follows necessarily from this, that the treatment, which may be proper in one case, may be most unfitted for another case; and, therefore, that in different cases, most opposite forms and methods of treatment become the true and proper methods. To no other reason than this, namely, the want of a proper recognition of the peculiar characters of the disease, can we attribute the various and opposing modes of treating pericarditis, which have been loudly extolled here, and as loudly decried there. In this must we seek an explanation of the extraordinary discrepancies, which the history of the treatment of this disease furnishes us with.

Pericarditis, let us recollect, is known to us in connexion with rheumatic fever, and it is known to us in connexion with Bright's disease of the kidney; but what rational being would pretend to apply the same treatment, indifferently, to the inflammation as associated with these two diseases? Broadly considered, the one is a disease of the strong and healthy; the other, a disease of the broken-down constitution. Treatment which might

rescue the patient from death in the one case, would perchance destroy his life in the other. The truth is, as we have already said, that every case must be judged of by its individual features, by its relations to co-existing diseases, and by the bodily condition of the patient. The general principles, applicable to the treatment of inflammation, hold good here; but the peculiar circumstances affecting the case modify, and altogether regulate, their application. How can we speak, in a comprehensive manner, of a particular method of treating pericarditis, when we are speaking of a disease which presents itself, now under an acute form, in persons of the most healthy and robust constitutions; and then again, as an obscure and chronic malady, in the weak, worn out, shattered constitution?

We have already seen, when describing the causes of pericarditis, that it was necessary to divide the disease into two general classes, *rheumatic* and *non-rheumatic;* and in accordance with what has been there advanced, I shall consider the treatment of the disease under those two separate heads.

The first sort, the *rheumatic pericarditis*, includes generally all the most acute forms of the disease. In such the local inflammation itself becomes the chief centre towards which the treatment is especially directed; for it is this local inflammation which immediately tends to the destruction of life,

and in the manner I have elsewhere described. The local character of the inflammation, therefore, and the consequences resulting from it, are the characteristics of rheumatic pericarditis,—its prominent features. The same cause which brings out the pericarditis is, perchance, the cause of the arthritic affections, and may be the cause of other local inflammations; but all such other co-existing and localized affections, are of diminished importance in comparison with the pericardial affection; once this has declared itself, it becomes the primary object of our treatment; the others are but secondary and subordinate objects.

On the other hand, in the *non-rheumatic* form of pericarditis, the local inflammation is rarely of other than secondary importance; its symptoms are ofttimes so little marked, so obscure, and produce, by their reaction, so little disturbance of the constitution, that the existence of the inflammation may altogether escape observation, if not carefully watched for and anticipated. Where non-rheumatic pericarditis occurs, death very generally ensues, but it is not the pericarditic inflammation, which is the chief destroyer of life in such cases. We know, indeed, that although the inflammation is ten times more severe in the rheumatic class of pericarditis, death is an exceptional result, whilst here it is the rule; and when we seek a clue to elucidate this apparent discrepancy, we find it at once in the

different nature of the maladies with which these inflammations are associated; acute rheumatism is not, *per se*, a fatal malady, but uræmia is, and so are very generally those forms of erysipelas, of scarlatina, of typhus, of pyæmia, of pneumonia, of pleurisy, in the course of which pericarditis appears, and which are erroneously said to be its causes.

Speaking generally, and in accordance with the foregoing remarks, we may say, that the treatment of the *non-rheumatic* class of pericarditis is the treatment of that particular general affection, with which it is associated; and that it has, therefore, quite a secondary reference to the particular local inflammation. Such is the rule; but of course it has its exceptions: for we now and then meet with cases of non-rheumatic pericarditis, in which the local inflammation takes on the most acute form. And again, on the other hand, we may say of *rheumatic pericarditis*, that though our consideration of its treatment may be partially guided by the diathetic character of the disease, yet that it is not, in the main, the treatment of acute rheumatism, but rather the treatment of inflammation of a serous membrane; its immediate and direct object being the local inflammation. In ignorance of its essential cause, we treat it as if it were truly a local and idiopathic inflammation of the pericardial serous membrane; we pass over the general dis-

order, and apply ourselves almost solely to this the local manifestation of it, and treat it on the ordinary principles, which are included under the head of the antiphlogistic treatment of inflammation. It is to the acute inflammation, as we find it especially typified in rheumatic pericarditis, that the following remarks on its treatment particularly apply.

Bleeding, in the treatment of pericarditis, was once highly extolled; at the present moment, however, its practice is rarely resorted to in this country. That its value was overstated by Bouillaud and others, no one can reasonably doubt; —that it is at this moment, and, as it were, by reaction, less resorted to than is well for the patient, there is good reason to believe. Its actual efficacy as an agent controlling the disease, data do not enable us succinctly or satisfactorily to determine; but in judging of this, the practitioner is not without help, for he has that best and safest of guides to direct him, namely, the general opinion of our best physicians. Their experience seems to tend to the conclusion, that, where the fever runs high, where the symptoms are acute, the constitution of the patient is good, and particularly when the inflammation is associated with rheumatism,—bleeding, general and local, carried to such an extent as to make an impression on the system, is proper; but that if, after this, the disease still progresses, the bleeding must not be persevered in.

When the heart's sounds are feeble or inaudible, and its impulse scarcely perceptible, we may be very sure that bleeding cannot be safely indulged in. Our best physical guides to a decision, where there is a doubt as to the propriety of bleeding, are, the strength of the impulse, and the loudness of the sounds of the heart.

When bleeding is advisable, it cannot be practised at too early a stage of the inflammation. There are occasions, when the general symptoms are such as to warrant us in resorting to its use, even before the physical signs have positively declared the existence of the pericarditis. It is well, indeed, to recollect that here, as in other thoracic diseases, the inflammation has already made much progress, before the physical signs demonstrate its actual seat. "Pain in the heart; excessive impulse of the heart; irregular action of the heart; any one of these, or any two, or all of them together, coming on in the course of acute rheumatism, make inflammation of the heart so nearly certain, that it would be folly to suspend the remedy by waiting for more certainty, and so running the hazard of having a more advanced and less tractable disease to deal with." (Latham.)

Whenever the local pain is great, local bleeding is almost invariably beneficial; and it may be practised independently of the general bleeding. We may resort to it, also, whenever the judgment halts

regarding the propriety of general bleeding. Caution, however, is requisite even in the local abstraction of blood, when the patient has previously undergone venesection; for just as the excitement of the heart's action is very great in the first stage of pericarditis, so likewise is its depression in the latter stage; and for this depression we should be ever carefully on the watch. Even a few leeches seem sometimes to produce alarming results. I have seen purpura hæmorrhagica and death quickly succeed the application of eight or ten leeches, in a case where bleeding was considered inadmissible; and I have also witnessed fatal choreal symptoms closely follow the application of a few leeches in a child; and in both cases was compelled, in reason, to associate the effects of the leeches with the immediate results which followed their application.

It has been supposed that leeches, applied to the inflamed joints, may reduce the pericardial inflammation, but of this the proofs are altogether wanting. Whether there really is any connexion, between the severity of the arthritic and the severity of the pericardial inflammation, is still matter of doubt. The influence which the one exercises over the other has yet to be determined.

Whether leeches or cupping be most adapted for the local abstraction of blood has been made matter of dispute; cupping is occasionally in pericarditis a very painful operation, but it has the advantage,

when skilfully performed, of more quickly obtaining the object sought than leeches, and of less worrying and fatiguing the patient; and less exposes him to the chances of being chilled by exposure to the air.*

Under all circumstances, great caution is required in the subtraction of blood, even when the inflammatory symptoms are violent: in pericarditis, the reaction of depression following upon the excitement is great, and it sets in early; indeed, it is just in those cases where the inflammation appears most violent in the first instance, that we should in particular watch for and expect the reaction of depression,—a depression, whose nature is such as sometimes unexpectedly to threaten the life of the patient. We must never forget, that an organ is involved in the inflammation, the integrity of whose functions is indispensable to life,—their cessation

* It might, perhaps, not be out of place here to insert a word of caution to the student in respect to the management of the physical examination of the patient. I believe there is no case in which physical diagnosis is required, where more gentleness and tenderness should be employed in its practice than in the case of acute pericarditis. The severe sufferings of the patient forbid all needless exposure of his person, and all manipulations which are not absolutely necessary for the satisfaction of the diagnosis. I cannot doubt, that the excitement, and the pain, and the exposure which frequent examinations necessarily entail, are often very prejudicial to the patient; and particularly in the instance of young and nervous females, who seem, among the lower classes, to be especially liable to the inflammation, and in its most acute forms.

bringing instant death; and that one of the effects of the inflammation and its products is, to induce a paralysed condition of the muscular structure of the heart.

Moreover, it is certain that bleeding will not, in all cases, arrest the exudation; on the contrary, in certain states of the body, it appears even to hasten and increase the pouring out of it. There is another danger, also, which may possibly be incurred by over bleeding. Endocarditis, as we have said, is very commonly associated with the pericarditis, and under such circumstances it would appear, that large bleedings, by promoting the tendency in the blood to the deposition of it fibrinous particles, increase the danger of permanent injury, to which the valves are exposed, through the deposition of fibrin upon them.

Mercury.—The induction of the specific effects of mercury has been considered by great authorities as of primary importance in the treatment of acute pericarditis; and although later experience rather leads us to the belief, that its influence in arresting the inflammation has been much exaggerated,* still the concurrent testimony of so many excellent practitioners in its favour scarcely justifies us in abstaining from its use; it is certain, that relief to all the symptoms very often ensues, when the signs of the

* We have seen fatal pericarditis commence when the patient was already suffering from severe salivation.

specific action of the mercury appear. When the mercury is thus administered, its action should be induced as early and as speedily as possible; for the disease is swift, and when it falls upon any part of the heart, it is apt to leave that part permanently damaged; and permanent damage of the heart is the first step to a series of fatal secondary disorders, which arise in different parts of the body. It happens however, unfortunately, that the mercurialization of the patient is not always easy of accomplishment, in cases of pericarditis. The mercury should be given immediately after the bleeding has been performed, for then it acts most efficaciously; it may be administered in the form of small doses of calomel mixed with opium, to prevent purging. If the calomel fail to act, or the stomach refuse to bear it, mercurial frictions may be used.

Mercury, even when given without any idea of inducing its specific action, is undoubtedly of very great service in pericarditis, through the action which it excites in the secreting organs. The importance of a proper regulation of the secretions is certain; and doubtless many may be disposed, and not without some show of reason it must be allowed, to think, that whatever good mercury effects, it effects in this way. It is much to be feared, indeed, that even at the present moment, very skilful physicians hold most opposite opinions respecting the use of this remedy in pericarditis. There

are those who invariably prescribe it, and there are those who never resort to its use. My own experience—and I have had considerable opportunities of watching the progress of the inflammation, both under, and free from, the influence of mercury—seems fully to warrant the conclusion, that the actual influence which the remedy possesses over the disease has yet to be shown; but that good results so often follow its moderate administration, as to justify its frequent use.

Opium.—I have already mentioned, that the pain and sufferings which afflict the patient during the progress of this disease are sometimes very grievous to bear; they are especially so, when severe arthritic affections co-exist with the pericarditis. We must remember, that the shock of violent and sudden pain will occasionally destroy life; and that a lesser degree of it, unceasing and severe, will likewise kill, by wearying out and exhausting the vital powers of the patient. Now I have warned the student, in speaking of bleeding, that the vital powers of the heart are apt to fail suddenly in pericarditis, through paralysis of its muscular structure: with double caution, therefore, should he watch and strive against the effects of this other exhausting influence—the pain.

In this, as in so many other diseases, long restlessness and sleeplessness, the results of pain and

suffering, are unfavourable symptoms. Sleep, then, must be procured; and, administered to this end, opium ofttimes acts with all its magic charms; indeed, it sometimes seems to have an effect which reaches beyond the immediate object sought for,—beyond the procuring of rest and the alleviation of pain. Judging from the manifest signs of improvement in the inflammation, which occasionally follows its administration, one might almost believe that it has some direct influence in arresting the disease. The quantity of opium administered, must be measured by the degree of pain suffered; and the large doses of it which even young children will take with impunity, in these cases, is surprising. Three or four grains of Dover's powder, given with a like quantity of the hydrargyrum cum cretâ every three or four hours, is an excellent manner of combining the opium and mercury: the amount of the Dover's powder and of the mercury being respectively diminished or increased, in proportion to the symptoms produced by them. In some cases, a grain of opium given every three or four hours, or even more frequently, is necessary, to bring ease to the patient.

The three remedies here spoken of—viz., local and general blood-letting, mercury, and opium—are the main therapeutical aids, on which we must rely, in our treatment of pericarditis. We must not, however, neglect general indications of treatment.

The secretions are to be duly attended to. The bowels should be regularly relieved, but frequent purging must be avoided; for the partial exposure to cold which is its necessary consequence, and the extreme pain and suffering which movement is apt to occasion in these cases (especially when the co-existing inflammation of the joints is severe), more than counterbalance, by the nervous excitement and general disturbance they produce, the good which the purging might be supposed otherwise to bring about, by the process of elimination. The action of the kidneys should be also carefully maintained.

It is very doubtful whether colchicum exerts any direct influence over the disease.. It may be administered, however (when there is no fear of depressing the heart's action, and when arthritis is present), on account of its admitted use in rheumatism: combined with alkaline salts, it serves to keep up the due action of the secreting organs. But I must remind the reader of what has been already said, respecting the special nature of rheumatic pericarditis, namely, that the severity of the local inflammation appears altogether to throw into the shade the specific character of the disease itself. Our treatment of it, in fact, as appears from what has gone before, is the treatment of an acute local inflammation; and, so far from being the same as the treatment of acute rheumatism, it in some material points differs therefrom.

Blisters often give great relief, even in the early stages of pericarditis, and may perhaps reduce the effusion at that period; but when the pericardial surfaces are covered with layers of lymph, we cannot hope that any immediate absorption of the fluid will take place. Stimulating embrocations and sinapisms, in the more chronic conditions of the disease, are also useful.

In every case of pericarditis, whether rheumatic or non-rheumatic, we should carefully watch for signs of the heart's failing powers. Weakness, in this disease, is apt to creep on very insidiously, and to manifest itself suddenly. This warns us to be cautious, in the first instance, that we do not over deplete the patient; and it also warns us not to delay too long the use of stimuli. There are cases, especially of the non-rheumatic class, in which stimuli seem requisite from the very beginning of the attack; the condition of the system, under which the pericarditis has arisen, demanding their use. This is a point to which Dr. Stokes has called especial attention, and, there can be no doubt, most wisely. He is "convinced that patients are often lost from want of stimulation at the proper time." The following are the particular signs, according to Dr. Stokes, which indicate a weakened condition of the organ: a feeble, intermittent, and irregular pulse, especially when it has not had these characters from the first; turgescence of the jugular veins;

change in the heart's sounds, particularly feebleness of the first sound; pallor, coldness of skin, œdema, and faintings.

Pericarditis, associated with rheumatic disease, is a very common disorder in the young; and it appears to select for its attacks those who are least fitted by bodily constitution to resist them. The disease is sometimes especially severe in young females: in such cases, the violence of the inflammation is shown by the abundance and rapidity of the pericardial exudation; and when accompanied by rheumatic and lung affections, it too often outstrips all our attempts to arrest its progress. Here, for obvious reasons, mercury and blood-letting are very ill-adapted remedies; but, on the other hand, opium often seems, in persons so constituted, to exercise a most beneficial influence. I have had occasion to observe the most marked good results follow upon the administration of this remedy, in cases of the description here referred to; and at the moment, when the local signs and general symptoms indicated that the disorder was at its height. The opium, in such cases, must be administered with an unsparing hand; it must be given, in fact, in such doses as are sufficient to produce its narcotic influence on the suffering body; to allay the exceeding pain, whether of the heart or of the joints, and the nervous agitations, which accompany the disease. I must confess, indeed, to have witnessed

with surprise, the recovery of patients thus treated —the marked and rapid recovery of them; and even when the signs of extensive cardiac and pulmonary inflammation existed. It is difficult to determine, how much of the good effects result from the allaying of the pain and nervous excitement, which in young, weakly-constitutioned individuals, are especially severe in this disorder; or if any benefit results from the direct influence of the drug over the inflammation; but of the good effects themselves, thus occasionally produced by the drug, I can scarcely venture to doubt.

Observation certainly leads me to the following conclusions: that rheumatic pericarditis is an inflammation attacking rather those of weak than of strong constitution; that it is much more common in the delicate and young, than in vigorous persons, at the prime or middle periods of life; that the degree of inflammation—that is, the general febrile re-action and the local exudation—is also greater in them than in the strong; and moreover, that the disease is more fatal. To lay down any specific rules of treatment, which shall be fitted to every case of pericarditis, is therefore irrational: as we have again and again repeated, every case must be considered according to its individual features. To speak of the treatment of pericarditis, as a treatment which is at once applicable to the disease as it occurs in a vigorous man at the prime of life,

and to the disease as it occurs in a weak, pale, sickly female, is simply to include most diverse objects under the same consideration; and yet, surely, this is just what they do, who speak and write of the disease and its treatment, as though the former possessed the same characters in every person it attacks, and as though the latter was to be in every case alike; and was alike fitted for every case.

From all which considerations, this corollary would seem to result, viz., that in the treatment of pericarditis, the administration of our remedies must be guided by the general condition and the idiosyncrasy of the individual attacked, rather than by the intensity or other peculiarity of the local inflammation.

Thus, our remedies in the acute stage of rheumatic pericarditis, whatever their kind, must be adapted and proportioned to the condition of the patient. We must not put our trust in any one remedy, nor rely upon any one particular method of treatment: the remedy and the treatment must be fitted by the skill and experience of the physician to the individual case in hand. In the strong and vigorous, for example, where the inflammation runs high, bleeding may be a right and proper remedy; but it is absurd to suppose that it can be equally so, in the weak and pallid artizan. We should remember that every person has, in a modified

sense, his own proper idiosyncrasy—a constitution differing from that of another; and therefore, that every case of pericarditis presents us with certain special objects for our consideration. In some persons, these objects are well and clearly marked; in others, again, they are obscure and ill defined. And herein—in seizing upon the special points of the individual case, and in modifying the remedies to meet their requirements—lies that peculiar practical tact which distinguishes the skilful physician.

But all the efforts of the most skilful may fail to arrest the progress of the inflammation; and frequent experience, indeed, warns us, that we must never be too sanguine in our hopes of guiding it to a successful issue. The reason of the frequent failure is worthy our consideration. Let us recollect that our treatment of pericarditis is purely experimental: we have no specific antidote to apply to it: every case treated is a fresh experiment tried. We bring to the experiment, it is true, the results of accumulated experience; and it is from these treasured results, that we are enabled to gain an insight into the workings and effects of remedies on the disease, and that we learn how to direct and apply them to its cure. But the conditions involved in the idea of a perfect method of treatment, it is impossible to satisfy, in any case: perfection in treatment would require, not only that the right remedy should be selected, but that it should be

applied at the right moment, and that a right degree of its active properties should be expressed from it; all which is manifestly impossible of attainment, in the present state of our knowledge. The thousand ever-varying accidents which modify and derange the functions of the different organs and parts of the body, unseen and inappreciable to our senses, present continual and insuperable stumbling blocks to our best-directed efforts and our well-tried experience, in the treatment of their derangements; and entirely preclude us from calculating, with anything approaching to mathematical precision, the results which may be anticipated from the remedies administered. We must not be surprised, therefore, if our most skilfully-applied knowledge — knowledge derived from a conscientious observation and an enlarged experience—is often applied in vain.

When the inflammation, then, still progresses, in spite of our efforts to arrest its steps, secondary consequences result from it, and offer fresh indications for treatment. The most immediate and serious of these are — increase of the pericardial effusion, which directly damages the muscular power of the heart; and congestions of the different organs and parts of the body, caused by the impeded circulation. Inflammation of the lungs and pleura, moreover, almost invariably complicate severe cases of pericarditis, as has been stated under

the head of its pathology; and it is only exceptionally, that endocarditis is absent during the progress of the inflammation. All these serious accidents multiply the list of evils, which so try the physician's skill in the treatment of pericarditis.

I shall have occasion, hereafter, to speak of the treatment of congestions of the organs, arising from impediments to the circulation through the heart. With regard to the pneumonia and pleurisy we may observe, that these intercurrent inflammations are generally subordinate in their importance to the pericardial affection, and that the treatment which applies to this, is more or less adapted to them; sometimes, however, they appear more immediately prejudicial to life than the heart disease, and in such case demand our especial attention. It is in cases of intercurrent pneumonia that we find tartar emetic often of great use in cardiac diseases. Unfortunately, when the lungs as well as the heart are seriously damaged, we cannot hope that any treatment will be of much avail. In such cases, the prostration of the patient forbids antiphlogistic treatment, and little more remains for the physician to do, than to support the failing powers of life, so as haply to give the system strength to struggle through the attack: at this stage of it, therefore, and amidst this complication of disorders, we cannot be too cautious in the application of powerful remedies.

Little advantage can be expected from any direct treatment applied to the lungs, so long as the cause producing the pulmonary disease is in action; the removal of the cause is naturally followed by a cessation of the disease. This may be said of the pulmonary disease in so far as it is a secondary disease, resulting simply from congestion, etc. But, in speaking of the pathology of pericarditis, it was remarked that the specific agent, which produces the pericarditis, seems often to operate likewise upon the lungs and the pleura, and other parts of the body; hence the pneumonia and pleurisy thus arising must, in such case, be considered as specific inflammations, not as conditions resulting secondarily from the heart disease; and their treatment is then, for the most part, merged in the treatment adopted for the heart disease.

The simple hypostatic congestion of the lungs, so often found after death, demands no especial treatment; it must be looked upon as the results of general debility.

Summarily, then, it may be said, that the treatment of rheumatic pericarditis is, in the main, antiphlogistic; that it has nothing specific in it; that it is the treatment of inflamed serous membranes generally: especial regard being had to the condition of the patient, and the particular situation of the inflammation. There being no specific remedy in which we can place our trust, we must be con-

tented wisely to employ in its cure, according to our skill, the different means here mentioned, proportioning them to the nature of the malady in each particular case, and the violence and character of the accompanying symptoms.

What I have to remark upon the *Treatment of non-rheumatic pericarditis*, may be anticipated from what has been said concerning its nature, and the causes which produce it. When the disease offers a purely inflammatory type, it must be treated on the antiphlogistic system here described; but it rarely does so, and seldom presents the severe symptoms of rheumatic pericarditis. It is more frequently of a low type, occurs at an advanced period of life, and is associated with adynamic diseases. Thus its treatment has a close relation to the treatment of the disease with which it is associated.

Non-rheumatic pericarditis is, for the most part, known to us in connexion with uræmic disease, which is of itself a fatal malady. The pericarditis here is one of the consequences of the uræmia, and quite secondary in its effects on the constitution, so that its treatment is subordinate to the treatment demanded by the uræmic disease. In so far as it produces local injury to the heart and impedes its functions it requires direct treatment, but a treatment always modified by the peculiar nature of the disease which is its cause.

We need scarcely allude to the treatment of the disease, when it occurs in connexion with scarlatina, pyæmia, typhus, etc. In such diseases its symptoms are so little marked, that the pericarditis is frequently not discovered during life; the treatment is involved in the treatment of those diseases.

Puncture of the Pericardium has been recommended and practised, in cases where large collections of fluids have taken place in the sac, and have resisted all ordinary methods of treatment employed for their absorption. The results of the operation, however, as far as we are enabled to judge of it from a limited number of recorded observations, cannot be considered satisfactory, or such as to warrant its being recommended for trial in pericarditis. Theoretical reasons suggest, that such an operation, practised under the circumstances which could alone justify its performance, can afford but small hopes of success. It must be remembered, that the pericardial effusion is not the disease, but only one, and not the most important, of its results; that when the effusion is persistent, the lungs and pleura are of necessity so affected by disease, as to render existence most precarious; and that when the effusion resists all ordinary treatment, and the patient still lingers on in life, there is always great reason to suspect the presence of some local cause of irritation in the pericardium: as, for instance, tubercular or carcinomatous dis-

eases, which, as we well know, are beyond all the power of medicine to remove.

It is right, however, to add, that the operation (as far as can be judged from the accounts given of it) does not in itself appear to be productive of any injurious consequences. The objection, therefore, to its performance, lies chiefly in this—that it holds out but little hope of success.

If it be ever advantageously employed, one would think that it must be so at an early stage of the disease, in cases where the lungs and other organs of the body are yet intact, and where large quantities of fluid matters have been suddenly exuded into the pericardium, and, mechanically compressing the heart, seriously interfere with its actions.

Cases of scurvy, I may add, have been described as occurring in one of the hospitals of Norway, in which large quantities of simple serous fluids—in one case, as much as eight pounds—were drawn from the pericardium, with complete success. It is hard to understand, however, why pleuritic and abdominal effusions did not exist, in such cases, as well as pericarditic.

Pericardial Adhesions.—Adhesions of the pericardium, generally speaking, do not offer any signs by which we can recognize their existence during life; but when we have observed the progress of the pericardial inflammation from its commencement, and have witnessed the gradual departure of

the friction sound, and of the other symptoms of pericarditis, in any particular case, we may be tolerably sure that adhesions have taken place.

It would appear, that complete union of the pericardial surfaces is, for a certain time, at least, compatible with integrity of the heart's action, and that the subjects of such a condition of the membrane have existed, even for years, without manifesting any symptoms of cardiac disease, and have died at last of other affections; but cases of this description are very rare, and as a rule, whenever old pericardial adhesions exist, some other abnormal condition of the heart is likewise present.

The abnormal conditions of the heart most frequently met with, in association with pericardial adhesions, are—its dilatation, hypertrophy, and atrophy; and each of these conditions has been supposed to result immediately, as a consequence of the adhesions: the hypertrophy, from the stimulus to muscular growth, caused through the impediment to the heart's action; and the dilatation and atrophy, from weakness and deterioration of its muscular structure, which arise from the injury to its nutrition, caused by the pericardial inflammation. It is very probable that these different conditions may all result from the pericarditis, as explained under the head of its pathology; but even though they were positively proved to be the immediate consequences of pericarditis, it must be

remembered that they are not peculiar to it alone, and are therefore, as far as diagnosis is concerned, not distinctive signs of pericardial adhesions.

Dr. Stokes has very fully discussed the question of the effects of pericardial adhesions; and his conclusions appear to represent fairly enough the present state of our knowledge on the subject. He says, that obliteration of the pericardium does not necessarily induce any manifest change in the condition of the heart; that where alteration of the muscular condition of the heart is found in connexion with this obliteration, it is not necessarily a state of hypertrophy, but is often one of an opposite nature; that the cases of valvular obstruction, and of adhesion of the pericardium, are not parallel, inasmuch as that in one case the heart is free to act, while in the other its motions are prevented or interfered with; that obliteration of other serous membranes is more often followed by atrophy than by hypertrophy of the subjacent organs; and that atrophy of the voluntary muscles is the ordinary effect of whatever interferes with their free action.

As regards the *diagnosis* of pericardial adhesions, we may remark, that no reliance can be placed upon the abrupt, jogging, tumbling, double motion of the heart, described as a sign by Dr. Hope; for it is the result simply of irregularity of the heart's rhythm. Nor is it necessary to do more than merely refer to depression and pulsation at the epigas-

trium during the heart's systole, which have been suggested as signs of pericardial adhesions. An irregular, small, unequal pulse, præcordial anxiety, dyspnœa, tendency to fainting, dropsical swellings, and many other like symptoms, though present when pericardial adhesions exist, are symptoms of many other conditions of the heart, and therefore not distinctive of this one. Certainly, wherever such symptoms remain, after an attack of pericarditis, or arise consequent to it, and no endocardial murmur is present at the same time, we may very fairly consider them as caused by pericardial adhesions.

A few years ago, Skoda pointed out signs which he considered indicative of pericardial adhesions; and these we will shortly mention. It must be premised, that where these signs exist, union is supposed to have taken place, not only between the pericardial surfaces, but also between the external parts of the pericardium, the neighbouring pleural surfaces, and the walls of the thorax.

His diagnosis is based upon the signs which show that the heart's apex is drawn *upwards and to the right* during the systole, being prevented from moving downwards and to the left.

The heart's apex gives no systolic beat: it is either not to be felt, or seems to cause a shock during the diastole.

During the systole, depressions are visible in

the intercostal space corresponding to the apex, and frequently in one or more spaces above it.

Systolic retraction of the left intercostal spaces, does not of itself enable us to diagnose adherent pericardium: it must also be shown, that, simultaneously with the retraction, the heart's apex is nowhere urged against the thoracic walls.

The systolic retraction of the lower half of the sternum, is a certain sign of adhesion of the heart and pericardium, and of their attachment to the vertebral column.

The limits of dull percussion remain the same during inspiration and expiration.

The nature of the sounds of the heart give us no aid in the diagnosis.

Of the *treatment* of pericardial adhesions there is nothing to be said: the process whereby they are brought about is not amenable to treatment. All that we can do is, to administer to the secondary consequences which result from the pericardial adhesions.

CHAPTER VII.

ENDOCARDITIS.—PATHOLOGY.

Inflammation of the Endocardium.—Fibrinous Coagula in the Heart.—Valvular Diseases.—Chronic Valvular Diseases.—Fibroid, Atheromatous, and Calcareous Degenerations.—Effects of the Inflammation on the Valves.

Inflammation of the Endocardium.—By the term endocarditis is understood, inflammation of the whole, or of a part, of the membrane which lines the valves and the internal surface of the heart. The inflammation is of especial interest to us, in consequence of the injuries which it inflicts upon the valvular apparatus. From these injuries very serious results (the existence of which is indicated to us by alterations in the sounds of the heart) almost constantly ensue.

I shall, in the first instance, describe generally the characters of the endocardial inflammation, and hereafter speak more especially of the particular valvular lesions which result from it.

For a proper consideration of this subject, it is necessary to keep in view the anatomical nature of the endocardial membrane, and the peculiar acci-

dental circumstances which influence and attend its inflammation. With regard to its anatomy: the membrane consists of a layer of epithelium resting upon a fibrous tissue, which again is separated from the muscular structure, by a layer of elastic and cellular tissue; in this cellular tissue, blood-vessels ramify, by which the nutrition of the fibrous and epithelial layers is carried on; and from which, also, the pathological products of the inflammation are especially derived. This cellular layer, therefore, may be considered as the main seat of the inflammatory action, and the chief source of the exudations met with on the surface of the membrane, or between its layers.

The peculiar circumstances which attend the inflammation are, the unceasing motion of the part inflamed; and the action of the blood constantly flowing over, and immediately in contact with, the inflamed surface and its exudations.

The anatomical signs of endocarditis—excluding its effects upon the valves—are, in the majority of cases, obscure and ill-defined; even those which are most frequently found to be its representatives, namely, opacity and thickening of the membrane, may result from other processes, of a non-inflammatory nature.

The deep red colour, which the internal surface of the heart so frequently presents to the observer, was, once, erroneously considered as an invariable

sign of inflammation of the endocardium. The fact is, that it very rarely is so; it cannot, indeed, be considered as such, unless when manifestly the consequence of an injected state of the blood-vessels; but we seldom have an occasion of observing the redness which results from congestion of the blood-vessels; for the congestion occurs during the first stage of the inflammation, and at this stage of its progress, death does not frequently happen.

The redness observed is, in most cases, the consequence of imbibition, by the tissues, of the colouring matter of the blood, which has escaped from the vessels, in consequence of alterations which the constituents of the blood have undergone within the vessels. When the true inflammatory injection is present, it is generally accompanied by this red staining of the tissues, and thus may be almost entirely concealed by it.

The redness of congestion—the true inflammatory redness—is described by Rokitansky, as constantly presenting a pale, rose-red colour, the tint of it being subdued by the superficial layers of the endocardial membrane: it assumes a stripe-like, ramifying appearance, corresponding to that of the vessels, and has not the appearance of a general saturation of the tissues.

After the stage of congestion, follows that of exudation. In our study of this stage of the inflammation, we must be guided in part by analogy,

and in part by demonstrated facts. The ordinary products of inflammation are deposited either upon the surface of the membrane, or within its tissues, thereby giving rise to thickening and opacity of it; sometimes they form milky, whitish, opaque, ill-defined patches, which, at their circumference, become gradually confounded with the healthy surface of the membrane. The membrane at those spots, if examined at an early period of the inflammation, is found loose and softened; it is swollen and roughened; has lost its shining appearance of health; it may be readily torn off from the parts beneath, in the form of velvety, shaggy, membranous shreds. False membranous formations are not, however, ordinarily found on the endocardium; they only exist under very favourable circumstances, some especial causes hastening their production. The reason of this is, that the fibrinous and purulent products exuded on the surface of the endocardium are carried away, as soon as formed, by the current of blood, into the general circulation; and thus they not unfrequently give rise to secondary disorders in the lungs, the kidneys, the brain, and other organs of the body, sometimes affecting and altering even the constitution of the blood itself.

Fibrinous coagula are occasionally found upon the endocardium as results of its inflammation; but it is certain, that they are also sometimes formed there, not as the consequences of exudation from

the membrane, but independently of any inflammatory process; in fact, as true depositions of the fibrin of the blood which, through some special exciting cause, is attracted to the spot where the coagulum is found attached. It is very probable that a slight degree of roughness of the surface, caused by inflammatory exudation, may induce a ready deposition upon it of the fibrin of the blood; in which case the exudation will become, indirectly, a cause of the formation of fibrinous coagula. These fibrinous bodies, thus deposited on the endocardium, undergo subsequent changes, which are of particular interest in relation to their effects upon the valves; and of these we shall speak more particularly when treating of valvular diseases.

Occasionally, as a rare result of endocarditis, abscesses are formed in the substance of the heart, whereby the continuity of its surface is broken, and even the walls of the auricles, or of the ventricles, occasionally, perforated. The valves, and the tendons of the papillary muscles, are also not unfrequently ruptured, through the action of the inflammation, and the tendons of the papillary muscles and the valves themselves fused together, or with the neighbouring parts.

Endocarditis occurs much more frequently, at least its action is much more severe, in the left than it is in the right side of the heart. The resolution of the inflammation is seldom perfect; wherever

we are able to diagnose with tolerable surety the presence of the inflammation, we generally find that some consequences of the inflammation remain after the inflammatory symptoms have passed away. The most important of these consequences are, in the majority of instances, injuries of the valvular apparatus of the heart. We may here incidentally observe, that, in all cases, the only *certain* proof which we possess of the existence of endocarditis during life, is derived from the signs, which indicate that some mischief has befallen the valves of the heart. Before the discovery of auscultation, endocarditis was a disorder, whose presence it was not possible to determine during life.

Valvular Diseases.— Endocarditis, as already said, is especially of interest in connexion with *valvular diseases;* its chief effects, indeed, appear to fall upon the valvular apparatus; and when this is not injured by the inflammation, the consequences of endocarditis, as a rule, become of minor importance. When the endocardium is attacked by inflammation at other parts than where it covers the valves, it does so generally in the immediate neighbourhood of the valves, the disease being propagated thence to the surrounding parts. Theoretical reasons, derived from a consideration of the peculiar functions of the valves, and of the different anatomical relations of the endocardium, where it forms the valves, and where it lines the walls of the heart,

have been proposed, to explain the fact of this especial liability of the valves to disease; but it is not necessary to do more than allude to them here.

Modern pathology teaches us, that diseases of the valves of the heart are not the products solely of inflammatory action, but that they are also, and perhaps in the majority of cases (at least in advanced periods of life), the consequences of a slow degeneration of the textures which form them, resulting from perverted nutrition, or natural decay. When the disease is of inflammatory origin, the valvular lesion is, for the most part, rapidly produced, and its production coincides with a general febrile condition of the body, and occurs more particularly at the vigorous periods of life. This we infer from the fact of the frequency with which endocardial murmurs, or altered heart's sounds—the only true indicators we possess of valvular lesions—are observed to arise during the progress of certain inflammatory fevers, and to persist after febrile action has ceased. The products of such inflammatory action exuded on or into the valves, may, and doubtless do, undergo changes and degenerations; and some of these degenerations assimilate precisely to the products of those diseases, which we assume to be of slow chronic growth, and which result from a slow disintegration of the natural structures.

The chronic forms of valvular disease are met

with, particularly, in persons of advanced life, or of unhealthy, broken-down constitutions—in those in whom the nutritive force is readily perverted from its standard of health, and is apt to produce materials lower in the scale of organization than those proper to the part. The calcareous, fibroid, and cartilaginous deposits, so frequently observed in and upon the inner coats of the arteries, and upon the valves of the heart in the aged, are marked illustrations of this.

Here, however, we must observe, that though, for convenience in practice, we make these divisions of valvular diseases, yet their original nature is, frequently, involved in much uncertainty. Death, happily, does not often give us an occasion of observing them in their first stages; we see them, generally, only when the original appearances of the endocardial inflammation have already passed away; when the lesion has become chronic, and has undergone still further pathological changes. Non-inflammatory degenerative processes may be engrafted on those, which were originally the consequences of inflammation, and thus inflammatory exudations in the valvular tissues, become at length converted into calcareous masses. Again, inflammatory exudations, when they roughen the surfaces of the valves, attract to themselves the fibrin of the blood as it passes over them through the heart, and in this manner are produced those fibrinous fringes,

and so-called polypous growths, which are frequently met with on the surfaces of the valves.

There are also conditions of the blood in which there is a peculiar tendency to the ready deposition of its fibrin; and it is possible, therefore, that such fibrinous deposits upon the valves may occasionally form, independently of any inflammatory action whatever of the valves. From which it follows that, in many instances, it is difficult to decide, from the mere anatomical inspection of the part, as to the nature of the original disease; all we can do, in such cases, is to refer back, where this is possible, to their past history, and therein seek the solution of the difficulty.

Effects of the Inflammation on the Valves.— The following are the most important consequences which result from inflammation of the valves:— Deposits upon, and within, their tissue of inflammatory exudations, rendering them thicker and more opaque than natural; deposition upon them of fibrinous particles derived from the fibrin of the blood, forming the so-called vegetations; their softening, fissuring, ulceration, perforation, and rupture; adhesions of them between themselves, and to the walls of the heart, and of the vessels; their shortening, contraction, and shriveling up; and, in the case of the auriculo-ventricular valves, adhesions between, and contractions and rupture of, their tendinous cords.

Such are the general consequences which we may trace, more or less distinctly and immediately, to the inflammation. Some of these pathological conditions, as they become chronic, undergo still further changes, and so at length, as we have said, become confounded with diseases of a non-inflammatory origin—with atheromatous and calcareous degenerations of the valves.

Many of the diseases of the valves, which we call *chronic,* are obscure in their origin, and in their progress slow and insidious: their history does not justify us in ascribing them to an inflammatory origin, for their growth is unaccompanied by any of the ordinary symptoms of inflammation. It is only in their advanced stages, indeed, that they become known to us; and then, by the local signs and general symptoms to which they give rise in the economy. They destroy the integrity of the valvular structures, so that the valves imperfectly perform their office; and thus arise those serious disturbances and disorders of the economy, to which we apply generally the term of heart diseases.*

These chronic, latent diseases consist, for the most part, in degenerations of the tissues of the valves, and chiefly of its internal membrane; they

* It must be remembered, that such degenerations are rarely ever confined to the valves alone; they affect the vascular system generally, in a greater or less marked degree; and perhaps may, hereafter, be found to have very wide pathological relations.

present themselves to us under the form of abnormal depositions of fibrinous, atheromatous, and calcareous matters, upon or within the membrane. The character, extent, and form of the degeneration determine, in each particular case, the amount of injury which the function of the valve suffers.

But however slow and lingering these diseases may be, they are still progressive; the process of degeneration once commenced, its course seems, ever after, onwards. The fact of the existence of the new formations in the tissue of the valves, is a sure sign that the natural vigour of healthy nutrition, proper to the part, is lost or altered. And more than this: these fibroid, atheromatous, and calcareous degenerations, have a wider signification for the pathologist, than their merely local consideration gives them; they indicate that not here alone, but elsewhere, in all parts of the body, there exists, in like tissues, a like tendency to such depositions and degenerations, in a greater or less degree. In other words, they tend to show, that these are but the local products of some general disordered condition of the body; that the disease has, in fact, an existence anterior to that of the mere deposit, and is not the result of mere local inflammatory action, but an accidental manifestation of the natural decay of the tissues, or of some general deterioration of the forces, which preside over their innervation, or their nutrition. In no sense, has modern pathology been of

more service to medicine, than in bringing out to view the correlations of the different local manifestations of disease, and in demonstrating the true nature of these diseased states.

It is not possible for us, as we have already said, to trace out the complete progress of any one of these particular degenerations, in any given case. During life, its existence is not revealed to us, until such time as it has probably wrought irremediable damage to the structure of the valve, and it is, therefore, in the latter stages of the disease only, that its anatomical nature comes under our observation. Accident, however, sometimes gives us an opportunity of witnessing what we may consider to be the condition of these degenerations, at an early period of their progress.

The nature of the fibroid degeneration of the valves is indicated by the name it bears: it consists in a deposition of a low, imperfect form of fibrinous exudation into, upon, or beneath, the endocardial membrane. It appears probable, that the deposition is often associated with a condition of the body, in which there appears a tendency to the escape of fibrinous exudations into certain membranous tissues, in different parts of the body. The deposit causes a gradual condensation of the tissues of the membrane, which thus often attains a considerable thickness; and it also frequently gives a completely cartilaginous appearance to the membrane, in which

it is exuded. Rokitansky describes, with considerable minuteness, another form of fibrinous exudation, consisting of "an excessive formation and deposition of the lining membrane of the artery, derived from the mass of the blood, etc., etc.;" and this, he considers, is afterwards converted into atheroma.

These fibroid exudations, by their disintegration into fatty materials, form atheromatous degeneration of the valves. The atheromatous deposit, again, may soften and cause destruction of the tissues around them; when undergoing this change, it is found in different degrees of consistency, "from that of boiled egg to that of pus," forming generally a thick pultaceous mass. On the other hand, the atheroma may harden, by the deposition within it of salts of lime, and be gradually converted into a calcareous mass—ossification—constituting calcareous degeneration. Calcareous degeneration probably never occurs as a primary local alteration, but is secondary to some other; it derives its name from its chief constituent, phosphate of lime, which is deposited in an amorphous form, in the exudation. It is scarcely necessary to add, that it is not identical with true bone, as might be supposed from the term ossification, which is generally applied to it.

It may be gathered, from the outline here given, of these degenerations of the valvular structure,

that much obscurity still hangs over their history, their origin, their relation to each other, and their relation to that state of the system at large, which is associated with their appearance. It was once affirmed, that these diseases invariably had their source in local inflammatory action; but the reverse of this appears much more probable to us. In part, they seem to be associated with certain general diseased states of the body; in part, perhaps, they may be regarded as the ordinary attributes of advancing age,—as the natural (if such a term may be used) mode of decay of the tissues.

Hence they are to be considered, generally, as not of inflammatory origin; and for the reason, chiefly, that they present themselves to us unassociated with any previous history of inflammation; which could hardly happen, if they had really resulted from inflammation; for although the diagnosis of endocarditis must be doubtful, so long as the valvular apparatus remains capable of performing its office,* still the excited action of the heart, and the general fever, and the accidental complication of other co-existing disorders, and the local symptoms of pain, etc., rarely fail to give us notice, when inflammation has seized upon any part of the

* The student must be again reminded, that we have no other *certain* proof of the existence of endocarditis than abnormal bruits, which result from defective conditions of the valves.

heart, and so to excite a suspicion, at least, of the presence of the inflammation. And, then again, when it happens that the valves are injured by the endocarditis, symptoms of disturbance in the circulation arise, and continue ever after; for, as far as we know, it is only in very rare cases, that a valve, once seriously injured by inflammation, ever recovers its integrity. As a rule, we find, when we inquire into the history of cases of valvular diseases, which can be traced back, with tolerable certainty, to some inflammatory attack of the heart, that the subjects of these diseases "have never been themselves," since the time of the attack. The injury was effectual and permanent, and the time of its incidence definite and clearly marked.

But the history of the degenerations of the valvular apparatus is very different; their beginnings are not made manifest through their effects, and their presence becomes known to us, then for the first time, when they have, after long, slow, and insidious advances, effectually damaged the valves. The degenerations, moreover, as we have already hinted, seem to be the attendants of old age, and to be associated with ill conditions of the system; whilst the inflammatory disease rather attacks those of vigorous health, and at the prime periods of their life.

There is, however, in all this, nothing incompatible with the idea, that the products of inflammation

may themselves, eventually, undergo these degenerative actions. Exudations, once poured out from the vessels, become subjected to a variety of influences external to themselves; and their future condition of existence, and their metamorphoses, must necessarily depend, in some degree, upon the nature of those influences: the idiosyncracy of the individual, in a word, will give a direction to the changes, which affect the exudation. A purely fibrinous exudation may thus pass into ordinary induration, forming a permanent cartilaginous structure in the part affected, or it may degenerate into atheromatous and calcareous depositions, and so on.

Defects of the valves, we may here add, result also from original malformations of parts of the heart. Thus, there may be one valve only at the aortic opening; or two of the valves may adhere together, and so effect its closure; or the three valves may be present, one of them being very small, and in a rudimentary state. Defects of the pulmonary artery's valves are, generally, associated with other serious organic defects, or disorders, of the heart or great vessels, such as are incompatible with prolonged existence; they are, therefore, rarely met with, except in the very young. Defects of the aortic valves often exist independently of other lesions of parts, and are met with at adult periods of life.

CHAPTER VIII.

ENDOCARDITIS.—VALVULAR DISEASES.

Vegetations on the Valves of the Heart.—The Mode in which the Valves are Injured by the Deposits.—Secondary Deposits.—Insufficiency, and Obstructive Diseases of the Valves.—Pulmonary Symptoms.—Abdominal and other Symptoms.

Vegetations on the Valves of the Heart.—These abnormal products, — fibrinous concretions, — so frequently met with on the valves and on the neighbouring surfaces of the endocardium, present themselves to us under a variety of forms: differing much in size, in number, in consistence, and in their general external appearances. At one time, they exist as minute granulations,—sometimes so minute as to be readily overlooked, if not carefully sought for, clustered together around the bases, or at the edges of the valves, or upon particular parts of their surfaces; then again, they are found fringing the borders of the valves, and projecting from between them, like cauliflower excrescences; or again, they have a more isolated character, and are attached, like polypoid bodies, to the valves, in size varying from that of a pea to that of a hazel

nut, and having, sometimes, thick, club-shaped, free extremities, which pass between and separate the borders of the valves, so as to prevent them coming into proper contact.

The consistence and the colour of these bodies depend upon their age; when recent, they bear a close resemblance to freshly coagulated fibrin, which they indeed really are; they are soft, easily broken down, and readily removed from their points of attachment. When of older date, they are firmer, denser, and more closely connected with the endocardium, requiring some force to tear them from their connexions to it; and when separated, they leave the surface of the endocardium roughened, deprived of its epithelium, and swollen in appearance. Their colour is at first reddish; but afterwards, as they become denser, they take a paler cast, the hæmatine of the blood being gradually removed from them.

These vegetations are found attached, under different forms, to all parts of the endocardium, and are sometimes spread, like small warty granulations, or condylomata, over a considerable portion of its surface; but their most common and ordinary situations are upon the valves and their tendons. They are almost invariably found fixed to those surfaces of the valves, against which the onward current of the blood more particularly impinges; their free and pendulous parts lying in the direc-

tion of the current. They are frequently met with on the valves of the left, and but rarely on the valves of the right, side of the heart.

These fibrinous vegetations appear, in the great majority of cases, to be the results of endocardial inflammation, and to be closely connected with alterations of texture caused by inflammation. The amount and extent of the deposition, in such case, bears a close relation to the intensity and character of the inflammation. Their mode of formation is not the same in all cases. They may result, simply, from the inflammatory exudation poured out on the surface of the membrane; or,—and this appears the most ordinary mode of their production,—the vegetations are formed, in part, by this inflammatory exudation, and, in part, by the fibrin of the blood circulating through the heart, which is attracted to, and deposited upon, the inflammatory exudation, or upon the roughened surface of the inflamed membrane. It is certain, that the blood, under particular conditions, has a tendency, of readily separating from it, its fibrinous constituents, and that those conditions obtain, especially, in the diseases which give rise to endocarditis. Whether the fibrin of the blood is ever deposited directly upon the valves, so as to form vegetations, independently of any co-existing inflammation of the endocardial membrane, appears doubtful. The frequent difficulty, there is, in ascertaining the presence

of the endocarditis; and the fact, that it may entirely disappear, and leave no trace behind it, and that we can, in most cases, trace the vegetations to an inflammation of the endocardium,—tend to lead us to an opposite conclusion.

Cases of endocarditis have been observed, which seem to justify the opinion, that fibrinous vegetations may, under favourable conditions, be entirely removed, and leave no trace of their existence behind them; their removal being effected by the disintegrating action of the blood, which flows over them, and by absorption at the points of their attachment. But such cases must be rare. More generally the vegetations remain, and undergo certain metamorphoses; their fluid portions are absorbed, they diminish in size, shrivel up, and become denser, and even take a cartilaginous character. They, perhaps, also undergo atheromatous and calcareous degenerations. When the fibrinous exudations have been very minute, they may, in consequence of having undergone absorption, leave no other trace, than that of a slight thickening of the membrane, at the part where they were situated, either along the base, or at the edge of the valve.

The mode in which the Valves are injured by the Deposit.—It is not necessary for me to describe here, at length, the manner in which the valves are rendered imperfect, through the agency of these pathological changes. We have seen, that these

changes are varied in their nature, and varied also is the manner in which they destroy the integrity of the valvular structures. But whatever be the nature of the disease, and whatever its mode of action, the consequences, in so far as they are of importance to the physician, are in all cases precisely the same; and those consequences are, interference with the proper performance of the functions of the valves.

When the existence of the valvular imperfection is determined by positive signs and symptoms, and we come practically to deal with it, we shall find the value of a right understanding of the pathology of these diseases. In accordance with the view above given of their nature, we shall regard the imperfection, either as the immediate result of some existing inflammation of the endocardium, or as the consequence of slow, chronic, degenerative processes. In the first case, our instant endeavours will be concentrated on the arresting of the local inflammation, on stopping its spread, and, if possible, on removing the damage it has already created; but if the general history and the present features of the case lead us to the conclusion, that the disease comes under the category of non-inflammatory and degenerative valvular diseases, then, as far as treatment, at least, is concerned, we shall regard the local disease as of secondary importance,—as an existing fact, indeed, beyond our

power to alter; and our attention will be directed towards the alleviation of the secondary diseases, which result from the valvular imperfection; and towards improving the general nutritive functions of the body, whereby we may hope to arrest the progress of the local degeneration.

A slight consideration, of the progress of these diseases, will enable us to comprehend the way in which they render the action of the valves imperfect. Inflammation softens the valves, partially destroys their texture, and renders them liable to ruptures and aneurismal bulgings. Its exudations into the textures of the valves give rise to fibrinous and cartilaginous formations, whereby they are rendered thicker, and less pliable and elastic than natural; thence, also, arise contractions of the valves, and of their orifices, and adhesions between themselves and the cardiac walls. The fibrinous vegetations and polypoid growths, which form upon the surfaces of the valves, and around their tendons, project between them, mechanically preventing their coming in contact, and, in this manner, obstruct the free current of the blood. The varied forms, which atheromatous and calcareous degenerations cause the valves to assume, and the destructive tendency of these degenerations, our pathological museums abundantly show: their complete description would be impossible. These degenerations affect the valves in the most extreme degrees: they

may be represented, simply, by small specks of atheromatous or calcareous matters, scattered through the tissue; and they may be so abundantly deposited within the valves, as to convert them into solid, or semi-solid, structures. The mitral valves, for instance, may thus, and by the adhesion of their borders, become a solid funnel-shaped cone, projecting into the left ventricle, the opening being situated at the apex of the cone, and scarcely admitting the entrance of the point of the little finger: the aortic valves may likewise be so united together, and fixed, and "ossified," as to render the aortic orifice scarcely large enough to admit the passage of a quill.

These calcareous masses, again, deposited within the valves, not unfrequently perforate them, and project from their surfaces: their growth, under such circumstances, takes place in different directions, and sometimes causes the valves to assume the most fantastic shapes. Upon these projections, when any local intercurrent inflammation arises, and, perhaps, independently of any inflammation, fibrin from the blood current has a tendency to be deposited. Frequently, indeed, we meet with complications of valvular diseases, arising in the same case; one kind of disease being, as it were, grafted upon the other.

We can readily understand, also, that the disease, which affects the valves, may spread into, or arise

at the same time in, the endocardium around the base of the valves. In such case, the mischief is aggravated; because the induration and contraction of the parts around, assist in diminishing the valvular orifice. This continuity of diseased structure is often strikingly manifested, in the manner in which it is seen passing from the roots of the aortic valves, and spreading into the anterior portion of the mitral valve.

Diseases in the right side of the heart, of such a nature as to render the valves imperfect, are rare; where they exist, they will be found of the same character, as those, here described, affecting the left side.

The products of the endocardial inflammation are, under certain circumstances, carried away by the current of the blood which is continually passing through the heart, into the systemic and pulmonary circulations, and give rise to disturbances in different parts and organs of the body. Disorders of the blood, purulent infection, and diseases, and disturbances in the functions of different organs, may thus be originated. Particular attention, also, has been called to the effects, which result from the disintegration and separation of particles of the fibrinous vegetations, described above, as forming upon the surfaces of the valves and endocardium. Particles of fibrin, thus separated, are, it is supposed, conveyed by the blood into both the pulmonary and systemic circulations, and there produce special

phenomena. The loose manner, in which the fibrinous vegetations are frequently found attached to the endocardium after death, and the facility with which they are separated by the slightest pressure, readily explain how this may take place.

Different sized masses of fibrin are thus carried into the blood vessels, and blocking up, or obstructing the circulation, produce effects, corresponding to the amount of obstruction which they cause, and to the particular organ affected by the arrested circulation. Disintegrated portions of the vegetations, resulting from the softening and breaking down of the fibrin, are also carried into the circulation, in the shape of fine granular matter, which, mingling with the blood, may give rise to various disturbances of the system, and to those symptoms, in particular, which we ascribe to contamination of the blood.

When the fibrinous particles are detached from the left side of the heart, they may be arrested in any of the branches of the systemic arteries; the vessels, in which they are particularly arrested, are those of the brain, the spleen, and the kidney—organs, naturally, receiving large supplies of blood. The tissue of the lungs will, of course, be most immediately affected by the fibrinous particles, which happen to be detached from the right side of the heart; particles, thus separated, may be carried into the pulmonary artery and its branches, causing

coagula to form within them ; which coagula again, by their presence, occasion various secondary disorders.

The cerebral, pulmonary, and other disturbances, which are supposed to result from these sudden, and more or less extensive, arrestments of the pulmonary and systemic circulations, and the disorders excited in the blood by the admixture with it of softened, disintegrated, and decomposed fibrinous particles, have been carefully detailed by Dr. Kirkes (*Med. Chir. Trans.* vol. xxxv). The subject requires still further investigation. The presence of secondary deposits, also, justifies the idea, that purulent matter may be, occasionally, secreted from the endocardial surfaces, as a consequence of its inflammation.

Insufficiency and Obstructive Disease of the Valves.—When the different pathological conditions of the valves, which have been described above, have reached a certain stage of their progress, they occasion impediment to the circulation of the blood through the heart, and in two different ways. First, they prevent the valves duly falling together, and therefore render them incapable of closing their respective orifices; the consequence of which is, that the blood is permitted to regurgitate through them: thus, for instance, when the mitral valves are defective, it flows backwards into the left auricle, during the heart's systole; and when the aortic valves are defective, from the aorta into the left

ventricle. Secondly, they obstruct the onward current of blood, by narrowing, in various ways, the orifices of the heart, so that the blood can no longer pass freely between them.

Defective action of the valves may be referred to altered conditions of the valves themselves, of their tendons, of their papillary muscles, and also of the heart's muscular walls. Defects of the valves are occasioned in many various ways; thus they are produced through their rupture, and perforation; through the deposit, within them, of fibrinous, atheromatous, and calcareous matters, whereby contraction, hardening, thickening, and rolling up of their edges, is occasioned; and through the deposit, upon them, of fibrinous vegetations. The action of the valves, also, is rendered imperfect by contractions, rupture, adhesions, and thickenings of their tendinous chords; and by all those diseases, also, which interfere with the action of the papillary muscles, and muscles of the heart; by rupture of the papillary muscles, and by extensive dilatation of the ventricles. Generally speaking, several of these causes act, simultaneously, in producing the insufficiency.

The most common causes of *obstructive diseases* are, rigidity of the valves, and of the tissues around their base, arising from deposits within the endocardium and the valves; and coalescence of the valves to each other, and adhesions of them to the

walls of the heart, or in the case of the aortic or pulmonary artery's valves, to the walls of the vessels.

Signs and Symptoms of Valvular Diseases.—The general effects which immediately result from these valvular diseases, in so. far as we regard them in their mechanical character, are much the same, whether they arise from insufficiency, or from obstructive disease, of the valves. These effects are, in a word, impeded circulation of blood through the heart, and its various consequences. The degree of these effects, however, varies much, according to the particular valve which is diseased, and the nature and extent of the injury which it has undergone; thus, a permanent cardiac bruit may indicate organic disease of a valve, and yet so slight may the injury be, that, under ordinary circumstances, no symptom betrays the presence of the mischief to the subject of it. A long life, indeed, is occasionally found to be compatible with a certain degree of valvular disorganization, in cases where the will of the individual is strong enough, and his social position such as, to give him power, on all occasions, to shun scrupulously whatever may act as exciting causes to a renewal of the diseased action, which produced the original mischief. And then, again, on the other hand, injury of a valve—its rupture, for instance—may be great and sudden, and such as rapidly to extinguish life.

These extreme conditions of the disease are, how-

ever, rare; and in most cases, a variety of symptoms, more or less marked, in proportion, as we have said, to the nature and degree of the valvular disease, indicate to us the presence of an obstruction to the circulation of the blood through the heart. These symptoms are, in fact, the indicators of the secondary diseases or disorders, which manifest themselves in different organs and parts of the body, as consequences of the obstructed circulation. The importance of a right understanding of these disorders, and the proper appreciation of their exact meaning, may be gathered from the fact, that, in the majority of cases of heart diseases, they are the immediate causes of death, and the especial objects of medical treatment. These general disorders, also, often indicate to us, by their insignificance or severity, far more than do the local auscultatory signs, the importance of the valvular disease.

We shall here, therefore, briefly enumerate the main general symptoms, which result from valvular diseases. These symptoms are manifested in the heart itself, in the lungs, the brain, the abdominal organs, and, generally, in the different parts and organs throughout the body.

Pulmonary Symptoms.—The intimate anatomical and functional relations, which exist between the lungs and the heart, explain why the immediate effects of an impeded circulation of blood through the left side of the heart should fall upon the lungs.

The blood no longer passes freely from these organs, along its wonted channels, into the left auricle of the heart, its passage being obstructed, either by the constricted valvular opening, or by the blood which regurgitates through the defective mitral, or defective aortic valves. The blood, thus obstructed in its course, accumulates in the lungs, and, then, these organs become congested, their function is deranged, and dyspnœa arises. Breathlessness is, in fact, ofttimes one of the first symptoms which indicate the presence of heart disease to the sub ject of it.

Congestions, pneumonia, bronchitis, so-called pulmonary apoplexy, œdema, emphysema, and their varied symptoms, are, all, the more or less frequent —immediate or secondary—results of valvular diseases of the heart; the extent of the particular disease, in any case, generally corresponding to the degree and amount of the valvular lesion. The disordered states and diseases of different organs, thus excited, have much in them which is peculiar. The bronchitis and pneumonia, for instance, have not the characters of (so called) idiopathic pneumonia and bronchitis; their origin, their development, and the course they run, are very different, and so likewise, is the nature of their inflammatory action. In many respects, we must regard them as efforts of nature to obtain relief by compensation; one organ, by increased activity, being called upon to

supply the defects of another. They indicate, moreover, to the physician, the particular direction which he should follow, in his treatment of the disordered states arising from cardiac diseases. The pulmonary hæmorrhage, the large abundance of bronchial secretion, the pulmonary œdema, and the serous effusions, which so often accompany these diseases, are all, natural efforts of the lungs to disembarrass themselves of the congestions which oppress their function. The suddenness with which congestions of the lungs, and even their consolidation, may occur in valvular diseases of the heart, revealing their presence in violent dyspnœa; and the rapidity with which, so long as the heart's action remains strong, they may disappear, under the influence of local depletion—strikingly illustrate their pathological nature.

Abdominal Symptoms.—But the respiratory are not the only organs affected by the heart disease. The abdominal organs, and the brain, likewise share in its consequences; indeed, there is no organ or part of the body which may not, in extreme cases, be brought, more or less, under the influence of the valvular disease.

Let us see how this happens. The pulmonary circulation being impeded, as above described, the blood cannot longer flow freely out of the right side of the heart, into and through the pulmonary artery; consequently, it accumulates in the right

ventricle and pulmonary artery, and thus presents an obstacle to the return of the blood from the venous system, through the venæ cavæ, into the heart. From these accumulations of blood, necessarily result congestions of blood, and their consequences, in those organs and parts to which the effects of such an obstacle are immediately communicated. The most important consequences, thence resulting, fall, in the abdomen, upon the liver, kidneys, and intestines: the large size which the liver sometimes, and rapidly, attains under these circumstances, is surprising; it may be felt extending low down into the abdomen, several inches beneath the edge of the ribs; and its decrease, under the treatment of the disease, may take place as rapidly, and be even marked out and followed by percussion, from day to day. The congestion of the kidneys may give rise to temporary albuminuria, which disappears when the congestion, which occasioned it, is removed. The stomach and intestines manifest various signs of disorders, resulting from impeded circulation: vomitings, hæmatemesis, and discharges of serum and blood from the bowels, are modes by which the oppressed circulation, in these cases, often finds relief.

When the disease of the heart is such, as to render the congested state of these organs a permanent condition, then, necessarily, other disorders must arise from these secondary disorders: digestion,

and nutrition, and absorption, as well as respiration, are deranged; and their permanent derangements entail conditions, incompatible with the due performance of their functions. They occasion dropsies, effusions into the serous cavities; and these, again, cause pressure, which, by its mechanical effects upon internal organs, tends to destroy life. The nutrition being disturbed, the blood is no longer duly supplied with its proper materials, and defective respiration prevents it undergoing aeration; so that not one, but all, the vital functions, are thus, directly or indirectly, disordered; each in its derangement tending to complicate the other, and to the increase of the general disorder, and so to the destruction of life.

The disturbances of the brain do not, generally, show themselves, until the valvular disease is much advanced and the circulation much embarrassed, except under the slight and passing forms of headaches and occasional giddiness. Of the more serious symptoms of cerebral disorders, of coma and convulsions, I shall have occasion to speak, more fully, hereafter.

CHAPTER IX.

THE SOUNDS OF THE HEART, THEIR CAUSES, ETC.

Character of the Sounds.—Variable in Health.—Causes of the Sounds.—Several Causes assist in their production.—First Sound.—Second Sound.—Modification of the heart's healthy Sounds.—Impulse of the Heart.

THE causes of the heart's normal, and of certain of its abnormal sounds, have a very intimate relation. The parts which, in their healthy state, occasion the former, are just those which, in their altered conditions, cause the latter sounds; and it is from these abnormal sounds, which may either partially supersede, or altogether replace, the healthy ones, that our diagnosis of the nature of many of the diseases of the heart is derived. The importance, therefore, of a correct understanding of the causes of its healthy sounds, becomes manifest. I shall, therefore, before entering upon the subject of endocardial murmurs, take a short review of the physiological history of the sounds of the heart, of their nature and their causes.

Causes of the Heart's Sounds.—It would be inconsistent with the objects of this treatise, to offer

any minute remarks concerning the causes which produce the normal sounds of the heart; but, at the same time, it is not possible to enter, successfully, on the consideration of its abnormal sounds, without dwelling, for a moment, on the sources of the sounds to which it gives rise during its healthy action.

Two sounds, the second rapidly succeeding the first, are heard, when the ear is placed over the præcordial region; scarcely any appreciable interval occurs between the first and the second sound, but the second is followed by a distinct pause. The sounds and the pause occupy the interval of time, which elapses, between the beginning of one beat of the heart, and the instant which immediately precedes the commencement of the next following beat. They correspond therefore to one pulsation of the heart. The *first sound* is that which is heard, very nearly synchronously, both with the impulse of the heart against the thoracic walls, and with the beat of the arteries in the neck and at the wrist; in character, it is dull and prolonged, and takes up (roughly speaking) about two-fifths of the whole interval, occupied by the two sounds and the pause. The systole of the ventricles, and the diastole of the auricles, take place during the period of this sound.

The *second sound*, which, as we have said, succeeds the first, without any well-marked intervening pause, is clear, well-defined, sharp, and of short

duration. It corresponds, in time, to a part of the ventricular diastole, and to the retraction of the heart's apex from the thoracic walls; and occupies about one-fifth, or rather more, of the entire interval before-mentioned. It is followed by the pause, which takes up about two-fifths, or rather less, of the whole interval; and during this pause, the auricular systole and the completion of the ventricular diastole take place. These divisions of the heart's action constitute, together, what is called its rhythmic movements. The second sound is, as a rule, heard loudest at that part of the thoracic walls which is nearest to the situation of the semilunar valves; the first sound, at that part against which the heart's impulse is felt to beat.

Sounds,* synchronous with and resembling these cardiac sounds, are heard, also, over the trunks of the great arteries in the neighbourhood of the heart.

The sounds of the heart are frequently found to vary in healthy individuals, in respect of their strength and clearness, in the extent to which they are heard over the thorax, and also with regard to the points of the thorax at which they may be heard loudest. In one person, we find them loud and ringing,—in another, scarcely perceptible; at

* As has been elsewhere observed, the word Sounds is used without any qualification, to indicate the healthy sounds of the heart. When its unhealthy sounds are referred to, they are spoken of as abnormal sounds, as murmurs, or as bruits.

one time, they are heard over every part of the thorax, and at another, scarcely reach beyond the cardiac region. They may be loud at the heart's apex, and weak at its base, and *vice versâ*. They vary, also, according to the age and sex of the individual, being clearer in women and children than in men; and they are affected by mental emotion. They are modified by the position of neighbouring organs; by displacements of the heart; by encroachment of the lungs over the organ, as in emphysema; by pericardial effusions; by a distended stomach; by tubercular diseases of the lungs, etc.

A consideration of these incidental modifications of the nature of the heart's sounds, which occur while the organ is healthy, will be found of the greatest importance in relation to the diagnosis of its diseased conditions.

Causes of the Heart's Sounds.—It may be observed, *in limine*, that science does not yet enable us to give a complete history of the causes of the sounds of the heart. The subject is one about which different observers still entertain very different ideas. Upon some, however, of its most important particulars, there is, happily, a pretty general coincidence in opinion. We may add (as in accordance with what we have said of the relations which exist between the causes of the normal and the abnormal sounds of the heart), that neither can a completely satisfactory explanation be given

of the causes which give rise to all its different abnormal sounds.

It will assist us much, in considering the sources whence the heart's sounds arise, to keep in view the chief physiological facts which attend, and are coincident in time with, the periods at which the two sounds occur. It must here be premised, that I accept, as indisputable, the proposition that the first sound coincides with the impulse of the heart; and that, when I speak of sounds of the heart, I include the two sounds, wherever audible over the præcordial region, and consequently those which proceed from the aorta and pulmonary artery.

The following are the phenomena, which are associated with the heart's action, during the period of the first sound:

1st. The falling together, and the tension, of the mitral and tricuspid valves.

2nd. The muscular contraction of the ventricles.

3rd. The impulse of the heart against the walls of the thorax.

4th. The rush of blood, forced by the ventricular contractions, through the orifices of the aorta and the pulmonary artery.

5th. The falling back of both sets of semilunar valves, against the walls of the aorta and pulmonary artery.

6th. The sudden distension of the aorta and pulmonary artery.

An *à priori* consideration of the nature of these different acts, which occur during the period of the first sound of the heart, naturally leads us to the conclusion, that there is not one of them, which is not capable of giving rise to some particular sound; and it so happens, indeed, that each one of them has, at different times, been adopted or suggested by writers, as a cause more or less efficient in the production of the first sound. A later and more extended observation, however, tends to show, that we must not accept any of these supposed causes of the heart's sounds in an exclusive and dogmatic matter. The most reasonable conclusion, indeed, appears to be, that the sounds of the heart are not the products of any one particular act, but that they are the resultants of many acts.

The reasons which justify such a conclusion appear to me unanswerable; they accord with our physiological knowledge, and they are corroborated by our clinical experience. It might be asked, indeed, How is it possible, that any one of these acts, above related, can be accomplished, without giving rise to audible vibrations? Can membranes and chords be suddenly and forcibly stretched, and remain toneless? Peculiar vibrations — *le bruit musculaire* — as we know, attend muscular contractions; how, then, can that powerful muscular organ, the heart, contract, and its muscular sound remain inaudible? Then again, can such a blow as

the impulse of the heart against the thoracic walls, during the heart's systole, less fail to produce an audible sound? The effects produced by the impulse may be judged of in the following way: let any one place his hand over the end of a stethoscope, when it is applied to the ear, and then tap upon the back of his hand, and he will find that, however gently he percusses, the contact of the finger will still be distinctly heard. And lastly, can fluids be forcibly driven through orifices smaller than the chambers out of which they are propelled? Can elastic membranous walls, such as those of the aorta and pulmonary artery, be suddenly distended, —and yet excite no audible vibrations?

These questions seem to admit of but one reply; the conclusion to which they inevitably lead being, that several causes concur in the production of the first sound. The results of clinical observation also bring us to the same conclusion. Thus, for example, we find that the sound may be altered in its nature; that it may have lost what it possessed of a sharp, valvular character, and have gained in its dull, rustling, muscular character; and that it may be altogether superseded and replaced by a bruit; or if the bruit be not loud, that it may be heard in combination with the bruit. The sound, again, may be reduplicated, as though there were a want of synchroniety in the acts which compose it. It may be added, that the beginning of the impulse of

the heart, and of the first sound, are not exactly coincident in time; the latter somewhat preceding the former.

It must be remembered, that when we are speaking of sounds of the heart, we are referring to sounds which, whatever their cause, have a double origin: there are two ventricles, two sets of valves, two great arterial trunks,—all of which act alike and simultaneously; so that, whatever sound is produced, either by them or by the currents of blood passing through the heart, must have a double, though a similar origin.

The sounds, also, heard over the præcordial region, and, as they are ordinarily called, cardiac sounds, arise not only in the ventricles, but also in the aorta and pulmonary artery. This is a very important fact; and it was first clearly laid down and illustrated by Skoda. He tells us, that "the ventricles, the aorta, and pulmonary artery, severally contribute to the production, both of the first and of the second sound of the heart." As proof of this statement, he calls attention to the following facts: the first sound, as heard over the base of the heart,—that is, near the roots of the aorta and pulmonary artery,—is frequently less marked than the second sound; whilst, on the other hand, at the heart's apex, the first sound is the most accentuated and prolonged. The sounds heard over the apex, that is, over the left ventricle, often differ materially in

strength, pitch, and clearness, from those heard over the lower sternum, that is, over the right ventricle. The sounds heard about the middle of the sternum, and at its right border, that is, over the aorta, often differ markedly from those heard to the left of the sternum, and on the same level, that is, over the pulmonary artery; and this is particularly the case in certain diseased conditions of the heart, when the passage of the blood through the pulmonary artery is obstructed.

Again, cases frequently occur, in which the sounds of the heart are plainly audible over the right ventricle, over the aorta and pulmonary artery at the base of the heart, whilst at the apex they are altogether replaced by a double bruit. A bruit, also, may be heard over the right ventricle, and yet both sounds be clear and good over the apex and base of the heart. And it very frequently happens, that both sounds may be audible over the right and left ventricles and the pulmonary artery, and a double or single bruit, at the same time, be heard along the aorta.

These and many other similar facts tend, evidently, to show the correctness of the important position maintained by Skoda, namely, that the ventricles, the pulmonary artery, and the aorta, severally assist in the production of the heart's sounds; and they also shew, that when we speak of the first sound heard over the aorta and pul-

monary artery, and of the first sound heard over the apex and the right ventricle of the heart, we are not speaking of one and the same sound, arising at one point only, and spreading from thence as from a centre. The first sound heard over the aorta must be attributed to those efficient causes of sound, which are in action nearest to the point where it is heard; and hence, the impulse of the heart may have little to do with its production, while the sudden distension of the coats of the aorta may be its chief source. And again, conversely, at the apex of the heart the impulse may count for much, and the distension of the aortic coats for little, in the sound's production. These facts may be readily applied in other instances; and their consideration is of great value in the matter of diagnosis.

When we consider these facts, and at the same time remember the number of physiological acts, already related, which take place in coincidence with the sounds, we may very safely admit, that the heart's sounds have a complicated origin. It is quite consistent, however, with this view, that *one* of the causes should have a prominent share in the production of the sound; and we find, upon a reviewal of the opinions held by different observers on the subject, that, for the most part, they concur in this, namely, that in some way or other, the valves of the heart, more than other parts of it, are concerned in the production of its sounds—that

to the valves, in fact, must be attributed the chief share in their production. This view is in accordance with the main characteristics of the sounds, with the results of experiments, and is strongly corroborated by this capital fact, derived from clinical observation, namely, that alterations of sounds are almost invariably associated with alterations of the structure of the valves. Some writers have even gone so far as to consider the sounds as altogether valvular in their origin.

We find, in fact, that when, for instance, the mitral or tricuspid valves are defective, a distinct first sound is rarely heard over the ventricle, whose valves are defective; the sound being, in such case, usually replaced by a murmur; it is still audible, however, over the aorta and pulmonary artery, and even over the ventricle whose valves are healthy.

The method, thus pursued by Skoda, in analysing the nature of the sounds heard over the præcordial region, is of every value in assisting our judgment in this matter, and as a means also of correcting and guiding our diagnosis of heart diseases. When again, for example, the sounds are distinct and clear over the left ventricle and the pulmonary artery, but are replaced by a double bruit over the aorta, we are certainly justified in the conclusion, that, at all events, the sounds heard did not arise in the aorta; and when they are replaced by a double bruit over the aorta and left ventricle, but

are clearly audible over the pulmonary artery, then the conclusion seems inevitable, that the sounds heard over the pulmonary artery arose in that artery itself.

It must be remembered, moreover, that the presence of normal sounds, as well as the absence of bruits, are, for the most part, requisite to indicate a sane condition of the different parts of the heart. Absence of both sounds and bruits, where other signs and symptoms of heart disease exist, generally indicates a very advanced stage of structural, and chiefly of valvular, changes.

Careful attention to the character of the heart's sounds—to their relative force and duration, as heard over its different parts both in health and in disease, may perhaps lead us, by their comparison, to a better knowledge of some of those morbid states of the heart—its fatty degeneration, for instance—whose physical diagnosis is attended with great difficulty during life.

The different reasons here given, seem clearly to lead to the conclusions, that each of the acts recited above, take a share in the formation of the first sound of the heart; that the sound is chiefly valvular; and that the falling together and tension of the mitral and tricuspid valves, play the most prominent part in its production. The exact share which is to be ascribed to the other assigned causes, it is impossible, in the present state of our know-

ledge on the subject, to do more than conjecture. We will, however, briefly sum up the reasons which force us to ascribe to each of them an agency in its production.

The closure of the auriculo-ventricular valves must, of necessity, be rapid and sudden; but the first sound does not, throughout its whole duration, bear the character of a sound derived from a tense vibrating membrane. It is prolonged, and, in part, heavy, dull, and muffled. And then again, the presence of a bruit coincident with the heart's systole, does not, invariably, supersede the first sound; for it sometimes happens, that the bruit and the sound are both heard together. Neither are the different acts referred to perfectly simultaneous; the closure of the valves must necessarily precede both the expulsion of the blood from, and the complete contraction of, the ventricles. The expulsion of the blood, too, is not an instantaneous act; the rush of it through the arterial orifices of the heart, though rapidly accomplished, must be, in a restricted sense, a continuous act. The vibration, again, of the coats of the aorta and pulmonary artery, resulting from their distension by the blood forced into them from the ventricles, is manifestly excited subsequently to the act last mentioned. Hence then, it appears, that though we commonly speak of the different phenomena which are concerned in, and connected with, the systole of the heart, as occurring simulta-

neously, yet they in reality follow, by however short an interval, the one upon the other. It is requisite for us to remember this, while we are considering, theoretically, the causes of the heart's sounds; for the knowledge of the fact enables us to explain many phenomena connected with the first sound of the heart, which would be incomprehensible to us, if we regarded it simply as the product of the closure of the auriculo-ventricular valves, or as the product of its impulse.

To render this view of the nature of the first sound more striking, let us compare the characters of it with those of the second sound, and we shall see how our opinion as to its nature is strengthened by the comparison. The second sound is short, and sharp, and even ringing, and, in all probability, is entirely valvular in its origin; resulting, in fact, from the flapping together, and the sudden tension, of the semilunar valves of the aorta and pulmonary artery. It differs very considerably from the first sound, in whose formation so many acts seem to concur, and, therefore, corroborates the conclusion, that many acts,—namely, the distension of the auriculo-ventricular valves, the impulse of the heart against the thoracic walls, its muscular contractions, the rush of blood through the arterial orifices, and the distension of the aorta and pulmonary artery,—that all of these give rise to audible vibrations, and thus form part of that sound which

attends the systole of the heart, and is heard over the præcordial region.

The Second Sound of the Heart.—The clear and uniform character of the heart's second sound, and the physiological acts which are associated with it, indicate, that its causes are of a much less complicated nature than those of the first sound—a conclusion which is strongly supported both by experiment and clinical observation.

The chief acts, which occur during the period of the second sound, are the following:

1. The valves of the aorta and pulmonary artery are suddenly closed, and rendered tense.

2. The distended coats of the pulmonary artery and the aorta contract on their contents.

3. The auriculo-ventricular valves separate, and fall back towards the walls of the ventricles.

4. The first part of the ventricular diastole takes place, and the blood passes into the ventricles from the auricles.

"The second sound heard over the base of the heart,"—*i.e.*, over the aorta and pulmonary artery,—says Skoda, "is undoubtedly produced by the shock which results from the regurgitation of the blood towards the semilunar valves, and which takes place during the ventricular diastole. The blood, which passes into the arteries during the heart's systole, is subjected to compression by the elasticity of their coats, and when the forcing power

of the heart ceases, is, of necessity, driven back towards the heart.

"The reflux of the blood is suddenly arrested by the semilunar valves, and the impulse which they receive in consequence, is communicated to the walls of the vessels; the sound resulting is not only audible over the aorta and pulmonary artery, but is frequently communicated along the carotid and subclavian arteries, and this, too, even in cases where the coats of the aorta have lost the conditions necessary for the production of sound. The correctness of this explanation of the cause of the second sound, heard over the aorta and pulmonary artery, is placed beyond doubt by physiological and pathological observations: the sound does not appear to have any other cause for its production."

This opinion is confirmed both by experiment and clinical observation; it has been shown, indeed, by experiment, that when the semilunar valves are held open during the ventricular diastole, the second sound ceases, and in its place is heard a bruit. The same fact is constantly matter of clinical observation, in cases where the aortic valves are rendered incompetent by disease. We find, also, that the second sound of the pulmonary artery is often remarkably loud, when the passage of the blood through its branches is impeded; as in the case, for instance, where disease of the mitral valves prevents the free return of the blood from the

lungs into the heart. And we can readily explain how this happens, if we take it for granted, that the second sound results from the act of closure of the semilunar valves. The blood, not being able to pass freely along the branches of the pulmonary artery, distends its coats; and their inordinate distension reacts, during the ventricular diastole, upon the blood within them, and is communicated to the valves, which are thus subjected to an unwonted degree of pressure at the moment of their closure; from this extra pressure, necessarily, results increase in loudness of the membranous vibrations, and therefore of the second sound.

It is more difficult to explain the cause or causes of the second sound, heard over the ventricles. *A priori*, indeed, we might conjecture, that vibrations, excited in the semilunar valves of the aorta and pulmonary artery, would be communicated to the ventricles; and such, indeed, appears in many cases to be the origin of the sound. But this explanation is not sufficient to account for the sound in every instance; thus, for example, it occasionally happens, that the second sound is heard very loud and clear at the apex, while at the same time it is scarcely audible at the base, of the heart; and then again, it may sometimes be distinctly heard at the apex, when the aortic valves are defective and a regurgitant murmur is actually audible over them. Skoda mentions a case of defective aortic valves,

where the second sound was heard louder over the apex of the heart, than at any other part of the præcordial region. Such cases are exceptional, but they naturally suggest the idea of the possible existence of some other cause or causes of the second sound, besides that of the vibrations arising from the sudden tension of the semilunar valves. Several other causes have, indeed, been at different times proposed, as more or less effective in the production of the second sound; as, for example, the falling back of the auriculo-ventricular valves against the walls of the ventricles; the flow of the blood over the fleshy columns of the ventricles of the heart during its diastole; and a diastolic impulse. The value of their agency, however, in forming the sound is very uncertain, and, in the present state of our knowledge, they can only be received as suggestions, awaiting further explanations.

In some of the cases, where the second sound is heard over the apex, when the aortic valves are defective, it is not improbable that it may be communicated from the pulmonary artery, whose second sound is, under such circumstances, often much louder than natural. The more clear and flapping the character of the second sound is, as heard over the ventricles, the greater is the probability that the sound is of valvular origin.

Varieties in the Characters of the Heart's Healthy Sounds.—It has been already observed,

that considerable differences occur in the characters of the sounds of the heart, as heard even in healthy individuals; and, as it happens that some of these variations become, under certain circumstances, indicative of structural diseases of the organ, it follows that we must be cautious in judging of such modified sounds, either as signs of health or as signs of disease. We must judge of them, in fact, only with the assistance, and by the light of, other diagnostic signs. It is not possible to give any distinct account of the healthy modifications of the heart's sounds; they are very numerous, and the conditions which they indicate are, for the most part, very obscure.

When the first sound, heard over the ventricles, has a clear, distinct, flapping character, we may generally predicate a healthy condition of the auriculo-ventricular valves, their large size, and fineness of their structure: the clear, ringing character of the sound is lost, when these valves are contracted, or their borders thickened.

The reduplication of the first sound, which is occasionally heard over the ventricles, is difficult of explanation; it has been attributed to a want of synchroneity in the contraction of the two ventricles, and to irregularities in the time of closure of the auriculo-ventricular valves. The second sound, heard over the ventricles and over the pulmonary artery and aorta, is also occasionally broken

and interrupted; a condition of it which may be ascribed to irregular closure of the semilunar valves.

Many other modifications, in the characters of the heart's sounds, are frequently met with, but in the present state of our knowledge no practical signification can be attached to them. The two sounds are occasionally found to vary much in their duration, and so also are the intervals of silence, which separate them. The sounds, moreover, may be of equal length, and of equal strength, over the ventricles, and over the arteries; the interval, also, between the first and second sounds, may be so short, as to be quite inappreciable; or, on the other hand, it may be so prolonged as to equal the natural interval, which succeeds the second sound.

Impulse of the Heart.—The beat of the heart's apex against the thoracic walls, which occurs during the ventricular systole, and is in health felt about the cartilages of the fifth and sixth ribs, in the fifth intercostal space on the left side, is called its impulse.

The *situation* of the impulse varies somewhat, even in healthy individuals. The heart, being loosely attached in the thorax, follows to a certain extent the movements of the body, tending by its gravity towards that part of it, which happens to be most dependent. Its impulse, therefore, will be felt more to the right or the left of one particular point, according to the position of the individual

examined; and, for the same reason, it is more marked, when he assumes an upright position, or when he leans forward, or lies upon his face, than when he lies upon his back.

Certain diseases of the heart, and abnormal conditions, also, of the parts around it, alter considerably both the force of its impulse, and its point of contact with the thoracic walls; thus, for instance, hypertrophy and dilatation, and atrophy of the heart, pericardial effusions, pleuritic exudations, pneumothorax, thoracic aneurisms, emphysema of the lungs, mediastinal tumours, enlargement of the liver, and particularly of its left lobe, peritoneal effusions, a tympanitic condition of the stomach and intestines, and hypertrophy of the spleen—all modify, in a greater or less degree, the force and position of the impulse.

It is always of importance, in the investigation of diseases of the heart, that we should ascertain, if possible, the position of the heart's apex; when we have discovered its point of contact with the walls of the thorax, we are enabled to judge, to a certain extent, of the relative position of the other parts of the organ. An altered position of the impulse, when discovered, is also a valuable diagnostic sign, in assisting us to determine the nature of the disease, which has displaced the heart. A knowledge, therefore, of the part of the thorax, against which the heart's apex impinges, enables us to judge of

the condition both of the heart itself, and of the different parts and organs around it.

The *force* of the natural impulse varies much in different individuals. In large, broad-chested, muscular persons, it is not so readily felt, as in those in whom the soft parts of the chest are less developed; sometimes, indeed, it is scarcely perceptible, especially in the recumbent position. During inspiration, also, its force is lessened; indeed, the lungs of some persons, when fully distended, almost completely cover the heart, and then, of course, break the force of the impulse. The impulse is generally well marked in women and children; and is, in all cases, felt best when the body is inclined forwards, and during expiration,—that is, when the heart, through the contraction of the lungs, is permitted to come more immediately, and completely, in contact with the walls of the chest.

The force of the impulse is increased by muscular exertion, and by mental emotions.

Its force is likewise both increased, and weakened, by various abnormal conditions of the heart, and of parts external to it. Hypertrophy; hypertrophy with dilatation; irritation of the organ, resulting from general febrile action; pericarditis; pericardial adhesions; and all impediments to the pulmonary circulation, increase its force.

The natural force is diminished, as in all chronic and wasting diseases, in cases of pericardial effusions,

in emphysema of the lungs, and when the thoracic walls are œdematous.

When the heart's impulse is perceptible in several intercostal spaces,—and it never is so, when healthy, in more than two,—or for some considerable distance along one intercostal space, we may be certain that the heart is enlarged. A strong and violent beat of the heart does not necessarily indicate its hypertrophy; it frequently happens, indeed, on the contrary, that a hard, sharp, and quick impulse, concentrated at one spot, feeling sometimes like the blow of a hammer, indicates a weakness of the organ; such a beat is met with often in weak and excitable females, and is apt to mislead the inexperienced observer, as to the condition of the heart, which produces it. The impulse of an hypertrophied heart is not concentrated, and has more the character of a diffused heaving, than of a sudden blow; and when combined, as it generally is, with dilatation, it raises the thoracic walls and the head of the auscultator; the thoracic walls sinking back again, during the diastole of the heart.

Even an hypertrophied heart, however, does not necessarily produce a heaving impulse; it may, indeed, when quiescent, yield a perfectly normal impulse. The impulse of an hypertrophied heart is also modified by the effects of pericardial effusion; when this is great, the increased impulse is lost. Consequently, no positive conclusions as to the con-

dition of the heart can be drawn from an impulse which is weak or barely perceptible.

Nor even when the impulse communicates a strong concussion to the thoracic walls, without raising them, can we be sure that the heart is hypertrophied, for such a concussion may result simply from the violent action of a healthy heart: whether hypertrophy exists or not, must in such case be determined by percussion, and other signs and symptoms.

Cause of the Impulse of the Heart.—The cause of the heart's impulse has not yet been fully and satisfactorily explained.

The many different theories which have been, at one time or another, offered as explanatory of this phenomenon, suggest to us the idea, that here, as in the case of the heart's first sound, more than one cause must be operating in its production—an idea which is strengthened by a consideration of the complicated nature of the acts, which are associated with the impulse.

It would be beside my purpose, here, to examine these theories in detail; and I shall therefore content myself with giving a particular account of that explanation of the cause of the heart's impulse, which I consider to be, in all probability, one of the most efficient agents in its production. Other causes, there is good reason to believe, aid therein; and these will, presently, be briefly alluded to.

The theory which I speak of is that which is generally known as Gutbrod's theory.* It has received the sanction of, and has been adopted by, many leading authorities in the German schools of medicine.

In this country, the theory in question has been subjected to some little ridicule; one author has facetiously called it the "sky-rocket" theory, because the cause, (as explained by it,) which produces the heart's impulse, is supposed to be identical, in action and direction, with that which raises the sky rocket in the air. The theory, notwithstanding, appears likely to survive the ridicule which it has excited. Many excellent observers† have adopted it; and until a better one be found, I shall consider it the most satisfactory explanation, yet given, of the phenomenon in question.

The following is an explanation of the principles upon which the theory alluded to is founded. When fluids—aeriform or liquid, it matters not which—escape out of any cavity which contains them, either as the natural consequence of their gravity, or through pressure exerted upon the walls of the cavity from without, or from their own ex-

* It is only due to Dr. Alderson to state, that as long ago as 1825, he propounded this explanation of the heart's impulse, and gave illustrations of it, identical to those now offered by Gutbrod and Skoda. The fact, we may observe incidentally, corroborates the probable correctness of the theory.

† I may mention one, than whom no person is more capable of judging of its worth—Dr. H. Davies.

pansion within its walls—the equality of the pressure of the fluid upon the inner surface of the cavity is disturbed. The pressure ceases to act—is lost, at that point of the cavity whence the fluid escapes, but is still equally exerted over every other part, and therefore at that part which is opposite to the point whence the fluid escapes; consequently, a less degree of pressure is exerted on the side from which the fluid escapes, than on the opposite side. Now from this inequality of pressure, it necessarily results, that the body containing the fluid tends to move in a direction opposite to that of the escaping fluid.

This tendency is the force which produces the recoil of fire-arms, and which sets in motion what is called Barker's mill; and is the force which, according to the theory we are discussing, is supposed to produce the heart's impulse.

Now careful calculations have been made to explain the force with which the blood escapes from the aortic orifice; and it appears to result therefrom, that the pressure upon every square inch of the inner surface of the left ventricle amounts to about four pounds; and upon every square inch of the right ventricle to one pound and a half. Hence, at each systole, that point of the heart—that is, its apex—which is opposite to the orifices of the aorta and pulmonary artery, will be driven in a direction contrary to that of the currents of blood passing

along those vessels, with a force equal to five and a half pounds. The different directions of the two currents naturally tend to move the ventricles into two different axes; but as the muscular power of the left is much greater than that of the right ventricle, the combined movement, thus imparted to the heart, is chiefly in the direction of the stream which issues from the aortic orifice.*

But then comes the question: Is such a force sufficient to account for the impulse? Dr. Davies has answered this, by saying, that there is certainly nothing unreasonable in our believing, that such a force, so rapidly exerted as it is, may impress a movement of recoil on an organ like the heart, loosely suspended, and having an average weight of about ten ounces. This theory, moreover, is not only supported by physical facts, but is consistent, in many particulars, with the results of clinical observation. Certainly, this may be said of it, that no other theory explains so well as it, the cause of the heart's impulse.

Clinical observation shows us, that an hypertrophied and dilated heart produces a strong impulse, and such a state of the heart combines, in an unwonted degree, the conditions which, according to the theory of Gutbrod, are requisite for the production of its vigorous recoil, namely, great mus-

* All this has been very satisfactorily worked out by Dr. Davies. "Lectures", etc., p. 250.

cular power, and a large flow of fluid from its cavity. The heart's muscular contraction is stronger, and the quantity of blood discharged from it at each systole greater, than natural. The impulse is also stronger than natural in simple hypertrophy without dilatation. But when the ventricle is hypertrophied and contracted, the impulse is weaker, and it is weaker in proportion to the degree of contraction, that is, in proportion to the diminished quantity of blood, which the ventricle can contain.

There is not, however, a constant increase of the force of the impulse when the left ventricle is hypertrophied and dilated, if, at the same time, the right ventricle retains its natural capacity; for, in such case, the blood which is discharged from the right ventricle at each systole, and passes on into the left ventricle, is not sufficient in quantity to enable the left ventricle to produce such an impulse, as would result, if it had received a full supply of blood.

The impulse, also, we find clinically, is, in a certain degree, proportioned to the size of the arterial orifice; when, for instance, this is much contracted, so as, in fact, to impede considerably the current of blood through it, the ventricles, even though hypertrophied and dilated, cannot contract thoroughly; hence their systole, in such cases, is not complete, and, consequently, the impulse is limited; and sometimes, indeed, it is so weak as to be almost imperceptible.

Such are the reasons which induce me to adopt the recoil theory, as affording a better explanation, than any other yet proposed, of the cause of the heart's impulse; but, as before remarked, it is very possible that other causes may also aid in its production.

The impulse has been ascribed: To a tilting forward of the heart's apex, supposed to result from the spiral movement of the ventricle, which is produced, during its contraction, in consequence of the peculiar distribution of the muscles of the heart; and also: To the lengthening of the aorta and pulmonary artery by the columns of blood, which are forced into them, at each systole, whereby the heart is projected downwards and forwards.

It may be very difficult to ascribe to these supposed causes their due effect, as agents in the production of the impulse; but we may, at all events, fairly believe, that they must, in some way or other, act either in the production of, or in modifying, the impulsive movement of the heart against the thorax. It must be confessed, indeed, that there are certain phenomena which are not explicable by the recoil theory, such as: "the double and triple impulse associated with a single beat of the pulse, and with simultaneous contraction of both ventricles; weakness of the pulse, conjoined with strong impulse of the heart, when the condition of its valves is normal; the slow heaving of

the thoracic walls, unattended by concussion, and other extensive disturbances of the heart, occurring during its systole"—(Skoda). All which facts render it tolerably clear, that more than one cause is associating in the production of the impulse.

CHAPTER X.

ENDOCARDITIS.—VALVULAR DISEASES.

Physical signs of Valvular Diseases.—Cardiac and arterial bruits.—Causes of endocardial bruits.—The parts where the bruits arise.—Organic and inorganic bruits.—Inorganic arterial bruits.

Murmurs, Bruits—abnormal sounds—heard over the præcordial region, synchronously with the movements of the heart, and replacing or superseding its normal sounds, indicate, with some few exceptions, that certain portions of the heart, or of its great blood-vessels, have undergone organic changes. These bruits are most important diagnostic guides, leading us to a knowledge of the existence of most of the chief forms of heart diseases—namely, of pericarditis, of diseases of the valves, of the lining membrane of the heart, and of the aorta and the pulmonary artery.

I have elsewhere given an account of the abnormal sounds, which are produced by pericardial disease; the attention of the reader will, therefore, in this place, be confined to a consideration of the bruits which arise within the heart, the aorta, and

the pulmonary artery—that is, to endocardial and arterial murmurs.

Of these bruits or abnormal sounds, it may be said, generally, that they are the consequences, and therefore the indicators, of abnormal conditions of parts within the heart, of diseased states of its valves, of the valvular orifices, and of the great arteries, in the immediate neighbourhood of the heart. To this general rule, however, there are exceptions; for cases are not unfrequently met with in which, though loud bruits have been heard during life, no structural changes are found, after death, to explain their cause.

Endocardial bruits present themselves to us under a great variety of characters, and have been described as having the different peculiarities of blowing, sawing, whistling, purring, rasping, bellows-like, and other sounds. There is no doubt, that endocardial bruits resemble all these, and many other varieties of sounds; but there is no advantage derived from our attempting to define, too nicely, all their particular resemblances; the character of the murmur, in any given case, is not a fact of much importance to arrive at; and, indeed, it is, to a certain extent, in most cases rather arbitrarily defined, depending very much upon the ear of the observer, and upon the particular state of the heart —whether excited or quiescent—at the time of the stethoscopic examination. Different observers fre-

quently give different descriptions of the same murmur, even likening it to sounds which have opposite characters.* Besides, the same endocardial lesion will, under opposite conditions of the heart's action, produce very different kinds of bruits, at different periods, to the same observer. Thus, a gentle blowing murmur, heard during the heart's quiet action, may quickly pass into a rough sawing murmur, when any sudden increase of its action takes place. Or, again, it not unfrequently happens, that a loud murmur is heard over the heart, when acting violently, in the case where no murmur at all is heard during its tranquil movements.

"It is vain to attempt to distinguish too particularly," writes Skoka, "between the different kinds of murmurs; indeed, it matters little as respects any conclusions which may be deduced from our observations, whether the murmurs are of a blowing, sawing, or rasping character.

"We often find a bellows murmur suddenly converted into a sawing or rasping murmur, through accidental increase of the heart's action. Murmurs arising within the cavities of the heart, and the arterial murmurs, not only become louder, but they also acquire a rougher, sharper, and more acute character; the reverse of this happens when the

* A fact which it is always well to bear in mind, when reading the stethoscopic account given of any cardiac or pulmonary affection.

heart's action is weakened, the murmur becoming muffled and very indistinct, or disappearing altogether."

The nature and degree of the endocardial injury cannot, therefore, be learnt from any character of the bruits thence arising. All that, perhaps, can be fairly said of them, under this head, is, that the rougher and more loudly vibratile the bruits are, the more well-marked, in all probability, are the organic changes which have occasioned them; but this is by no means an unexceptional rule, for in some of the worst forms of endocardial disease, in their latter stages, the endocardial bruits disappear altogether. The all-important point in diagnosis is, first, to ascertain with certainty the existence of the bruit, and then, to discover the point within the heart, or great vessels, where it arises.

Causes of Endocardial Murmurs.—In discussing the causes of the healthy sounds of the heart, we found good reason to believe, that these sounds result, in great part, at least, from the vibrations excited through impulses imparted to the valves; and that they are, in some way or other, connected with the perfect action of the valves. This opinion is strongly corroborated by the fact, that endocardial bruits, which either entirely supersede, or partially replace, the normal sounds, are almost invariably found to be associated with some diseased or defective condition of the valves, or of the val-

vular orifices of the heart. When these parts are disorganized, the current of blood no longer runs its smooth, uninterrupted course through the heart; the stream is obstructed, or its direction is altered, so that it may even take a backward course, or have an eddying movement imparted to it. Vibrations are thus excited, through the unnatural friction of the blood against the walls of the ventricles, or of the great arterial trunks, or against the valves; and these unnatural vibrations occasion the murmurs.

The pathological changes, which give rise to these unnatural states of the current of blood within the heart and the great vessels, and consequently to its murmurs, are: Structural changes of the valves and valvular orifices (whatever the nature of the diseases producing them), whereby the flow of blood through them is obstructed; structural changes, which render the valves and orifices defective, and so permit of the regurgitation of blood through them; the presence of fibrinous and calcareous concretions upon the endocardial membrane in the neighbourhood of the valves, or upon those surfaces of the valves which meet directly the onward current of the blood; concretions, which form irregular projections upon the endocardial surfaces, but do not necessarily obstruct, in any material degree, the current of blood, and perhaps occasion bruits simply by producing mere ripples in its stream.

We have already observed, when describing the pathological anatomy of valvular diseases, that the valves of the right side of the heart are much less frequently affected than those of the left; and from this it necessarily follows, that valvular murmurs —the consequences of such diseases—are, comparatively, rarely met with here.* Practically, indeed, we almost entirely exclude from our consideration murmurs of the right side of the heart; their presence being so exceptional. Endocardial murmurs, consequently, are chiefly known to us as the results of diseased conditions of the aortic and mitral valves.

The part where the Murmur arises.—Very particular rules have been laid down, for our guidance, in the diagnosis of valvular diseases; and the signs minutely described, which should enable us to ascribe to each murmur its true seat of origin. In the main, we may certainly trust to the guidance of these rules, and in the great majority of cases, it is probable, that the careful observer will have little difficulty in fixing the true point of origin of the murmur heard; especially if the murmur be single, and the heart's action not very rapid. The diagnosis

* It must be understood, that in this statement, I speak only of such diseases as render the valves incompetent, or produce constriction of the valvular orifice. I believe, that the tricuspid valves, though they may be competent for their office, will rarely be found perfectly healthy in their structure, when the mitral valves are much diseased.

is greatly facilitated by the circumstance, that the tricuspid and pulmonary artery's valves are almost altogether excluded from our consideration, as being sources of the murmur.

It is only right, however, at once to warn the student, that these rules are not universally applicable, and that he will frequently meet with anomalies, in this part of the history of cardiac murmurs. This he may well expect to do, if he recollect what has been said respecting the causes of the sounds of the heart, and of the obscurity which, in many particulars, still envelopes the subject. If we are not able to give a complete account of the causes of the heart's healthy sounds, we may fairly argue, how can we expect to have possession of the complete history of its morbid sounds? Happily, such a knowledge is not essential for our guidance, in the practical application of our art to the treatment of cardiac diseases; and upon this fact I would lay especial stress at the present moment, when so much attention and minute consideration are given to the differential diagnosis of valvular diseases of the heart.

It is of infinitely more importance for us to ascertain the fact, that a murmur indicative of organic lesion of some of its parts, exists in the heart, than to know *where* the murmur takes its rise. The fact, of the existence of the lesion, being determined, our treatment of the whole train of general

symptoms which exist, either in coincidence with, or as the consequences of, the cardiac lesion, becomes clear in its indications; and it is in no way enlightened by a knowledge of the exact situation of the disease, which produces the symptoms. The reason of this becomes manifest, when we recollect, that in cases of chronic valvular diseases, we are not called upon to treat the valvular disease itself, but to deal with the symptoms, which are its natural consequences; and that in acute diseases of the valves,—associated with rheumatic fever, for example,—the situation of the disease in no way whatever guides or regulates our treatment of it.

Obstructive diseases of the valvular apparatus—constrictions of their orifices—as a rule, give rise to more severe general symptoms, or in other words, to greater impediments to the circulation of blood through the heart, than diseases where the valves are simply defective, that is, in which regurgitation takes place. But then, again, the valvular defect may be so great, as to render the regurgitation as much a source of impediment, as obstructive disease usually is. Hence, in such cases, where the grand fact—the impeded circulation—is manifest, the cause thereof is surely of very secondary importance. Whether the murmur be mitral or aortic, whether it be diastolic or systolic, can matter but little to the practitioner; its nature in such case neither influencing nor directing his treatment.

Not that I would desire, by anything here said, to undervalue the advantages of a careful differential diagnosis of endocardial diseases; the very habit of scrupulously investigating their physical characters, leads the student, necessarily, to a more intimate acquaintance with their nature. What I desire to inculcate is this, that a too curious investigation of physical signs is apt here, as in the case of all other diseases wherein physical diagnosis is concerned, to make the observer less attentive to the general signs and symptoms, than their real importance demands, and thus to induce him to give more weight to the physical signs, than they reasonably merit. The local physical sign is most valuable, as an indicator of the heart disease—but, in the *treatment* of the disease, it is, perhaps, the least important of the coexistent signs and symptoms. Thus, a murmur may demonstrate the presence of disease in the heart, and no other signs or symptoms exist to corroborate the fact; in such case, the value of the murmur is great, because it tells us that the heart is affected, and consequently enables us to anticipate and guard against future evils; but it points out no special treatment, and the injury it indicates (supposing all other signs and symptoms of cardiac disease absent), demands none. Again, the same murmur may exist, and, with it, a train of symptoms, all indicating great obstruction to the circulation; in this case also, the

value of the murmur is great, because it points out to us the original source of the disorders, but in itself it indicates no especial treatment; to these disorders, which result from the cardiac disease, our treatment is entirely directed.

Inorganic Murmurs.—Before proceeding to give the particular signs which, in general, enable us to decide as to the nature of any murmur, I would premise, that there are two great sources of error, which may mislead us, in judging of the value of cardiac murmurs as indicators of structural diseases of the heart. In the first place, murmurs, unassociated with any organic changes of parts, are frequently observed to arise in the heart and arteries; and secondly, most serious organic changes may be present, and yet give rise to no cardiac murmurs whatever; whence it follows, that neither is the absence of murmur any certain indication of absence of valvular disease, nor is the presence of murmur, *per se*, a sure sign of the existence of organic disease. These two facts are enough to show the student, how necessary it is, in all cases of heart affections, to seek elsewhere, as well as in the heart itself, for guides to a knowledge of their nature.

The true history of the first class of these affections, namely, of inorganic murmurs,—that is, of murmurs heard over and arising within the heart and the arterial trunks, without perceptible changes existing in the organic structure of these parts, has

yet to be written; their cause, or causes, are certainly only conjectural, at the present moment, and their points of origin are not yet determined.

It appears not improbable, that inorganic murmurs, heard over the heart synchronously with the ventricular systole, are in most, perhaps in all cases, not truly cardiac, but arterial or venous murmurs; in fact, that they arise either in the large venous trunks, in the aorta, the pulmonary artery, or at their arterial or venous orifices, and not within the heart itself; such seems to have been the opinion of Dr. Hope, and it is that of most writers on heart diseases. In the very great majority of cases, it is certain, that inorganic murmurs have the characters of systolic aortic murmurs; and only very exceptional instances give colour to the belief, that mitral systolic murmurs are of inorganic origin. My own belief is, that mitral systolic murmurs invariably indicate a defective condition of the mitral valves, and are never inorganic, and that, as before said, inorganic murmurs heard over the heart are either arterial or venous in their origin.

The following are the grounds upon which such belief is founded. Inorganic murmurs, heard at the præcordial region, have the characters, in general, of arterial murmurs,—they are soft and blowing. They exist, very frequently, in cases in which murmurs may, at the same time, be readily excited in some of the large vessels of the body,—in the cer-

vical vessels for instance,—and in which the blood itself appears to have undergone certain changes, either qualitative or quantitative; or in cases, where the proper relations which, in health, exist between the vessels and their contents, are lost. Inorganic murmurs, moreover, are always systolic, never diastolic: it is impossible, indeed, to conceive the existence of a diastolic cardiac murmur, without co-existing defective organization of the heart's valves or orifices. Again, a mitral systolic murmur is necessarily a regurgitant murmur, and a regurgitant murmur, unassociated with some defect of the mitral valves, is a condition, in the present state of our knowledge, scarcely comprehensible.* Mitral—so-called—inorganic murmurs, also, are admitted, by those who believe in their existence, to be of very rare occurrence, whilst arterial inorganic murmurs are notoriously common. Certainly, in the present incomplete state of our knowledge of the history of cardiac murmurs, it would

* That certain spasmodic contractions of the papillary muscles may, at times, occur, and so prevent the closure of these valves for a certain period, I can neither believe nor deny, having no data to guide me to an opinion on the subject; but, assuredly, there is nothing of a spasmodic character in the continuous, uninterrupted, smoothness of ordinary inorganic murmurs. The murmurs, which are occasionally heard in cases of chorea, and which apparently disappear after a time, are of too obscure a nature to be taken into consideration, as examples of inorganic mitral murmurs. At the same time, I feel the difficulties which surround this subject too fully, not to admit the possibility of the existence of such a murmur.

be most unwise to accept rare and doubtful cases, as demonstrative of particular facts. Moreover, these inorganic murmurs are rarely ever heard elsewhere than at the base of the heart; seldom do they reach below the nipple, and are, perhaps, never audible at the heart's apex.

For these reasons, I feel compelled to admit the conclusion, that all so-called inorganic cardiac murmurs heard over the heart, are of arterial origin, that is, arise either within or at the orifices of the aorta and pulmonary artery, or in the large venous trunks.

The *causes* of these inorganic murmurs are, at present, unknown, and all that can be said thereof is hypothetical. They arise in the course of various diseases, being very frequently present in chlorosis, in anæmia, and in diseases which produce an impoverished condition of the blood; they often arise also after great loss of blood. Occasionally, also, systolic basic murmurs are present in the acute stages of many inflammatory diseases—in typhus, in the exanthemata, in pneumonia, in acute rheumatism, and other disorders. The name of *hæmic* has been given to these murmurs, and with some show of reason, inasmuch as they appear, for the most part, to arise in connexion with those diseases in which there generally exists a manifest deviation from the healthy constitution of the blood. This, however, is mere hypothesis; for it has not yet

been shown what that peculiar condition of the blood is, from which the murmurs result; and moreover we know that the blood in chlorosis, and in acute pneumonia, and in typhus, must differ in each case very considerably in its constitution; and yet, under all these morbid states, as we have seen, the presence of cardiac murmurs is occasionally observed.

These kinds of murmurs, being very frequently found associated with a spanæmic condition of the blood, have been attributed to its watery constitution; the supposition being, that the particles of the blood, under such circumstances, are readily thrown into vibrations. This is pure supposition; and, indeed, we are continually meeting with cases of anæmia and chlorosis, in which no such murmurs exist,—so often, in fact, as to forbid us associating together the condition of the blood and the murmurs, as cause and effect.

It has also been supposed, and with much more appearance of probability, that the murmurs may result from the sudden and quickly performed ventricular contractions of the heart, which are so often observed in spanæmia; the violence and rapidity with which the blood is thus driven through the arterial orifices, producing the same results as would ensue from contraction of the orifices under ordinary conditions of the heart's action. This view derives some support from the fact, that anæ-

mic cardiac murmurs, heard when the heart's action is violent, frequently disappear when the action is tranquillized; and from this fact, also, that systolic cardiac murmurs appear, occasionally, to arise in healthy persons, simply as the consequence of violent excitement of the heart,—an explanation which may perhaps account for the murmur occasionally heard in the course of certain inflammatory disorders, as above mentioned.

It appears, indeed, that the most probable suggestion or plausible hypothesis that can be offered, as to the cause of these murmurs, is this: viz., that, through some reason or other, the due relation as regards pressure, which exists in health between the blood and the heart and vessels containing it, is lost. It cannot be denied, that whatever produces an alteration of the healthy pressure, or of the force of the contraction of the heart or of the arteries upon the blood, must necessarily occasion an impediment to its circulation; and that abnormal sounds may result from such impediments to the passage of the blood, through the orifices of the heart, and along the larger arteries, seems very probable. The causes which produce the undue relation above spoken of, we can but guess at; they may reside in the blood itself, or they may result from altered innervation of the heart, or of the coats of the arteries.

Inorganic Arterial Murmurs.—Murmurs are

frequently audible in the roots of the vessels near the heart, when the blood is in a spanæmic condition; in such states, indeed, it occasionally happens, that smaller arteries, even the radial, when gently pressed upon by the stethoscope, will give rise to loudish murmurs. These arterial murmurs correspond to the diastole of the vessel, that is, to the systole of the heart, and are never continuous, like venous murmurs; they are rendered more sharp and clear by the pressure of the stethoscope. Unlike organic murmurs, they are not localized to the neighbourhood of any particular part, but are audible generally over arteries in different parts of the body.

In considering these arterial bruits, it must be remembered, that murmurs may often be readily excited, by the pressure of the stethoscope, in the larger arteries,—in the subclavian and carotid arteries, for example,—in persons, who, to all appearance, are perfectly healthy; and the same effect is also not unfrequently produced, by so fixing the muscles of the arm and neck, as to create a pressure upon those vessels. Kiwisch asserts, that the so-called placental bruit of pregnant women arises in the epigastric arteries, and not in the placenta; and that it disappears when these arteries are compressed near to their origin.

It is often difficult, and sometimes impossible, to decide, in certain cases, whether the murmur heard

is of organic or inorganic origin, there being little or nothing in the character of the murmur itself, by which its nature can be determined. In the majority of cases, however, by attending to the facts above stated, we may come to a tolerably certain conclusion as to its nature; where there is difficulty in the diagnosis, there is, happily, rarely ever any in the treatment of the morbid condition which it indicates; for the accompanying symptoms, and the history of the case, generally indicate clearly enough what this should be.

CHAPTER XI.

ENDOCARDITIS.

Endocardial murmurs (organic).—Seat of the murmurs.—Systolic murmurs over the left ventricle of the Heart.—Regurgitant mitral murmur.—Diastolic mitral murmur.—Systolic aortic murmur.—Regurgitant aortic murmur.—Systolic tricuspid murmur.

ALL abnormal sounds, arising from disorganization or visible alteration of structure, which are heard over the cardiac region, and proceed from, or arise within, the heart itself, at its orifices, or within the trunks of the aorta or pulmonary artery, and are conveyed thence into the heart, are usually included under the head of organic endocardial murmurs.

There are certain general rules which may be laid down, respecting the diagnosis of these endocardial murmurs, and these we will now describe. We find that endocardial murmurs are loudest at those parts of the thoracic walls which are nearest to the points whence they take their origin; to this rule, however, there are exceptions, as in the case when the lungs, or some adventitious body,

intervene between the heart and the thoracic walls, and so naturally diminish the force of the sounds. The murmurs correspond very closely, in time, to the systole and diastole of the heart; but their rhythm often has no correspondence with the healthy rhythm of the heart's sounds: one murmur may be lengthened, and the natural intervals of repose between the heart's sounds prolonged or shortened. And then, again, the natural duration of the sounds may be totally perverted; the diastolic, in certain cases, becoming the longer, — a very prolonged murmur,—and the systolic the shorter, so as to render the interval between them imperceptible. They are conveyed most readily in the direction of the current of the blood which produces them: a systolic aortic murmur, for example, extends more freely upwards along the aorta; a diastolic aortic murmur, most freely downwards, towards the ventricles: either of such murmurs may, however, be conveyed upwards and downwards at the same time. But though the murmurs pass most freely in particular directions, it is well to remember, that the vibrations, which excite the sounds, spread in all directions, and therefore in an opposite direction to the current of blood; so that murmurs, when intense, may pass even to a considerable distance from their seat of origin, in a direction contrary to the current of blood.

The murmurs, again, may be heard simultaneously

with the heart's healthy sounds,* which are then modified in their characters; or they may entirely replace and supersede these sounds; or one of the heart's sounds may commence as a murmur and finish as a healthy sound. Organic murmurs, also, are persistent, not temporary, like the inorganic sort. To this rule, however, there are apparent exceptions; for instance, the cause producing the murmur is occasionally so slight, that, under ordinary circumstances, no murmur is excited, but the least hurrying of the heart's action at once reveals the defect. Cases are frequently met with, in which we suspect the existence of heart disease, but find no corroborating endocardial murmur, until the patient has been made to excite the heart's action, by hurrying up and down the room, or mounting a staircase: a fact well worthy attention, in the particular instance of examining a person for a life insurance. In the latter stages of heart diseases, also, when the heart's actions are enfeebled, and its cavities distended and oppressed with the blood, which they are unable to propel onwards, murmurs, previously existing, very frequently cease to be heard.

* We may as well repeat here, to prevent any chance of confusion, that when we use the term *sounds* of the heart, we refer to its healthy sounds. The distinction in nomenclature is clearly faulty, for murmurs are sounds. Some latitude of expression, however, must be permitted, through imperfection of language.

Seat of the Murmur.—The chief facts which it is requisite for us to consider, in determining the point of origin of any particular murmur, are, the parts of the thorax at which the murmur is heard loudest, and the direction in which it is conveyed. Now, as almost all cardiac murmurs arise at, or about, the orifices or the valves of the heart, and are, *cæteris paribus*, heard loudest nearest to their points of origin, it becomes a matter of importance that we should, in every case, as far as possible, ascertain the position of the valves and the orifices. This, unfortunately, it is often difficult to ascertain with exactitude; for, as we know, casual circumstances, even in health, tend to alter the position of the heart; and when the shape and figure of the organ is altered by disease, its position, and the relation of its individual parts to the thoracic walls, become still more deranged.

It is useless, therefore, to attempt to fix too nicely the exact position of the several parts of the heart; but we shall find, in the point where the apex of the heart beats against the thoracic walls, a most important mark to guide us in determining the relative position of the other different parts of the organ. This point can almost always be ascertained; and it represents the situation of the apex of the left ventricle. Consequently, as a general rule, a cardiac murmur, heard loudest at that point, must be ascribed to the left side of the

heart; and in like manner, a murmur heard loudest over the lower part of the sternum, must be attributed to the right side of the heart. Murmurs, heard loudest about the centre of the sternum, and passing upwards a little to the right of it, belong to the aorta; and those passing to the left of the centre of the sternum, that is, in the direction of the pulmonary artery, to the pulmonary artery.

Systolic Murmurs over the Left Ventricle of the Heart.—A murmur, heard about the apex of the heart, and in a direction upwards, over its left side, indicates: either imperfect closure of the mitral valves, and consequently, regurgitation of blood into the left auricle; or, irregularities or roughnesses —from calcareous or fibrinous deposits—upon the surface of the endocardium near the valvular orifices, or upon the ventricular surfaces of the aortic valves, whereby obstructions to the free current of blood from the ventricle into the aorta, are produced. The murmur, moreover, may arise from both these causes combined,—that is, from the coexistence of regurgitant mitral, and obstructive aortic diseases. Skoda gives another cause of this murmur, namely, "a rapid current of blood, forced from the left ventricle, in a direction opposite to the blood flowing from the left auricle,"—a cause manifestly hypothetical, but still worthy of attention, inasmuch as such currents, artificially produced in tubes, are found to give rise to murmurs.

When the systolic murmur results from *imperfection of the mitral valves,—systolic or regurgitant mitral murmur,*—it is heard loud at the apex of the heart, and in the direction of the mitral valves (the position of which must be judged of, from their necessary relation to the point where the apex of the heart is found to beat), and thus upwards, and to the left of the nipple; at the same time, the murmur may be heard very faintly, or not at all, over the situation of the aortic valves, and up the sternum; it is generally, however, audible over a large portion of the præcordial region, when very intense, and when the heart is much enlarged. Both cardiac sounds over the aorta may, at the same time, be heard clear and healthy; and so, both sounds over the lower part of the sternum,—that is, over the right ventricle and over the right base of the heart; should the murmur, however, be heard in this situation, it will be only faintly so, in comparison with the murmur which is heard over the left ventricle. Regurgitant mitral murmur, also, entirely supersedes, or partially replaces, the first sound at the apex. It is not unfrequently audible, more or less distinctly, in the lower part of the interscapular space.

When the murmur results from the friction of the blood against the roughened surfaces of the endocardium, at and around the arterial orifice and its valves, or from constriction of the arterial ori-

fice, it is heard but indistinctly, or not all, at the apex of the heart, becoming more distinct, however, as we proceed upwards from the apex towards the mid-sternum,—that is, towards the site of the aortic valves, which is its point of intensity, and from whence it is conveyed upwards, in the direction of the aorta, either partially or wholly replacing the first aortic sound. Thus, the point of intensity of such a murmur, arising in the left ventricle, lies quite in a different direction to that of a mitral regurgitant murmur.

In some rare cases, it appears that a systolic murmur, having characters resembling those here given of a regurgitant mitral murmur, may arise simply from the friction of the blood, during the contraction of the ventricle, against vegetations situated on the ventricular surfaces of the mitral valves, or against coagula fixed in the columnæ carneæ. In such cases, we may derive assistance in our diagnosis, by attending to the condition of the second sound of the pulmonary artery.

The condition of the second sound of the pulmonary artery has, indeed, been proposed and adopted by Skoda, and other German pathologists, as a test by which we may invariably decide, whether the ventricular murmur depends merely upon friction of the blood over the roughened surfaces of the ventricle, or of the ventricular surfaces of the valves; or whether it is the actual result of

mitral regurgitation. According to these observers, the second sound is invariably increased in intensity, whenever mitral regurgitation really occurs; and the increase of its sound is accounted for in the following manner: "When the closure of the mitral valves is not complete, a portion of blood, at each systole, regurgitates from the left ventricle into the left auricle, causing distension of the left ventricle and of the pulmonary veins and arteries, so that increased efforts on the part of the right ventricle become necessary, in order to force the blood onwards through the distended vessels: the pulmonary artery, thus strongly dilated, presses with the increased force of its elasticity upon the blood within it, and drives it more suddenly and forcibly backwards than ordinary, against the semilunar valves, during the heart's diastole, whereby the second sound of the pulmonary artery is intensified." (Skoda.) Skoda, indeed, lays it down as a rule, that we are not to conclude, from the mere fact of a murmur being heard in the left ventricle during the heart's systole, that the mitral valves are defective, unless we at the same time ascertain, that there is an increase in the intensity of the second sound of the pulmonary artery.

Notwithstanding Skoda's authority, I venture to suggest, that this cannot be admitted as an absolute test of mitral deficiency, for I believe that such deficiency frequently exists without any perceptible

increase of the second sounds of the pulmonary artery; at the same time, in the majority of cases, where the *defect of the valves is considerable,* the second sound of the artery is, without doubt, generally intensified. In such cases, however, it is well to remark, that the regurgitation is so considerable, as, at the same time, to produce severe oppression of the respiration; wherefore oppression of the lungs, and intensification of the second sound of the pulmonary artery, appear to exist together, and are thus corroborative signs of the defect of the mitral valves.*

Upon the whole, the diagnosis of mitral systolic

* The sounds of the pulmonary artery are to be sought for in the second left, those of the aorta, in the second right, intercostal space, about a finger's breadth from the sternum. In health, the strength, pitch, timbre, and duration of these two sets of sounds are generally alike, but occasionally they vary. Whatever produces increased tension of the pulmonary artery, produces increase of its second sound. So Hamernjk writes of the second sound of the pulmonary artery. I have paid some attention to this second arterial sound, and may say of it, that I have never been able to decide, whether, in health, the second sound of the aorta, or of the pulmonary artery, is the louder. I have found the intensity of the two sounds vary much in different persons, being sometimes greatest in the aorta, and sometimes in the pulmonary artery. I believe the value, as a diagnostic sign, of this increase of the second sound of the pulmonary artery, has been much overrated by the Germans; but, as above said, I do not deny that, in most cases, where there is *considerable* mitral regurgitation, the sound is really intensified. In these cases, however, as above said, marked oppression of the lungs also co-exists, and tends to point out the nature of the murmur, equally as the increased second sound; and therefore reduces its value as a diagnostic sign.

murmur—the most frequently met with of organic murmurs—is plain and clear, and it is only in the rare cases above alluded to, that there is any chance of an error being made by the careful observer.

Both ventricles, particularly the right ventricle, are generally hypertrophied and dilated when the mitral valves are defective; consequently, the extent of præcordial dulness is found on percussion to be greater than natural; and so, also, is the impulse of the heart. The character of the pulse varies considerably in such a condition of the heart; when the regurgitation is considerable, it becomes weak and irregular.

The cause of hypertrophy and dilatation of the right ventricle in such cases is evident enough; but the cause of the hypertrophy of the left ventricle, as a consequence of such disease, is not so clear. It is possible, that the defective supply of blood, which passes into the aorta at each ventricular systole, may be a stimulus which incites the heart to extra exertion, and thereby induces hypertrophy; moreover, when the pulmonary obstruction is so considerable as to produce engorgement of the systemic veins and general serous effusion, impediment to the circulation of the blood through the capillary vessels necessarily results, and may thus give rise to hypertrophy of the left ventricle.

Occasionally it happens, that *neither Sound nor Murmur are heard over the left ventricle during*

its systole. Such a state gives us no indication as to the condition of the mitral valves: for the normal sound may be absent when the closure of the valves is perfect. A murmur may also fail to be heard when the valves are defective, and permit regurgitation of blood through them; and for the reason, in both cases, that those other causes are absent which are absolutely necessary for the formation of the sound in the one case, and of the murmur in the other. In such cases, we must investigate the condition of the second sound of the pulmonary artery and of the respiration, and take note of the general symptoms, and of the state of the right ventricle, which generally becomes hypertrophied under the effects of long standing mitral valvular disease.

Diastolic Mitral Murmur.—A murmur—heard over the left ventricle, and along the thoracic walls in the same direction as a systolic mitral, that is, from the apex of the heart up towards the left of the left nipple, and coinciding in time with the diastole of the heart—results from one of two causes, namely, either from the presence of roughnesses upon the auricular surface of the mitral valves, or upon the internal surface of the auricle around the valves; or from constriction of the mitral valves. Its most ordinary cause is constriction of the valves.

This murmur is not of frequent occurrence; and

when present, its diagnosis is often attended with considerable difficulty. Some authors appear almost to ignore the existence of such a murmur. Dr. Latham speaks of it as a sort of clinical curiosity, and remarks, that "it would almost seem that the mitral orifice could be the seat of only one murmur, and that murmur the systolic."

Nevertheless, there can be no doubt whatever as to the existence of the murmur; and I believe that the misconceptions which have arisen concerning it, depend upon the difficulties accidentally attending its diagnosis. The consequences resulting from advanced obstructive disease of the mitral orifice, and manifested in the heart itself, are necessarily severe; they are direct, and incessantly in action; and they are severe in proportion to the degree of constriction of the orifice.

These consequences are, rapid and tumultuous action of the heart; changes, often very considerable, in the rhythm of its movements; and such alterations in the character of its sounds, as may readily permit of the one being mistaken for the other, and consequently of the systole being confounded with the diastole of the heart. Another circumstance which complicates the diagnosis of diastolic mitral murmurs is this, namely, that in obstructive disease of the mitral valve, the first—the systolic sound—is, as may readily be conceived, on account of the condition of the valve, almost

always abnormal; it is, in fact, as far as my own observation goes, invariably associated with, or superseded by, a murmur in all cases where the constriction of the mitral orifice is considerable.

In such cases, in order to arrive at a correct diagnosis, it is absolutely necessary *to fix the exact time of the heart's systole*, by feeling its impulse at the same time that its sounds are auscultated; when this is done, the observer will not have much difficulty in determining with which movement of the heart the murmur is synchronous; that is, provided its action is not excessively rapid, for in such case the most practised ear may be unable to unravel the complication of sounds heard.*

The characters of a diastolic mitral murmur will naturally correspond and vary in their intensity with the degree of constriction of the mitral orifice, and with the power of the heart to carry on the circulation. In the latter stages of such a disease of the heart, the murmur may, and I believe generally does, altogether disappear; and the reason of this is, that the muscular power of the heart is no longer capable of forcing such a stream of blood through the orifice, as shall suffice to create audible

* I may observe, that in no kind of valvular diseases does the action of the heart seem more amenable to the influence of digitalis than in this. Consequently, in these cases this medicine affords us an excellent means of analysing the sounds and murmurs of the heart.

vibrations.* In the advanced stages of this, as in other cases of valvular disease, the loudness of the murmur must, to a certain extent, be looked upon as a proof of the still existing vigour of the heart, and its disappearance as the reverse, and therefore as an unfavourable sign.

When the murmur is well marked, it is loud and prolonged, and sometimes audible over the whole of the præcordial region; it is loudest, however, at the apex, and in a direction leading thence up the left side of the heart. At the apex of the heart, when the action of the organ is strong, a distinct vibratile thrill is perceptible to the touch,†—fré-

* I witnessed a striking instance of this fact lately, in the case of a little girl, in whom, two years before her death, I had diagnosed mitral obstructive disease. The diastolic murmur was constantly present up to within a few days of her death. The murmur then disappeared, and in its place *a tolerable good first and second sound were distinctly audible over the left ventricle.* After death, the point of the little finger could be scarcely passed into the contracted mitral orifice; other parts of the heart were healthy. The following are the notes which were taken of this girl's case nearly two years before her death: "Slight frémissement perceptible at the heart's apex; the bruit heard immediately precedes the heart's impulse, which seems to conclude it; the bruit is not heard along the aorta or over its valves; the pulse in the carotids, and its branches, is very weak. Difference of opinion has been held as to the nature of the murmur, though none as to the fact of the bruit preceding the beat. Slight exertion produces difficulty of breathing; and lately, hæmoptysis has taken place. If this be not a diastolic mitral murmur, it seems to me impossible to give any account of it." (*Edin. Month. Journ.*, Jan. 1854.)

† "Un bruissement particulier, difficile à décrire, sensible à la main appliquée sur la région précordiale, bruissement qui

missement cataire. Sometimes the murmur is high-pitched and even musical; sometimes it is deep and rough. It may be so prolonged, as to occupy the whole of the period of the diastole, the interval, and a part of the systole of the heart: so that the natural rhythm of the heart's movements is lost. In such case, the impulse appears to follow immediately upon the cessation of the murmur, or, rather, it seems to be its conclusion, and to wind it up, as it were. The impulse is brief and rapidly accomplished, and, after a very short pause, is followed by the recurrence of the murmur.

The heart is, in these cases, hypertrophied; the

provient, sans doute, de la difficulté qu'éprouve le sang à passer par un orifice qui n'est plus proportionné à la quantité de fluide," etc. (Corvisart.)

"When the contraction of the mitral orifice is great," says Hamernjk, "the second murmur is long and loud; and some portions of it are louder than others, producing the hum of a spinning-wheel, and has thus been taken for a double sound; such a second (diastolic) murmur is protracted, and ends in the systole; there is a distinct pause between the systole and diastole of one complete heart's movements, but none between the diastolic and recurring systolic murmurs."

"It is especially," says Skoda, "in cases like these that vibrations are felt when the hand is laid upon the præcordial region,—the frémissement cataire described by Laennec."

"It is a common opinion," says Professor Jacksch of Prague, "that a fremitus which is felt at the apex of the heart, and which accompanies the diastole, is a pathognomonic sign of an obstructive mitral orifice."

As the account I have given of this murmur differs much from that of many stethoscopic authorities in this country, I have thought it well to show that it is backed by the observations of the above-cited authorities.

hypertrophy affecting, in a particular manner, its right side and its left auricle. The præcordial dulness is, therefore, found increased on percussion, and especially in the direction of the right ventricle. In consequence, we may fairly suppose, of the small quantity of blood which passes through the constricted mitral orifice into the left ventricle, and the small quantity which is therefore propelled into the aorta at each ventricular systole, the aortic sounds (and so, also, the arterial pulse) are weak, though normal in their characters; a circumstance which renders, by comparison, the increased intensity of the second sound of the pulmonary artery more than ordinarily remarkable. The left ventricle may be of normal size.

Such are the physical signs observed in typical cases of the disease; and they are the same as those which are present, in a more or less well-marked degree, in all cases of diastolic mitral murmur. The prominence and intensity of the signs, however, are necessarily proportioned to the amount of obstruction to the circulation, which the constricted mitral orifice produces; when, therefore, this is slight, the signs may not be prominently marked.

It may not be out of place to remark here, that in all the cases which I have seen of this particular valvular disease, the general symptoms corresponded remarkably to the nature of the physical defect. The difficulty of breathing was generally consider-

able, and was very readily increased by exertion; it was much greater, in fact, than is generally observed in regurgitant mitral disease: the left ventricle was not much hypertrophied, and its impulse, though thrilling, not very strong; the arterial pulse was remarkably weak, and, indeed, sometimes even difficult to feel in the cervical arteries: hæmoptysis was common. Rest and quiet, in an especial manner, afforded relief and comfort.

When neither diastolic sound nor murmur are heard over the left ventricle, we must, Skoda tells us, form our opinion as to the condition of the mitral valve, by judging of the nature of the systolic ventricular sound, and of the second sound of the pulmonary artery: if the systolic sound be good and clear, and the pulmonary artery's sound not increased, then we have no reason to believe that the valves are unsound. But if there be a murmur instead of the healthy systolic sound, and if the artery's second sound be increased, then the absence of the second sound may be attributed to defect of the mitral valves, without constriction of their orifice, though it does not, in such case, preclude the existence of such constriction.

Again, when the mitral opening is constricted, the mitral valves are, at the same time, very often defective, but the systolic murmur thence arising is generally weak, short, and often inaudible: in exceptional instances, however, the reverse of this is

the case, the diastolic being weak and short, and the systolic prolonged. The difference depends, adds Skoda, in all probability, upon the form and direction of the constricted canal, and upon the situation of its roughened surfaces.

When, moreover, the movements of the heart are rapid, the systolic may be so confounded with the diastolic murmur, that one prolonged murmur alone is heard, commencing with the systole, continuing through the first part of the diastole, and broken only by the very short interval which corresponds to the quiescent state of the ventricle.

Absence of sounds and murmurs over the left ventricle, during the systole and diastole of the heart, do not indicate any organic disease of the mitral valves, unless the pulmonary artery's second sound be increased.

Systolic Aortic Murmur.—The absence of a murmur over the aorta, during the heart's systole, does not necessarily indicate a healthy condition of the aorta. If the first sound be indistinct, or duller than natural, and the heart's impulse of natural strength, we may conclude that the coats of the aorta are not in a normal condition.

The presence of a systolic murmur, in place of the first sound, over the aortic valves, and the first portion of the aorta,—that is, over the centre of the sternum, about opposite to the third intercostal spaces, and some little way thence up along the

right border of the sternum,—indicates a roughened condition of the inner surface of the aorta, or of the ventricular surface of the aortic valves. When very loud, it indicates constriction of the aortic orifice, resulting from fibrinous deposits upon or around the valves, or from hardening and stiffening of the valves, through calcareous degenerations in and upon them. Fibrinous coagula, impeding the passage of the blood from the ventricle, have also been supposed to create, on rare occasions, systolic aortic murmur.

There is, often, nothing in the character of this murmur to distinguish it from the systolic murmur of anæmia, or of other inorganic systolic murmurs, or from the murmur which often accompanies cyanosis, and certain aortic aneurisms; in such cases, however, the accompanying symptoms will rarely fail to enable us to decide as to the nature of the murmur.

Anæmic murmurs may be occasionally present, in cases of organic valvular diseases of the heart, and thus complicate the diagnosis. In advanced stages of organic valvular diseases, an anæmic condition of the blood often arises; and under such conditions, I have found even the radial artery readily giving rise to a murmur, when the stethoscope was placed over it; and I have seen the fact in men as well as in women. The possibility of an anæmic bruit co-existing with organic valvular

diseases, is therefore well worthy the recollection of the physician,—the possibility, for instance, of an inorganic aortic murmur co-existing with an organic mitral murmur.

Systolic aortic murmur is not heard, or but very faintly, at the apex of the heart. The second sound over the left ventricle is also generally rather indistinct; and for the reason, that the state of the valves which produces the systolic murmur naturally interferes with their proper action, and therefore renders imperfect the second aortic valvular sound.

When the aortic murmur is heard louder towards the top of the sternum than over the aortic valves, we may be sure that the disease provoking it lies rather in the aorta itself than at its valvular orifice.

Hypertrophy and dilatation of the left ventricle are the common consequences of constricted aortic orifice, and of disease of the aorta; the amount thereof will depend on the degree of obstruction which is offered to the circulation, and on the age of the disease.* The pulse, also, varies with the

* When we are considering the physical signs and symptoms of valvular diseases, it is requisite in all cases, as far as possible, to take *the age* of the disease into our calculation. Unless this is done by the student, he will often find, that many of the signs and symptoms, which he had been led to believe were proper to a particular murmur, absent; thus, for instance, he may find the most marked aortic, or mitral murmurs, unaccompanied by signs of hypertrophy of the heart, or by any marked degree of disturbance of the respiration, etc. On reflection,

degree of constriction: when this is great, the pulse is small, but hard and wiry.

Aortic Regurgitant Murmur indicates a defective condition of the aortic valves, whereby the blood is permitted to regurgitate from the aorta into the left ventricle, during the ventricular diastole. Its point of greatest intensity is over the aortic valves, that is, about the middle of the sternum, and opposite the third intercostal spaces; from thence the sound, with the current of blood which produces it, is carried downwards, in an opposite direction to a systolic aortic bruit. The correctness of the remark of Dr. Walshe, that this diastolic murmur is often heard very loud over the lower part of the sternum, and even at the ensiform cartilage, I have again and again had occasion to verify; the sound appears also, generally speaking, to be conducted downwards along the sternum, that is, in the direction of the right ventricle, rather than in that of the left ventricle; which is contrary to what is usually supposed to happen. I have frequently heard a loud regurgitant aortic murmur at the

however, he will see how this naturally happens. Hypertrophy requires time for its production; whilst the injury, which occasions the murmur, may be effected in a few days. In like manner, we may argue of other secondary symptoms which result from valvular diseases, and which are represented as signs of those diseases. Their presence, and their, more or less, well-defined characters, depend very much upon the age of the valvular disease. This is a point, affecting diagnosis, to which attention has not been sufficiently directed.

lower part of the sternum, when the second sound, probably of the pulmonary artery, was clearly audible at the apex of the heart, and unmixed with any murmur. The reason why the regurgitant current does not rather convey the diastolic murmur towards the apex of the left, instead of towards that of the right ventricle, is not clear.

Occasionally, the diastolic aortic sound commences as a murmur, but terminates as a natural aortic second sound; in such case, it has been supposed that the murmur arises from vibrations, excited by a diseased condition of the coats of the aorta during their recoil, and that the natural sound in which it terminates results from closure of the valves.

The first aortic sound may be healthy in cases of diastolic aortic murmur: but, as may be readily supposed, in the majority of cases it is altered, and for the reason that when the valves are so disordered as to permit of regurgitation, they almost always at the same time present some obstruction to the current of blood which flows through them during the heart's systole.

Hypertrophy and dilatation of the heart result from aortic valvular regurgitation and from disease of the aorta, and the signs and symptoms of these conditions of the heart will be therefore present. The state of the pulse in aortic valvular regurgitation is peculiar: the artery is suddenly filled, but

rapidly and abruptly collapses under the finger; the phenomenon results doubtless from the defective condition of the aortic valves, which permits regurgitation to take place.

Tricuspid Valvular Murmur.—In accordance with what has been said respecting the pathology of valvular diseases on the right side of the heart, we find that murmurs arising here are, comparatively speaking, rarely met with. *Diastolic Murmurs* of the right side of the heart—that is, murmurs arising from regurgitation of blood through the pulmonary artery's valves, or from the passage of the blood into the right ventricle out of the right auricle during the ventricular diastole—may, practically, be excluded from our consideration.

A *Systolic Tricuspid Murmur* will be heard loudest at the lower part of the sternum; it has generally the character of a smooth—not overloud—blowing murmur. It arises from two causes: either from regurgitation of the blood through the tricuspid valve; or from friction of the blood, during the heart's systole, against roughnesses—fibrinous or calcareous deposits—on or around the ventricular surfaces of the valves; the latter, however, must be an excessively rare cause of the murmur, and hardly needs attention. When tricuspid regurgitation takes place, the blood must accumulate in the right auricle, the venæ cavæ, etc.,

causing distension of the jugular veins, and occasionally giving rise to what is called the venous pulsation. It is certain, however, that regurgitation takes place much more frequently without, than with, an accompanying murmur. The diseased conditions which produce the murmur in the case of the mitral valves are very rarely present here, and yet tricuspid regurgitation is very common; the regurgitation, therefore, seems to arise simply from enlargement of the tricuspid orifice and defect of the valves,—conditions which do not appear in this case necessarily to occasion a murmur.

The murmur, when present, may be occasionally marked by a co-existing mitral murmur; and it must be remembered, that hypertrophy and dilatation of the right side of the heart and tricuspid regurgitation, naturally result from the causes which produce the mitral murmur; so that the co-existence of a murmur over the right side of the heart, and distension of the jugular veins, do not of necessity indicate disease of the tricuspid valves.

Structural diseases of the *pulmonary artery* and of its valves, such as give rise to arterial murmurs, are exceedingly rare. Systolic murmurs, it is true, are not unfrequently heard over, and seem to proceed from, the pulmonary artery; but the cause of such murmurs is obscure, and they, in all probability, result from influences acting extrinsically to the artery itself. A systolic murmur of this kind has

been frequently noticed in cases of tubercular disease of the apex of the lung.

The following symptoms have been observed in exceeding constriction of the orifice of the pulmonary artery. Loud, prolonged, systolic murmur; loudest in the third intercostal space, close to the sternum, and heard along the lower border of the third left rib for the space of one to two inches; the murmur being inaudible about the top of the sternum and its right upper border; slightly audible over the aortic valves, and downwards over the right ventricle; the first sound being replaced by the murmur at the base of the heart, but remaining audible at the apex.

CHAPTER XII.

ENDOCARDITIS.

Valvular diseases and their Secondary disorders, treatment of.—Rapid progress of the disease.—Early application of remedies necessary.—Treatment in the main resembles that of Pericarditis.—The difficulties attending it.—Chronic valvular diseases, their treatment.—Secondary pulmonary disorders, treatment.—Treatment of secondary abdominal disorders.—General treatment.

THE treatment of acute valvular disease, which is necessarily comprised under that of acute endocarditis, must be conducted on the same principles as those which guide us in the treatment of acute pericarditis. The near relation of these diseases has been already spoken of; their frequent co-existence; their origin from, or connexion with, the same exciting causes; and the anatomical similarity of the inflamed structures.

In detailing the treatment of pericarditis, I have advised the use of bleeding, mercury, and opium, as being, in that disease, the means recommended to us by the greatest experience as best fitted for its cure, due caution being taken in their proper application. These, then, are also the chief reme-

dies, combined with the other adjuvants there mentioned, which are to be employed in the treatment of endocarditis. For the mode of employing them, I must refer the reader to the details given under the head of pericarditis.

In the treatment of endocarditis, it may be said, that if any remedy is to serve us, it must be one that is powerful in its effects, and quick in its action; one that will tell rapidly on the disease, without seriously damaging the constitution of the patient. There is less time for delay here, than in the case of pericarditis: the pericardial membrane may receive a permanent injury; lymph may be deposited upon it, and partial adhesions of its surfaces may result, and yet no damage to the general working of the heart eventually occur; the inflammation may pass away, and the organ be left capable of well performing its duties. Not so is it with the endocardium; the force of the inflammation falls upon the valves, whose smallest injury is of the most serious importance; and a few hours of the inflammatory action exercised upon them, may ruin them for ever. Hence it is, that our treatment should, as far as possible, even anticipate the mischief which we dread.

The first certain proof we have of the presence of valvular disease, is the substitution of unnatural murmurs for the ordinary healthy sounds of the heart; but then, unfortunately, when this fact

reaches us, we are sure that the inflammation has already progressed in its work of disorganization; and that our remedies now come late in the field to combat the malady. The valve may be already irretrievably damaged, beyond the *vis medicatrix naturæ* or the art of medicine to repair. What follows from this? Plainly, that we should endeavour, by a most careful watching of signs and symptoms, in all those affections with which we find endocarditis frequently associated, to be beforehand, as it were, with the inflammation,—to be busily employed in the treatment of the valvular disease, before that final test, the valvular murmur, gives us the positive proof of its existence. And certainly, in the majority of cases, there are signs and symptoms from which we may obtain that lesser degree of knowledge,—that is, the knowledge that injury to the valves is imminent; and obtain it, indeed, with such an amount of assurance, as to justify us in acting as if the positive proofs—namely, the valvular murmurs—had demonstrated the existence of the endocarditis. We are surely justified in so acting, when, for instance, during an attack of acute rheumatism, we suddenly find the heart's action become violent and irregular; its sounds altered in character, though still to be called healthy sounds; præcordial pain, or tenderness on pressure over the heart; high fever, and irregular pulse: in such a case, there can be no

need to wait for a cardiac bruit, to decide our curative proceedings. To anticipate thus the endocardial inflammation, in its early aggression, becomes a proper trial for the skill of the physician.

Bleeding, local or general, as the case may warrant, to subdue the heart's violence in action; mercury, specifically used to stop the inflammation, or merely to act so as to keep the different secretions free; opium, to allay the pain and give repose; purgatives, diuretics, and alkalis for their particular purposes,—these are the general remedies which experience places to our hand as most to be trusted for the object desired. I say as most to be trusted: their actual value—the real power which they exert over the inflammation—has yet to be shown.

It is only fair to add this word of caution to the student. The most skilful physician must ever watch the rise and progress of pericardial and endocardial inflammations with dread, and apply his remedies with dread, lest they prove powerless to resist the inflammation. The treatment here recommended, aiming as it does at fulfilling not one but many several indications, and skilfully applied according to the particular symptoms and idiosyncrasy of the individual case; containing nothing within it of the specific, but resting entirely on the ordinary principles of medicine,—this treatment we consider the rational, and, in the present state of our knowledge, the true treatment of endocard-

itis. But it constantly fails us; how often, indeed, can we flatter ourselves that it has arrested the inflammation, and saved a valve from permanent injury?

The difficulties which beset the treatment of these inflammations, may be gathered from the fact of the discordance in opinion which exists concerning it among different physicians. I will not here enlarge upon this subject; but have said this much, because experience compels me, through its lessons, to warn the student against over-enthusiasm in the effects of his remedies; too often will he find, that the hopes and confidence of to-day, derived from some happy issue which the disease has found under his hands, will be belied by the reverses which await him to-morrow. Of particular, specific methods of treatment, I will only say that I believe them to be altogether ill-founded and illogical; they ignore entirely what I presume to consider are the proper principles which should guide our treatment,—principles based upon a due consideration of the individual features of each particular case; upon a skilful appreciation of the peculiarities of the disease itself, and of the idiosyncrasy of the individual attacked by it; upon a rational view of its pathological characters; and upon the results of experience.

When inflammation has once fallen upon a part, and left it damaged, that part appears ever after to

be particularly liable to a recurrence of the attack. This is the case, to a remarkable degree, in the instance of valvular diseases. And thus it is, that the treatment of secondary valvular diseases is often attended with much difficulty and uncertainty. For example, rheumatism and febrile action may cause a patient to apply for relief to his physician, who at once, perhaps, discovers the existence of a cardiac bruit; but how is he to judge of it? Is it the consequence resulting from some former inflammation? or is it the result of inflammation at present existing, and still in action? In the one case, energetic treatment may be required; in the other, gentle and palliative remedies only.

The fact is, however, that, generally speaking, we may arrive at a tolerable degree of certainty in these cases,—enough, at least, to guide us to their treatment. And for this purpose, the previous history of the patient, and his present condition, must be our guides: if he has suffered from former attacks of rheumatic fever, and been more or less ailing since the attacks, — subject to occasional cough, shortness of breath, and palpitations; and if, in addition to the cardiac bruit, there exist signs of hypertrophy of the heart, then, in such case, there can be little doubt that the valvular lesion is not of recent date.

But it is quite true—and, indeed, the case is common enough—that fresh disease, a repetition of

the inflammation, may befal the already injured valve; and here the difficulty of diagnosis becomes considerable. We have no longer that sign, which was originally pathognomonic (inasmuch as it arose under our immediate observation) of the endocarditis, to help us, namely, the cardiac bruit; for this already exists, demonstrative of the valvular injury. We must, therefore, in such a case, judge of the presence or absence of local inflammation, and of its degree, when it exists, by the presence or absence of general febrile symptoms, and by the quiet or excited action of the heart, and by whatever other symptoms there are, which have been described as indicative of acute endocardial inflammation.

If the evidence is sufficient to lead us to satisfactory proof of the existence of inflammation, then we must proceed in our treatment as in an ordinary case of acute valvular disease; but with this difference,—that the particular condition of the patient must be carefully taken into calculation, that is, the fact of his heart having been already organically diseased, in consequence of which, the different functions of the body are more or less disturbed and weakened. If caution in treatment was required in the original instance of the disease, still more is it necessary now; no rules for application of the treatment under such circumstances can be laid down; each individual case must be judged of by its own particular characters. This

much, however, may be said, that the general disordered condition of the body and its organs, resulting from previous attacks of the endocarditis, will be found to require the attention of the practitioner, rather than the renewed inflammatory attack which has befallen the valves.

Chronic Valvular Disease: Treatment. — We now come to consider the treatment of chronic valvular diseases, or rather, of the secondary disorders which necessarily result from them. When the fact of a valvular imperfection is established, by its proper diagnostic signs, and the endocardial inflammation which attended its origin has passed away, the local injury is no longer the especial object of our treatment; it has reached a condition beyond our power to control. The pulmonary, the abdominal, and the cerebral disorders, to which the valvular disease gives rise, are those which now claim our attention; and so, likewise, are the disordered conditions of the muscular tissue of the heart itself, and of the general circulation.

Hypertrophy and dilatation are the chief effects which result from the valvular diseases to the heart itself. Of these morbid states, we have elsewhere given the history. Passing by, then, the treatment of the secondary morbid conditions of the heart, we find that the treatment of chronic valvular disease involves two especial indications,—namely: first, by prophylactic means, to endeavour to guard against

a recurrence of the endocardial inflammation; and, secondly, to minister to the accidental disorders which arise in different parts of the organism, as consequences of the valvular disease.

The fulfilment of the first indication is to be attempted, by the adoption of ordinary prophylactic measures proper in such case: abstinence from whatever excites the heart's action inordinately; careful attention to the diet and clothing; avoiding exposure to sudden changes of temperature, to wet and cold, and all those influences which appear to render the body liable to rheumatic seizures; moderate but restricted exercise, and the due maintenance of the excretory functions of the body;—these are the chief particulars to be attended to. Few, however, of those affected by heart disease, are able to subject themselves to such favourable conditions; the bulk of humanity must labour and toil; and daily labour and toil necessarily entail exposure to influences hurtful to the disordered body; and thus it is that, in the great majority of instances of cardiac affections, the progress of the disease is continual, and the severity and complication of the symptoms ever on the increase.

Where the condition in life of the patient is such as to enable him to take advantage of all proper hygienic aids, and when, at the same time, the valvular disease has not reached beyond a certain degree, it happens occasionally that life is passed

with comfort, and in freedom from most of the ordinary consequences of such disease. It is surprising, indeed, how frequently we meet with cases, in which, though loudish and persistent bruits surely indicate organic disease of the heart, few and but slight symptoms are present to enforce the existence of the disease on the minds of the patients themselves. The fact is encouraging for treatment; but without treatment—that is, proper prophylactic treatment—we cannot hope that such a favourable condition of things can long exist.

Nothing surely can be more injudicious, than suddenly informing a person, who is ignorant of the fact, that he is the subject of organic disease of the heart; the mental emotion thereby caused has been known, in many instances, to produce the most injurious consequences; but still, in all cases wherever the stethoscopic signs leave no room for doubt concerning the existence of heart disease, it is the duty of the physician to watch over the subject of it, and to place him, if possible, under a duly regulated mode of life. Such a person must ever after be looked upon and treated as an invalid; it is impossible to believe that any one so affected can follow out the ordinary pursuits of active life, for any length of time at least, with impunity; and most assuredly a small accident is in him sufficient to wake up the inflammation once again, and once again to add fresh injury to the valves.

Such is the general system of management which must be adopted with regard to the heart disease itself. But there are other indications for treatment. The valvular disease, as we have seen, is the fruitful source of disorders and disturbances in the different organs and parts of the body; and when once the disease has produced a certain degree of disorganization in the valves,—when it has given rise to a certain amount of imperfection in function,—these disorders become permanently fixed, and display themselves by the presence of a train of more or less severe symptoms. The treatment of these disorders now becomes specially important; and the relief of the disturbed and oppressed organs the particular objects of our attention. Here, happily, it is certain that medicine can do very much in giving temporary relief, and in so aiding the powers of life as to lengthen the days of the patient, and in diminishing his sufferings. The treatment of the disorders resulting from organic valvular diseases we will now speak of.

Pulmonary Disorders: Treatment.—Pulmonary affections are the most constant and numerous of the secondary disorders which result from valvular diseases, and they are the most important which the physician is called upon to treat. We may say, generally, of these disorders, that the treatment is palliative, inasmuch as the exciting cause of them (that is, the valvular disease) is in con-

tinual operation, and beyond remedial art. The treatment has one immediate and special object in view, and that is, to free the respiratory organs from the obstacles which, within themselves, oppose the due performance of their functions.

But though the main indication for treatment of these disorders be clear, the proper method of carrying it out is often hard to choose; for the question involved is a complicated one, and many considerations, including points of much nicety, present themselves to us and demand careful attention, as being guides to a right and proper method of effecting the object desired. These considerations are, in the main, the nature of other co-existing secondary disorders, as of the organs of the abdomen and of the brain, and the condition of general health of the patient; and by these must our manner of treatment of the pulmonary affections be in great part regulated.

The pulmonary affection is most varied in degree in different individuals, and in the same individual likewise at different times. The affection may be, at one time, represented by disturbances of a transient character,—by a passing congestion of the lungs; and, at another time, by the most formidable disorders. The injury of the valves may be such, that, under the ordinary conditions of a tranquil life, the subject of it is free from the usual symptoms which mark its existence; it being only

under the influence of great excitement, or of unwonted exertion,—that is, when the blood is suddenly forced with increased violence towards the heart,—that the organ fails, and that its defective parts reveal their existence, and congestion of the lungs arises. In cases of this nature, the treatment is simple and hopeful, even though the lungs be for a time much oppressed, and the function of respiration much disturbed. When the heart's action is strong, and the bodily health vigorous, and the other organs of the body free from disorder, the mere cessation of the extra-excitement, with quiet and repose, often suffice to restore the lungs to their wonted condition, especially if aided by gentle local depletion.

But how different is the prognosis, and how different the treatment, when the valvular disease has occasioned extensive structural changes in the lungs! The oppression of the lungs here is no longer the result of simple congestion; the disordered condition is of long date, and its cause—the valvular disease—is permanently in action, and under all conditions of the heart's action, whether tranquil or excited. Moreover, other disorders now complicate the case: abdominal congestions, serous effusions, and cerebral disturbances; the patient is weak, pallid, and anæmic, and the action of the heart feeble, fluttering, and intermittent. No case more tests the skill of the physician than this.

Amidst the complication of local disorders and the general condition of the body, the true treatment is sometimes hard to find, and any treatment, indeed, often most difficult of application.

It is impossible to lay down any particular rules which shall comprise all cases of this description, for each individual case requires an especial consideration. We must remember, however, that what we are now aiming to obtain is temporary relief, not permanent cure, and that this relief must be sought at the least possible expenditure of vital power, and of the least shock to the constitution. Bronchitis, pneumonia, œdema, and congestions of the lungs, with pleuritic effusions, these are, in the main, the pulmonary disorders which present themselves to us as consequences of organic valvular diseases.

There are two objects which we have to pursue in carrying out their treatment: one is, endeavouring to diminish the force and activity (as we cannot remove it) of the producing cause; and the other, attempting to remove the local impediment to the respiration which results from that cause, and is represented by some one or more of the above-mentioned disorders. The first indication we fulfil by carefully regulating the diet, and especially the amount of fluids taken by the patient. Large quantities of fluids taken into the circulation necessarily throw an inordinate burthen on the heart, by in-

creasing the bulk of the blood, and at a time when, from its diseased condition, the power of the organ does not suffice to carry on even its ordinary duties. This is a point which should be very carefully attended to. We may attempt, also, to tranquillize the disturbed and irregular action of the heart, and strengthen it by appropriate remedies; it is surprising often to witness the good effects thence resulting, that is to say, the returning power of carrying on the circulation which the heart gains when its rapid, feeble, and yet tumultuous and ill-regulated action has been tranquillized. For this purpose, no remedy appears more efficacious than digitalis, which, when combined with the tincture of sesquichloride of iron,* and given in bitter infusion, forms one of our most valuable remedies, especially in cases where dropsical effusions have already taken place.

The dread of using digitalis in heart diseases still seems to linger in the minds of many practitioners, and I think I may venture to say, from a very large experience of its use, quite unnecessarily; any possible danger resulting from it may surely be anticipated, if the cautious practitioner will watch its effects with only ordinary care.

Thus by tranquillizing and regulating the action of the heart, we attempt to diminish or relieve the

* I mention this particular preparation of iron, because I believe that there is no other equal to it in efficacy and certainty.

pulmonary disorder. We also attempt to relieve the disorder, by removing the local impediments to the respiration which exist in the lungs themselves; and these consist chiefly in congestions of blood; in serous effusions into the pleuræ and into the air-cells and tissue of the lungs; in inflammatory-pneumonic and bronchitic exudations; and in accumulations of mucus in the bronchial tubes.

The exudations of mucus and the effusions of blood, which so often in these cases take place from the mucous membrane of the bronchial tubes, are frequently sources of great relief to the congested lungs, and we may gain valuable hints from them in treatment. So long as the patient has strength to eject them, no mischief may result from these accidents; but should his powers fail, he may be subjected thereby to the danger of suffocation; it is better therefore not to trust too much to this natural effort of relief, and thus throw too great a task upon the lungs themselves. Consequently, a few leeches from time to time, blisters, mustard poultices, stimulating liniments, should be applied to the thorax; due care being ever paid to the state of the general strength. Even in the very last stages of these pulmonary congestions, we need rarely fear the application of a few leeches; the almost constant, and often very striking, relief which they afford, encourages us to persevere to the last in their use.

The local abstraction of blood is, perhaps, less generally resorted to in these chronic pulmonary disorders than is advisable. The weak pulse, which may deter some persons from its use, is really no indication against it; on the contrary, the pulse will be found to rise as the pulmonary congestion is removed. There are conditions, common enough in cardiac diseases, in which, I believe, abstraction of blood is the only means by which life can be preserved; for instance, we not unfrequently meet with serious organic diseases of the heart in patients who, although constantly suffering in some degree under symptoms of their disease, still sustain for a length of time a tolerably vigorous constitution; suddenly, however, through some unusual exciting cause, violent symptoms of pulmonary congestion arise, and unless relieved, rapidly destroy life. On examining such cases after death, we find, that the immediate cause of death was the pulmonary engorgement alone, not dropsical exudations into the tissues and the serous cavities. Here it appears evident, that the relief, if it is to be of any avail at all, must be direct and immediate; and what remedy can be more so than the local abstraction of blood?

Cases of this kind are to be carefully distinguished from those in which the pulmonary congestion is gradually produced, by the pressure of serous exudations in the pleura upon the lungs, and by œdema

of the lungs, etc.; here the disease is irremediable, and the pulmonary congestion the inevitable forerunner of death.*

The pneumonia and serous exudations are less amenable to treatment. The pneumonia is generally of an hypostatic, asthenic, character; and therefore rather the consequence of a long-continued congestion of the pulmonary tissue, than of any especial inflammatory attack. In the former case, we can do little more than attempt, by general treatment, to sustain the strength, and, at the same time, by local derivatives, temporarily to relieve the congested state of the lungs. The supervention of an acute attack of pneumonia justifies more active treatment, but then, always most carefully adapted to the state of the system at large.

Œdema of the lungs partakes of the general dropsical condition of the body; it is a sign of weakness, of advanced disease, and a very bad sign; it comes far on in the progress of the disease, and is an indicator of its fatal termination; it is therefore little amenable to treatment; not at least to any local treatment. Here we are placed between two dangers; if we attempt to remove the

* I might, perhaps, here recall to the memory of the student the experiments of the late Dr. John Reid on asphyxia. He found, when the heart's action had ceased, in consequence of arrested respiration, that it was once more renewed, when the distended jugular veins were opened, and blood allowed to escape from the oppressed organ.

dropsical disorder, we are compelled to use remedies which weaken the general system, and therefore have a tendency to produce that very condition whose removal is attempted. In all these cases, stimulants are absolutely necessary. Even when the signs of inflammation are distinct and positive, we must freely use them, if the heart shew signs of depression and feebleness. In this condition of the heart itself, much more than in the condition of the lungs themselves, must we derive our indications for treatment; the power of the heart, rather than the pulmonary inflammation, or the œdema, or the bronchitis, deciding the nature and direction of our remedies.

In all cases, the treatment should be as simple as possible. Its main indications are clear; namely, to relieve symptoms, and sustain the bodily strength; but the mode of carrying them out, as we have said, is often very difficult. The greatest nicety is required in selecting the proper remedy, and in rightly applying it: a few leeches may give great relief, and in the same case, a dozen leeches may destroy life. In the latter stages of heart diseases, indeed, the balance of life and death is so finely hung, that the least counterpoise may disturb it for good or for ill.

Abdominal Disorders: Treatment.—Numerous disorders of the abdominal organs arise as consequences of obstructed circulation through the heart. The liver, from its intimate connexion with the

heart, especially resents the obstruction, and its enlargement is the immediate consequence. The rapid manner in which this organ sometimes enlarges, under these circumstances, is surprising; and so also is the rapid manner in which it diminishes again under treatment. When thus enlarged, it compresses the right lung upwards, and so interferes with the pulmonary functions; and in like manner, it may impede the action of the heart. The digestive and assimilative, as well as the secretive functions of the abdominal organs, suffer greatly from the continued congestions of blood, which these diseases of the heart give rise to. They make themselves known to us, among other ways, by disorders of the digestion and the nutrition. Hæmatemesis, hæmorrhage from the bowels, and diarrhœa, also from the same causes, not unfrequently occur, and often give great temporary relief to the patient. The congestion of the kidney diminishes the secretion of urine, and sometimes renders the urine albuminous, and gradually induces changes in its structure. Thus, these secondary abdominal disorders in themselves tend to destroy life.

Under these circumstances, our endeavour in treatment must be, after having provided, as far as possible, for the removal of the original cause of disease, to relieve the different organs of the congestions which oppress them, and to re-establish their proper secretions. We should especially pay

attention to the liver, and endeavour to maintain a free flow of bile; for we must remember, that the chief decarbonizing organs of the system—the lungs—are, in all probability, already seriously disordered, so that the proper function of the liver being rightly performed, is here of especial consequence. A few leeches applied over the organ, when hard and tender to the touch, generally give great relief; so also do the use of salines and mercurials. The gastric disorders arising from the congestion of the portal system, are very difficult to treat; but their importance in deteriorating the powers of life, by preventing nutrition being properly performed, is evident enough. They give rise to various symptoms of dyspepsia.

The congested state of the intestinal canal and of the kidneys, which is more or less their permanent condition in advanced stages of cardiac disease, not only impedes the proper assimilation of the food and the secretion of the urine (producing constipation and a diminished flow of urine), but it also interferes considerably with the application of the remedial measures which are requisite. By increasing the intestinal and renal secretions, we endeavour to get rid of the serous—dropsical—effusions which are pressing upon different important organs of the body; but on account of the congested states of these organs, it is necessary that we should be extremely cautious, both in the use

of purgatives and of diuretics, at least in the latter stage of heart diseases. The use of drastic purgatives should be avoided as far as possible, for their depressing effects on the nervous powers are very great, and they frequently occasion considerable injury to the mucous membrane of the bowels. The same may be said of the indiscriminate use of diuretics, in their effects upon the kidneys. The more gentle the means by which the different secretions can be maintained and increased, the more sure and beneficial is the result.

We must remember, that these congestions and serous effusions are accidents which, for the most part, are the attendants of heart diseases in their latter stages, and that, therefore, we have to deal with the system already enfeebled and worn out by long pre-existing disease; so that, in almost all cases, we must support and strengthen the body, while endeavouring to maintain the flow of its different secretions. The milder warm cathartics, blue pill, colocynth, and aloes, in different combinations, and saline medicines, are the best adapted for the relief of the intestines. The use of elaterium, croton oil, etc., should be reserved for those cases in which all other remedies fail to give relief; and when used, their administration should be carefully watched, and the system at the same time well supported.

There cannot be a doubt, that if due care and attention were paid to the general state of their

health, by the subjects of heart diseases, in the early stages of these disorders, much subsequent suffering would be avoided, and life prolonged. Unfortunately, it is not in the power of the many to bestow this care upon themselves, and, in the great majority of the cases which come into our hospitals, the subjects of them present themselves for treatment then only when they are actually prevented by physical impediments—the results of their bodily disorders—from further carrying on their daily work: that is to say, when the different disorders of the body, produced by the cardiac disease, are already far advanced, and therefore less amenable to treatment than they would have been at an earlier period.

It would be far more beneficial to the patient, that we should be called upon to anticipate these secondary disorders, than to remove them when established. I have already remarked upon the great benefits which may be derived from attention to proper prophylactic measures. These measures consist simply in due regulation of the diet, and in promoting the proper performance of the different secreting functions. The diet should be generous and nutritious,—sufficient, but not abundant: of fluids, great care should be taken that too large quantities are not imbibed; in fact, the usual quantity should be diminished, for repletion of the vascular system acts injuriously on the absorbing

powers, and also accelerates the heart's action, and aggravates the cardiac disease.

The starving system,—the system of Valsalva,—once highly recommended and long adopted in the treatment of cardiac disorders, is entirely pernicious, and now generally abandoned; it was founded on a complete misconception of the pathology of these diseases. Happily, it is now not necessary to do more than merely refer to this method of treatment. No specific laws can be laid down for the diet; it must be regulated according to the habits and condition of body of the individual affected. The manner in which the secretions may be best kept duly at work depends much upon the constitution—the idiosyncrasy—of the individual, and still more upon a due consideration of the progress which the disease has made when we are called upon to treat it. In an early stage, while the congestions are still slight, the secretions are readily managed; but at a time when their action is of greater importance, that is at later periods, when effusions have to be got rid of, the secreting organs are least capable of performing the work required of them. The secretions are locked up, as is metaphorically said of them; and this disturbance of the functions of the different organs causes them to react injuriously the one on the other. The case that so frequently meets us, where the bowels are confined, the liver torpid, the pulmonary

exhalant function ill performed, the urine dark-coloured and scanty; the skin hot, dry, and tense from serous effusions into the cellular tissue; and the vascular system generally, and the heart itself, oppressed by the fluids,—demonstrates to us the necessity of guarding, by anticipatory measures, against the occurrence of this condition of things. It is a condition most difficult to treat, now that it is fully established, but it is one that might have been provided against to a great extent, had it been anticipated.

In a case of this description, the first object is to relieve the congestion of the heart, the lungs, and the liver, and, until this is done, we can scarcely hope so to establish the renal and intestinal secretions, as to enable them to discharge the serous effusions. The action of the skin should be promoted by diaphoretics and vapour baths, and warm purgatives given to excite the flow of bile; small quantities of nourishing food being at the same time administered. By the use of expectorants, also, the pulmonary secretions may be re-established.

When the congestions are partially relieved, we may venture on diuretics, and so endeavour to excite the action of the kidneys: when these organs are not structurally diseased, the greatest benefit is frequently derived from the proper performance of their functions; and even when the urine is albuminous, and the structure of the organs degene-

rated, we may still derive this benefit through them. The injury which it has been supposed the diseased kidney suffers from the action of diuretics is problematical; and the fact is, that they are now much more freely given than was once the case, the good being manifest, and the injury very doubtful. As a tonic and diuretic in these cases, no medicine which we have tried equals the combination of digitalis and the sesquichloride of iron, given in bitter infusion.

The injuries which the cerebral organs suffer in consequence of heart diseases have been considered elsewhere.

CHAPTER XIII.

HYPERTROPHY OF THE HEART.

Pathology.—Natural weight, size, etc., of the heart.—Cause of hypertrophy.—Hypertrophy of the left side of the heart.—Hypertrophy of the right side of the heart.—Dilatation of the heart.—Active, Simple, Passive dilatation.—Causes of dilatation.—Consequences of hypertrophy and dilatation.—Action on the brain.—Physical signs of hypertrophy and dilatation.—Enlargement of the thyroid gland, etc.

By hypertrophy of the heart is understood, an increase of the natural amount of its muscular tissue; the structure of the muscular tissue, thus increased, being either perfectly healthy, or more or less degenerated. Hypertrophy may be partial, or it may affect every portion of the organ; it is in most cases associated with dilatation of the heart's cavities.

In order to arrive at an estimate of what may be properly called hypertrophy of the heart, it is necessary that we should possess some measure, by which to judge of its size and weight in health. In fixing such a standard, however, we must be contented with something approximative to the truth. Investigations into this subject show that the medium

weight of a healthy adult heart ranges between eight and ten ounces, and that the weight is constantly progressive with age—at least, in the male.

The measurements given by Bizot, of the average thickness of the walls of the heart's cavities, are as follows :*

	English inches. In men.	In women.
Thickness of the walls of the left ventricle at the base	0·43	0·36
Ditto at the middle	0·45	0·39
Ditto near the apex	0·31	0·28
Thickness of the septum of the ventricles at the middle	0·43	0·39
Thickness of the walls of the right ventricle at the base	0·16	0·15
Ditto at the middle	0·11	0·11
Ditto near the apex	0·08	0·08

And these are his measurements of the size of the valvular openings of the heart :—

	English inches. In men.	In women.
Width of the left auriculo-ventricular orifice	4·29	3·61
Ditto of the right	4·81	4·18
Width of the origin of the aorta above the valves	2·74	2·49
Ditto of the pulmonary artery	2·79	2·60

The cavities of the right side of the heart have also,

* These measurements of Bizot must be considered more accurate than those given by Bouilland. I have taken them as reduced in Dr. Stokes' work to English inches. The following are Bouilland's measurements :—

Thickness at base of	left ventricle	6 to 7	lines.
,, ,,	right ventricle	2½	,,
,, ,,	left auricle	1½	,,
,, ,,	right auricle	1	,,

like the orifices, a greater capacity than those of the left side.

The above measurements may be supposed to represent, with tolerable accuracy, the thickness of the walls of the heart, when its cavities are distended to their natural size; I say, distended to their natural size; and it is important to bear this in mind, whilst considering the subject of hypertrophy, for the reason, that when the healthy heart is much contracted, the walls of its ventricles necessarily become thicker than natural—that is, than the standard measurements above given of them. This contracted state of the heart, which is sometimes met with after death, has, indeed, been looked upon as abnormal, and the name of concentric hypertrophy has been applied to it; but the truth is, that the term hypertrophy is here—in most cases, at least—misapplied; the so-called hypertrophy being simply thickening of the ventricles, resulting from contraction of the heart. This condition of the heart is observed occasionally in anæmia and phthisis, and invariably, according to Cruveilhier, in the bodies of persons guillotined.

On the other hand, again, when the walls of the heart are of their natural thickness, and its cavities dilated, the heart—according to the definition of hypertrophy above given—must be considered as hypertrophied, inasmuch as that, in such case, the bulk of its muscular tissue is necessarily increased.

From which it would appear, that we cannot judge accurately of hypertrophy of the heart by measurements of its walls alone. We find, in one case, where the heart is contracted, that the ventricular walls may be thicker than natural, and yet quite healthy: and then again, when the heart is dilated, that it may be hypertrophied, although its walls are of a standard thickness. The most satisfactory mode, therefore, of judging of the hypertrophy of a heart, is by a consideration of its weight, as well as of the measurements of its parts.*

* The following observations on the weight of the heart in health and in disease are taken from a very instructive paper by Dr. Peacock. (*Edin. Monthly Journ.* 1854.)

"The average weight of the healthy heart in males, between 20 and 55 years of age, is 9oz. 8dr.; that in females of like age, 8oz. 13dr. The calculations, however, are arbitrary, for it is not easy to say at what size a heart ceases to be healthy.

"The average weight of the healthy heart in those who die of chronic and wasting disease is less than in those who die after a short illness. In cancer of the stomach and chronic affections of the liver, the heart will be found to weigh occasionally only five or six ounces; and in large men who have died suddenly, it may be twelve ounces. The heart increases in weight with advance of life; but, perhaps, in very advanced age undergoes a decrease.

"In phthisis, the weight is less than natural, but its wasting is counteracted by the impeded respiration causing its hypertrophy. In chronic bronchitis, it ordinarily acquires a considerable size; and in diseased kidney there is a tendency to enlargement.

"In twenty-four cases the heart weighed more than twenty ounces; and in all these, except two, there was present marked aortic or some other valvular disease. In one of the two, the heart weighing twenty ounces, there was slight atheromatous disease of aortic and mitral valves and of the aorta; and in

The following are the different kinds of hypertrophy of the heart generally adopted by authors:

1. *Simple hypertrophy*, which consists in a thickening of the walls of the heart, the cavities remaining of their natural size.

2. *Eccentric hypertrophy.* Here the cavities and the walls of the heart are both enlarged.

3. *Concentric hypertrophy.* In this form, the size of the cavities are diminished, and the walls increased, or of a normal size, or diminished in size (?).

When the hypertrophy is uncomplicated, it consists simply in an increase of healthy muscular fibres, the fibres presenting the ordinary appearances of the healthy muscular tissue.* But the hypertrophied tissue is sometimes partially degenerated, and then it presents, at those parts, the appearances characteristic of the particular degeneration which it has undergone. The proper walls of the ventricles, and the columnæ carneæ and papillary muscles, are generally all enlarged together, but sometimes one of these parts more so than the other. The complicated columnæ of the right ven-

the other, weighing forty ounces, chiefly hypertrophy and dilatation of right ventricle; there was no valvular disease, and no cause of death given."

* Cases in which the enlargement depends upon fatty growths on the heart are, of course, excluded from this calculation. I have thought it useless to make a subdivision of hypertrophy into *true* and *false*.

tricle are often remarkably increased, and out of proportion to the walls of the cavity; they are so large, prominent, and numerous, as sometimes to separate the cavity into several divisions. The nervous ganglia and the coronary arteries, also, increase in size at the same time.

The ventricles are more frequently hypertrophied than the auricles; and the left ventricle more frequently than the right. Dilatation of the cavities of the heart is almost invariably associated with hypertrophy of its walls; for the cause which induces the latter condition, naturally promotes the former likewise. Dilatation with hypertrophy of both the ventricles, is the condition of the heart most commonly met with in cases of advanced hypertrophy. With regard to the auricles, it may be observed, that their hypertrophy, unless when well marked, is readily overlooked, and not easily defined. Their exact measurements, moreover, have not been satisfactorily given. The extent to which hypertrophy of the heart may reach, can be gathered from this,—that the left ventricle has been known to have acquired a thickness of two inches, and the heart itself to weigh as much as five pounds.

When the hypertrophy of the heart is considerable, the natural form of the organ, and its position in the thorax, are altered. It loses its usual conical shape, taking a globular, and even a squarish figure, and becoming broader than it is long; the

ordinary prominent appearance of its apex being at the same time entirely lost. The position of the heart also is changed: it lies more transversely than natural, reaching away towards the left lateral region of the thorax; so that its beat, instead of being felt to the right of the left nipple, is felt far away to the left of it, and if the hypertrophy be great, below the sixth rib.

Hypertrophy of the heart may, practically speaking, be looked upon as a condition resulting from the presence—either within or without the heart—of some hindrance to its freedom of action; such a hindrance as the healthy natural organ is unable, for any long time, to strive against successfully,—that is, so as duly to supply the wants of the system. To overcome the impediment, the heart is, in the first instance, stimulated to extra exertion, and then, according to the principle *ubi stimulus ibi fluxus*, the stimulus provokes an increased supply of nutritive materials, by which the muscular tissue is multiplied and strengthened. Here, as in the homely instance of the blacksmith's arm, the increased demand for force gives birth to an increase of the nutrition which generates the force.

Hence it happens, that hypertrophy of the heart, though in itself an unnatural condition, is regarded by the physician rather as a wise effort of nature, striving to compensate for the defective condition of other parts, than as a disease tending to the

destruction of life. The importance, therefore, of a proper understanding of hypertrophy is manifest enough. The time has been, when the whole efforts of his art were employed by the physician, in the vain attempt to reduce this hypertrophy; or, in other words, in endeavouring to counteract the necessary provision which nature had, for an especial occasion, established. A better pathology teaches us to regard the hypertrophy—if not in all, at least in the very great majority of instances—as a secondary derangement—as the consequence of some other diseased condition; and teaches us also, that it is this other diseased condition, towards which our attention and our medical resources must be mainly directed.

We are perhaps hardly justified in speaking of simple uncomplicated hypertrophy. The existence of such a disease is problematical. Certainly, as a general rule, the hypertrophy may be traced to a distinct cause; and when we in any instance fail so to trace it, we may ascribe the failure, not to the non-existence of a cause, but rather to our inability to discover it. "Hypertrophy and atrophy of the heart," says Dr. Latham, "dilatation and contraction of its cavities, seldom, perhaps never, take place, but where some disease or unsoundness has previously existed, either in the heart itself or in other parts of the body, from which they are derived as a natural and necessary consequence; I say, per-

haps never, because the instances are very few in which such disease or unsoundness is not either already known during life or discovered after death; and in those very few where it is not apparent, either during life or after death, the probability is greater that we had not penetration enough to find it out, than that it did not exist."

Causes of Hypertrophy.—The above remarks naturally lead us to consider the diseased conditions which excite the hypertrophy, in other words its causes. These we shall find to exist both within the heart itself, and in parts external to, and even distant from, the organ. They consist, for the most part, of mechanical impediments to the circulation of the blood. In some few cases, the hypertrophy results from causes which we are not able clearly to trace out.

The chief causes of hypertrophy of the heart are found to exist within the organ, and consist in diseases of its valves, and in abnormal states of its orifices. Defective aortic valves, permitting regurgitation of the blood into the left ventricle during its diastole; constriction of the aortic orifice, impeding the free passage of the blood from the left ventricle during its systole; deficiency of the aortic valves, associated with constriction of the aortic orifice; defective mitral valves, permitting regurgitation of the blood from the left ventricle into the left auricle,—all these abnormal conditions occa-

sion impediments to the circulation of the blood through the heart, and their immediate effects are communicated directly to the left side, and indirectly to the right side, of the heart.

I have already said, that the left ventricle is the part of the heart most frequently hypertrophied, and this accords with the fact that diseases of the valves (such diseases, at least, as render them defective) are frequent on the left, and comparatively rare on the right side of the heart. When they exist, diseases of the valves and orifices of the right side of the heart occasion hypertrophy of the right ventricle, after the same manner as the diseases on the left side cause hypertrophy of the left ventricle.

But notwithstanding that the valves of the right side of the heart are rarely diseased, we find the right ventricle very often hypertrophied and dilated. The chief causes of this are the impediment to the pulmonary circulation which arises from chronic diseases of the lungs, or from defects of the valves in the left side of the heart. The latter are, perhaps, the most common causes of hypertrophy and dilatation of the right ventricle. Their action is exerted indirectly in the following manner. The disease of the mitral or aortic valves hinders the free passage of the blood from the lungs into the left side of the heart, and thus obstructs the pulmonary circulation; the consequence of this is, that the blood is thrown back upon the pulmonary

artery and right ventricle, and the right ventricle stimulated to extra exertion, in order to overcome the obstruction; and thus are occasioned its hypertrophy and dilatation.

After the same manner we may trace back, even still further along the current of the circulation, the consequences of valvular diseases of the left side of the heart, and, in doing so, we shall see how a hindrance to the passage of the blood through the aortic orifice may at length communicate its effects, even to the systemic capillary circulation at the most remote parts of the body. The impediment to the blood arising from aortic valvular disease, which we have already traced back as far as the right ventricle, is readily communicated to the blood passing from the right auricle, and from this again to the venæ cavæ, and so backwards along the venous system, even to the capillary circulation. Nor are the effects of the impediment lost even here. In consequence of the capillary circulation being obstructed, the blood does not flow readily along through the arteries; fresh force is required to drive it on. The heart should naturally supply the required extra force; the heart which, in the case supposed, is already labouring with extra exertion to overcome the obstruction at its aortic orifice. A new obstruction, secondary to and the consequence of the first, thus presents itself, and the left ventricle is again called upon for

a renewed increase of its efforts, at a moment when it is least capable of affording them.

Mechanical impediments to the passage of the blood through the aorta and large arterial trunks, also, are causes (external to the heart) which give rise to hypertrophy and dilatation, and their effects are the greater the nearer they are situated to the heart. They may result from contraction of the arterial trunks, whatever the causes, whether through pressure from without, or from disease within; from aneurisms, and dilatations, and atheromatous diseases, whereby the natural elasticity and contractile power of the vessel is destroyed.

Impediments, moreover, to the pulmonary or systemic capillary circulations, also, give occasion to hypertrophy and dilatation of the heart. Whatever interferes with the pulmonary circulation acts directly as a cause of active dilatation and hypertrophy of the right ventricle; thus, all diseases of the lungs which suddenly obstruct the pulmonary circulation, by destroying, or rendering useless for a time, a large amount of lung-tissue,—emphysema, cirrhosis with enlarged bronchi, particular deformities of the thorax, pleuritic effusions, pneumonia,—all these, and perhaps other affections, must be considered as sources of hypertrophy and dilatation of the right side of the heart. But no cause acts more effectually through the lungs in inducing this hypertrophy and dilatation of the

right side of the heart, than constriction of the mitral orifice.

In cases of uræmia, where hypertrophy of the left ventricle exists without disease of the valves or of the large vessels, the hypertrophy may be attributed to the impediment to the systemic capillary circulation, which results from the uræmic condition of the blood. The hypertrophy has been, but with less reason, in such cases ascribed to the stimulant action of the uræmic blood upon the heart itself.

What effect pericardial adhesions have in producing hypertrophy of the heart is not very clear. I have already alluded to this subject. Skoda and Rokitansky both assert that dilatation of the heart results from these adhesions, not hypertrophy.

Hypertrophy and Dilatation of the Heart, and diseases of its valves and orifices, are pathological states of the heart so closely blended together, that it is scarcely possible in practice to describe the consequences which ensue to the system from the one apart from the other. I shall, therefore, before proceeding to speak of the consequences of hypertrophy, describe shortly the pathology of dilatation of the heart and of its valves.

Dilatation of the heart is described as associated with different conditions of its walls:

1. With hypertrophy of the walls.
2. With normal thickness of the walls.

3. With thinning of the walls.

The first condition is that most commonly met with, called *active dilatation.* It is, indeed, merely one form of hypertrophy,—excentric hypertrophy,—but it is placed under the head of dilatation, because the dilatation predominates over the hypertrophy, being the most marked of the two pathological states. Active dilatation generally commences at one part, and gradually extends to the rest of the heart. The auricles often become the seat of active dilatation, especially when the auriculo-ventricular orifices are much constricted.

The second condition is *simple dilatation,* the cavities being dilated, and their walls of normal thickness. Here, however, it must be remarked, that, though the walls of the cavities be of normal thickness, the bulk of the heart's muscular tissue is increased, and the heart therefore heavier than natural: it is, in fact, hypertrophied.

The third condition is *passive dilatation,* the walls being relaxed and attenuated, and the cavity enlarged.

Occasionally, partial dilatations of a single cavity are met with, associated with alterations of the texture of the tissue forming its walls. They are known as *partial aneurisms* of the heart.

Of the forms of dilatation of the heart above-mentioned, the third, namely, *passive dilatation,* is the one which most demands our attention here.

The two first come more or less under the head of hypertrophy of the organ. In passive dilatation, the muscular tissue of the heart is soft and flabby, it easily tears, and is often found to have undergone partial fatty degeneration. The walls of the dilated cavity, when divided, collapse; if healthy and merely distended by the blood, they contract when the blood is removed, and become firm on the cavity regaining its natural size. Hereby we are able to distinguish between true dilatation and simple distension. When the auricles are dilated, they probably are also always more or less hypertrophied; their simple distension is a very common occurrence, and, in the majority of instances merely an accident belonging to the death struggle. Passive dilatation generally affects both ventricles together; the left ventricle is sometimes reduced to a third of its natural thickness, and at some points, particularly about the apex of the heart, its walls are occasionally so attenuated as to be diaphanous; the fleshy columns of the heart being separated and forced apart, leaving in the intervals the membrane of the heart only; the membrane there actually alone forming its walls. The right ventricle, however, is more liable to extreme thinning than the left. The septum of the heart is less affected than other parts. Dilatation gives the heart a peculiar globular appearance; it enlarges it in its transverse direction; the natural conical form of the organ is

entirely lost, and it looks almost as broad at its lower part (apex), as it does at its base.

The muscular substance of the heart is generally altered in its colour as well as in its consistence, in passive dilatation; sometimes it has a dirty rusty colour, and looks as if half boiled; and sometimes is covered with fat, having undergone, in parts, fatty degeneration. The alteration in colour depends upon the degeneration of the tissue, and the staining of the tissues results from imbibition of the colouring matter of the stagnating blood.

The orifices of the heart are frequently dilated in unison with dilatation of its cavities; that orifice in particular being dilated, which is most closely connected with the dilated cavity. The valves themselves, and their tendinous attachments, are generally enlarged at the same time, so as to enable them still duly to perform their functions; but they are not always so; and then regurgitation of blood occurs, although the valves themselves be healthy.

The *causes* of *dilatation of the heart* are, for the most part, the same as those which occasion hypertrophy of the heart; what has been said, therefore, concerning the causes of hypertrophy, applies generally here. Why, in certain cases, the dilatation should predominate over the hypertrophy, is not so evident. Perhaps, in the majority of such instances, we may attribute the fact to the altered or degene-

rated condition of the heart's muscular tissue, which, as we have seen, is so frequently associated with dilatation, at least, in its most marked—the passive—form. This altered condition of the muscular tissue, may be produced by an attack of inflammation to which the endocardium or pericardium has previously been subjected; or we may ascribe it to an originally ill-constitution of the heart, or to some low condition of the body, arising from great losses of blood, or from wasting diseases, etc. All of these causes tend to weaken the firmness and cohesion of the muscular tissue, and some of them to alter its anatomical structure; and hence, when any impediment to the ready performance of the heart's functions arises, in cases where any of these causes happen to be in operation, the muscular walls of its cavities, instead of overcoming, by an increased vigour of contraction, yield to, the extra pressure within them, and dilate. Had the tissue been healthy, hypertrophy would rather have resulted from the impediment.

How the muscular tissue is affected by the inflammation, which we have supposed to induce its softening and degeneration, is plain enough. The chief causes of dilatation, as before mentioned, are, mechanical impediments to the free circulation of the blood; and I have shown how these may act directly or mediately on any cavity of the heart. Now the chief of these mechanical impediments are

connected with the heart itself, and result from diseases of its valves and of its external membrane. These diseases, again, are the results of inflammation, of endocarditis, or of pericarditis; and endocarditis and pericarditis affect the muscular tissue, and in a greater or less degree. In some cases, we may imagine that they so affect its nutrition as to render the complete return of its tissue to a healthy state impossible: they leave it damaged, and in that state which, as we suggested above, readily gives rise to dilatation of the heart, when impediments to the circulation obstruct its freedom of action.

It is in this manner that pericarditis and endocarditis are said to be causes of hypertrophy and dilatation of the heart. For obvious reasons, we may understand, that the muscular tissue is more liable to be affected in cases of pericarditis, where the disease is extensive and of long duration, than in cases of endocarditis.

Deposition of fat upon the heart, and between its muscular fibres, may give rise to passive dilatation of the heart, by causing atrophy or attenuation, or degeneration of the muscular tissue.

Consequences of Hypertrophy and Dilatation.—The most important pathological conditions associated with hypertrophy and dilatation of the heart are, congestions and extravasations of blood in different parts and organs of the body, together with

effusions into the serous cavities, and general dropsical symptoms. These morbid states, however, are, for the most part, the consequences of the same causes as those which produced the hypertrophy and dilatation,—namely, the obstructions to the circulation of blood through the heart,—and will therefore be more properly spoken of in the chapter which treats of these obstructions.

It is evident that hypertrophy and dilatation of the heart must be, in themselves, productive of certain effects,—that is, must give birth to certain pathological conditions in the system, quite independently of the causes by which they themselves were provoked; but it is very difficult to state, with precision, what the effects are, inasmuch as they are inextricably mixed up with those resulting from the causes producing the obstructions to the circulation. We must, therefore, arrive at a knowledge of these effects rather by a process of reasoning, than by any positive demonstration that can be obtained of them.

It is not possible to doubt, for instance, that an hypertrophied left ventricle has a direct and injurious influence on the brain: we can hardly imagine that the blood can be incessantly driven onwards, and with violence, into the delicate vessels of the brain, without exerting in them an unusual distending power, and therefore also, a certain degree of pressure upon the brain. "Hypertrophy of the

left ventricle," says Dr. Burrows, "must be admitted as a powerful predisposing, or even exciting cause to apoplexy and sudden hemiplegia. Simple hypertrophy of the left ventricle may continue a long time, and it will cause an increased activity in the general circulation: the blood will be thrown into the cranium with more than usual force; there will be a more rapid transit of the blood through the cerebral arteries. But, after a time, this constantly increased force of the left ventricle will have the effect of dilating the cerebral arteries, and thus of overcoming the healthy elasticity of their tunics. Congestion of the cerebral arteries now ensues, and the coats of the dilated vessels no longer protect the surrounding cerebral substance from the inordinate momentum of the blood propelled from the left ventricle. Apoplectic coma is now very likely to ensue, from some sudden accidental increase of vascular pressure on the brain. Cerebral hæmorrhage is also likely to occur; for we know, that in advanced periods of life, this hypertrophy of heart is often associated with disease of the coats of the arteries of the brain."

I would observe, however, that this *vis a tergo* of the hypertrophied heart, which forces the blood towards the brain, is not nearly so powerful an agent in the production of serous effusions, cerebral congestions, and extravasations of blood, as is any obstruction seated in the heart, which impedes the

return of the blood from the brain; and we must also recollect, that in almost all cases of hypertrophy of the left ventricle, whatever acts as the cause of the hypertrophy, sooner or later acts also indirectly as a cause of obstruction to the return of the blood to the heart. Hence it follows, that the brain, in hypertrophy of the left ventricle, is sooner or later almost invariably subjected to two injurious influences,—namely, the extra impulse of the heart which forces the blood towards the head, and the obstruction (seated in the heart, and acting through the venous sinuses, the jugular veins, and the superior vena cava) to the return of blood from the head.

When, therefore, under these circumstances, the brain suffers damage, it becomes impossible to decide how much of the damage is to be attributed to the obstruction, and how much of it to the violence and force of the arterial circulation produced by the hypertrophied ventricle.

The force of the arterial impulse also is considerably modified by the nature of the cause producing the hypertrophy, and so likewise is the degree of venous obstruction. Thus constriction of the aortic orifice will act indirectly as a powerful cause of obstruction to the return of blood to the heart, but it will diminish the direct effects of the contractions of the hypertrophied ventricle (which it has itself provoked) upon the brain. Still greater, in such

case, would the obstruction be, if, at the same time, together with constriction of the aortic orifice, there existed deficiency of the mitral valves, permitting regurgitation of the blood through them; and still less forcible would be the direct effects of the hypertrophy on the brain. The arterial impulse is likewise modified by the defective, as well as the constricted, condition of the aortic valves. The regurgitation of the blood through the aortic orifice manifestly diminishes the force which would be excited by it on the brain, through the action of the hypertrophied left ventricle, if the valves were sound; and it must be remembered, that where the left ventricle is much hypertrophied, the aortic valves are almost invariably diseased.

The fact would seem to be, that the direct action of the hypertrophied heart on the different organs is less injurious than the venous obstruction which generally accompanies it. Its pathological importance in this respect therefore, has, perhaps, been somewhat exaggerated. It would be very difficult to prove (and, reasoning from analogy, scarcely possible to believe), that the hypertrophied action of the heart ever sufficed of itself to rupture a healthy vessel of the brain, or even to produce coma and apoplectic symptoms.

Hypertrophy of the right ventricle of the heart was looked upon, both by Laennec and Dr. Hope, as a cause of pulmonary hæmorrhage, of apoplexy

of the lungs, as it is called. The violence of the contraction was such, in their opinion, as "to overcome the tonic power of the vessels", and hence ensued their rupture and extravasation of blood into the pulmonary tissue. Now, here again, the suggestion already offered, concerning the direct effect of hypertrophy of the heart on the capillary circulation of the brain, may be applied, and still more forcibly. The cause producing the hypertrophy of the left ventricle acts directly as obstructive to the return of blood from the lungs; the consequence of which obstruction is hypertrophy of the right ventricle, arising from its efforts to overcome the impediment to the pulmonary circulation. Now, surely, the direct opposition here offered by the obstruction to the return of blood into the left side of the heart must be considered as much more likely to effect rupture of the pulmonary vessels, and so occasion pulmonary hæmorrhage, than the hypertrophy simply of the right side of the heart. The degree of the hypertrophy is a measure of the pulmonary congestion; it is a provision arising from the necessity of the case and adapted to it. Can any one imagine that hæmorrhage would ever take place if the obstruction were removed?

Certainly, the increased impulse given to the circulation of the blood through the lungs by the hypertrophy, must aid the tendency to the rupture of the pulmonary vessels, and thus far may be con-

sidered a cause of hæmorrhage when it occurs; but it is clearly quite secondary to that of the obstruction to the circulation.

We cannot practically consider the consequences of dilatation of the heart apart from its hypertrophied state, except in the case of that form of it called passive dilatation. The consequences of passive dilatation are those which result from a weakened condition of the heart; we shall not refer particularly to them now, for they necessarily are modified by the cause which excites the dilatation, and on this subject we shall have more to say further on.

Hypertrophy and Dilatation of the Heart: Physical Signs.—The local physical signs which indicate to us the existence of hypertrophy and dilatation of the heart consist in alterations of its healthy sounds, in respect of their loudness, clearness, and duration; in alterations of the natural force of the heart's impulse, and of the situation where, and of the extent of surface over which, it is perceptible; and in alteration of the natural extent of dulness produced by percussion over the præcordial region. In considering these particulars, it is necessary always to remember, that the healthy heart in different individuals presents often great variations in its sounds and in the degree of its impulse; and, moreover, that many of these alterations of sound, impulse, etc., which, under certain circumstances, become signs of disease, frequently arise

as consequences merely of temporary derangements of the heart's action.

Hypertrophy and dilatation of the heart increase the extent of præcordial dull percussion sound, and in proportion to their degree. When the dilatation exceeds the hypertrophy, the dulness increases chiefly in the transverse direction; and when the reverse of this occurs, it increases in the direction from above downwards, that is, of the long diameter of the heart. The dulness may reach over many inches of surface—indeed, when a very considerable degree of hypertrophy and dilatation exist together, over as much as five or six inches; whilst in health the dulness does not extend beyond an inch and a half to two inches. The dulness arising from hypertrophy and dilatation of the heart, may be distinguished from that which results from pericardial effusions, by an examination of the history of the case, by the attendant symptoms, and by the fact, that the dulness of hypertrophy increases in a direction downwards and towards the left side; the dulness arising from effusion rather upwards. The resistance felt on percussion also is much more marked in pericardial effusion than in hypertrophy.

It must not be forgotten, while considering this subject, that pneumonic and tubercular infiltrations in the lungs, where they surround the heart, may render the præcordial percussion sound abnormally

dull; and, on the other hand, that the dull percussion over an hypertrophied heart may be diminished by an emphysematous condition of the lungs, which causes their borders to overlap the heart to an abnormal degree;—a condition, in fact, which we find very frequently associated with enlargement of the right side of the heart. Under such circumstances, indeed, the natural amount of dulness is very frequently actually diminished. The same results may ensue when, through any cause, the thoracic cavity is rendered larger and wider than natural, so as to allow great expansion of the lungs.

Percussion does not allow us to distinguish between hypertrophy and dilatation of the heart; both states give a like percussion sound. The dulness increases laterally when the right ventricle is hypertrophied and dilated; and rather in the direction of the heart's length, when the left ventricle is hypertrophied and dilated, and the right ventricle of normal size.

When the ventricles of the heart are hypertrophied and dilated, its *impulse* is almost invariably increased in force; frequently, indeed, to such an extent, that the forcing heaving beat of the heart resulting therefrom, is perceptible to the eye as well as to the hand, and raises the head of the auscultator, when placed upon the chest, The impulse, however, may be weak, and even imperceptible, notwithstanding the heart be hypertrophied;

for rapidity and completeness of the heart's contractions are necessary, to produce a strong impulse; and these conditions are not always present in cases of hypertrophy and dilatation of the heart.

An hypertrophied ventricle is not of itself sufficient to produce a strong impulse; in fact, when the ventricle is hypertrophied and contracted, the impulse of the heart is weak. The impulse of a simply hypertrophied heart does not produce the strong, slow, extensive heaving of hypertrophy and dilatation; it has more the character of a quick, sharp blow.

The impulse of the heart is stronger than natural when its walls are dilated without thinning; but it is weaker when the walls at the same time are abnormally attenuated: a ventricle thus dilated, has not sufficient force to expel its contents rapidly and completely.

When the right ventricle is hypertrophied and dilated, the impulse is often very great about the xiphoid cartilage, and at the lower portion of the sternum, and sometimes is communicated to, and strongly perceptible, over a considerable portion of the right side of the liver. When the left ventricle is hypertrophied, the impulse is felt more towards the left side; but it must be recollected, that the cause which excites hypertrophy of the left ventricle, sooner or later produces, in a secondary manner, hypertrophy of the right ventricle also. If one

ventricle be much hypertrophied and the other of normal size, the impulse will be but little increased in force.

Hypertrophy and dilatation of the heart, associated with considerable contraction of the aortic orifice, may give rise to a feeble impulse; and for the reason, that the heart's contractions in such case are not complete. When the contraction is such as to admit of the complete expulsion of the blood from the ventricle, then the impulse is forcible and prolonged.

On the subject of the *treatment* of hypertrophy and dilatation of the heart, we need not linger. For what may be said hereon, I refer the reader to the treatment of valvular diseases, and of the secondary disorders thence resulting. We have seen that hypertrophy and dilatation of the heart are almost invariably, if not invariably, the consequence of valvular and other diseases; their treatment, therefore, necessarily becomes a part of the history of the treatment of such diseases.

Before concluding this chapter, I may add a few words respecting a series of symptoms which appear to have some especial connexion with an hypertrophied and dilated condition of the heart. I refer to the swollen state of the thyroid gland, the violent action of the cervical arteries, and enlargement of the eyes, found in association with permanent excitement of the heart. This affection has been

particularly investigated by Dr. Graves and Dr. Stokes; and it is from the account given of it by the latter, that the following summary of its history is gathered. I will only observe, that the subject is one which still requires further investigation; the conclusions which have been arrived at concerning it, being based upon a rather slender series of observations.

The condition above stated, of irregular, rapid, and increased action of the heart, associated with throbbing of the cervical vessels, and enlargement of the thyroid glands and the eyeballs, is unattended with any sign of cardiac inflammation. It is generally seen in females, connected with hysteria and uterine disturbance; rarely in men; and occurs at any age above puberty. The symptoms have remissions, which appear to depend upon the state of the heart's action. The enlarged thyroid offers a throbbing pulsation, and the usual physical signs of aneurismal varix. These signs may be partial or general, varying in intensity at different parts of the tumour, and at different periods of the disease; with the progress of the disease, as the gland becomes more solid, they partially subside. Venous murmurs may be heard occasionally, over and around the tumour. The enlargement of the eyeball varies at different periods, and is not necessarily attended with any loss of vision. The essence of the disease, according to Dr. Stokes, consists in

functional disturbance of the heart, which is followed by organic changes; the morbid states which have been observed in fatal cases, are dilatation and hypertrophy of the heart, enlargement of the inferior thyroid arteries, and dilatation of the jugular veins.

CHAPTER XIV.

FATTY DISEASES OF THE HEART.

Fatty growth on the heart.—Symptoms.—Fatty degeneration of the heart.—Pathology.—Symptoms.—Diagnosis.—Prognosis.—Treatment.—Rupture of the heart.—Symptoms.—Treatment.—Atrophy of the heart.—Displacements of the heart.—Causes.—Consequences.

Fatty Growth on the Heart.—A certain amount of fat is found, in health, deposited in and around the heart, and chiefly along the course of the coronary arteries and their branches. Sometimes this fat accumulates to a morbid extent, that is, in such abundance, as by its presence to interfere mechanically with the free action of the organ. This morbid accumulation of fat has received the name of *fatty growth* on the heart, to distinguish it from another morbid condition of the heart, namely, fatty degeneration of its muscular tissue. The fatty growth is more developed on the right than on the left side of the heart. It may be associated with a perfectly healthy state of the muscular structure, and this even when the fat has been deposited between its muscular fasciculi. Generally speaking,

however, when the growth of the fat is considerable, the muscular tissue is at the same time more or less altered, the fibres being softened and atrophied, and encroached upon by the fat around them.

There is no absolute connexion between general obesity and fatty growth on the heart; but the two conditions are frequently found to co-exist, so that when other symptoms lead us to expect the presence of fatty growth on the heart, the existence of general obesity strengthens the diagnosis. Obesity presents itself under two opposite forms: sthenic and asthenic. *Sthenic* being simply hypertrophy of the fatty structure, occurring in the prime of life; and *asthenic*, the growth of fat, which occurs in advanced life, in broken-down constitutions, connected with decay of the vegetative life, with depravation of the blood, and with weakness of the eliminating and assimilating processes. In the sthenic form, the fat is accumulated chiefly about the base of the heart; in the asthenic, it collects towards the apex, and is deposited at the expense of the muscular tissue,* rather than accumulated upon it. The existence of the asthenic form is more to be suspected in those who, having been always thin, become suddenly fat in the decline of life, than in those who have been always fat.

* "Plus un cœur est surchargé de graisse, et moins, en général, ses parois ont d'épaisseur." Laennec, *Ausc. Med.* iii, 167.

Of sthenic obesity, we may say: That it exerts a predisposing influence to fatty degeneration, but that it may long exist unassociated with any structural change. Asthenic obesity is, on the other hand, closely associated with fatty degeneration, and where the one condition exists, we may suspect the co-existence of the other likewise. (H. Jones. Ormerod.)

Symptoms.—A considerable amount of fat may be deposited upon the heart's surface without interfering with its functions; but when large collections of fat take place upon, or are infiltrated into, the muscular tissue, an atrophous condition of the muscular fibres at least results. Fatty degeneration is thus frequently associated with fatty growth on the heart. Hence we can hardly speak of the symptoms of fatty growth apart from symptoms of atrophy, or degeneration of the muscular tissue of the heart. We do not, in fact, possess a clinical history of the symptoms simply of fatty growth on the heart to which we can trust. The cases described by the older writers are valueless, inasmuch as we have no proof that in them the muscular fibres were intact and sane. The symptoms present in one case, well observed by Dr. Walshe, were: sensation of oppression at the præcordium, difficulty in walking quickly, cold extremities, feeble pulse, giddiness, weak cardiac impulse, extensive dulness on percussion over the heart, whose sounds

were weak; in fact, the symptoms were those of a heart impeded in the performance of its functions.

The *Treatment* of this disease, when there is reason to believe that it is associated with fatty degeneration, is that of fatty degeneration of the heart. When from the general obesity and otherwise healthy condition of the body, fatty growth on the heart alone may be supposed to exist, the treatment must be such as is proper for the reduction generally of the fatty condition of the body, and this consists mainly in regulation of the diet and in exercise.

Fatty degeneration of the Heart.—This form of fatty disease differs entirely, in its pathological characters, from fatty growth on the heart. Here the intrinsic change consists, not in a mere deposit of fat on or between the fibres of the heart, but in a morbid condition of the muscular fibre itself; its sarcous elements being converted, wholly or partially, into fatty matters. When the muscular fibre, thus diseased, is viewed under the microscope, we find that its sharp, well-defined outlines are lost, that its striæ have disappeared, and that oily granules occupy the place of the striæ and their intervals. These changes seem to take place gradually, and we therefore meet with the disease in different stages of progress; when the disease is far advanced, whole fibres appear fused together,

and lose all trace of resemblance to the natural tissue. The degeneration may be partial, and it may be very general; it more frequently affects the tissue of the left than that of the right ventricle of the heart; but both ventricles are generally affected together. The auricles appear to be rarely injured by the degeneration.

When the degeneration is extensive and has reached a somewhat advanced stage of its progress, the naked eye detects alterations in the physical appearances of the heart. Instead of the bright red colour of health, it presents a pale, fawn, yellow, dirty, mottled aspect. The firmness of its structure is lost, it is soft and friable, and is torn or ruptured with great facility; its ventricular walls are loose and flabby, and fall together when incised, even though hypertrophied. When rupture of the heart occurs, it is almost invariably in connexion with its fatty degeneration.

Fatty degeneration of the heart is met with in persons of all ages, but occurs most frequently at advanced periods of life. All classes of society are subject to it; the luxuriously fed and the ill-nourished appear equally obnoxious to its slow and insidious attacks.

Sometimes the fatty degeneration constitutes the only perceptible diseased condition existing in the body after death, but more generally it is associated with other morbid states: with fatty states

of other organs and parts, with diseases of the kidney; with atheroma of the arterial trunks and their valves; with hypertrophy—but more frequently dilatation—of the heart; with pathological indications of previous attacks of pericarditis and endocarditis; with emphysema of the lungs, etc., etc. The fatty degeneration appears in some way to be especially associated with ossification of the coronary arteries; the two diseased conditions being very frequently found existing together. Often, indeed, where the heart's tissue is at one part healthy and at another part degenerated, that part which is degenerated is found connected with a diseased branch of the coronary artery. It is possible, however, that the two diseases are not related as cause and effect, but are both rather the results of the same general deteriorating influence.

Fatty degeneration is thus found to be co-existent with many and very different diseases of the heart, and of other organs of the body, and, occasionally, to be the only morbid condition present. It cannot be said, indeed, that any manifest connexion has yet been traced between fatty degeneration and other diseases. The frequency of its occurrence, and the difficulty of ascertaining its relations to other diseases, may be inferred from the fact, that in one hundred and forty-three cases in which the heart, taken indiscriminately after death, was examined microscopically by Dr. Ogle, it was found

degenerated one hundred times. In many instances, we are forced to regard it as a local disease.

The cause of this degeneration—this atrophy of the muscular fibre of the heart—this morbid state of its nutrition—this conversion of its elementary contents into oily particles—the mode of commencement of the metamorphosis—whether the cause be local or general—are facts which science has yet to elucidate. Dr. H. Jones, who has paid much attention to this subject, believes the cause of the disease to consist in a lowered vitality of the affected tissue, and does not regard general or constitutional as its chief causes, for the reason that these general causes very frequently exist when it is absent, and *vice versâ*, that the disease is present when those causes are absent.

Corvisart was well aware of the distinction to be drawn between fatty growth on, and fatty degeneration of, the muscular tissue. Modern pathology, by the aid of the microscope, has rendered the demonstration of the fact more positive, but has added little to his practical exposition of it. He says, speaking of "degeneration graisseuse" of muscular tissue, that inasmuch as the parts affected preserve their natural shape and size, the alteration cannot be attributed to any accumulation of fat *on* the degenerated parts. And, again: the fatty nature of the change in the muscles is evident, because they offer many of the physical and chemical pro-

perties of fat, and because weakness of the muscular power is the necessary result of the degeneration, and stands in a direct ratio to its extent. This morbid condition, he tells us, though he himself had never seen a case, had been observed in the muscular structure of the heart by different physicians. He draws a marked distinction between this condition and fatty growth: "This fatty transformation," he says, "must be distinguished from another pathological state, which appears to resemble it . . . . that state, in which the heart is found oppressed, and, as it were, stifled by an enormous quantity of fat, by which it is entirely enveloped." (*Mal. du Cœur*, c. iv, art. 3.)

Symptoms of Fatty Degeneration.—We have seen that the pathological condition which marks the existence of this disease of the heart, consists in the degeneration of its muscular fibres. Thereby the physiological action of the fibre is diminished in force or destroyed, and the contractile power of the heart, consequently, more or less enfeebled, in proportion to the extent of the degeneration which its tissue has undergone. In accordance with this, we find that the chief symptoms attending this condition of the heart, are those which indicate an enfeebled condition of its contractile power.

These symptoms are: general debility, incapacity for exertion; shortness of breath, sometimes slight, sometimes very severe; weakness of the digestive

powers; stifling sensations, with flutterings, occasionally felt over the heart, accompanied with more or less pain. The pulse is weak, and so also the impulse of the heart; the heart's sounds are feeble, and there is an absence of cardiac bruits.* The cardiac pains, when united with shortness of breath and syncope, form that combination of symptoms which represents angina pectoris; and there can be no doubt that fatty degeneration of the heart existed, and was overlooked, in many of the cases of angina formerly described, in which no lesion was discovered after death. The cerebral disturbances attending fatty degeneration of the heart, vary between a passing giddiness and complete coma; they are, for the most part, but slight and transient, and quickly pass away. They may be attributed to the weakened action of the heart, which, under slight disturbing influences, becomes incapable of properly carrying on the cerebral circulation. Sometimes the attacks are severe, and then the patient may fall as if struck by apoplexy; but the weak pulse, the pale features, the unaltered pupils, the absence of stertor, the advanced age of the patient, serve to distinguish the attack from true apoplectic seizure. Moreover, the attack is rarely followed by paralytic symptoms.

The value of "arcus senilis" as a sign corrobora-

* They of course may be present, but then they have no necessary connexion with the fatty degeneration.

tive of fatty degeneration, has been overstated. The great frequency with which it is met, in the most healthy individuals in advanced life, justifies us in considering it as little more than a natural consequence of old age. What its value may be in earlier life, has yet to be shown.

Diagnosis.—There are no positive signs which indicate the presence of this disease; but in its advanced stages, and when it occurs at a late period of life, the rational signs of fatty degeneration are, for the most part, well marked, and leave little doubt concerning its nature in the mind of the careful observer. In early life, and when the degeneration is slight, the diagnosis must be conjectural, and thus the disease may be readily overlooked. It must be observed, that fatty degeneration of the heart, in an advanced stage even of its progress, has been found in persons, after death, in whom during life no symptoms had excited suspicion of its existence; perhaps, however, symptoms might have been discovered in these cases had they been carefully sought for* by medical observers.

Diagnosis of this disease must be mainly based upon a consideration of the symptoms and signs which result from, and are indicative of, an en-

* How frequently it happens that patients, with lungs in an advanced stage of disorganization, will tell us, that they have never suffered from any disturbance of the respiratory functions!

feebled heart,—enfeebled through diminution of its muscular power, and so rendered incapable of duly regulating the movements of the blood, and supplying the wants of the system. These symptoms have been already described. Judging by their light, and by the absence of those auscultatory and percussion signs, which positively indicate the existence of organic disease of the heart, we may in the great majority of cases arrive at a tolerably sure decision as to the nature of the disease.

We have already given reasons why, practically, no distinction can be drawn between the signs of fatty growth on the heart and its fatty degeneration.

The *prognosis* of the disease must be unfavourable. Whenever it has arrived at such an advanced stage as, by symptoms, to indicate its existence, we may be sure that the degeneration of the muscular tissue is great, and, as far as we know, its repair is impossible. Our prognosis must be drawn from a consideration of the severity of the symptoms; of the social position of the patient, whether it enables him or not to submit to strict hygienic rules; and from the effect which treatment seems to have in arresting the progress of the degeneration.

Treatment.—We have already said, that the relation of fatty degeneration to other diseased conditions of the body is obscure; nevertheless, we must, in practice, regard the local disease as the

consequence of some depraved condition of the assimilative powers, whether acting immediately upon the heart or generally throughout the body. Certain it is, that the disease is often present in those who have suffered under lingering and wasting diseases; that it is generally associated with various abnormal states of different organs of the body; and that, for the most part, it arises as the vigorous age of life passes away. It is an atonic disease.

The following, in accordance with these views, are the main objects towards which our treatment must be directed: first, to prevent the further progress of the degeneration of the tissue (its repair being impossible), by removing all causes which tend to exhaust and weaken the bodily or mental powers, and to improve the nutritive materials of the blood; and, secondly, to regulate the habits of life of the patient, so as to protect him, as far as may be, against the dangerous consequences which such a condition of the heart ever makes imminent over him.

For the first object, a generous and moderately stimulating diet, pure air, moderate exercise when it can be taken without inconvenience, avoidance of large quantities of fluids, due regulation of the digestive organs; tonic medicines, particularly steel in bitter infusions,—are to be recommended. The particular nature of the diet must be decided by

the habits of the patient, and his powers of digestion. The use of oils has been proscribed; but, I think, on the very insufficient grounds of their fatty nature. I believe I have seen cod-liver oil, combined with steel, followed by very satisfactory results in supposed cases of fatty degeneration of the heart.

The paroxysmal pains demand antispasmodics; when very severe, small local abstractions of blood and blisters sometimes relieve them. The cerebral and syncopal symptoms must be treated by internal stimuli, and by the application of counter-irritants to the surface of the body.

Violent bodily exertion and mental emotions should be carefully avoided: all acts, in short, which call upon the heart for sudden and unwonted energy of function. The heart, it must be remembered, is inordinately weak; it may possess power enough to carry on quietly the ordinary circulation, but a slight extra strain upon it throws the organ suddenly off its balance, and ofttimes so as to endanger and even destroy life.

Rupture of the Heart.—When rupture of the heart is not caused by external injury, it is for the most part known to us as one of the consequences of fatty degeneration of its muscular tissue. The accident may, indeed, result from other causes, such as from an abscess in, or aneurism of, or hæmorrhagic effusion into, the walls of the heart;

from their ulceration, dilatation with attenuation, etc., but these causes are very exceptional. Under all circumstances, we may be sure that whenever rupture takes place from any internal cause, the muscular tissue is not in a healthy condition. Corvisart, indeed, tells of some rare cases, where rupture had occurred through violent excitement in persons perfectly healthy: but in such cases the muscular tissue of the heart was, in all probability, partially degenerated; it is impossible to imagine that muscular rupture, such as occasionally occurs to the abdominal muscles in tetanus, can happen to the healthy heart.

Ruptures of the heart are more frequent in the male than in the female sex, and occur generally at advanced periods of life; in these particulars they resemble fatty degeneration of the organ. All parts of the heart are subject to laceration: the ventricles, the auricles, the septum, the papillary muscles and their tendons; but of all parts, the ventricles are most liable to the accident, and the left ventricle more so than the right. The rupture is in most cases single, but occasionally it is represented by several small lacerations; the passage it forms is generally direct, but now and then it runs obliquely and sinuously through the walls of the cavity. The rupture may occur gradually, the blood insinuating itself between, and progressively forcing its way through the softened muscular

fibres, but this probably happens rarely, the rupture being generally complete at once. The most frequent seat of the laceration is about the middle of the anterior part of the left ventricle, near the septum; rarely is the posterior part affected. Laennec made the remark, that the rupture seldom occurs at the heart's apex, where the walls have least force. It is impossible to decide whether the rupture takes place during the systole or the diastole of the heart. Ruptures of the valves and their tendons are probably in almost all cases the results of endocardial inflammation.

The immediate cause of the rupture may in most cases, though not in all, be traced to some violent or sudden strain upon the heart's action, such as in its enfeebled deteriorated state it is unable to sustain. But the main cause is to be sought in the diseased condition of its muscular structure.

Death is generally, but not always, the immediate result of the rupture. Corvisart refers to a case of ruptured heart caused by external injury, where the patient survived until the twenty-third day the infliction of the injury. The sufferer often lingers on for many hours, and appears to die at last not so much from loss of blood (for this can rarely be very great, on account of the firm membranous nature of the pericardium), as from the pressure exerted upon the heart, and the arterial trunks arising from it, by the blood which has escaped

into the pericardium. By this pressure its action is so interfered with, that it becomes incapable of carrying on the circulation. When death does not follow immediately upon the occurrence of the injury, we must suppose that the escape of blood from the heart takes place gradually and at intervals; and in such case the rent in the heart's walls is probably oblique, or it may be partially or temporarily obstructed by a coagulum. Cases like that given by Corvisart, and in which firm coagula have been found in the opening, suggest the possibility of the rent being permanently healed under favourable circumstances.

Symptoms.—The symptoms will rarely ever enable the physician to predict with certainty the nature of the injury which has occurred; for they are such as may result from other diseases of the heart and its great vessels. They indicate direct, sudden, and serious interruption to the heart's action. When death is not instantaneous, the patient complains of violent, oppressive, and stifling sensations at the præcordia,—of pains shooting through the chest; he is very pale; his skin is cold, and covered with clammy sweat; the pulse weak and fluttering, or, it may be, regular; the countenance expressive of intense anxiety and suffering; faintings, syncope, and convulsions sometimes supervene before death occurs. Dr. Stokes remarks with perfect truth, that precisely similar symptoms to these may attend

death resulting solely from a weakened heart. Little faith can be attached to increased præcordial dulness on percussion in cases of rupture of the heart.

Treatment.—Towards remedying such an injury as rupture of the heart, medicine has little to offer. And besides this, while life lasts, we can never be certain of the real nature of the injury which has produced the symptoms. The urgency and character of these symptoms can alone guide our treatment. To sustain, by stimuli, the system under the violence of the shock which it has undergone; to relieve the obstructed circulation of blood through the heart, and at the same time to tranquillize its action,—these are the indications which the physician must endeavour to fulfil, when opportunity is given him. In the majority of cases, the effusion of blood is rapid, and life is at once destroyed; so that it is only in those cases where the effusion takes place gradually, that we can entertain any small hopes of success.

Atrophy of the Heart.—This condition of the heart consists mainly in a diminution of its size. "In atrophy," says Dr. Latham, "the muscular substance is less red than natural, its carneæ columnæ less developed, and its proper fibrous texture less distinguishable. But there is still the appearance of a muscle shrunk and withered, as if from an insufficient supply of nourishment." (*Lectures*, vol. ii, p. 179.) Like the voluntary muscles, the

heart diminishes in size and in power, when long subjected to causes which produce general wasting of the body. Atrophy of the heart is, in fact, observed in connexion with wasting diseases, as in carcinomatous affections and occasionally in phthisis. The valves of the heart, as well as its muscles, have been found thin, contracted, and atrophied. Ossification and adhesions of the pericardium have been spoken of as sources of atrophy, but their exact relations with it have not been fully made out.

There are no auscultatory signs pathognomonic of this condition of the heart. The pulse at the wrist, and the heart's impulse, are feeble; the area of præcordial dulness is diminished perhaps, but such a sign, as diagnostic of the atrophy, is manifestly here of no account. This condition of the heart, indeed, can hardly be classed as a disease, being rather one of many consequences resulting from a wasting disease; and it will probably remain undiscovered until after death. Its *treatment* is necessarily merged in, and has a distinct reference to that of the wasting disease which has produced it.

Displacements of the Heart.—The attachments of the heart allow considerable freedom to its movements, and permit of its being readily displaced from its natural position in the thorax, by pressure exerted on it from without. It may be displaced towards either side, and it may be forced upwards or downwards. The most common cause of its

lateral displacement is pleuritic effusion. Effusion, of whatever nature, occupying the cavity of one of the pleuræ, compresses the lung, and pushes the heart towards the opposite side of the thorax. Simple hydrothorax, however, rarely produces displacement; for, unlike pleurisy, it is generally double, and the fluid being simultaneously and equally collected in the two pleuræ, presses alike on either side of the heart, and so retains the organ in its proper site. As occasional causes of its displacements in different directions, may be mentioned—solid and fluid tumours, aneurisms, extensive emphysema, and pericardial effusions.

The heart is forced upwards in ascites; and also, as it has been said, may be drawn upwards and towards one side, in consequence of the diminution of one of the lungs, produced, for instance, through pleurisy or tubercular disease—the fluid having been absorbed in the former case, leaving a permanently compressed lung; and a large tubercular cavity having contracted and cicatrized in the latter. The heart, in such cases, passes towards the affected side. It may, however, be fairly suggested, that the enlargement of the healthy lung—which has become unduly developed, to compensate for the loss of the spoilt lung—may at all events aid in the displacement of the heart, by pushing the organ towards the injured side.

The most common cause of its displacement

downwards is double emphysema: single emphysema, which must be a very rare affection of the lungs, would push the heart laterally. When thus displaced, the heart is felt strongly beating at the epigastrium. But it must be remembered, that emphysema of the lungs, and enlargement of the right side of the heart, are constantly associated together, and for obvious reasons; hence, we have an explanation of the epigastric pulsation, independent of any displacement of the heart; the pulsation here felt being produced by the hypertrophy of the right side of the heart. It is needless to enumerate the possible displacements of the heart through pressure of morbid growths.

The displacements of the heart do not, as a rule, cause any alteration in its sounds; but it is conceivable, that such an altered direction may be thereby given to its arterial trunks, as to occasion a bruit so long as the displacement lasts: such a case has been related by Dr. Walshe. It is possible also, that the character of the first sound may be modified, when the impulse of the displaced heart no longer adds its natural share to the production of the sound. The sounds will naturally be heard loudest, and the heart's impulse be felt strongest, at that part of the thoracic walls beneath which it lies; and thus, and by the percussion signs, we are enabled to ascertain the fact of the displacement and its direction.

It must not be forgotten, that its hypertrophy causes apparent displacement of the heart: when much enlarged, for instance, its apex reaches away to the left of the nipple, and may be felt beating in the left lateral thoracic region—the heart taking a transverse direction.

It is difficult, if not impossible, to calculate the effects produced on the circulation and respiration, through displacements of the heart, for the causes themselves—the pleuritic effusion or the emphysema, for example—which produce the displacements, in almost all cases, create disturbances of those functions: the effects of the displacements, and of their causes, are thus inextricably mixed up together. *A priori* we may reasonably argue, that displacement of the heart must interfere with the integrity of its movements, and that consequently it must, in a greater or less degree, add to the embarrassment which the circulation and respiration already suffer, through the cause which produced the displacement; it is, however, surprising to find, in practice, how greatly the heart may be pressed from its natural site, without apparently adding, in any marked manner, to the difficulties of the circulation or the respiration; and this, too, even when the displacement takes place suddenly. This fact is very striking in certain cases of chronic empyema, where the change has taken place slowly; but then the heart adapts itself gradually to the

pressure, and its duties are diminished,—the quantity of blood circulating through the lessened capacity of the aerating surface of the lungs being proportionably diminished.

The treatment of these displacements is, of course, the treatment of the particular morbid conditions which produce them, and will therefore not occupy our attention here.

CHAPTER XV.

ANGINA PECTORIS.

Pathology.—Connexion with fatty degeneration of the heart, and disease of the coronary arteries.—Resemblance between the symptoms of the diseases.—Its nature.—Treatment.

THE exact condition of the heart which occasions that peculiar and striking combination of symptoms, to which the name of angina pectoris has been given, has not yet been positively determined; but the obscurity which once hung over the pathology of the disease has been, in great part, removed by the researches which have thrown so much light into the history of textural degeneration of the heart.

The obscurity which thus involved the nature of this disorder arose from the circumstance, that, though the heart was manifestly its seat, no peculiar abnormal conditions of the organ were discovered to which it could be especially traced. The angina was found to accompany every one of the known diseases of the heart, and it was found to be present in cases in which, after death, the organ

to all appearance seemed perfectly healthy. Later investigatious, however, to which we have alluded above, tend to show that organic disease of the heart is, in all probability, never absent when the symptoms of angina pectoris are present ; and, also, that there is no pathological condition of the heart to which we may more reasonably ascribe those symptoms, than that of fatty degeneration of its muscular tissue,—a disease which, though long ago described by authors when seen in its exquisite forms, required the modern aid of the microscope to demonstrate its existence in its less developed condition. Before the microscope lent its aid to medicine, it must, therefore, have been continually passed over unrecognized by pathological observers.

I have said, that angina pectoris had been observed to exist in association with all the then recognized abnormal states of the heart. It has been, indeed, at various times, ascribed by different observers to each one of those abnormal states ; that particular diseased condition being selected for its cause, with which the observer, in his own experience, had happened to see it most frequently associated.

One particular abnormal state of the heart, however, namely, ossification of its coronary arteries, was found more frequently than any other to exist in connexion with symptoms of angina ; and was consequently generally considered to be the cause

of the angina. Now, it so happens, that a very close relation exists between ossification, or partial obstruction, of the coronary arteries, and fatty degeneration of the muscular tissue of the heart: the latter condition being very frequently associated with the former. The fatty degeneration is, indeed, sometimes confined to that particular portion of the muscular tissue, to which an ossified branch of the coronary artery is distributed.

The connexion of angina pectoris with this fatty condition of the heart is likewise confirmed by the symptoms of the disorder. The symptoms of a well-marked case of fatty degeneration much resemble those of angina pectoris. Sudden, violent, rending, oppressive pain at the epigastrium, extending more or less over the thorax, and even down the left arm; a weak and almost imperceptible pulse; shortness of breath, amounting to choking and suffocation; occasional syncope and coma, and even death itself,—are all symptoms alike of fatty degeneration of the heart and of angina pectoris. And there are other points of resemblance between them. Both disorders are rare, comparatively speaking, in females; both occur chiefly at advanced periods of life; both may be associated with every kind of organic disease of the heart; an attack in both is generally induced by excitement of the mind, or extra-exertion of the body,—by causes which suddenly excite and in-

crease the heart's action. In both, during the intervals of the attacks, the patient seems tolerably free from disorder; in both, one attack is pretty surely, sooner or later, followed by another, and on each occasion recurs at shorter intervals and with an increase of severity; both have, sooner or later, but one issue, and that a fatal one. The treatment in both is alike; antispasmodics and diffusive stimuli during the attack, quietude of mind and body, nourishing diet, and careful regulation of the animal functions during the intervals. Therefore, that there is some close connexion between the symptoms called angina and fatty degeneration of its structure seems more than probable; but that all cases of angina depend upon fatty degeneration is not certain.

The characters of an attack of angina pectoris, its sudden onset, and the apparently total disappearance of the disease at its close, have induced observers to consider it as spasmodic; in fact, to ascribe the symptoms of the angina to a spasm of the heart. This view of its nature is, however, purely hypothetical, and certainly it is hard, *à priori*, to understand, how such a thing as spasm of the heart—that is, a spasmodic contraction of its muscles—can be compatible with existence. A much more satisfactory and reasonable account of the angina suggests itself upon a consideration of the general symptoms of the disease, and a compa-

rison of them with those pathological conditions of the heart, with which, as above related, we almost invariably find the disease associated, namely, with fatty degeneration of the muscular tissue of the organ.

Here we have weakness of power arising from an irremediable change of structure, and can readily imagine how, under such circumstances, an extra call upon that power may induce incapacity, more or less complete, of function. For the ordinary purposes of tranquil life, the power of such a heart may suffice to carry on the circulation; but when an extra stress is laid upon it; when, either through bodily exertion or mental excitement, an unwonted quantity of blood is forced towards the heart, its powers prove unequal to the effort required of it, the blood accumulates, the circulation is more or less arrested, respiration is necessarily affected, and then the train of painful suffocative symptoms—called angina pectoris—ensue; and coma, and syncope, and death, if at last the heart be unable to struggle out of the violence of the attack. And be it remembered, "bodily exertion is the most frequent and the most certain exciting cause. In the vast majority of cases upon record, the first paroxysm has arisen while the man was making some strenuous effort." (Latham.)

And thus, the pathology and the whole history of angina tend to shew, that it is the result of a

suddenly arising incapacity of the heart to carry on the circulation effectually: and that the incapacity depends upon weakness of power: and that the weakness of power is the result of the degeneration of the muscular tissue of the organ, or of its atrophy.

That angina may be the product of spasm of the heart, and of nervous affections, it is impossible to deny, and great authorities have given their names to such an opinion; but it is hardly necessary, in the present state of our knowledge on the subject, to call those agencies into question. The effects of nervous influence on the heart in health are manifest enough, but no one now-a-days can believe—for what facts are there which warrant the belief?—that any nervous influence can so excite or affect the heart as to produce the symptoms of angina, unless the structure of the organ be itself in some way seriously compromised, that is, unless the excitement act upon the heart already diseased. The pathology of the disease, and its history, are quite opposed to the idea of the angina being a spasm of the heart; they both tend to demonstrate the existence of an opposite condition. The angina occurs generally in the leucophlegmatic, and in those advanced in life: the heart, after death, is almost invariably found softened, flabby, fatty, attenuated, and dilated, and never spasmodically contracted, such as it has been seen, for instance, in tetanus.

And, after all, do we not meet with some of the chief symptoms of angina in the advanced stages of most heart diseases, whatever their kind, when the disease has arrived at that stage at which the heart's action begins to fail, the blood to accumulate in it, the respiration to become suffocative,—in fact, when the death struggle has commenced? Surely, there is here the same rending pain, the same constriction over the heart, the same choking respiration, and then the syncope and the coma. The difference between the two cases is in this,—that the angina arises suddenly, and suddenly passes away; in these other heart diseases, it is the continuation of a struggle, and the violent termination of it. But there is in this nothing to contradict the idea, that the actual state of the heart which produces the symptoms may be alike in both, so long as the struggle lasts. In the one case, the heart for a season regains its former state, the paroxysm passes away, and the patient recovers; but he recovers only to be again and again the victim of his malady,—each attack being more severe and more readily excited. In the other case, the paroxysm is the last effort of the enfeebled heart. In the one case, the struggle occurs at intervals; in the other, it is continuous. In the one, it is the result of actual degeneration and destruction of the muscular fibre; in the other, the muscular tissue

remains healthy, but its nervous energy, or contractile power, is destroyed. In both, the resulting consequences—the attack—are alike.

We may observe, moreover, that an attack of angina pectoris, attended with all its characteristic signs, is a thing which rarely meets the physician's eye: Heberden speaks of it as being "not extremely rare." Neither can there be any doubt, that the number of recorded cases of so-called angina pectoris is much fewer now, since science has advanced the pathology of heart diseases, than it was some few years ago. "Many cases," as Dr. Stokes observes, "denominated angina pectoris by one physician, would be called cardiac asthma by another."

From what has been here laid down, it naturally follows, that there is little to be said concerning the treatment of the disease angina, as such. In most instances, the attack itself has passed away before the physician can interfere. Its nature, however, is such as evidently to demand for the patient the immediate administration of diffusive stimuli,—among which, warm brandy and water, the compound spirits of sulphuric æther, and aromatic spirits of ammonia, rank first in utility and efficacy. If the pain be great and lasting, opium in full doses, repeated at short intervals, until relief is given, should be administered; for it must be remembered, that pain itself will kill at last: gene-

rally, however, the attack has passed away before the opium has had time to act. Stimulant applications to the epigastrium and the feet, and external frictions, must be also used during the attack.

When the attack has passed away, the condition of the organs, and the general state of health of the individual affected, should be taken into consideration by the physician; and upon this consideration his proceedings will be founded. In most cases, the treatment should be that which supports and invigorates the system generally, and tends to counteract or arrest that ill-nutrition, which is supposed to be disintegrating or weakening the tissues of the heart. The disease is atonic, not sthenic; and he who has once suffered from it is ever after liable to a repetition of the attack; for the symptoms indicate the existence of a disease whose cause is irremediable. All sources of mental excitement, and violent bodily exertion, must be carefully avoided, for these are by experience found to be the chief provoking agents of an attack. Intercurrent diseases or disorders of the body—valvular and other diseases of the heart, for example, dyspepsia, gout, pulmonary affections, and many other disorders—will demand particular attention, and may modify the general treatment.

To sum up the treatment in a few words: during the attacks, the heart's embarrassed action must be promoted and sustained by the most energetic and

rapidly-acting stimuli; and in their intervals, any accidental disorder cared for, and the general health preserved, by strict attention to proper hygienic rules; all promoting and exciting causes of disease being most carefully avoided.

CHAPTER XVI.

CYANOSIS.

Nature.—Pathological states of the heart accompanying it.—Chiefly a congenital affection.—Symptoms.—Effects on different organs.—Physical signs.—Treatment.

THE term Cyanosis, strictly speaking, implies a blue discoloration of the skin; and in such sense, all diseases which prevent the return of the blood from the systemic circulation, and produce discoloration of the skin, through venous congestion, may be classed under this head. The use of the word, however, is more restricted, and serves to indicate the existence of certain defective anatomical conditions of the heart, in which the blueness of the skin is one of the most constant and striking phenomena; the defective organization of parts being more or less connected with original malformation, and therefore congenital.

The discoloration has generally been ascribed to the fact of the admixture of arterial and venous blood, which takes place in consequence of the existence of some unnatural communication between the two sides of the heart, or between its

great vessels. Some obscurity, however, still hangs over the subject. More recent observation tends to the conclusion, that the blue discoloration does not depend so much upon this admixture of blood as upon the venous congestion which is associated with it—a conclusion, which is supported by the fact, that cases of defective organization of the heart, permitting of free mixture of the arterial and venous blood, are constantly met with, in which cyanosis in not present, provided the pulmonary artery is free and patent; and by this fact also, that whenever cyanosis exists in a marked degree, constriction of the pulmonary artery is almost invariably associated with the defect of the heart. It is certain, moreover, that no lesion can act more powerfully and directly, as a cause of systemic venous congestion, than contraction of the pulmonary artery, or of its orifice. Cases also of marked cyanosis have been observed, where no communication existed between the two sides of the heart, and therefore no admixture of blood was possible.

Thus it appears, on the one hand, that the most complete admixture of the arterial and venous blood may occur—as in the case where the heart possesses only a single ventricle — without producing cyanosis, provided the pulmonary artery be free; and again, on the other hand, that marked cyanosis may attend a constricted pulmonary artery,

when no communication whatever exists between the two sides of the heart.

The most probable conclusion to be drawn from the facts before us, when they are carefully analyzed, seems to be, that the chief promoting cause of cyanosis is obstruction to the systemic venous circulation, produced by contractions of some one or more of the heart's orifices, and, in particular, by contraction of the pulmonary artery or its orifice. Free mingling of the venous and arterial blood within the heart, may promote the cyanosis; but the intensity of the cyanosis and accompanying symptoms, seem to bear a distinct relation, not to the extent of communication which may exist between the two sides of the heart, but to the degree of impediment which the above-mentioned constrictions oppose to the circulation.

There does not appear to be anything in the general history of the disease which contradicts these views. That the mingling of the venous and arterial blood may assist in the production of the cyanosis, seems probable enough; but it is not easy, or perhaps possible, to decide what is the actual share it takes therein. It has been asked, as an objection to the opinion that cyanosis is commonly the result merely of venous obstruction, how it happens that "the most intense venous obstruction may arise in the adult, without inducing cyanotic congestion?" The question may be answered by

saying that the two cases do not admit of comparison: the intense venous obstruction here spoken of, is most probably the result of rapidly-induced disease; while the cyanosis, being of ancient date, the quantity of the blood, and the demand for its supply, have gradually accommodated themselves to the condition of the body. Moreover, it is very probable that, in the case of cyanosis, the extreme vessels of the surface have undergone physical changes, and have become gradually enlarged, so as to admit of ready and rapid distension, to a degree which can be hardly supposed to attend an ordinary case of venous obstruction.

The most ordinary pathological conditions of the heart met with in association with cyanosis are, the permanent opening of the foramen ovale, and of the ductus arteriosus; the openings being such as to permit of the free passage of blood between the two auricles in the one case, and between the pulmonary artery and the aorta in the other; for we must remember, that both these parts may remain unclosed to a certain extent, without giving rise to any abnormal sign whatever, provided the orifices of the heart and the trunks of the great vessels be not contracted. Patency of the ductus arteriosus seems always to co-exist with an open foramen ovale, and with perforation of the ventricular septum. Besides these, we meet with the following conditions in connexion with cyanosis:

deficiency of the intra-ventricular septum; malposition of the origin of the aorta; the origin of the pulmonary artery and aorta from one ventricle, the heart being in such case formed of one auricle and one ventricle only; transpositions of the great vessels,—the aorta arising from the right, and the pulmonary artery from the left ventricle; the origin of the aorta and pulmonary artery from both ventricles simultaneously, the semilunar valves being in such case very defective and ill-formed.

Together with these abnormal conditions of parts, there almost invariably co-exists contraction of some one or more of the orifices of the heart, or of its great vessels; of which, constriction of the pulmonary artery is the most frequently met with. These contractions seem to determine, in many, if not in all cases, the existence of the cyanosis, and the admixture of the blood through the abnormal opening: as, for instance, through the foramen ovale and ductus arteriosus; and they do so by obstructing the current of blood. When these parts remain open to a certain degree, and no constriction of the orifices exists, admixture does not seem to take place.

Cyanosis is for the most part, but not entirely, a congenital affection. It has occasionally been known to appear at a late period of life; and in such case, has been explained by the supposition, that some violent action of the heart has suddenly enlarged

an already existing patency of the foramen ovale; or that the intra-ventricular or intra-auricular communication has resulted from rupture or ulceration of the septa between the auricles or the ventricles. It has been known also to follow as the consequence of a violent blow over the heart.

Symptoms.—The discoloration varies much, both in its intensity and in the extent to which it affects the surface of the body. It may be limited to certain parts, and it may be general; it is most striking in those parts which present naturally a ruddy hue, as the cheeks, the lips, the nails, the inside of the lips, etc. It varies much also in the same individual at different times, becoming much more marked than ordinary when the heart's action is excited by muscular exertion, or mental emotions, or when any intercurrent pulmonary affection produces impediments to the respiratory function. The temperature of the surface of the body is generally lower than natural, and the physical and moral energy of the patient defective. The extremities of the fingers are said to take the clubbed form so commonly observed in phthisical subjects.

These symptoms vary much in degree in different individuals: a fact explicable by the circumstance of the different kind of abnormal changes from which they result, and the extent which the abnormal changes have reached, and of the degree

of obstruction to the circulation thence resulting. In one person, the discoloration may be, under ordinary conditions, scarcely perceptible; in another, constantly well marked; in one, the physical and moral powers seem to retain their usual energy, even to advanced age; in another, these powers are always feeble, and life fails at an early period. In the one case, we may suppose that the abnormal condition is such, that the circulation meets with little obstruction in the heart under ordinary circumstances, and that the mixture of the venous and arterial blood occasionally occurs under extraordinary circumstances only; and in the other, that the physical defect causes great obstruction to the circulation, and is constantly in action. Where the physical defects are considerable, life seldom is prolonged.

Symptoms of pulmonary obstructions and, in fact, of congestions of the organs of the abdomen and brain, are frequently observed as attendants of the cyanosis. The pulmonary affections shew themselves under the forms of catarrh, hæmorrhage, and bronchorrhœa. The attacks of dyspnœa come on generally in paroxysms, and are not constant. The heart is also enlarged, and its impulse rapid and extensive, and its palpitations violent; faintings, convulsions, and, occasionally, sopor and partial coma, indicate the effects of the disease upon the brain.

The *physical signs* must necessarily correspond to, and therefore vary with, the physical defects which have produced the cyanosis; they present to us nothing specially diagnostic of the nature of those defects. Hypertrophy, with dilatation of the right ventricle of the heart, is almost always present, the thickness of its walls being sometimes greater than that of the left ventricle. A systolic bruit is often heard about the base of the heart, and occasionally a thrill is perceptible at the same part; both the bruit and thrill probably arise in such case from the constriction of the pulmonary artery's orifice. It is possible, also, that a bruit may arise from the passage of the blood through the foramen ovale. Bruits may also be present, arising from constriction of the arterial and auriculo-ventricular orifices.

Treatment.—Of this there is little to be said. Inasmuch as the cause of the disease is irremediable, all that the physician can do is by prophylactic treatment to endeavour to ward off the affections of the lungs and the brain, etc., above described, which result from the physical defect provoking them. Our chief object, for which purposes, is to instruct the patient to avoid over much muscular exertion and mental excitement (which, from the nature of the complaint, he is instinctively inclined to do); to be strictly cautious in his diet, and attentive to the ordinary hygienic rules regarding

clothing, food, and exercise. The particular secondary disorders which arise, namely, congestions, etc., of different organs, must be treated according to the principles indicated under the head of valvular diseases.

CHAPTER XVII.

DISEASES OF THE AORTA.

Pathology.—Consequences which result from them.—Aneurisms.—Aneurisms of the thoracic aorta.—Their situation and form.—Their effects upon the parts around them.—Their diagnosis.—Physical signs.—Auscultatory signs.—General symptoms.—Dysphagia.—Pain.—Pressure upon the vessels around.—General health.—Course.—Prognosis.—Treatment.

THE present state of our knowledge enables us to say very little, which can be turned to any practical account, concerning *inflammation of the aorta.* Its existence, as a disease, is hardly demonstrable during life, and the signs which it leaves after death are involved in much obscurity. With respect to the pathology of the inflammation of the inner membrane of the artery, we may refer the reader to what has been said concerning the pathology of endocarditis. Certain deposits and thickenings which are met with in the vessel, and alterations of its innervation and contractile power, may be considered as the occasional resultants of such inflammation.

Atheromatous, cartilaginous, and calcareous

deposits, are very frequently met with in the aorta, and the conditions under which they occur appear to be similar to those which give rise to like abnormal deposits in and around the valves of the heart, whose pathology has already been treated of. A certain degree of atheromatous degeneration appears to be almost constantly present in the aorta at advanced periods of life, so much so as to make the change appear rather a natural mode of decay of the tissues, than as the consequences of an active disease; and aneurism, we may here incidentally observe, is a disease not of youth but of advanced periods of life. These deposits sometimes occupy continuous large portions of the inner surface of the vessel; and when the deposit is extensive, and of a calcareous nature, it may even convert the vessel into something approaching to a tube, with firm unyielding walls; very large scales of calcareous matter spreading over and lining the inner surface of the vessel.

Now all these different deposits, whether the consequences of degeneration or inflammation, undoubtedly have the effect, in a greater or less degree, of injuring the proper tissues which form the vessel; and of destroying or diminishing the elasticity proper and natural to it. The elasticity of the vessel is destroyed in two ways; namely, by the presence of the hardened deposit itself, and by the effects exercised by the process which produces

the deposit, upon the elastic and other tissues of the vessel, injuring their nutrition and innervation, —injuring, in fact, their structure.

The consequence resulting from the loss of elasticity of its coats is *dilatation of the aorta*—enlargement of the calibre of the vessel, particularly of the first and second portions of its arch, which is most directly and especially exposed to the dilating force of the blood thrown into it during the heart's systole, and is more subject to disease than other parts of the aorta. The dilatation of the vessel generally affects its whole calibre, but is most marked, perhaps, in the upper part of the arch, there where the current of blood impinges with most force; sometimes the vessel is weaker, and so more readily yields at some points than others, and little pouches, as it were, may be seen projecting from the external surface of the generally dilated aorta. The large arterial trunks which rise at a right angle from the first part of such a dilated aorta, almost always participate in the dilatation; the left subclavian, which passes off at an acute angle, does not so participate.

A certain degree of obstruction to the circulation results from such loss of elasticity and dilatation of the aorta; and thence arise, to overcome and compensate for the obstruction, hypertrophy and dilatation of the heart. The whole office of forcing the blood onwards is now thrown upon the heart.

In health, the contractile power of the aorta materially aids in carrying on the circulation ; the aorta is distended by the blood which is thrown into it during the systole of the heart, and then, during the diastole of the organ, its contractile force is brought into play ; re-acting upon the blood within it, the elastic vessel forces the fluid onwards in the direction of the circulation, the backward flow of blood into the ventricle being prevented by the closure of the semi-lunar valves.

Dilatation of the aorta, we may suppose, arises thus from the forcible and distending action of the blood which is thrown into the diseased vessel during the contractions of the left ventricle of the heart—the vessel itself having become, as it were, passive and paralysed through the injury which its contractile powers have received from the inflammatory or degenerative processes, which gave rise to the abnormal deposits.

Obliteration of the aorta need not detain us here ; it must be considered as wholly or partially a congenital malformation.

The aorta when healthy is found unnaturally contracted in certain cardiac and pulmonary diseases ; in those, for instance, in which a due amount of blood is not thrown into the vessel at each systole of the heart ; as when there is obstruction to the entrance of the blood into the left ventricle, or to the pulmonary circulation in chronic diseases, etc.

Pressure, also, from without, may reduce the calibre of the aorta ; consolidated exudations of lymph, for example, may compress its ascending portion, and so also tumours, abscesses, etc., situated in the vicinity of the vessel. The calibre of the vessel has also been found diminished by an excessive growth of calcareous matter projecting into it, and occasioning portions of the fibrin of the blood to become entangled about and deposited upon its roughened surfaces.

There are no particular physical signs or symptoms, pathognomonic of these diseased conditions of the aorta ; they do not, of themselves, give rise directly to any signs by which their presence can be diagnosed : indirectly, they occasion hypertrophy and dilatation of the heart, and in the manner above described, by producing obstruction to the cardiac circulation ; these, therefore, are the chief signs which indicate the existence of those conditions to us.

Roughnesses on the inner surface of the aorta, when considerable in amount, will occasion arterial bruits ; but it must be remembered, that atheromatous or calcareous degenerations can hardly affect to any extent the first portions of the aorta, without likewise involving more or less its valvular apparatus, and so giving rise to systolic or diastolic bruits. Occasionally, however, a systolic bruit is heard louder about the upper part of the sternum

than it is over the midsternum, that is, near the aortic valves. Such a bruit may be found associated with dilatation of the aorta and with roughness of its internal surface, without any co-existing disease of the aortic valves or the aortic openings: in which case, we must ascribe the bruit to the diseased condition of the first portions of the aorta; the blood in its passage over the roughened surfaces giving rise to the bruit heard.

Aneurism of the Thoracic Aorta.—It is not easy to give a distinct definition of the term aneurism; for all enlargements of the aorta are, in one sense, aneurismatic enlargements, and therefore dilatation of the aorta, as above described, might be included under that head. Practically, however, there is very little difficulty in coming to a right understanding as to the meaning of the word aneurism; we can readily enough determine the nature of the disease when it is placed before us as an object of pathological anatomy. Considerable dilatation of the whole circumference of a portion of the aorta must certainly be called an aneurism, whatever its form, whether globular or fusiform; so also must all distinct bulgings or enlargements of the vessel which affect only a part of its circumference.

No practical advantage can be gained by dividing and subdividing these different enlargements of the aorta into a variety of genera and species, according

to the form and position which they possess in relation to the vessel; for it is certain that aneurisms may arise at any portion of the aorta, and at any points in its walls, though some parts are more frequently than others subject to its invasions.

An aneurism, therefore, is a partial or general bulging of any portion of the artery. Now the very fact of this enlargement of the vessel conveys with it the idea of some imperfection in the condition of the tissues of the vessel, which permit of such an enlargement. We may, indeed, set it down as a rule, that the coats of the vessel where such aneurismal swelling appears, cannot be in a normal state. Pathology, moreover, has taught us, that the existence of aneurism in any part of the arterial system, for the most part indicates a disordered condition of the vascular system generally throughout the body. The surgeon well knows, that in the case of those particular aneurisms which are the objects of his operations, it is not so much the mere aneurismal swelling of the part, as the general diseased condition of the affected artery on which he operates, and of other parts of the arterial system, which he dreads to encounter. He fears to find the vessel diseased at the point where he operates, and that there may likewise be lurking, yet undiscovered by him, aneurisms of the arteries in other parts of the body. Such weakness of the vessel at one point indicates a weakened condition

of the arterial system everywhere throughout the body.

Thoracic aneurisms present themselves to us under different forms or shapes, and are found at different parts of the aorta. Their most common situation is in the ascending and transverse portions of the aorta; much more rarely are they met with in its descending portion. The reason of this may reasonably be ascribed to the circumstance, that the parts of the aorta nearest to the heart are more frequently found in a diseased condition than those at a distance; and that when the aorta is generally and extensively affected, the disease is more pronounced in that portion of the vessel which is immediately connected with the heart. It is not necessary to linger over a description of the different shapes which aneurismal tumours may assume, for these are evidently accidental circumstances in no way affecting the pathology of the disease. I will only observe, that the swelling may embrace the whole circumference of the tube, and that it may project from some one part only of its walls.

The condition of the walls which constitute the aneurismal tumour differs much in different cases. The three coats of the vessel, which in health form its proper tunics, may remain unbroken and form the covering of the tumour; or the inner membrane may be torn or destroyed, and leave the two

outer tunics of the vessel to form its walls; or the sac may be constituted of the inner and outer tunics, the middle coat being removed by disease; or the inner and middle coats of the vessel may be destroyed, and the aneurismal sac formed of the outer membrane only; or the outer coat may be destroyed, and then the sac consists of the two inner tunics of the vessel; and, lastly, the two inner tunics may be partially or totally ruptured, and the blood escaping therefrom may separate the outer tunic from them, and so produce a large diffused and extensively spreading tumour, called dissecting aneurism. The contents of the aneurismal sac are either fluid or solid; almost always we find within it layers of coagulated fibrin and fluid blood, the blood of course communicating with that circulating in the artery. Sometimes the layers of coagulated fibrin are so thick as nearly to fill up the sac, and render it solid; and in some rare cases, small aneurismal tumours of the aorta have thus seemed to have been cured. The layers of fibrin are drier and firmer, the nearer they are to the walls of the sac; those lying in contact with the blood are generally loose and soft, and have a shaggy-looking surface. Coagulation of the fibrin within the sac, seems to be favoured by smallness of the aperture of communication which exists between the vessel and the interior of the sac projecting from it.

It may readily be imagined, that with the progress of the disease, and the enlargement of the tumour, the condition of the walls which form the aneurism alter, and that they undergo considerable changes. One kind of aneurismal pouch (as above described) may thus become converted into another kind. Indeed, it is not easy to make clear and distinct the anatomical divisions of the sac after death, when the disease is of some standing; for, as we have already seen, the arterial coats are in such cases almost always much altered from the healthy state, independently of the changes which they have suffered through the progress of the aneurismal swelling; the divisions, therefore, above given of the constitution of the sac in aneurisms must not be looked upon as unexceptional, or as of any great service to us in practice.

The mere pathological anatomy of thoracic aneurisms, which we attain a knowledge of only after death, is of very little avail to us during life. From the nature of the case it necessarily happens, that then only when the disease has made considerable progress, and is in fact completely established as such, do we discover its existence during life. Of much more importance to us than the form and constitution of the aneurismal sac, is a knowledge of the effects which it produces on the parts and organs with which it comes in contact during the progress of its growth. From a consi-

deration of these effects we mainly form our judgment, as to the existence of a thoracic aneurism, previously to the moment when physical signs appear, and render the diagnosis more sure.

Thoracic aneurisms, as we have already mentioned, are most frequently met with in the ascending portion and arch of the aorta, and are situated almost invariably on the anterior or lateral surfaces of the vessel. In growing, they naturally tend to enlarge in that direction which affords least opposition to their expansion. When the aneurism is small, it may excite no disturbance in parts around it; and thus it not unfrequently happens, that an aneurism is discovered after death, whose existence had never been suspected during life. Now it may be said, generally, that all the different effects produced by the growth of a thoracic aneurism depend upon the same cause,—namely, the mechanical pressure of the tumour exerted upon the organs and parts around it. This pressure may displace the parts around the tumour; or it may interfere with their functions; or it may destroy and injure their structures: it may excite inflammation in them, and its consequences; and by irritation of nerves, give rise to spasmodic and reflex muscular actions, and to great pain.

By pressure on the trachea, or one or other of the bronchi, respiration is interfered with, and cough of a very irremediable nature excited. This

cough seems, in many cases, to result directly from the irritation of the recurrent nerve, produced through the stretching of it by the tumour. The tumour not unfrequently perforates the trachea or one of the large bronchial tubes, and sometimes suddenly bursts into them, and so causes instant death by suffocation. By its pressure upon the lungs, the tumour excites inflammation of, and effusion of lymph into, the pulmonary tissues; sometimes it destroys the tissue, and gives rise to pulmonary hæmorrhage, thereby producing death. It also occasionally excites extensive pneumonia and pleuritis, which carry off the patient. By its pressure on the œsophagus, difficulty of swallowing is produced, and the tumour may, indeed, thus cause the symptoms of œsophageal stricture;* the pressure on the œsophagus may at length produce ulceration and destruction of a portion of the tube, and destroy life by occasioning a fatal hæmorrhage.

The tumour may also press upon the large roots of the arteries which take their rise from the aorta, and partially obstruct the circulation of blood through them, by distorting them and pushing them from their proper places, or causing a deposition of fibrinous plugs within them; and so, also,

* This is one of the many cases which warn the surgeon of the great consideration required, in practising catheterism of the œsophagus: the mischief which may result from such interference in these cases is manifestly extremely great.

it may prevent the return of blood to the heart, by obstructing, more or less completely, the large venous trunks which open into the organ. The office of the thoracic duct may likwise be destroyed by the pressure of the aneurism upon it. When the tumour advances towards the surface of the body, it destroys, by its incessant pressure, the bones and soft parts which form the walls of the chest. Inflammation of the tracheal and bronchial membranes may also be mentioned as direct or indirect results of the presence of the tumour. When the aneurism takes its rise in the aorta near the heart, it may press upon the walls of the cavities of the heart itself, and also force the heart aside from its natural position in the thorax. It follows from all this, that thoracic aneurism may interfere with or arrest entirely, the functions of the parts with which it comes in contact, and may also destroy and disorganize the structures subjected to its pressure.

The heart itself may remain healthy, but generally we find it hypertrophied, when there is an aortic aneurism present. We must remember, that the very cause which gives occasion to the aneurismal tumour—viz., a diseased condition of the walls of the aorta—is of itself sufficient to provoke hypertrophy of the heart, in consequence of the obstruction to the circulation resulting from the loss of the elasticity of the coats of the artery. Hypertrophy of the heart would also be promoted

by the impeded pulmonary circulation. Of course it might exist coincidentally with the aneurism, through accidental defect of the valves of the heart. Furthermore, it would appear that the aortic valves are generally more or less implicated in all diseased conditions of the first portions of the aorta.

Diagnosis.—A careful consideration of the signs and symptoms which result from the obstructions and injuries of the different parts and organs, produced by the aneurismal pressure, assists us much in our diagnosis of the cause producing them; but however well marked any of them may be, they cannot in any case be held as pathognomonic of the presence of aneurism. The existence of small aneurismal tumours may, as before observed, readily pass unnoticed; and it not unfrequently happens, that even large aneurisms give rise to no general or local symptoms, and so escape detection during life; or they may declare their presence only at the moment when they destroy life by bursting internally, either into the lungs, or into some one of the organs or cavities of the body. It is only when we can feel, and, as it were, see the tumour, that we can positively affirm its existence,—then, when it has made its way towards the surface of the body, and projects from beneath the skin, heaving and beating synchronously with the systole of the heart. It is therefore from these direct physical signs of aneurism alone, that we can attain certitude in its dia-

gnosis; and such physical signs present themselves only in the very advanced periods of the disease.

Previous to the existence of some one or more of these signs, then, our only means of arriving at a diagnosis of the disease is from a consideration of the disturbances produced by it in the functions of the lungs, the heart, etc.; and there are certain peculiarities in the mode of appearance, the progress and relations of these disturbances, which often enable us to predict the existence of the aneurism with a high degree of probability, even before the physical signs appear.

Physical Signs of Thoracic Aneurism.—The most remarkable sign, when it exists, is that of a prominence—generally of an obtusely conical form—of some part of the thoracic walls: the portion which bulges has a relation to, and therefore to some extent indicates, the point where the aneurism takes its origin from the aorta. An aneurismal prominence situated about the right and upper part of the sternum, indicates especially a connexion of the tumour with the ascending portion of the aorta. When the transverse portion of the arch is the seat of the aneurism, then the tumour is developed in a direction upwards towards the top of the sternum, and perhaps rather towards the right of it. Aneurisms situated on the descending portion of the thoracic aorta, rarely give rise to any prominence of the thoracic walls anteriorly; they usually en-

large into that side of the thorax—the left—down which the aorta passes, and sometimes they destroy the ribs and vertebræ, and present themselves behind in the dorsal region; but such development is comparatively rare. These are the general directions which thoracic aneurisms may be said to take in the progress of their growth; but there are, of course, many exceptions thereto. Aneurisms on the posterior surface of the arch will enlarge backwards; and particular conditions and mode of growth of an aneurism of the ascending aorta may cause it to appear to the left of the sternum.

The aneurism, as it advances, not only destroys the soft parts which form the parietes of the chest, but it likewise gradually erodes the bones, by the constant pressure which it exercises upon them. The cartilaginous portions of the thoracic walls resist destruction longer than the bones, probably from their being of a more elastic and yielding nature. The skin remains long unchanged, but it at last likewise succumbs under the pressure of the advancing tumour: it becomes thin and red, and sometimes ulcerates, and then the sac may burst, and, with a gush of blood, the patient is instantly destroyed. The thinness of the skin and of the anterior walls of the sac, at the prominent part of the tumour, is really, at times, something fearful to feel and witness, especially when the pulsation of the aneurism is strong: one dreads that every beat

of the heart will cause the blood to rupture the sac. It is, however, surprising how long the fatal moment may be averted, even under such circumstances, by judicious care and management.

Auscultatory Signs.—The sounds heard over the aneurismal tumour vary considerably in different cases; and of the cause of this difference we may readily find an explanation in the particular formation of the aneurismal sac, and in the nature of its contents. In some instances, the mouth of the sac is small, or partially obstructed by fibrinous coagula; in others, it is large and open, permitting the free entrance and exit of the circulating blood. At one time, the sac is almost entirely blocked up by layers of coagulated fibrin, so as to become, as it were, a solid tumour; and at another, its contents are nearly altogether fluid. The position of the tumour, and the direction in which it grows, also materially affect the free passage of blood into and out of it; the tumour may be large and the neck of the sac twisted, and so the current of blood into it obstructed. The strength of the heart's action; the position of the opening of the sac relatively to the direction of the current which is forced from the heart; the partially elastic or completely inelastic nature of the walls of the sac; the distance of the tumour from the walls of the chest;—these, and other like circumstances, necessarily affect the character of the sounds which may arise in the

aneurismal tumour. Of course, in all this, it is taken for granted that the sounds heard in association with such tumour, arise either within the tumour or at its mouth, where it opens into the aorta.

Double and single murmurs and sounds, corresponding almost exactly in time with the systole and diastole of the heart, may be heard over the aneurismal sac, and may exactly resemble those which arise in the heart. The systolic murmur may be of various kinds,—soft and blowing, and harsh and grating; the diastolic murmur is softer, generally speaking, and less intense than the systolic, and is less rarely present than the systolic. This fact, which may be explained by considering the nature of its probable cause, which is the passage of the blood, during the diastole of the heart (and the *systole* of the aneurism), out of the sac, through its narrow opening, into the aorta: the force which expels the blood out of, is less than that which drives it into the sac. Moreover, the roughnesses on the aorta, about the mouth of the sac, which the blood may encounter during its passage into the sac, would not present the same obstructions to its exit therefrom. Sometimes it is not impossible that the murmur may be produced by the pressure of the sac itself on the aorta, or on some large vessel arising from it. Every variety and combination of sound and murmur may thus

be heard over aneurisms. On the other hand, the tumour may yield no sound whatever. According to Dr. Stokes, indeed, the presence of a murmur is exceptional in cases of thoracic aneurism; that is, it is more frequently absent than present.

The auscultator almost invariably feels an impulse communicated to his head, through the stethoscope placed over the aneurism, at each systole of the heart; an impulse as strong as, or even stronger than, that felt over the heart. Scarcely any impulse is perceptible over the thoracic walls, between those points where the impulse of the heart and that of the aneurism are felt.

Summarily, we may say of the auscultatory sounds of thoracic aneurism, that, in general, a more or less pronounced murmur accompanies either the systole or the diastole of the heart, or both; that the normal aortic sounds may alone be heard; that sounds and murmurs may both be heard; and that both sounds and murmurs may be very indistinctly or not at all heard. There is therefore no auscultatory sound, *per se*, pathognomonic of aneurismal tumours.

It is well also to remark here, that the valves of the heart are very apt to be diseased in cases of aortic aneurisms,—the same degenerating process which causes the injury of the artery, producing the faulty conditions of the valves, and consequently that the cardiac bruits special to such conditions

may occur coincidentally with the existence of the aneurism, and passing up along the aorta, may give the idea that they arise in the aneurism, instead of at the aortic orifice.

Percussion Sounds.—The percussion sound of an aneurismal tumour, where it comes in contact with, or projects through the thoracic walls, is dull, whether the contents of the sac be fluid or solid. The extent of the dulness will depend upon the size of the tumour, and the amount of its circumference which comes in contact with the thoracic walls. It is necessary to remember, in estimating the degree of dulness as an indicator of the size of the aneurism, that the lung is very apt to be consolidated in the neighbourhood of the tumour, in consequence of the inflammation excited in its tissue by the pressure of the tumour upon it. The dull percussion is most striking when it is found at parts where the sound is clear in health; and so to the right of, and upper part of the sternum: lower down, and to the left of the sternum, the dull sound becomes obscured by the pressure of the heart, etc.

A powerful impulse is communicated to the hand, when laid over the part against which the tumour impinges; and sometimes a distinct vibratory thrill is likewise felt, coincident with the systole of the heart. Also, when the tumour has passed through the thoracic walls, a back stroke is occasionally perceived, following immediately upon the impulse.

When the aneurism is situated at the arch of the aorta, the impulse of the tumour is generally felt most strongly above the notch of the sternum, when the finger is pressed into the hollow there.

General Symptoms of Aortic Aneurism.—These, in the main, are the same as those which represent to us organic disease of the heart. They arise from a similar cause—viz., obstruction to the free performance of the pulmonary and cardiac functions: in so far, therefore, as the disorders of the heart and lungs which arise from the presence of the aneurism are concerned, there is nothing special to be said concerning them.

Thoracic aneurism may long escape detection, producing neither physical nor general signs; and may even attain a considerable size without provoking them. Generally, however, symptoms, though obscure, early indicate the presence of something abnormal within the thorax. With the advance of the tumour, a dry, whistling, painful cough there may be; dull, heavy, stifling, fixed pain within the thorax, reaching perhaps down the arm. One position of the body may be more conducive to ease than another; for in certain attitudes, it would seem that the aneurism, by its weight, presses upon and interferes with the functions of parts: it may press upon the trachea, and cause dyspnœa; or upon the intercostal nerves, and occasion pain; or again, on the lungs themselves, in a manner which admits of

relief by change of posture. Paroxysms of pain and dyspnœa may thus arise from pressure on the trachea, and from stretching of the recurrent laryngeal nerves; and this pain and dyspnœa, when from the advanced growth of the tumour the pressure on the bronchi or trachea, and the irritation of the laryngeal nerve, has become incessant, are most fearful sources of distress to the patient: when thus suffering, he seeks relief in strange and most constrained attitudes, and retains them for long periods of time.

Dysphagia.—Difficulty of swallowing occurs when the tumour presses upon, or even comes slightly in contact with, the œsophagus; of course the degree of difficulty will vary much according to the amount of pressure exercised by the tumour upon the œsophagus—according to the nervous susceptibility of the individual, and the position he may assume. Sometimes the pressure excites even ulceration of the œsophagus, and thus, and from rupture of the sac, blood may be discharged, passing perhaps first into the stomach, and thence rejected by vomiting, thereby producing a false kind of hæmatemesis.

When the trachea, larynx, and recurrent nerve are compressed by the tumour, the voice is often much altered: it may be weak, hoarse, trembling, husky, and even almost lost. At the same time the breathing is much interrupted, and a painful ringing dry cough present.

The pain suffered by the patient varies in kind and in situation ; it has a reference to the position of the tumour, and to the cause from which the pain results. A gnawing, rending, constant pain is felt in the back, when the tumour is pressing on the vertebræ and the nerves arising thence. When the nervous plexuses of the neck are involved, the pain reaches down the arms, and is chiefly felt in the neck. Then, again, pain may result from pleuritis excited by the pressure ; from irritation of the intercostal and cutaneous nerves ; from periosteal inflammation ; and from spasms of the diaphragm caused by irritation of the phrenic nerve.

Pressure of the aneurism upon the large venous trunks occasions congestions and œdematous conditions of parts, particularly of the upper extremities and of the head and neck. Œdema of the lower extremities is rarely observed. The first results which ensue from the pressure on the veins are congestions of the integuments of the head and neck, which give them a puffy, tumefied, livid appearance, the veins at the same time being swollen and prominent ; and then follows œdema of the same parts, and sometimes of the arms and shoulders and upper part of the chest likewise. The pressure when exercised on one particular vein has been known to produce local œdema of the parts to which that vein is distributed.

The pulse in thoracic aneurism varies much in

its characters: inequality of its force in the radial arteries is very significant where other symptoms of aneurism are present; we may then fairly attribute the inequality to a partial obstruction of one of the large vessels springing from the aorta.

The general health suffers, in most cases, as the disease advances; but in some rare cases the health of the patient remains good up to the very time that he is destroyed by the rupture of the tumour. The patient wastes; and the cause of this is chiefly, it would seem, the pain which he suffers, the wearing out, exhausting pain. From the pain, loss of appetite results. Occasionally inanition is the consequence of the pressure of the tumour upon, and obliteration of, the thoracic duct.

When the most prominent of the signs and symptoms related above are present in any given case, we can have little doubt as to the existence of an aortic thoracic aneurism. A pulsating tumour occupying the position of the first portion of the aorta, dull to percussion, yielding a double or single murmur, and communicating a thrill to the hand; united with difficulty of respiration and deglutition; and with cough, pain, and change of voice—leave little room for doubt as to its nature. But it is well to be aware, that there are many circumstances which, in special cases, render the diagnosis difficult; in fact, that there are diseases which simulate aneurism, and sometimes in a re-

markable manner. Thus, pulsation may be communicated to a tumour not aneurismal, by the beat of a healthy subjacent aorta; and such tumour may press upon the aorta and give rise to murmurs, and it may press upon the trachea and œsophagus, and cause difficulty of respiration and deglutition. Also, there are many morbid conditions, as well as that of aneurism, which may cause dull percussion about the aortic arch and along the course of the descending aorta in the back. In almost all these cases, however, the attendant circumstances, and the history of the disease, enable us readily to surmount the difficulty of diagnosis and fix the true nature of the morbid condition, which thus simulates an aneurism. Much greater is the difficulty of diagnosis when there is an entire absence of all physical signs of the presence of a tumour; but even then a careful investigation will oftentimes enable us, by searching into the history and nature of the disturbances which present themselves, to come to a tolerably sure conclusion as to their cause; for though the disturbances caused by aneurism may be the same, in kind, as those which result from other causes, still, in their attendant circumstances, in their antecedents and consequences, they generally possess marked points of difference.

If, for instance, violent hæmoptysis should suddenly occur in an individual who had long suffered from anomalous thoracic symptoms, and who yet

had no other sign of disease, little doubt would exist as to the aneurismal nature of the disease.

The situation of an aneurism of the innominata, will assist in distinguishing it from aortic aneurism: it occurs higher up, behind the right clavicle; it less frequently affects the respiration and deglutition, and more frequently produces local symptoms, through pressure on the nerves and vessels of the right arm.

The *course* which thoracic aneurisms run is, with very rare exceptions, ever progressive; and their termination is fatal. Sometimes the disease for a time seems to pause and linger; and occasionally it would appear that even a complete cure, by occlusion with fibrin, of a small aortic aneurism may take place. Aneurism may destroy life immediately, through rupture of its sac and sudden loss of blood; or by gradual exhaustion of the system,—the escape of blood being small, and again and again repeated. It may be poured out, also, into the pleura or the pericardium, into the bronchial tubes, or the œsophagus, or into the pulmonary tissue itself, etc. Aneurism, again, may prove fatal by exciting pulmonary or pleuritic inflammations,—by pressure upon the trachea and lungs, and upon the œsophagus. Death may also result from inanition, as a consequence of pressure upon the thoracic duct; and from the exhaustion and loss of appetite caused by pain, restlessness, and nervous irritation. The most

common cause of death is rupture of the aneurismal sac.

Treatment.—From what has been said of the course of an aortic aneurism, it follows, that we cannot hope by treatment to effect a cure of the disease; but by careful management, the physician may prolong the life of his patient, and alleviate the great sufferings which so frequently accompany thoracic aneurism. The treatment, in its main characters, must be of a general nature; the diet should be nourishing and yet not stimulating; excitement, mental as well as bodily, must be most carefully avoided; and bodily exercise should be of the gentlest kind. In other words, the heart should be maintained in a quiet state of action, and the powers of life duly supported. How often has it happened, that the lives of persons suffering from aneurism have been cut short by sudden rupture of the aneurismal sac, resulting from violent action of the heart, brought on by passion or some great bodily effort!

No one now-a-days adopts the method of treatment recommended by Valsalva: the venesections he advised for the cure of aneurism, are on all hands admitted to be most injurious and dangerous, and opposed to all rational pathology. In sacculated aneurisms our desire naturally is, to induce and promote the deposition of fibrin: by the layers of fibrin deposited in the aneurism the sac is strength-

ened, and an attempt at cure is so far begun; but it is worse than useless to attempt to produce such coagulation of the blood by venesection. The state of anæmia which may be produced by repeated bleedings, is just the condition we should least desire to see the patient fall into, being that which would give least hopes of his ultimate recovery. In a strong, vigorous person, in whom the heart's action is excited, an occasional bleeding, done for the express purpose of tranquilizing the circulation, may be of service; and, in fact, a few leeches now and then applied over the aneurism, in most cases give considerable temporary relief to the patient. This is especially the case when the respiration is much impeded, through the pressure of the tumour upon the lungs, and when the heart's action is easily excited.

Those medicines, also, will often be required which tend to tranquilize the circulation and soothe the sufferings of the patient: digitalis and opium, hydrocyanic acid, and conium, may be especially mentioned for this purpose. The bowels should be kept free by laxatives: constipation, indeed, is always to be particularly avoided, on account of the straining at stool which it frequently occasions. Gallic acid and acetate of lead have been highly recommended, as promoting coagulation of the blood; but their effects in this way are far from certain.

Local applications give great relief to the patient: cold applications—even ice—laid on the tumour, afford sometimes much comfort, and are said to promote coagulation within the sac. When the tumour is at all prominent, it will be requisite to apply over it some mechanical covering; on the one hand, to protect it from external injury, and on the other, to support and somewhat restrain the progress of the tumour.

FINIS.

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